EXPERT REPORT

U.S. v. BP Exploration & Production, Inc. et al.

Toxicological Impact of the MC252 Blowout, Oil Spill, and Response
Submitted on Behalf of the United States

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I. Executive Summary

The objectives of this report are to address two major questions regarding the potential for toxicity damage from the *Deepwater Horizon* (DWH) spill in 2010: (1) was there potential for toxicity damage to aquatic life at the surface interface, upper surface waters, and shoreline and shallow estuarine waters; and (2) was there potential for toxicity damage to organisms in the deep layers where plumes of dispersed oil were trapped?

While every spill is unique (location, volume, type of oil, temperature, vulnerability of species and habitat), a series of past experiences demonstrates that oil spills typically damage habitat and are toxic to marine species. DWH crude oil contains PAHs, similar to other crude oils throughout the world, where studies have demonstrated their chemical toxicity, particularly to early life stages. Given the volume of oil spilled from the well in 2010 (4.2 million US barrels into the environment),[1] the long release time, the depth of release, and the large volume of dispersants applied (at depth and on the surface), based on pre-spill scientific knowledge from earlier spills, extensive toxicity damage to marine life from the spill was expected by many scientists, including me. There were, however, unique aspects to the DWH spill that are very important, each of which I considered in my analysis to assess whether the expectation of harm should be adjusted in light of the nature of the *Deepwater* spill.

- First, it is a record setting spill for US waters, the largest spill on record.
- Second, a record amount of chemical dispersant was applied, both at depth and at the surface.
- Third, there were effectively two spills; an upper surface water spill that affected the shoreline, surface interface, and the upper water column where observations and literature from previous spills are relevant, and the submerged plume of dispersed oil at depth, affecting bottom habitat and organisms, where previous spills are not as informative.

Release of oil at depth plus addition of chemical dispersant both at depth and on the surface caused oil dispersions that efficiently solubilized toxins, making them biologically available at depth (contaminated plumes in the deep waters) and in the upper water column, as well as at the sea surface and shoreline.

In drafting this report, I reviewed previous spill literature, some of which I was personally involved in, as discussed below, and examined DWH published studies, comparing the historical spill literature with the evidence and conclusions in the DWH published studies. *Deepwater Horizon* literature generated after the spill indicates damage to habitat, fouling of

[1] United States' Proposed Findings of Fact for Quantification Segment of the Phase Two Trial (Rec. Doc 12048-1), Section VI.

birds and marine mammals, fouling of the shoreline, the presence of oil in water at depth and the upper water column, and chemical toxicity. Based on my review of the pre-spill literature and the DWH literature, I have concluded that there is good reason to believe there was significant potential for toxicity damage to marine life from the DWH spill.

II. Expert Background and Methodology

This report is prepared by Stanley D. Rice (Ph.D. Comparative physiology, 1971). My career with the National Marine Fisheries Service (1971-2012) was focused on oil toxicity and impacts; my first assignments were to draft potential impacts of spills in the marine environment for the TransAlaska Pipeline Environmental Impact statement and to initiate a new research group of biologists and chemists focused on oil toxicity and impacts. I supervised and managed this group over the next 40 years, which produced many research papers. I have observed and consulted for my agency response options and strategies for several spills (Ixtoc I, Exxon Valdez, Selendang Ayu, and Kuroshima), and I have conducted and managed damage assessment projects for Exxon Valdez, Selendang Ayu, and Kursohima. However, the majority of my career was focused on the damage assessment of the Exxon Valdez oil spill, particularly the long term impacts over the 20 plus years following the spill. I have authored/co-authored over 100 peer reviewed publications on a variety of spill impact issues, ranging from oil persistence to impacts on fish embryos, sea otters, and killer whales. I have authored/co-authored several review papers and was the lead editor of the Trustee sponsored symposium reporting on the Exxon Valdez spill. As a professional government biologist, I have counseled federal and state agencies in the past on Exxon Valdez impacts, issues pertaining to the Oil Pollution Act of 1990, Alyeska marine terminal issues, and provided advice to the National Oceanic and Atmospheric Administration (NOAA) during the early stages of response and NRDA of the DWH spill. Appendix C contains a bibliography of publications I have authored or co-authored since 2000.

In preparing this report, I have relied primarily on published scientific literature, including reports on the DWH spill and also previous well-studied spills, because these papers are peer reviewed. In addition, I have examined abstracts and presentations at conferences, but do not rely on them as much, as they are not peer reviewed. Further, I have also examined data provided to and by BP as part of the Natural Resource Damage Assessment and the Deepwater Horizon litigation; however, I do not rely heavily on these materials because they are overwhelmingly large, have not been analyzed and summarized into a format and volume conducive for review, and are part of an ongoing NRDA, so drawing final conclusions about the data at this point would be premature.

III. Organization of the Report

Prior to addressing the question of toxicity potential from DWH spill, Section IV of this report provides a general overview of oil chemistry and the ecological toxicity of oil and dispersants, including methodologies used to evaluate ecological toxicity. Section V describes the arc of scientific lessons learned from pre-DWH oil spills that provide the foundation for evaluating potential harms from the DWH spill. My analysis of the potential toxicological effects of the DWH spill is presented in Section VI. Conclusions are presented in Section VII.

IV. "Oil and Dispersant Toxicity 101"

A. "Oil Chemistry 101"

1. Oil Composition in General

Oil is composed of hundreds of thousands of organic compounds, making oil complex to analyze, to understand, and to summarize. Composition is important; it is the composition that determines the physical properties of oil (how viscous the oil is) and the chemical toxicity of the oil. The viscosity matters, in turn, as this determines the energy needed to physically mix oil into the water and the responsiveness to dispersants. Viscosity also affects how severe the fouling can be of surface species such as birds or intertidal fauna, and shoreline habitats. The composition varies between oils of different origins, but the major classes of compounds (including classes like the prevalent alkanes and aromatic compounds) are usually represented in all crude oils, but the proportions of specific compounds will vary to the specific oil. Many if not most of the compounds in oil are not particularly chemically toxic (like methane, and the prevalent alkanes), but they all contribute to the crude mess that can foul organisms and shorelines.

Alkanes are the simplest of the oil compounds and the least toxic. They are saturated straight-chain aliphatics and are prevalent in crude oil. These compounds are easily metabolized for energy, and most are easily degraded by microbes. The alkanes are carbon chains of variable lengths with single covalent bonds between carbon atoms and hydrogen at all other locations (each carbon atom has four bonds; Fig. 1). Examples are methane (1 carbon atom), pentane (5 carbons), and octane (8 carbons).

Aromatics are a more complex set of oil compounds, the key feature of which is the 6 carbon aromatic ring, with double bonds; it this aromatic ring with double bonds that makes these compounds reactive (and toxic). There are many toxic compounds in oil, with different

structures, but there is consensus in the scientific literature that aromatics are the compounds most responsible for chemical toxicity from crude oil spills based on their toxicity, solubility, volatility, and prevalence within crude oil. **Mono-aromatic hydrocarbons** (benzene, toluene, ethylbenzene, and xylenes) are single ringed and are commonly known as **BTEX**; these are single 6-carbon ring compounds distinguished by substitution of methyl (CH_3) or ethyl (C_2H_5) groups on the ring (Fig. 1). **Polycyclic aromatic hydrocarbons (PAHs)** have two or more benzene rings (Fig. 1). For example, naphthalene has two rings, phenanthrene three, and chrysene four. The structure of each of the aromatic compounds matters: : solubility, volatility, and persistence all affect the toxicity of a compound, as discussed below in "Oil Toxicity 101."

2. Deepwater Horizon Oil Contained the Same Classes of Compounds as Other Crude Oils

The Deepwater Horizon Oil contained the same classes of compounds as other crude oils, including the BTEX and PAH compounds (Liu et al. 2012). At the time of release, more than 50% of the hydrocarbons were low molecular weight (methane and C_2-C_{11}) (Ryerson et al. 2011; Liu et al. 2012). Like other crude oils, trace metals in DWH oil included iron, aluminum, manganese, cobalt, nickel, copper, zinc, chromium, vanadium, arsenic, and lead. (Liu et al. 2012). At trace levels these are seldom of concern, but the volume of oil spilled does raise some concern about exposure to metals like metals like copper, zinc, chromium, arsenic, and lead.

3. Oil Weathers When It Is Spilled

"Weathering" is a short-hand way of indicating that the composition of oil changes as oil is subject to various natural processes. Hydrocarbon volatility and solubility are generally dependent on molecular characteristics (Fig. 2). Oil compounds are not stable in water, and weathering (processes that change chemical composition) starts immediately upon release. The structures of the compounds affect differently the rates of solubility, volatility, and degradation. The rate at which BTEX and PAHs move from whole oil into water is dependent on molecular weight: smaller, less-substituted molecules are lost most rapidly and larger compounds move slower out of the oil (Short and Heintz 1997). This thermodynamically-driven process explains the characteristic changes in composition as oil weathers and is well documented (Short and Heintz 1997). See Fig. 3 for an example of PAH changes due to weathering. At the surface, the lighter molecules can be lost to the air because of their volatility (BTEX for example). In the water the lighter molecules have greater solubility and will be lost into the water at faster rates than larger molecules. Understanding the impact of particular oil requires detailed chemical analyses repeated over time and space to understand both concentration and composition of toxic compounds.

4. Oil in Water Is Bioavailable to Marine Life

Oil hydrocarbons in water are biologically available (bioavailable) to marine life. Although the concentrations of oil in water may be very low (like parts per billion), organisms can still easily absorb significant quantities of oil because hydrocarbons are much more soluble in the lipids of a marine organism: oil hydrocarbons can move across membranes and become trapped in the lipid part of cells. As a result, concentrations within an organism can be 1000 times higher than the water concentrations (Carls et al. 2004), thus achieving internal dose levels that can be toxic. In some invertebrates, and all vertebrates, there are metabolic pathways to degrade the oil hydrocarbons (like "P450" enzymes), thus making it more difficult to assess the uptake of the toxic components. This is particularly true in most fish: the parent hydrocarbons can be degraded quickly, while the metabolites, with unknown toxicity, are difficult to measure and it is difficult to assess the significance of the exposure or the uptake loads.

B. "Dispersants 101"

Oil dispersions are the creation of droplets of oil that can be driven down into the water column, thus removing oil from the surface. Dispersions can be natural, such as the formation of droplets of oil through physical action such as wave action or the violent release from the riser pipe, or chemical dispersions can be created by application of dispersant chemicals. Figure 4 shows an example of chemical dispersion of oil into water. Both types of dispersions were significant in the DWH spill.

The use of chemical dispersants is a response strategy used when preferred options such as mechanical pick up (skimming) or burning are either impossible or insufficient to fully address the extent of an oil spill. Dispersing the oil into the water column has the advantage of removing oil from the surface, thus lowering the damage potential from physical fouling to shoreline habitat and surface organisms, but has the downside effect of increasing the toxicity threat to organisms within the water column. The motivation behind chemical dispersant use is that small droplet formation improves the rate of natural oil removal processes such as dissolution into the water column, volatilization at the surface, biodegradation by organisms that consume hydrocarbons, and sedimentation resulting from interaction with suspended particulate material.

Chemical oil dispersants are mixtures of solvents (like nail polish), surfactants (like soaps), and other additives that are applied to oil slicks to promote droplet formation when the system is mixed by wave energy (Michel et al. 2005). Solvents are included primarily to

promote the dissolution of surfactants and additives so the dispersant mixture is homogeneous and they affect dispersant viscosity and solubility in oil. Solvents can be very toxic, as learned when "first generation" dispersants were used on the Torrey Canyon spill.[2] The primary active component in modern dispersants are the surfactants, which have one end of the molecule that is soluble in water (hydrophilic), and another part of the molecule that is soluble in lipids (lipophilic) and dissolves easily into the oil, thus promoting the formation of oil droplets when energy is applied. Additives are included for various purposes, such as improving surfactant dissolution into oil and increasing dispersant stability.

Dispersants are effective within constraints; there must be sufficient mixing energy to be effective (wind, wave, current), however higher energy winds can cause dispersant drift, so it does not reach target oil (Michel et al. 2005). Dispersants are most effective when applied to fresh oil, and least effective when the oil has weathered to a highly viscous state, or when it is a mousse (water/oil is about 50/50), probably because the surface properties of the oil prevent the lipophilic portion of the surfactant molecules to penetrate.

Adding dispersants increases toxicity by promoting oil droplet formation, thus increasing the rate at which PAHs move into solution and thus increases their bioavailability. Physical mixing energy generally increases droplet formation and is necessary for dispersants to be effective. Mixing energy and chemical dispersants increase the surface area of oil in contact with water, reducing oil droplet size, thus promoting the speed at which PAHs solubilize into water and increasing aqueous total PAH concentrations. More PAH in the water increases the toxicity of the solution. By allowing more sparingly soluble toxins from oil to dissolve, dispersants increase oil toxicity. This increase comes primarily from the increased bioavailability of the PAHs, not from a change in the oil or the direct toxicity of the dispersant. (Wu et al. 2012; Adams et al. 2014; Martin et al. 2014).

C. "Oil Toxicity 101"

As discussed earlier, the aromatic hydrocarbons are considered the fraction of oil most responsible for the toxicity because this fraction is relatively high in concentration within the oil, relatively toxic, and have the ability to solubilize (dissolve) into the water column and thereby expose living organisms. Although there are many other toxic compounds in oil, they

[2] The Torrey Canyon spill in England in 1967 came ashore; dispersants were heavily used in the inshore, and shoreline damage was extensive. The dispersants were an early generation type, and were more like a solvent than less toxic soap, and caused direct toxicity, particularly to the intertidal fauna. The direct toxicity was because the dispersant composition had significant concentrations of toxic compounds and added to the toxicity of the spilled oil, which supplied a rationale case against using dispersants on oil spills.

are often present in lower concentrations or have lower solubility. The aromatic fraction typically has percentages between 1-10% of the total oil mass in crude oils.

The smallest aromatics, the BTEX, are relatively volatile and water soluble compared to the larger aromatics; the BTEX leave the oil mass most quickly by volatizing in air or dissolving in water. From an inhalation perspective, BTEX are the most dangerous and toxic compounds; when inhaled, they can enter the blood stream very quickly, and move rapidly to neural and respiratory tissues.

Aromatic toxicity generally increases with ring number and with alkyl substitution, solubility decreases, volatility decreases, and the ability of microbes and other fauna to metabolize and excrete by-products decreases with additional rings and substitution. Although the toxicity generally increases with increasing rings and substitutions, bioavailability often decreases because the larger compounds are not as soluble, thus limiting exposure quantities. Consequently, the toxicity risk shifts from acute (immediate) toxicity mechanisms involving the 1-3 ringed aromatics, to more of a chronic risk that is longer term and involves the larger 3-5 ringed compounds. The larger compounds are still very toxic, but their bioavailability is seldom sufficient to cause acute toxicity; however, their concentrations in the environment can be sufficient to affect many different toxicity mechanisms, just not quickly. Further, larger more complicated PAH compounds persist in the environment longer because of the difficulty in breaking them down; hence their toxicity and risk persist longer in the environment. Dispersions of oil, with increased surface area, enhance the solubilizing of the larger complex PAH, but they can also contribute to toxicity independent of solubilizing PAH when the droplets themselves come into direct contact with sensitive tissues (e.g. when droplets are eaten, or clog gill like structures).

1. Fouling versus Toxicity

Fouling is the physical coating of oil on an organism's pelage, feathers, or other surfaces and has been observed since the first oil spills. Spill mortalities usually include the birds and intertidal organisms coated by oil. All oil compounds contribute to the organic mess that can foul an organism, particularly the birds and marine mammals with feathers or fur, and the intertidal fauna when oil comes ashore. Fouling can have several effects- from smothering (lack of oxygen), to compromises of the insulation (feathers, fur), and also a toxic effect (toxins are internalized by preening of feathers or fur for example).

In contrast to physical fouling, **chemical toxicity** affects the ability of the organism to survive because body tissues and cells are chemically disrupted by toxic compounds in the cells

and membranes. Chemical toxicity ranges from rapid acute toxicity, to death on a longer time scale, to sublethal effects that may still affect populations.[3] In the short term, acute toxicity involves rapid disruption of neural and respiratory systems, often leading to a narcosis,[4] followed shortly by death. In the long term, cells and other organ systems may be affected, causing a decrease in function, and if severe enough, death. Other effects may be sublethal, meaning cells and tissues may be affected, may need repair, and will decrease the organism's survival by impairing growth, ability to forage, or avoidance of a predator. The population may still be impacted even without a mortality caused by direct chemical toxicity. Any impacts beyond rapid acute mortality are challenging to detect, take time to play out, and may go undetected even though the population is affected over time. Some impacts may not affect the survival of an individual, but affect the ability to reproduce or viability of the offspring, and consequently affect the population over time.

2. Toxicity mechanisms

There are a range of toxicity mechanisms, each with a range of impacts. For acute toxicity, the effects are quick, and the mode of action is likely on neural or gill membranes, where the ability to function is quickly impacted, and the organism dies. Narcosis is often observed in the minutes prior to death. Acute toxicity bioassays[5] usually target this relatively easy end point to quantify the relative differences in toxicity between different toxins. However, there are many different toxicity mechanisms, and these have variable effects on organisms ranging from depressed growth (energetic costs), to impacts on tissues (lesions in the liver, skin, or elsewhere), to impacts on tissues (such as gill lamellae fusion), to cellular impacts (for example abnormal cells, carcinogenesis), to impacts on DNA. If the dose is low enough, or transient, some of this damage may be repairable, but at a cost; some damage will shorten the life expectancy, by lowering the fitness the organism, which translates to lower probability of survival, more difficulty in finding prey, and/or more difficulty in avoiding predation. The net result of lowered fitness is fewer adults to reproduce and lower recruitment of juveniles into the spawning population.

Oil toxicity is complicated by two parts of the equation: chemistry and structure of the toxins, and the biology of the organisms. As discussed above, oil is composed of many different

[3] The term "sublethal" can be misleading, as it implies a "safe" exposure level. While "lethal" levels define exposure levels that are obviously harmful, sublethal levels do not define levels that are safe. They only define doses that are not acutely lethal on the short term, and do not describe exposure levels that are safe/harmful on the long term to the individual or population.

[4] Narcosis is a state of stupor, drowsiness, or unconsciousness produced by chemical exposure.

[5] Bioassays are procedures for determining the toxicity of compounds or mixtures of compounds using graded concentration series and multiple test organisms. Biological end points, such as death, are used to measure the toxicity.

compounds, each with varying degrees of toxicity to a variety of different biological structures and processes. The biological sensitivity of a given organism is impacted by a long list of variables too, from level of cellular integration, to life stage, reproductive stage, fitness, and habitat niche. Bioassays measure the "net result" of mixing toxic chemicals with sensitive biological organism, and summarizing the result as an "LC50".

Early life stages (embryos, larvae) are generally believed to be very sensitive to PAHs from oil. Uptake of PAHs at this life stage is rapid, reaching equilibrium with environmental concentrations in minutes. There are many sensitive tissues and developmental processes that can be impacted, and repair mechanisms are poorly developed. This literature has progressed significantly since the Exxon Valdez oil spill, and this research theme was further developed after the DWH spill, and continues to support the observation that early life stages are very chemically sensitive. Basically, it does not take much chemical disruption to have a range of negative impacts on developing embryos, from deformities and early death to poor growth, reduced survival, and reduced adult populations.

3. Measuring Toxicity: Bioassay Methods

Oil toxicity bioassays are a valuable tool that provide insight into comparative oil toxicities and comparative sensitivities of different species and life stages. Because several of the studies of the toxicity impact of the DWH spill are based on oil bioassays, I describe the performance of bioassays, their evolution, and their limitations below.

a) The Basics of Bioassays

Bioassays (biological assays) are laboratory-based experiments commonly employed to understand the toxicity of a substance or mixture of substances. They generally are focused on the dose required to kill half the test organisms (known as the "LC50") in a specific period of time, typically 4 days. Separate groups of organisms, chosen at random for each dose, are exposed to a series of toxin concentrations ranging from 0 (the control) to a level sufficient to cause all animals to respond in that dose (such as death). Intervening doses are included to yield graded multiple partial responses, hence providing statistically valid results. With the exception of the toxin, assay conditions are designed to support organism growth and survival. Assay results are often summarized by a single number, such as the LC50 (median lethal concentration).

Oil bioassays are more complicated than single compound assays because oil typically contains many aromatic toxins, each differing in structure, solubility, and toxicity. Early oil bioassays adopted the short-term acute bioassay approach. This was essentially the study of a

single response mechanism (acute death) to non-specific narcotic depression of cellular function by compounds such as benzene, toluene, and xylene. Comparison among first generation assays was difficult because they were not easily reproduced, as the physical properties of different oils and mixing affected the chemical compositions of aromatics in the exposure solutions. Without supporting chemical analyses, doses were often reported on the quantities of oil added (parts per thousand), rather than the quantities of toxic compounds in the exposure solution (usually in the parts per million or less).

b) The Evolution of Oil Toxicity Bioassays

Oil toxicity bioassays have evolved considerably over time. First generation oil bioassays (1960s-1970s) were focused on acute toxicity, with minimal chemistries in support of the tests. Concentrations were often reported as "oil added", making comparisons between tests, oils, and organisms very difficult as the methods were usually different. The difficulty of replicating tests was recognized early, and more attention was given to standardizing mixing methods and supporting chemistries. Acute toxicity was still the focus, but with better tests; a focus on BTEX and 2-ringed aromatics emerged as the toxic components of most concern. Negative organism responses were generally expected to occur in the parts-per-million range of total mono-aromatic and di-aromatic hydrocarbons.

The next generation of oil bioassays (post Exxon Valdez) discovered that embryos could be affected at the low parts per billion PAH range, much lower than tests with adult life stages. Tests were often longer, with more complicated end points, and the results often required following the success of the organism long after the exposure ended. These bioassays typically had better chemical analyses in support of the tests, and focused on the early life stages, generally of fish, because these are the most vulnerable and represent the weakest link in ecosystems.

c) The Complexity of Oil Toxicity Bioassays

Oil exposure in spills are complex events that constantly change over time; oil exposures in laboratory bioassays are also complex. In both cases, the exposure levels are never stable, and are easily affected by weathering processes that work to change both the concentration and composition of the oil. In bioassays, these processes are recognized, and researchers attempt to minimize the instability by controlling mixing time and energy, aeration,[6] temperature, and other factors so that the tests are reproducible from one batch to the next.

[6] Aeration is the process of bubbling air (or oxygen) into test containers. This procedure is generally not recommended in oil bioassays because it accelerates the loss of toxic components and changes the concentration rapidly.

However, it is difficult to control these processes, and detailed high quality chemical analyses of the exposure solutions are required during the test to describe and verify the chemical dose regime during the test, thus enabling comparisons across species, and across laboratories.

Bioassays are most relevant if they include exposure levels that are environmentally relevant, but the concept of "emulating the real world" is not practical. Scale matters, and laboratory tests cannot reproduce a test at a scale that is environmentally relevant at the ecosystem level. The same weathering processes that change concentration and composition also operate in the environment and laboratory tests, but never at the same rates or scale. Bioassays are best utilized when they are conducted with high quality procedures, to compare across species, life stages, oils, laboratories, or specific variables - but not to predict ecosystem level impacts and interactions.

d) Oil Toxicity Bioassays Likely Overestimate the Survival Rates of Exposed Organisms in the Wild

As complex as oil bioassays are, they are conducted in simplified and controlled environments; whereas the wild environment is always changing, from day to night, from day to day, and from season to season. Organisms in the wild need to survive chemical toxicity challenges, but also the challenges from starvation and predation. Quality bioassays with supporting chemistries will give us insight into the comparative toxicity of oils, and factors affecting toxicity, as well as insight into comparative sensitivities of different species and life stages, but they will likely be poor predictors of an exposed organism's success in the complex wild environment. Toxic death can be predicted; predicting survival in the wild at sublethal exposure levels is a profoundly more difficult and complex task.

Thus, although laboratory bioassays provide useful data on the adverse effects of oil, they do not emulate the stresses of the environment and likely overestimate the survival of exposed organisms in the wild. Except for the toxins, bioassay conditions are a protected microenvironment, without starvation or predators. Bioassays often describe doses that lead to death, but they often are unable to describe accurately or with certainty what exposure levels are safe.

V. An Overview of Changes in the Oil Spill Paradigm

Our understanding of the effects of oil spills has changed over the past fifty years. That change in understanding has influenced how we respond to spills in the first instance and how we assess the damage caused by spills.

Traditionally, there was a focus on the fouling of birds and shorelines, which were generally immediate, obvious and visible effects of the spill, in contrast to long term impacts and impacts to organisms below the surface. Thus, the handling of oil spills was driven largely by the impacts of the visible oil on shorelines and to surface species (e.g., birds and marine mammals). Typical strategies were to get oil off the surface, prevent it from reaching shorelines, and remove it from beaches after it strands. During pre-Exxon Valdez spills such as the Ixtoc blowout in 1979 (during which approximately 3 million barrels of oil were spilled over many months into the Gulf of Mexico), there was limited understanding of the extent of toxicity of oil to embryos at lower levels. Toxicity was thought to exist only at parts per million levels and the persistence of oil was underestimated.

In the aftermath of the 1989 Exxon Valdez spill, however, embryo toxicity studies were performed and published which indicated an increase in fish embryo mortalities, triggering a change in the oil response and assessment paradigm to a concern and focus on the effects of a spill on early fish life stages. That change was cemented by the 2007 Cosco Busan spill, which led to the detection of cardiotoxicity effects to herring embryos both in the field and in laboratory controlled studies. The embryo toxicity studies from both Exxon Valdez and Cosco Busan detected damage at very low levels of PAH, and marked a change in the approach of detecting long term oil damage to fish from a spill. The extreme sensitivity of embryos was well documented, as well as the cause/effect relationship. From these studies, it was apparent that even low environmental concentrations of PAH were potentially harmful to organisms and biological processes, and could impact important organisms beyond those species at the surface or at the shoreline. These studies established a scientific base from which DWH toxicity potential could be – and has been - further examined.

A. The Exxon Valdez Oil Spill (1989)

The Exxon Valdez Spill in Alaska caused a paradigm shift in how we measure damage in oil spills, particularly the long term persistence and long term effects. At the time it was the largest spill in US waters, and happened in a relatively pristine habitat where human influence was limited. Ultimately, this spill became the most studied spill in history, and documented long term persistence of oil buried a few inches below the beach surface and long term damage to several species (e.g. pink salmon, killer whales, sea otters). Like other spills, surface species were oiled during the exposure period of the first couple of months; approximately 500,000

birds and 4,000 sea otters were killed, all estimated from collected carcasses.[7] 1,300 miles of shoreline were coated with oil. Oil recovery was very low, less than 10%, except for the oil that was lightered off the vessel and was not spilled. The damages to the shoreline and to the surface species were immediately apparent, and all immediate effects could be predicted from experiences in previous spills.

Soon, however, the suite of Exxon studies focused on damage assessment began to find "surprises" (see review by Rice 2010; Ballachey et al. 2014):

- **Elevated pink salmon embryo mortality** was detected in oiled streams up to four years after the spill, an unprecedented finding. A series of long term exposures in a controlled hatchery setting detected low part per billion PAH effects on embryo survival, growth, and abnormalities – thus providing evidence of the extreme sensitivity of fish embryos to oil constituents at levels 2-3 orders of magnitude below what had been expected. Field studies found oil along the banks of the spawning channels, and an exposure mechanism was found. Later, embryos exposed to low concentrations of oil were tagged as juveniles and released to the environment, and were evaluated when they returned a year later as adults to spawn. Low part per billion level exposures (5 and 18 ppb) to pink salmon embryos were found to reduce adult survival by 20 and 40%, respectively.

- **Long-term population effects were detected in killer whales and sea otters.** There were studies on long-term population effects in killer whales and sea otters, made possible by population baseline studies prior to the spill. Killer whale numbers in two pods were 40% fewer within a year after the spill, although no carcasses were recovered. Recovery has yet to occur for one pod, while the other pod lacks reproductive females and will become extinct. In the case of sea otters, subpopulations in much of Prince William Sound began to recover in the years following the spill, but not in the area of the most severely oiled shoreline habitats. In 2001 (12 years after the spill), extensive shoreline studies discovered liquid oil a few inches below the surface in over half of the beaches examined in the heavily oiled areas, thus linking an exposure pathway to the local struggling sea otter subpopulation. Evidence of chronic oil exposure

[7] The bird numbers are high compared to most spills, because of the large area oiled by the spill, convergence currents that brought oil and birds together, and because several large rookeries and feeding areas were within the spill zone.

from digging pits in oil contaminated beaches and lack of recovery continued through about 2 decades.

- **Surprises and enigmas continue even 25 years after the spill.** The delayed impact to and recovery of the Prince William Sound population of herring has been both a surprise and an ongoing enigma. Herring reproductive success was impacted in 1989 (1989 year class had the lowest recruitment on record), but scientists determined that were a delayed population crash of many year classes in winter 1993 that has never been explained satisfactorily. No other herring population in Alaska has suffered a crash in the years immediately following the spill, or as steeply as the Prince William Sound population. Similarly, there is no satisfactory explanation of the continued lack of recovery by herring even though their exposure directly to oil is long over. Herring are an important forage species, similar in niche to the gulf menhaden, and are important to the general health of the ecosystem. It is difficult to judge the ecosystem as fully recovered until the herring population has recovered.

The Exxon Valdez oil spill damage assessment indicated that embryo toxicity can occur at low parts per billion of PAH, oil can persist in the shoreline for decades, and several species have suffered long term population effects and will likely never be the same. The damage assessment also led to the understanding that long-term intense study does not always lead to answers about the complex response of the environment after an oil spill, however, as indicated by the failure to explain the delayed crash in the herring population. Collectively, the long term impacts to sea otters and killer whales, coupled with the finding of oil persistence and the significant lowering of the dose required to affect embryos (from ppm to ppb), have changed the oil spill paradigm. Damage assessments on spills since the Exxon Valdez now track persistence of oil and long term damage, rather than a primary focus only on collecting carcasses to assess the damage from a spill. Impact on herring spawn in San Francisco Bay were examined after the Cosco Busan spill, for example, and detailed studies following the DWH spill have been shaped in part from the studies of the Exxon Valdez.

B. The Cosco Busan Spill (2007)

The Cosco Busan Spill was a relatively small bunker oil spill in San Francisco Bay, but embryotoxicity impacts were detected in herring spawn in oiled areas three months after the spill (Incardona et al. 2012). This study was initiated in part because of the impacts to herring following the Exxon Valdez, and the overlap of oiled sites and spawning habitat in San Francisco bay. Like the exposures to embryos in the Exxon Valdez crude oil studies, cardiotoxicity effects

to herring embryos were detected in the field and in laboratory controlled studies; low tissue levels of PAHs were detected in herring from oiled sites.

While investigating the impacts to herring from the Cosco Busan Spill, the additional factor of phototoxicity was apparent at some of the oiled sites. (Incardona et al. 2012). Cosco Busan bunker oil is similar to crude oil, but with a significant portion of the lighter compounds removed, leaving a higher proportion of multi ringed PAHs. Direct sunlight (UV) increases toxicity by changing ("activating") multi-ringed PAH structures, and is more of a problem to organisms that are less pigmented and transparent (such as fish embryos and larvae), because the UV can penetrate into cells with absorbed PAHs within them; this is known as "phototoxicity". This is not a problem at depth, or in turbid waters, as UV has very limited potential to penetrate. Under certain conditions, however (like when oil exposed embryos/larvae are in the top three feet near the surface, or exposed when the tide is out), the increase in toxicity can be an order of magnitude - hence it can be a serious issue to these sensitive life stages with little or no capacity to avoid the sunlight. A significant phototoxic effect was detected in the intertidal spawn compared to the spawn below the surface where turbid waters shielded embryos from UV exposure and the impacts of phototoxicity (Incardona et al. 2012). Two years later, the study was repeated at oiled and control sites; toxicity impacts from the spilled oil were no longer detected.

Like the embryo toxicity studies from Exxon Valdez, the Cosco Busan embryo toxicity studies detected damage at very low levels of PAH. The extreme sensitivity of embryos was well documented, as well as the cause/effect relationship. From these studies, it was apparent that even low environmental concentrations of PAH were potentially harmful to organisms and biological processes, and could impact important organisms beyond those species at the surface or at the shoreline. Thus, the Exxon Valdez and Cosco Busan studies established a scientific base from which DWH toxicity potential could be – and has been - further examined.

VI. Potential for Toxicity Damage from the DWH Spill

Simply put, for there to be a potential for toxicity damage, several criteria need to be met: significant volume of oil spilled, over a period of time, entry of toxic PAH components into the water, reaching habitats that support important species and sensitive life stages. Published reports from DWH researchers document that these criteria have been met.

A. Volume and Scope of the DWH Spill

1. The Volume of Oil Spilled and Dispersant Applied Were Record Breaking for US Waters

Approximately 4.2 million barrels of oil and gaseous hydrocarbons were released into the Gulf of Mexico over an 87 day period at about a rate of 53 thousand barrels per day (Phase Two Trial Findings of Fact; Crone and Tolstoy 2010; McNutt et al. 2011; Allan et al. 2012).[8] The DWH source oil contained approximately 3.9% PAHs by weight, one of the principal toxic classes, thus about 190,000 barrels of PAHs were released (Allan et al. 2012; Reddy et al. 2012). An oil plume rose through the water column and surfaced, but more than 2 million barrels of oil and methane remained in the deep sea (McNutt et al. 2011), thus also exposing organisms typically isolated from oil spills. In contrast, the Exxon Valdez spill, which caused biological damage for more than twenty years (Rice 2010; Balachey et al. 2014), was 1/20 of this volume and previously held the record for volume spilled in US waters. In 1979, the Ixtoc I blowout near Ciudad del Carmen in the Gulf of Mexico released nearly as much oil (3.3 million barrels) over a longer time period, but the release was in relatively shallow water, about 50 M. Damages were detected with the Ixtoc spill (Jernelov and Linden 1981; Rabalais et al. 1981; Rabalais and Flint 1983), but the spill effects were understudied and relatively little scientific information was produced for a spill of such large size.

In addition to the oil release, approximately 1.8 million gallons of chemical dispersant were applied during the DWH spill, and this release of chemical dispersant constituted one of the larger chemical spills in U.S. waters (Lehr et al. 2010). Some 18,000 barrels were applied subsea and 26,000 barrels at the surface (Lehr et al. 2010), presumably enhancing oil dispersions that facilitate solubilizing toxins and increasing their biological availability. The effectiveness of subsurface dispersant application is unknown as the proportion of escaping oil actually treated is unknown (Lehr et al. 2010).

2. The Spill Occurred over a Long Period of Time

The protracted release time (and volume) increased the spatial and temporal extent to which DWH oil would be in contact with organisms. The DWH spill continued for an unusually long period of time (87 days) and the volume released per day was greater than for any spill that continued for a week or more. Typical oil events are characterized by spillage over a single date, sometimes for several days, but it is a rare event that has spillage continuing past a week

[8] Five million barrels were released from the well, but 810,000 barrels were collected on site in the later part of the spill. See Phase Two Trial Findings of Fact.

or two. Once spilled, oil may persist in the environment for decades. However, only six reported spills in the world continued longer than the DWH spill. One of these was the Ixtoc I oil spill, which released nearly as much oil but over 294 days. The DWH release rate was thus greater than the Ixtoc release rate and it was about three times greater than the 1910 land-based Lakeview Gusher in California.

3. The Spill Covered Significant Geographical Areas: Water, Deep water, and Shorelines

The area oiled was extensive. The oil slick produced by the Deepwater Horizon oil spill covered as much as 29,000 square miles, an area about the size of South Carolina, on May 24, 2010, with the extent and location of the slick changing from day to day depending on weather conditions; a total overall oil slick footprint was estimated to be 68,000 square miles.[9] By June 21, 2010, the National Oceanic and Atmospheric Administration (NOAA) had banned fishing in about 36% of federal waters, or 86,985 sq mi of the gulf.[10]

The DWH spill was unique because the release point was 1500 m below sea surface, therefore the oil and gas entered cold seawater under high pressure and was hot (~180 F), creating ideal conditions for a physical dispersion; Lehr et al. (2010) estimated 10 to 30% of the oil was mechanically dispersed. Dispersants were applied at depth, and presumably enhanced the dispersion process of moving the oil into small droplets. Most importantly, the small droplets increase the bioavailability of PAHs to fauna in the water column. Some exposure may be through direct contact with droplets, such as oral intake by predatory organisms or the contamination of gill-like filaments in filter feeding organisms. However, the primary exposure route is through the increased solubilizing of PAHs that is promoted by the increased surface area with droplets, and the retention time in the water column of the droplets before they reach the surface.

Oil retention at depth, which was primarily caused by the formation of dispersed droplets of oil, was unprecedented in this spill, particularly at the scale observed (2 million barrels remaining at depth in large plumes). The retention of oil at depth was a function of the size of the oil particles (and buoyancy) which promoted the weathering of oil (transfer of PAHs and other compounds into the water). The retention of plumes of oil dispersed particles at depth fueled speculation of toxicity damage at depth, and was supported by surprisingly high

[9] Cleveland, Cutler, "Deepwater Oil Spill," Encyclopedia of Earth (2010, updated 2013) at http://www.eoearth.org/view/article/161185/

[10] "NOAA Expands Fishing Closed Area in Gulf of Mexico" at http://www.noaanews.noaa.gov/stories2010/20100621_closure.html

PAH concentration measurements (discussed later in the section on toxicity damage potential at depth). Consequently, the deep habitat and fauna were exposed to toxic components.

B. Oil Was Detected in the Shoreline and Upper Water Habitats

1. Shoreline Oil

About 25% of the shoreline was visibly oiled (Fig. 5) within the affected area, 1100 of 4386 miles (Michel et al. 2013). These estimates are from SCAT surveys (Shoreline Clean-up Assessment Team), and are conducted to identify areas appropriate for shoreline clean-up, not to evaluate the environmental harm caused by the spill. These surveys indicate the large area that was impacted by the spill to some degree, and correlate well with the extent of the slicks (Fig. 6). Louisiana shoreline had the most oiling, about 61% (Michel et al. 2013). Oil was remobilized multiple times on some beaches as beaches went through normal erosional and depositional cycles (Michel et al. 2013; Wang and Roberts 2013).

While the spatial distribution of oiled shoreline was extensive, indicating the potential for toxicity exposure, it is more difficult to determine the significance of the exposure from the SCAT surveys alone. Five types of contamination were observed: tar balls, tar patties, tar cakes, oil sheet, and stained sand (Wang and Roberts 2013), but they vary dramatically in terms of oil volumes, bioavailability, and toxicity potential. For example, heavily weathered tar balls are nearly inert, with little exposure potential unless they have liquid oil centers that can release oil if punctured.

There are quantitative reports of exposure and toxicity damage. Seven months after coastal oiling began, total petroleum hydrocarbon concentrations in the surface 2 cm of heavily oiled marsh soil ranged up to 510 mg/g (Lin and Mendelssohn 2012). Heavy oiling caused nearly complete mortality of the saltmarsh plants *Spartina alterniflora* and *Juncus roemerianus*. The latter was more sensitive; moderate oiling significantly lowered live above-ground biomass and stem density. Killifish exposure and impacts in oiled marsh were detected in 2010 by Whitehead et al. (2012), and confirmed in the following year by Dubansky et al. (2013).

2. Oil Was Detected in the Upper Water Column

Surface water (upper 50 m) was extensively oiled by DWH and as result was contaminated with PAHs (Fig. 7). More than 300 of the 650 samples collected from the top 50 m in May contained measureable amounts of oil (NRDA publically available data). Contamination in this habitat is important, as this is the productive biological habitat, home to many fishes, as well as important spawning habitat. Contamination of this habitat is not

surprising, as the oil had to rise through these waters, in the form of dispersed droplets and slicks that were formed were often hit by surface application of more dispersants. The concentration of the oil is important, and the number of oiled samples indicates there were multiple opportunities for drifting embryos and larvae to encounter PAHs.

C. Concentrations of PAHs Were Significant in Many of the Oiled Water Samples.

The PAH concentration was ≥ 0.5 ppb in about half of the oiled water samples (NRDA publically available data), hence many of these samples approach the threshold concentrations that may harm developing fish embryos. These samples were taken from a large geographical area; oil was detected in surface seawater in a roughly 22,000 km2 area in May (NRDA publically available data). This increased to about 45,000 km2 in June and 100,000 km2 in July. Oiled water samples were located in the vicinity of the DWH well in May. By June oil had also spread to the shoreline and this continued in July and August. By October, most oiled water samples were located inshore, though a few offshore surface water samples contained oil through the remainder of the year.

Thus, oil concentrations that can be harmful to sensitive life stages (e.g., less than 1 ppb) were present in the upper 50 m, over a wide geographic area, from May through July (NRDA publically available data). The distribution of oily water was uneven, but substantial, with multiple opportunities for developing embryos and larvae to encounter oil in their spawning habitat. Small embryos and larvae have extremely fast equilibrium times to absorb PAHs in the "uneven patches" of oil water (in minutes); hence uneven patchiness is not as important as the geographical spread of the oily water which determines the number of opportunities to encounter PAHs.

D. Oil Composition Was Similar to Other Toxic Oils

DWH oil contains saturated hydrocarbons (74%), aromatic hydrocarbons (16%), and polar hydrocarbons (10%) (Reddy et al. 2012), similar to other crude oils. The toxic aromatic fraction includes the BTEX (single ring) compounds was about 12% and PAHs were about 4%. (Allan et al. 2012; Reddy et al. 2012). Thus, DWH oil is comparable to other crude oils in PAH composition and is not unique, with the caveat that PAH composition changes dramatically as oil released into the environment and weathers. Side-by-side comparison reveals that PAHs present in DWH oil, Exxon Valdez oil, and others are compositionally similar, thus DWH oil poses similar toxic risks (Fig. 8).

E. Application of Dispersants Increased the Toxicity of the DWH Oil

Studies into the impacts of effects on the water column from dispersant use in the DWH spill response are ongoing. Chemical oil dispersant is at least one order of magnitude less toxic than oil to most fauna, possibly more when the part per billion sensitivities of embryos are considered. The median lethal concentration (LC50) of Corexit 9500A is about 42 to 130 uL/L (parts-per-million) in mysid shrimp (*Americamysis bahia*) and an estuarine fish, Inland silverside (*Menidia beryllina*), respectively (Hemmer et al. 2011). Chemically dispersed Louisiana crude oil was more toxic; LC50s were 5.4 to 7.6 mg TPH/L for mysids and silverside, respectively, where Corexit 9500A was the dispersant and TPH is total petroleum hydrocarbons (Hemmer et al. 2011). These authors found mechanically dispersed Louisiana crude oil was more toxic, 2.7 – 3.5 mg/L for the two test species. However, the probable toxins, PAHs, likely comprise only a small fraction of the reported TPH and the authors did not characterize PAH composition in their bioassays. Dispersants are less toxic than the PAHs in the oil, and they were used at levels that were much less than the volume of oil. While the increase in PAH toxicity due to the large volume of dispersants applied during the DWH is thus not yet precisely quantified, it is clear based on existing literature that dispersants increased the toxicity of the oil spilled.

F. Demonstration of Toxicity from Important DWH Studies

Many species were likely affected to some degree, but the most compelling evidence lies with embryo toxicity studies on both inshore and off shore species. The pre-DWH literature has been documenting the extreme sensitivity of fish embryos over the last 2 decades to PAHs, and DWH researchers took advantage of those studies and focused on fish embryos (studies embryos of bluefin tuna, yellowfin tuna, amberjack by Incardona et al. 2014; on mahi-mahi by Mager et al. 2014, and on killifish by Dubansky et al. 2013).

1. Pre-DWH Embryo Toxicity Studies

That embryo damage takes place at low ppb (parts per billion) concentrations of PAH became apparent during study of the Exxon Valdez oil spill, with the observations of elevated embryo mortalities in the wild pink salmon (Bue et al. 1996; Bue et al. 1998) and confirmed later in controlled laboratory exposures at ppb concentrations of PAH that created the same embryo mortalities. Part per billion exposures to PAHs are low, and are environmentally relevant, meaning they can be achieved under spill conditions. The embryo damage syndrome (pericardial and yolk-sac edema) was known much earlier, (e.g., Linden 1978) but by the time the Exxon Valdez spill occurred the environmental problem compounds were more clearly

defined; mono-aromatic hydrocarbons were lost to the atmosphere in a few days, leaving the more persistent and toxic PAHs to cause lasting damage. Research attention turned to PAHs and led to a series of studies demonstrating embryo toxicity at low part-per-billion (µg/L) aqueous total PAH concentrations (Marty et al. 1997; Heintz et al. 1999; Heintz et al. 2000; Carls et al. 2005). Most important, the laboratory studies, with detailed chemistry and low exposure levels, confirmed the findings in the field, where elevated embryo mortalities were found in pink salmon redds (Bue et al. 1996; Bue et al. 1998). The population modeling estimated a loss of 2 million returning adult salmon (Geiger et al. 1996). Further, the oil exposures in the field had to be "indirect" and low, but because of oil persistence, PAHs were still available in some intertidal streams for several years (Murphy et al. 1999), and the detailed laboratory exposures confirmed that low environmental doses could cause the embryo toxicity to pink salmon. Embryo toxicity studies with Pacific herring also found low part per billion levels of PAH were damaging to embryos (Carls et al. 1999). Since then, the extreme sensitivity of embryos from several different species to low concentrations of PAH have been independently verified by others (Hawkins et al. 1990; White et al. 1999; Birtwell and McAllister 2002; Rhodes et al. 2005; Farwell et al. 2006; Olsvik et al. 2011), including work originating with the DWH spill (Incardona et al. 2014; Mager et al. 2014).

Since Exxon Valdez, many studies have not only found the pericardial yolk sac edema in fish embryos, but have examined the developmental toxicity in detail, both biologically and with different constituents of oil (Marty et al. 1997; Carls et al. 1999; Incardona et al. 2004; Incardona et al. 2006; Hodson et al. 2007; Incardona et al. 2009; Hicken et al. 2011; Incardona et al. 2011; Scott et al. 2011; Turcotte et al. 2011; de Soysa et al. 2012; Fallahtafti et al. 2012; Brette et al. 2014). These functional studies have identified cardiac development as a key process impaired by PAHs, particularly 3-ring (tricylic) compounds, causing anatomical malformations and functional defects that likely diminish cardiac output in association with bradycardia and arrhythmia. Cardiac function develops early in the embryo, and the normal development of many organs is dependent on the proper blood flow with nutrients from the yolk and oxygen from the surface for proper development. At higher exposure levels, embryos will develop severe cardiac failure from edema and jaw deformities (Hodson et al. 2007; Incardona et al. 2009), and are unlikely to survive past the yolk sac stage to become free swimming and feeding larvae (Carls et al. 1999). At lower exposure levels, there will be gradations of cardiac edema and impact, but the impacts, such as poorly shaped heart, can persist to adulthood (Hicken et al. 2011). Lower exposures can produce a lower functioning heart that leads to slower development and wide spread but low level damage throughout the embryo, all lowering the chances for survival in the environment through impacts on growth rates (Heintz et al. 2000; Carls et al. 2005), swimming ability (Hicken et al. 2011), and reduction of marine survival (Heintz et al. 2000).

Embryo toxicity is different from acute narcosis toxicity: (1) the mechanisms at the cellular level are different, (2) dose levels that cause embryo toxicity are orders of magnitude lower, and (3) the impacts are more difficult to detect as the embryo can often be alive for a period of time during the yolk resorption phase, which delays until later the critical time and challenges for survival when all of the organ systems become co-dependent, and have to function at a higher integrated level to forage and avoid predation. With acute narcosis, the long standing toxicity paradigm focused on 1 and 2 ring aromatics (Peterson et al. 2003); in embryo toxicity, the focus is on the 3 ring PAH, and larger, and the observable consequence is usually not evident for some time as the embryo continues to develop. Recent studies have also shown that the nervous system may be a target of oil toxicity in fish, as heavy fuel oil exposure (composition similar to crude oil with the lighter fractions removed) in marine fish embryos led to abnormal projections of cranial and peripheral nerves (Irie et al. 2011; Kawaguchi et al. 2011).

Constituents in crude oil, particularly the three ringed PAHs, cause embryo damage characterized by cardiac damage, edema, malformations, hemorrhaging, anemia, cell death, reduced growth, heritable reproductive effects, and impaired fitness (Marty et al. 1997; Billiard et al. 1999; Carls et al. 1999; White et al. 1999; Billiard et al. 2002; Barron et al. 2003; Brinkworth et al. 2003; Incardona et al. 2004; Incardona et al. 2005; Colavecchia et al. 2007; Dubansky et al. 2013). Pure compound bioassays replicate these problems and help narrow down specifically which compounds are problems, such as a study by Incardona et al. (2004), which clearly identified fluorenes, dibenzothiophenes, and phenanthrenes as causal. Another approach to understanding the identity of damaging compounds is to fractionate polluted source material; for example, Sundberg et al. (2005) extracted PAHs and other compounds (such as polychlorinated biphenyls, PCBs) from polluted sediment and fractionated the extract into multiple subfractions; the fraction mainly composed of PAHs was more teratogenic than the fraction containing di-cyclic aromatics and PCBs when injected into trout embryos.

2. DWH Fish Embryo Toxicity Studies

a) Incardona and Mager Studies

Two DWH studies have confirmed that low part per billion PAH exposures caused cardiotoxic effects to Gulf fish species, and corroborate the earlier work on pink salmon and herring relative to Exxon Valdez and Cosco Busan. Incardona et al. (2014) observed toxic effects to developing hearts of Bluefin tuna, yellowfin tuna, and amberjack at low exposure levels of DWH PAH (Figs. 9a and 9b). These controlled laboratory exposures used environmentally relevant exposure levels (see *infra* Section IV.C.). The observed heart defects were not

surprising because previous tests compared the embryo toxicity of both Alaska North Slope crude oil and DWH oil to zebra fish embryos; pericardial edema and a suite of other effects occurred at similar dose levels, and the PAH compositions were similar. This is consistent with the low part-per-billion observations determined for fish exposed to Exxon Valdez oil, and other studies such as Cosco Busan spill.

The significance of low level part per billion exposures to sensitive developing embryos was evident in the tests with mahi-mahi when juveniles were reared post exposure and swimming performance was tested (Mager et al. 2014). Embryos were affected at low levels (1.2 ppb PAH for 48 hours) with increased rates of pericardial edema. However the most significant effect was measured later after rearing to the juvenile life stage, where swimming performance was by reduced by 37%. Brief exposures (1 day) of juvenile mahi-mahi to water-accommodated fractions of oil (30ppb total PAH) also were effective in reducing their critical swimming speed by 22% (Mager et al. 2014). Fast swimming speeds are critical for foraging, and during the juvenile stages, which are very important for avoiding predators. All of these apex[11] fish predators with sublethal cardiac damage incurred during the embryo life stage would have a lower probability of surviving to juvenile stages and on to adult stages.

b) Low part per billion PAH Levels Are Environmentally Relevant

The low part per billion PAH exposure levels found to be toxic to fish embryos in the laboratory (Incardona et al. 2014, Mager et al. 2014) were also found in the upper 50 m of the surface waters from May through July 2010 (NOAA NRDA publically available database). This is not surprising given the large surface footprint of the spill and the volume of dispersants applied at depth and at the surface, over a long period of time. The physical nature of the spill, including application of chemical dispersants, made the solubilizing of PAH from the dispersed oil droplets a high probability. Further, Incardona et al. (2014) reports that given the large footprint of the oil spill, over a lengthy time period, there was considerable overlap with the upper spawning habitat for these species and others. These are all important target species, but they also are surrogates for the hundreds of species not examined, which also contribute to the ecosystem at various trophic levels, from forage fish to predators.

[11] Apex predators are those at the top of their food webs, for example, lions, polar bears, and killer whales. For fish, it is the fast swimming predators like tunas.

c) Inshore Fish Habitat Impacted, for At Least a Year

Biological damage and oil exposure was observed in killifish at oiled sites (Grand Terre, LA); gene expression of thousands of genes were profoundly different between oiled and unoiled sites (Whitehead et al. 2012). Killifish collected from an oiled site (Grand Terre, LA) demonstrated proof of oil exposure with up-regulation of P450 genes in liver and gill tissues that corresponded with the arrival of oil. Fish from the oiled site had divergent gene expression in liver and gill tissue coincident with the arrival of contaminating oil (and different from the samples prior to oiling and from samples from non-oiled reference sites). The significance of divergent gene expression is difficult to assess; this is an emerging new technique that is extremely sensitive. Of the 3296 genes examined, about half varied in response to the arriving oil, and many were still divergent 2 months after the peak arrival of oil. While gene expression differences were different between oiled and unoiled fish, persisting for at least two months, there is no assessment that indicates the survival or fitness was decreases by the exposure, but that is suggested. The fish sampling design was excellent; fish were collected prior to oil land fall, at peak land fall, and two months post peak landfall of the oil at Grand Terre and at non-oiled reference sites.

A year later, much of this field test was repeated (with continuing genomic effects), but with the addition of laboratory exposures of killifish embryos to sediments from Grand Terre (Dubansky et al. 2013). Genomic response continued at the Grand Terre oiled site compared to reference sites; a stronger response was detected in gill-specific genes compared to those in liver tissue, perhaps reflecting the organ's direct contact with the environment. Oil contaminated sediments collected at Grand Terre were exposed to embryos, and the sensitive embryo toxicity impacts on developing hearts was also found in developing embryos; a year post spill, the sediments still contained enough PAH to impair embryo development.

G. DWH Crude Oil Is Not Unique in Causing Embryo Toxicity at Low Levels

Using zebrafish embryos, Incardona et al. (2013) explicitly demonstrated DWH and Alaska North Slope Crude Oil had similar PAH compositions, and similar effects on cardiotoxicity, cytochrome P450 induction, and morphological defects. The origin of the oil did not matter; the effects were largely indistinguishable by oil source and generally correlated with PAH composition. These results are predictable because PAHs are the common toxic currency among oils. The geologically similar Ixtoc I crude oil was highly toxic to the pelagic eggs of red drum (*Sciaenops ocellatus*) (Rabalais et al. 1981). Embryo toxicity with DWH at low concentrations has been confirmed in other studies and species, such as gulf killifish, bluefin

tuna, yellowfin tuna, amberjack, and mahi tuna (Dubansky et al. 2012; Whitehead et al. 2012; Dubansky et al. 2013; Incardona et al. 2014; Mager et al. 2014)

H. Toxicity Potential in Deep Water as a Result of the DWH Spill

The potential for toxicity damage to this deep habitat was proved by detection of high oil concentrations, as well as some impacts. This habitat is very difficult to study, requiring specialized equipment (submersibles), resulting in a habitat and impacts that are understudied. Previous blowouts, like Ixtoc I in 1979, were in much shallower water, with the oil rising quickly to the surface without the formation of dispersed particles at depth. Oil effects literature from shallower habitats indicates the likelihood of toxicity damage, even if specific organisms and early life stages from deep water species have not been tested.

1. Oil Was Detected in Deep Water

Potentially damaging hydrocarbon concentrations were observed at depth in Gulf of Mexico water. A persistent, continuous subsurface plume of oil extended 10 to more than 35 km from the well head at about 1100 meters (Camilli et al. 2010; Diercks et al. 2010; Hazen et al. 2010). The hydrocarbon maximum at 35 km was only 53% less than that at 5.8 km from the source, thus the plume likely extended considerably beyond survey bounds. Publically available NRDA data indicate that the plume extended about 500 km in a NE – SW direction by September; PAH concentrations were ≥ 0.5 µg/L or greater in some samples over this distance .

High PAH concentrations of up to 189 µg/L were observed in plumes between 1000 and 1400 m extending at least 13 km from the DWH well in May 2010 (Diercks et al. 2010). These concentrations were likely acutely toxic; Diercks et al. (2010) concluded that with dispersants present the effects on the deep sea ecosystem may have been severe. About 6 to 7% of all BTEX leaked from the well was required to support the plume.[12]

The subsurface oil plume generally migrated southwest and covered an area >73,200 km^2 in the deep Gulf of Mexico, as estimated by dissolved oxygen anomalies (Du and Kessler 2012), (Fig. 10) as well as fluorometry measurements (JAG 2012),(Fig. 11). The oxygen anomaly was caused by microbial growth; methane was likely the dominant hydrocarbon controlling respiration rate (Du and Kessler 2012). Chemical dispersant apparently accelerated carbon respiration but there were saturation dispersant quantities above which no further increase in respiration was observed (Du and Kessler 2012).

[12] Natural seeps could not be the source because even if all natural seeps in the Gulf of Mexico were flowing into the plume, they would explain less than half the BTEX observed (Camilli et al. 2010; JAG 2012).

2. Oil Was Detected in the Benthic Seafloor Habitat

About 24.4 km^2 of seafloor habitat around the wellhead was strongly impacted and additional 148 km^2 was moderately impacted (Figs. 12a and 12.b); recovery may take years, decades, or longer (Montagna et al. 2013). The deep-sea plume was as much as 200 m thick and 2 km wide in some locations, providing a potential mechanism of DWH hydrocarbon transfer to deep-sea communities (Camilli et al. 2010; Montagna et al. 2013). Coincidence of the plume identified by Du and Kessler (2012) (Figs. 10) and the benthic impact area (Montagna 2013, Figs. 12a., 12.b), suggests the actual impact area is larger than estimated by Montagna et al (2013).

3. Biological Impacts Were Detected at Extreme Depths

Visual evidence of damage to deep water corals and brittle stars was detected 11 km southwest from the well head (White et al. 2012), at a depth of 1370 m. The supporting chemistries of the corals and surrounding sediments provide compelling evidence the damage was related to DWH oil. In another study, two other communities of corals were found to be impacted by the DWH oil, at 6 and 22 km from the wellhead (Fisher et al. 2014). At the 6 km site, over 90% of the corals were impacted. Corals, with their filter feeding structures, are vulnerable to contamination from small dispersed oil droplets. Impact to other known coral beds was extended out and the depth and the direction of the damage are consistent with known plumes of oil from DWH. Further, this damage will be long term, as the dead coral was carbon dated to be approximately 460 years old (White et al. 2012). Other studies are suggestive of damage to megafauna near the well head (0.5, 2 km), where populations appear to be suppressed, but pre-spill population numbers do not exist, thus demonstrating some of the difficulties of assessing impacts at these extreme depths.

4. Toxicity Damage to the Benthic and Deep Water Habitats Was Likely

Oil was detected in high concentrations. Plumes of dispersed oil were detected many kilometers from the well head; there was a large footprint of area potentially impacted by the plumes. Damage assessment studies at depth are limited, but damage to deep water corals and other megafauna were detected. Given the high concentrations of PAH detected, the area covered by plumes, and the length of exposure from these plumes, toxicity damage is very likely. Filter feeders like corals will be particularly vulnerable to dispersed oil droplets. Because some of these fauna are very slow growing, such as the corals that are hundreds of years old, full recovery back to pre-spill conditions will likely take centuries.

VII. Conclusion

There are multiple overlapping compelling reasons to believe there was significant potential for toxicity damage from the DWH spill. An enormous quantity of oil was spilled (about 4.9 million barrels), far larger (about 20 times) than the next largest spill in US waters (Exxon Valdez), where oil persistence and biological damage were documented for over 20 years. Release of oil continued an unusually long period of time (87 days), thus increasing the potential for exposure, both spatially and temporally. High pressure release at depth caused mechanical dispersion (and this was augmented by unprecedented release of chemical dispersant at depth), thus providing an efficient and effective way of dispersing the oil into small droplets, thus enhancing the retention of oil at depth and enhancing the movement of PAHs into the water column for bioavailability. Large areas (and volumes) of the Gulf of Mexico were oiled, including shoreline habitat, surface water, deep water, and the benthos.

DWH crude oil contains PAHs, similar to other crude oils throughout the world, where studies have demonstrated their chemical toxicity, particularly to early life stages. PAH compounds were detected in the water column, confirming exposure potential to early life stages. Early life stages, such as developing embryos, are easily damaged, as shown by DWH studies as well as with other oils, thus concern is focused on the weak link in the life cycle of fish and invertebrates. Based on previous spills, as well as from the early papers on DWH, the damage at the surface, shoreline, and to embryos is expected, and corroborated. There is significant overlap between the area oiled (concentrations near 1 ppb) during the spill months and known spawning habitat of many off shore fishes.

In contrast to other spills, there was significant and unprecedented chemical contamination in the deep waters, with plumes of dispersed oil tracked out for several kilometers, over a significant length of time. High concentrations of PAH were detected at depth, and biological damage to corals and brittle stars were detected. Damage to deep corals has been detected at three sites (from two studies). The filter feeding strategy of corals makes them particularly vulnerable to exposure through the capture of dispersed oil droplets. Pre-spill information is generally not available, so population impacts are difficult to detect and assess. Linking oil exposure with dead corals by chemical detection of DWH oil in the surrounding sediments is consistent with the path and depth of plumes of dispersed oil; together the evidence is compelling for toxic damage in the deep waters. Detecting damage at extreme depths is a difficult task, and it is likely that more damage occurred than was detected. Unfortunately, the damage at depth will likely be very long term, possibly taking centuries for corals to recover to pre-spill conditions because corals grow very slowly and live hundreds of years.

Given the large volume of oil, over a lengthy period of time, and the introduction of oil particles into the water column, the potential for toxicity damage was expected. Studies have documented exposure at depth, in the water column, surface, and shorelines. Studies have corroborated toxicity impacts at depth, in the water column, at the surface, and the shoreline. As research into the natural resource damage assessment continues, the potential to find other toxic effects of DWH crude oil on marine life in the Gulf similar to toxic effects observed from exposure to other crude oil is very likely.

VIII. Figures

Fig. 1. Representative hydrocarbons typically present in crude oil. Carbon atoms are located at each juncture. Hydrogen bonds are not depicted except in substitution examples.

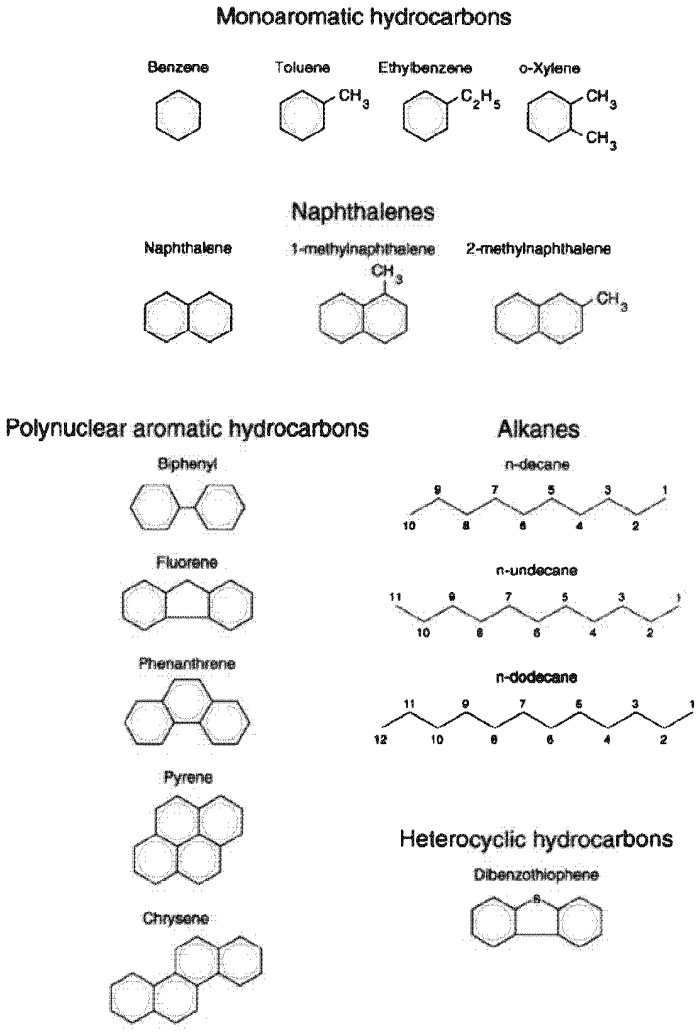


Fig. 2. Relationship between log Kow and molecular weight. The octanol-water partition coefficient is the ratio of the concentration of a chemical in octanol and water at equilibrium. Octanol is an organic solvent that is used as a surrogate for natural organic matter such as oil and is used in many environmental studies to help determine the fate of chemicals in the environment. The octanol-water partition coefficient has been correlated with water solubility. The molecular weight range illustrated spans PAH masses typical in crude oil. A few example compound names are noted in the figure, naphthalene (N0), fluoranthene (FLU), C4-phenanthrenes (P4), and C4-chrysenes (C4).

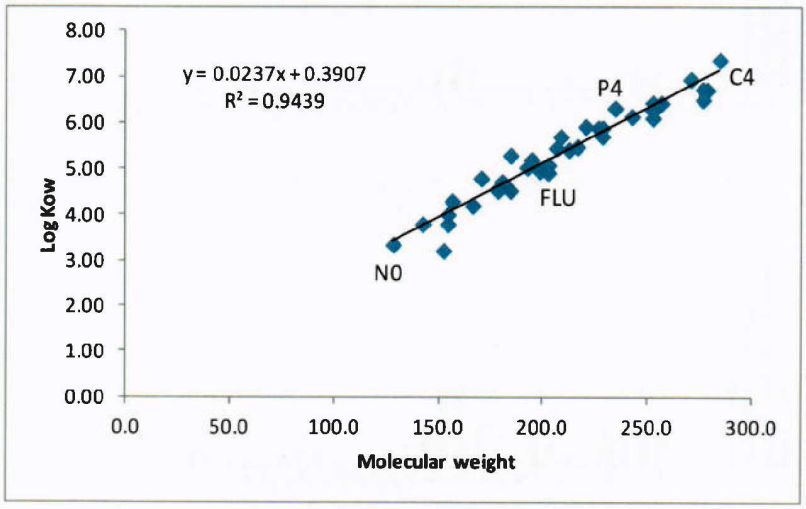
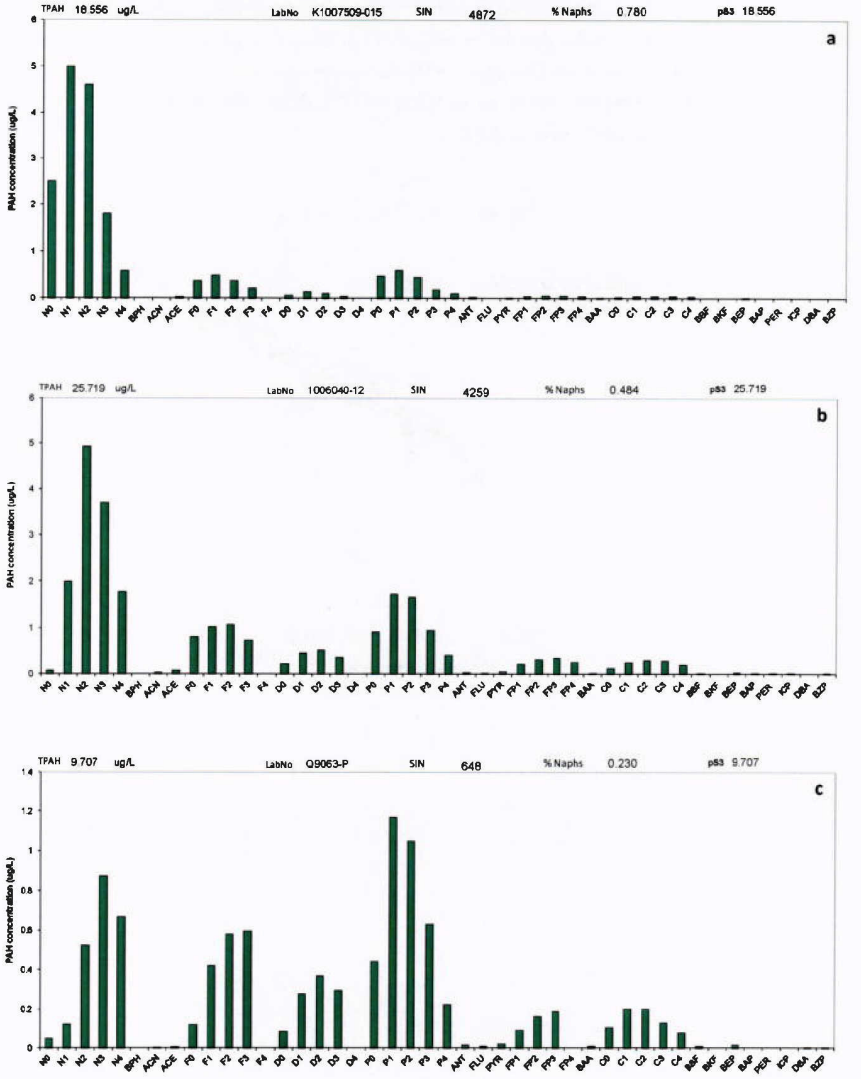
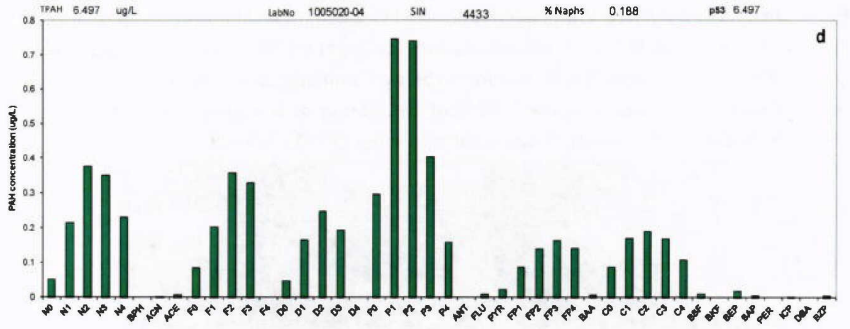
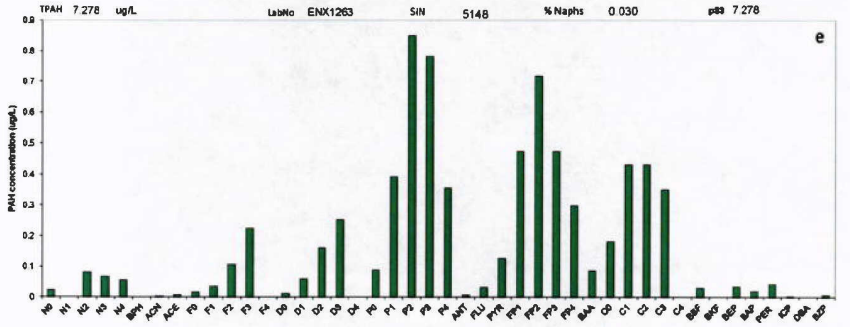


Fig. 3. Typical PAH weathering change. Example from oiled DWH water samples. Note the relative disappearance of the light ends (far left) and the relative increase in percentage of the larger heavier compounds (right side).







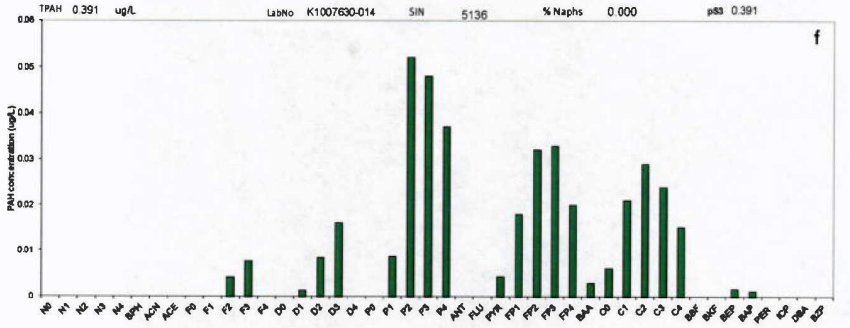


Fig. 4. Chemically Enhanced Water Accommodated Fraction (CE-WAF) showing application and mixture of Corexit 9500A with Southern Louisiana Crude at a 1:10 ratio of dispersant to oil (2.5 g/L dispersant to each bottle) and resulting dispersion 4 minutes later. Hemmer, et al. "Toxic Soup? Methods for Determining the Toxicity of Oil Dispersants and Dispersed Crude Oil," US EPA ORD, NHEERL, Gulf Ecology Division, Bates Number EPF225-002498

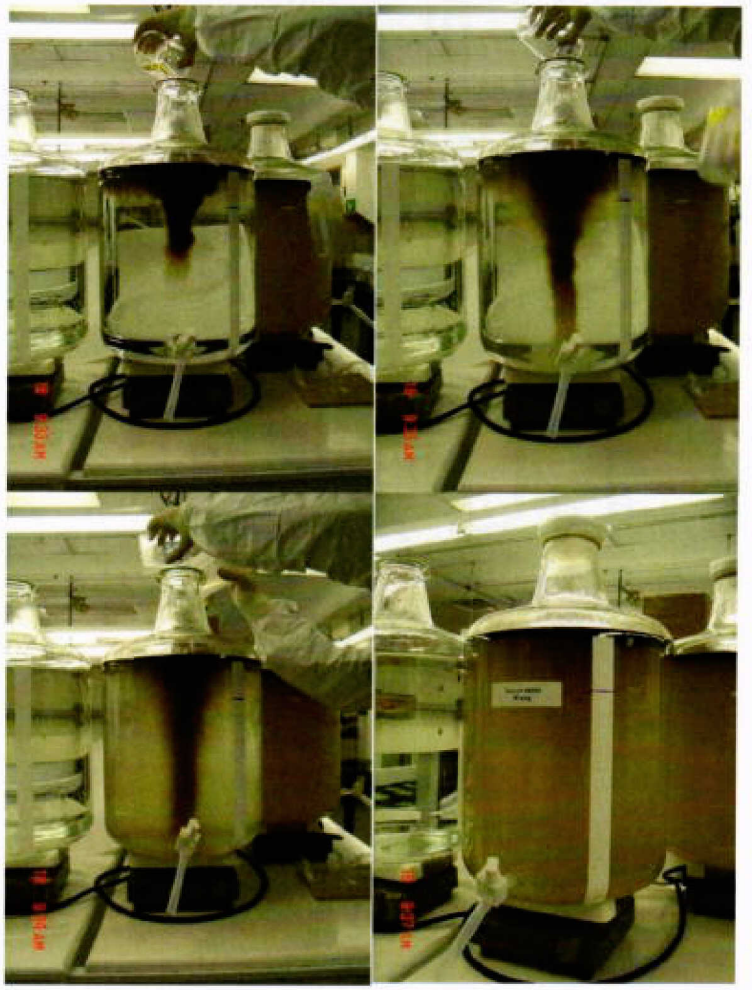


Fig. 5. Shoreline oiling as estimated visually from the Shoreline Cleanup Assessment Teams (SCAT) (Michel et al. 2013). The top panel summarizes the highest degree of oiling observed (2010). The middle panel is 1 year post-spill, and the bottom panel is 2 years post spill. Red depicts the heaviest oiling; blue represents no visual oiling observed. The Table below provides lengths of visibly oiled shoreline.

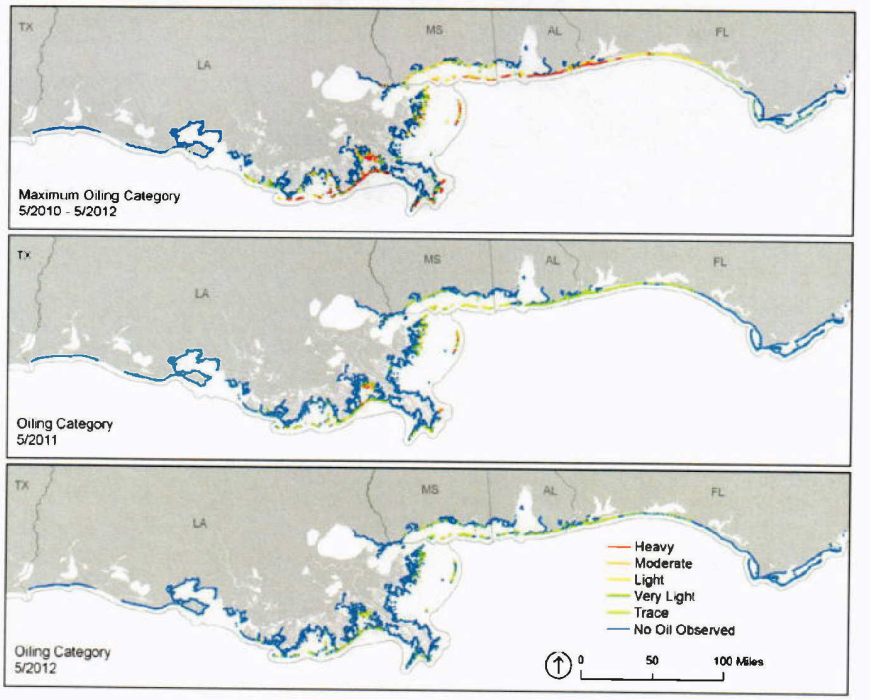


Table z (Michel et al. 2013). Oiled shoreline lengths (km) by visible oiling category at maximum oiling conditions, one year (May 2011), and two years (May 2012) post spill[1].

Length (km)	Total Surveyed	Heavy	Moderate	Light	Very Light	Trace (~1%)	Total Oiled	No Oil Observed
Maximum Oiling	7058	360	222	637	322	232	1,773	5,285
One Year Post-	6967	22.4	56	178	131	459	847	6,120
Two Years Post-Spill	7057	6.4	17.5	91.6	83.7	488	687	6,370

Values rounded to nearest whole km. when greater than 100 km.
[1]Shoreline oiling along the Texas coast was surveyed only once and using a slightly different approach, with a reported 58 km of trace oiling. doi:10.1371/journal.pone.0065087.t001

Fig. 6. Relationship between oiled shoreline and maximum slick extent in 2010. (Source: publically available ERMA data.)

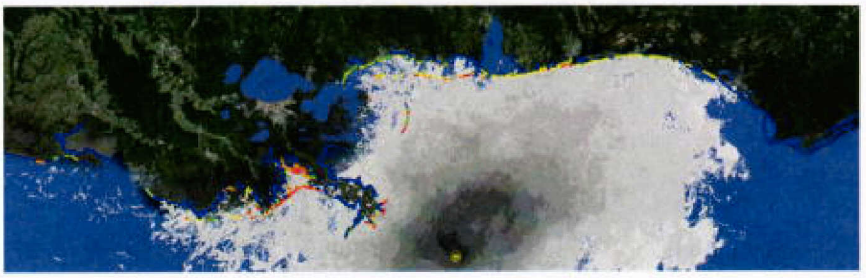


Fig. 7. Confirmation of oil in water samples taken in 2010 from the top 50 m (top panel), and comparison of oiled water samples from the top 50 m with the rest of the water column (bottom panel). Oiling in the top panel was confirmed by two modeling procedures; Sample in blue by source oil modeling and percent accepted as oiled based on modeling, PAH concentration, and PCA (green, top panel). The total number of samples analyzed each month is listed along the x-axis; note that effort differed by month, and that the sampling was not random. The bottom panel indicates the depth distribution of oiled samples; surface (0-50 m, blue) and subsurface (gray). All chemistries were taken from the publically available NOAA NRDA database.

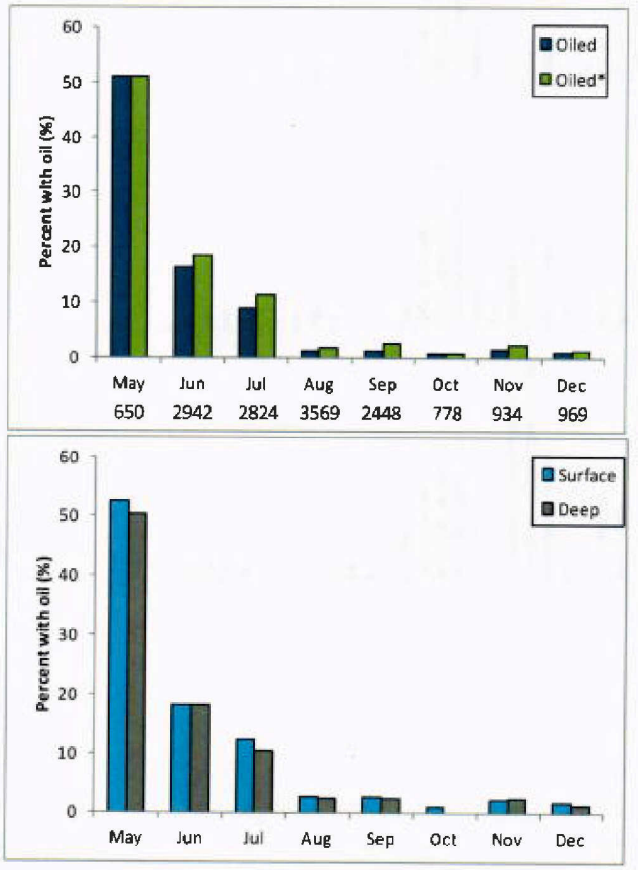
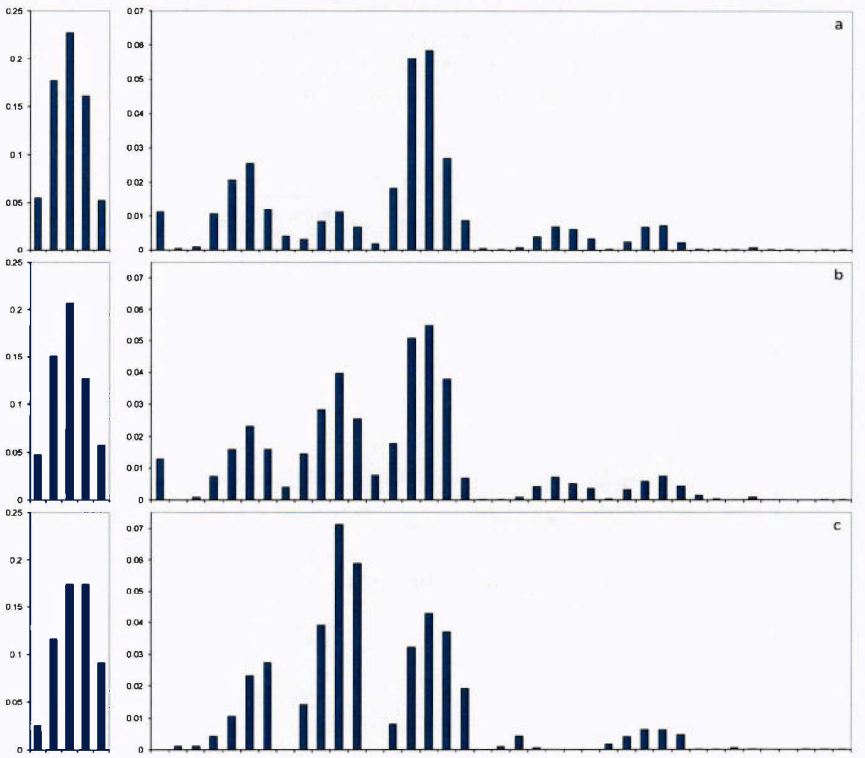


Fig. 8. PAH composition in DWH oil and other crude oils: a) DWH source oil (NIST), b) Exxon Valdez crude oil (Alaska North Slope crude oil), c) Iranian Heavy crude oil (Jung et al. 2013). The y-axis is a proportion of individual PAH, and expressed as a percentage of the total PAH The x-asis is progression from 2 ringed PAH to 3 and 4 ringed PAH at the right.



Figs. 9.a, 9.b. Observed gross morphology of hatching stage bluefin, yellowfin, and amberjack tuna larvae exposed to MC252 in 12.a (top); oil-induced circulatory failure and corresponding edema in bluefin, yellowfin, and amberjack tuna embryo hearts in 12.b (bottom). Incardona et al. 2014.

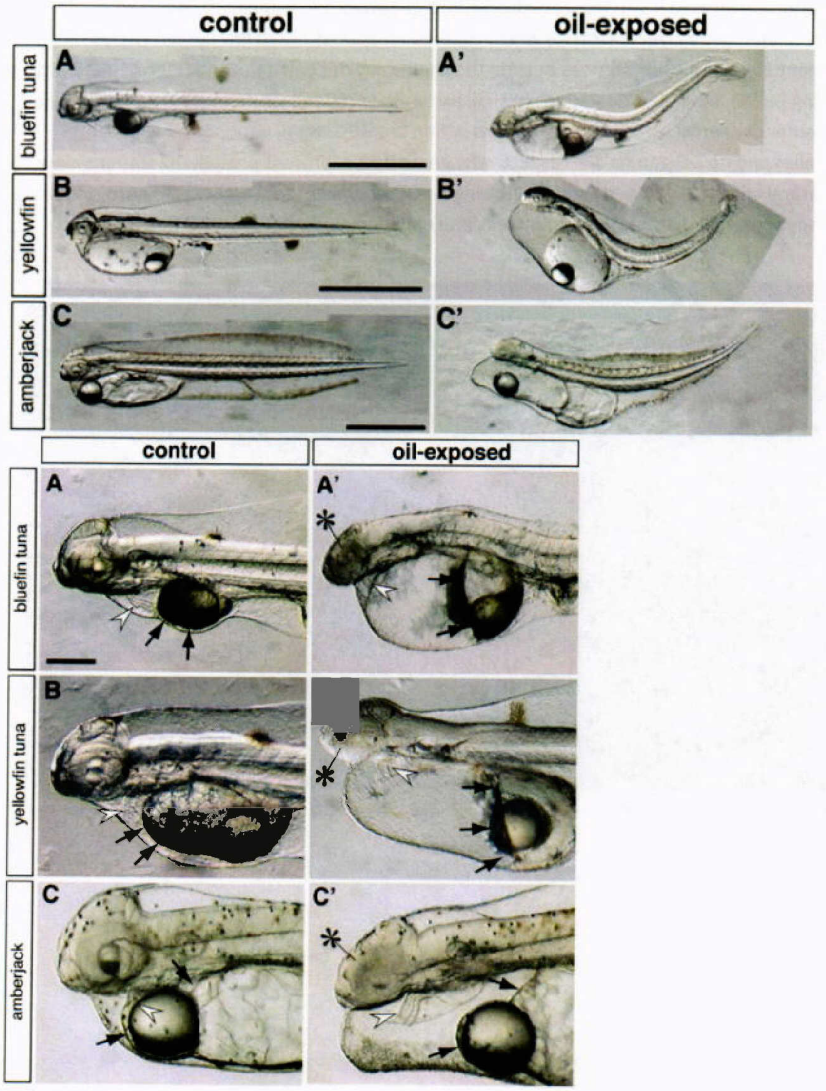


Fig. 10. Deep and intermediate plume location as evidenced by anomalous dissolved oxygen (DO) levels. (Du and Kessler 2012). "A red star represents the wellhead; pink triangles represent natural seeps (MC118, GC600, and GC185); yellow circles represent 936 during-spill DO stations in an area impacted by hydrocarbons dissolved and trapped in deep and intermediate plume layers (>700 m) which covered an area of 73200 km^2; green circles represent 354 during-spill stations outside the chemically defined deep and intermediate plume showing no DO anomalies or stations of shallow depth (<700 m); white (n = 19) and orange (n = 20) diamonds represent during-spill stations within the Mississippi Canyon showing DO anomalies and no DO anomalies, respectively; green dots represent prespill DO stations close to the natural seeps (GC185 and GC600) showing no DO anomalies (see Figure 2B); purple squares represent postspill DO stations within the Mississippi Canyon showing no DO anomalies."

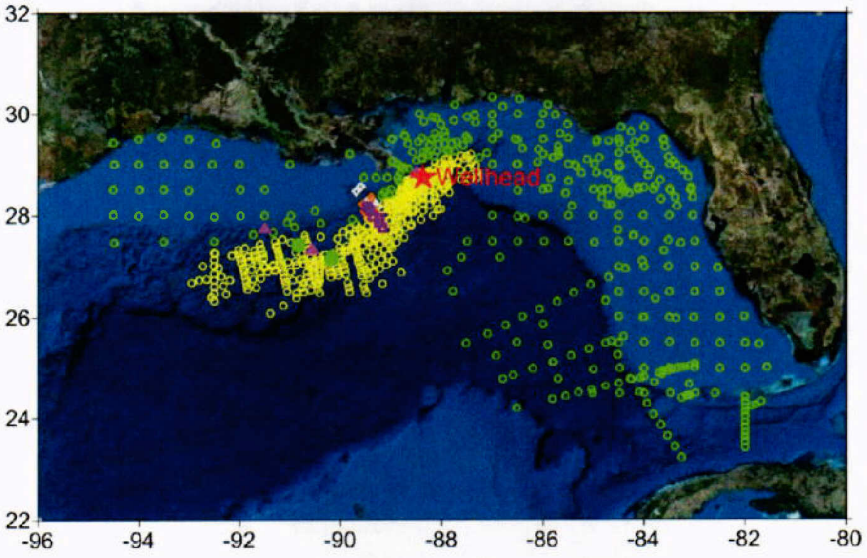
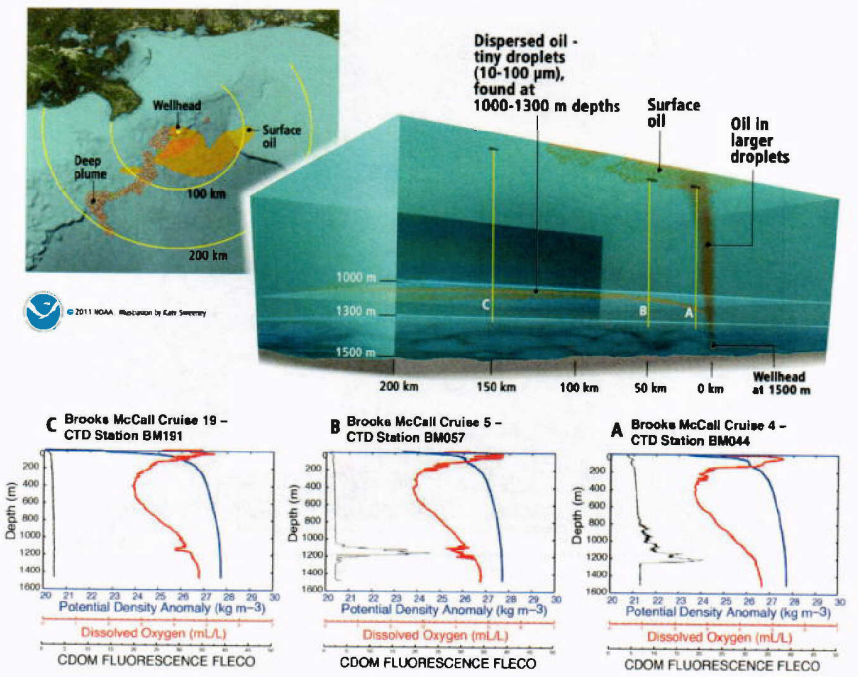
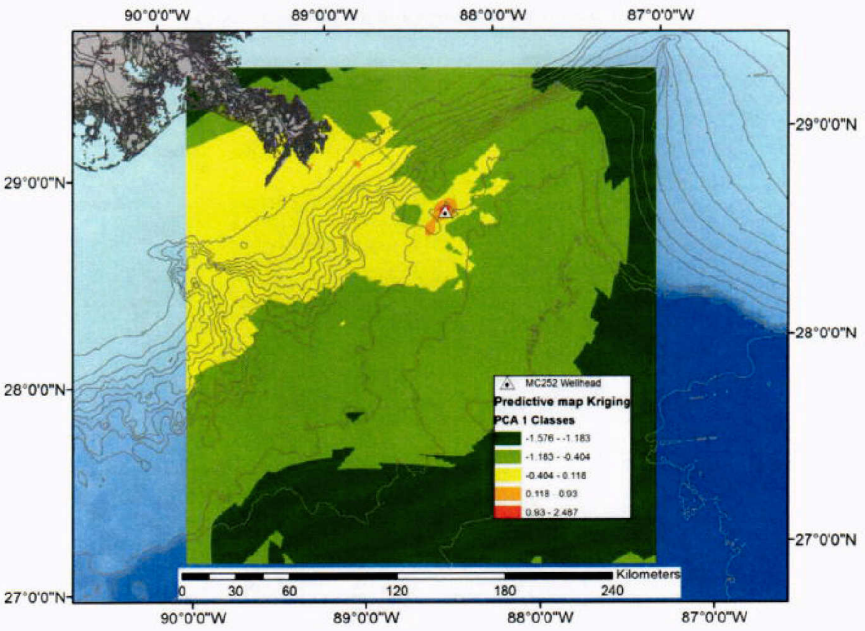
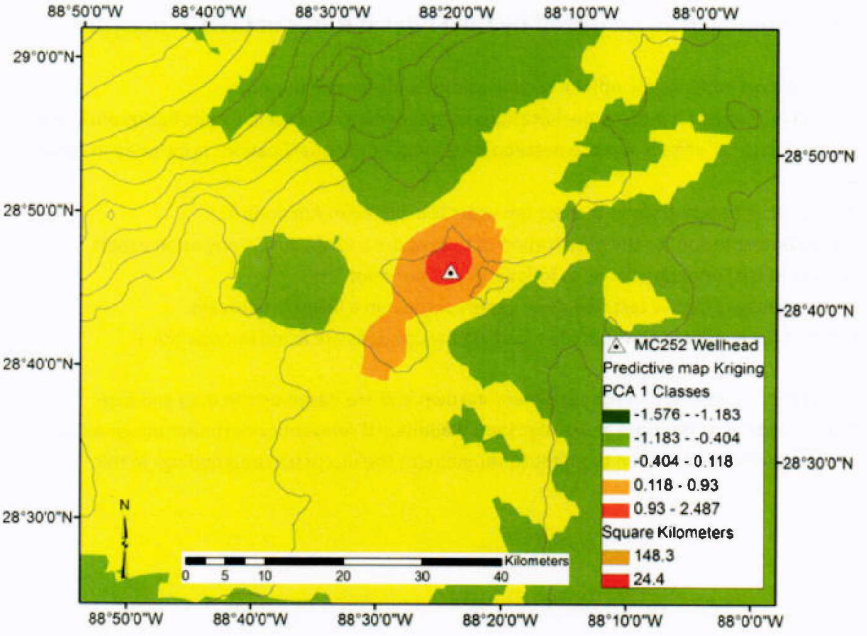


Fig. 11. "Representation of the oil spill as it existed just before the wellhead was shut-in on July 15, 2010. The map view shows the surface plume (based on the NOAA forecast of 7/13/2010) and an estimate of the position of the deep dispersed oil. The perspective view illustrates the vertical position of the deep plume at an approximate depth of 1100 m. The three profiles illustrate how fluorescence as measured by CDOM fluorometers and DO2 vary as the plume is carried away from the wellhead by a mean west-southwest current. The approximate positions of the profiles are indicated in both the map view and the perspective view." (JAG 2012 at 10).



Figs. 12.a, 12.b. Benthic area affected in vicinity of Deepwater well (Montagna et al. 2013). "Interpolated area of deep sea impact based on PC1 station scores. The interpolated area shown covers 70,166 km^2 of which 167 km^2 (orange) are considered moderately impacted and 24 km^2 (red) are considered severely impacted." Second image shows close-up of wellhead area.

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28°50'0"N
28°40'0"N
28°30'0"N
MC252 Wellhead
Predictive map Kriging
PCA 1 Classes
-1.576 - -1.183
-1.183 - -0.404
-0.404 - 0.118
0.118 - 0.93
0.93 - 2.487
Square Kilometers
148.3
24.4
N
0 5 10 20 30 40
Kilometers

IX. Information Required by the Federal Rules of Civil Procedure

1. This report contains my opinions, conclusions, and reasons therefor.
2. A general statement of my qualifications is contained in Section II, Expert Background and Methodology, at page 5. A more detailed statement of my qualifications is included in Appendix B.
3. A list of all publications in the last ten years is included in Appendix C.
4. My compensation for the preparation of this report and any testimony as an expert witness at trial or deposition is as follows: $ 250 per hour.
5. I have not previously testified as an expert witness in a litigation process.
6. The facts and data I considered in forming my opinions are listed in Appendix D.

The opinions expressed in this report are my own and are based on the data and facts available to me at the time of writing. Should additional relevant or pertinent information become available, I reserve the right to supplement the discussion and findings in this report.

APPENDIX A: REFERENCES

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APPENDIX B: RESUME FOR STANLEY D. RICE

Retired, 30 Nov 2012, From:
NOAA Fisheries, Alaska Fisheries Science Center
Auke Bay Laboratories as TSMRI
17109 Point Lena Loop Road
Juneau, Alaska 99801-8626.

[redacted]

E-mail jeeprice907[redacted]

Research Expertise:
Toxicology: Oil effects, oil chemistry, embryo toxicology, chemical and biological biomarkers, pollutants in Alaska (PAH, TBT), Risk and oil development in the Arctic; Spills: Ixtoc, Exxon Valdez, Kuroshima, Selendang Ayu, Deep Water Horizon spill in Gulf of Mexico.

Biology: Forage fish biology, herring biology, humpback whale predation on herring, energetics of forage fish, comparative physiology, salmon biology, sea otter and killer whale biology. 130 peer reviewed publications are listed in the bibliography.

Management Expertise:
Program Management: 30 plus years of program management, including integration of chemistry, biology, budgets, personnel, team building. Outside funding sources include for program include, but not limited to: OCSEAP, EVOS, Gulf Watch, Integrated Herring Program, North Pacific Research Board, Dept of Interior (BOEM), Prince William Sound Regional Citizens Advisory Council.

EDUCATION
B.S. 1966, Biological Science; Chico State University, Chico, California
Secondary Teaching Credential, Chico State University, Chico, California (Lifetime)
M.S., 1968, Biological Science; Chico State University, Chico, California
Ph.D., 1971, Physiology; Kent State University, Kent, Ohio

PROFESSIONAL EXPERIENCE
1971-Nov 2012: Marine Biologist/Toxicologist at Auke Bay Laboratory, Juneau Alaska.

1986-Nov 2012: Program Manager, Habitat and Marine Chemistry at the Auke Bay Laboratory, Alaska Fisheries Science Center.

- Program Leader, Habitat Alaska Fisheries Science Center. Oversee tasks ranging from ShoreZone habitat mapping of nearshore to long term impact studies of natural environmental change, ecosystem change through energetics, to energetics of prey and marine mammal response to changes in forage, to contaminant impacts on species and ecosystems; genetics task
- Principal investigator for specific tasks on the Exxon Valdez
- Damage assessment studies in the early years of the spill, on herring and pink salmon, and intertidal zone. Long term studies tracking oil persistence, and connecting persistence with chronic effects to intertidal zone fauna, pink salmon, herring, Sea Otters, and Harlequin Ducks.
- Principal Investigator on OCSEAP studies in the 1970-early 1980s, dealing with toxicity research themes
- Principal Investigator for Environmental Impact Statement on TransAlaska Pipeline in early 1970s. Drafted parts of EIS.
- Herring steering committee for EVOS; lead drafter of herring restoration plan for EVOS
- Lead NOAA scientist in proposing two long term ecosystem studies (20 years, in 5 year blocks) to EVOS Trustee Council that began in 2012. I continue to consult on both studies (ecosystem monitoring; herring program).

1975-present: Affiliate Professor, University of Alaska Fairbanks, Juneau Center, School of Fisheries and Ocean Sciences. Currently on one Master Thesis committee, defending May 2104.

Fall 2013: Taught Marine Pollution Biology course at UAS.

1990-2014: Served on the Science Board for the Oil Spill Recovery Institute in Cordova (funded organization through the Oil Spill Pollution act of 1990.

1974-present: Testified at State and National legislative levels of various contaminant legistlation issues: (Kachemak buy back, double hull tankers, Tri-butyl tin restrictions, OPA 90, water quality implementation, OPA 90 renewal, and EVOS "re-opener" resolutions; Testified in British Columbia on potential oil development impacts in Dixon Entrance).

1993-1996: Lead editor organizing and publishing the first Trustee sponsored symposium proceedings of Exxon Valdez effects. Contributed to three NRC reviews of oil effects. Committee member on several theses at UAF, LSU, and Simon Fraser; Master's and Ph.D. level NOAA Best Practices Management Award 1998; NOAA Bronze Award 2002, NOAA Distinguished Career Award 2012

Public Service:

5 years on the Southeast Boy Scout Council board of directors
33 years of coaching football in Juneau Alaska; 7 at youth levels, 26 at high school level
Head Coach of Juneau High School football program in 1988 and 1989
Currently Head Coach, Thunder Mountain High School, 2013 and 2014

APPENDIX C: BIBLIOGRAPHY FOR STANLEY D. RICE

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CYP1A Inducers in Prince William Sound, Alaska" (2008) 66 Marine Environmental Research 487-498.

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APPENDIX D: CONSIDERATION MATERIALS

Expert Report of Stanley Rice: Appendix D
Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

Bates, Exhibit, TREX, or Other Description
BP-HZN-2179MDL01773121-BP-HZN-2179MDL01773210
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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

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US_PP_RICE002714-US_PP_RICE002727
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US_PP_RICE003019-US_PP_RICE003024
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US_PP_RICE003137-US_PP_RICE003145
US_PP_RICE003146-US_PP_RICE003153
US_PP_RICE003154-US_PP_RICE003183
US_PP_RICE003184-US_PP_RICE003188

Expert Report of Stanley Rice: Appendix D
Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

US_PP_RICE003189-US_PP_RICE003199
US_PP_RICE003200-US_PP_RICE003204
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US_PP_RICE004358-US_PP_RICE004459
US_PP_RICE004460-US_PP_RICE004474
US_PP_RICE004475-US_PP_RICE004483

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Consideration Materials
(Documents Cited in Report are Consideration Materials even if Not Listed Below)

US_PP_RICE004484-US_PP_RICE004491
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