EXPERT REPORT

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

REBUTTAL TO BP ROUND 2 REPORTS

Submitted on Behalf of the United States

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September 26, 2014

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I. Introduction

This report was prepared for the United States and includes my responses to the Round 2 Reports of Dr. John R. Tunnell and Dr. Damien Shea[1] who addressed my opening Round 1 Report on the Toxicological Impact of the MC252 Blowout, Oil Spill, and Response.[2] In my response to each report I have replied in detail to their numerous specific points rebutting my Round 1 Report. Based on these considerations, as well as those made in a Round 2 Report that I prepared together with Dr. Don Boesch rebutting the Round 1 Expert Reports of Drs. Tunnell, Taylor, and Shea and Capt. Paskewich, I draw the following conclusions:

1) The Round 2 Reports of Drs. Shea and Tunnell do not raise valid criticisms or new evidence that would require me to alter the conclusions of my opening Round 1 Report.

2) The Round 2 Reports of Drs. Shea and Tunnell do not resolve the criticisms raised in the Boesch-Rice Round 2 Report.

3) The evidence presently available in the scientific literature supports a conclusion that the actual and potential harm caused by the Macondo well blowout was serious; however, the full quantification of that harm remains to be determined.

Dr. Shea's Round 2 Report attacks the toxicity interpretations by Dr. Boesch and myself, on several themes, but primarily on the grounds that his methods are "highly conservative," correct, and the peer-reviewed literature relied upon by Dr. Boesch and myself used inappropriate "novel" methods. As demonstrated below, these attacks are not only unfounded, they ignore decades of peer-reviewed and tested data in favor of a model that cannot account for advances in scientific knowledge or methods, nor can it explain BP's own test data. BP's experts continue to try to trivialize evidence of actual harm to the Gulf through inappropriate averaging of water chemistry and mischaracterization of sampling results.

I have organized my response to Dr. Shea's rebuttal report along his lines of organization, and address each topic in turn:

"II. THE UNITED STATES' ENVIRONMENTAL EXPERTS RELY UPON FLAWED TOXICITY METHODS TO SPECULATE ABOUT POTENTIAL HARM

III. THE ENVIRONMENTAL IMPACT FROM SURFACE OIL WAS FAR LESS THAN THE UNITED STATES CLAIMS

[1] I note at the outset that Drs. Tunnell and Shea responded to my Round 1 report, often making similar points on overlapping subject matters. I have primarily focused my responses on Dr. Shea's Round 2 Report, as he is the primary critic of my work. However, to the extent that more than one BP expert rebutted my assessment of harm, this report serves as a response to all of them (e.g. my response to Dr. Shea's discussion of corals may serve to rebut points raised in Dr. Tunnell's report as well).
[2] I refer to reports submitted by myself, Dr. Shea, or Dr. Tunnell that were submitted on August 15, 2014, as Round 1 reports and my joint report with Dr. Boesch and the reports of Drs. Shea and Tunnell submitted on September 12, 2014, as Round 2 reports.

IV. DRS. RICE AND BOESCH INCORRECTLY IMPLY THAT DISPERSANTS CAUSED A SIGNIFICANT INCREASE IN TOXICITY

V. DR. BOESCH'S DISCUSSION OF A "DIRTY BLIZZARD" IS THEORETICAL; ACTUAL DATA DEMONSTRATES LIMITED IMPACT ON THE SEA FLOOR"

II. Dr. Shea's Allegation that Dr. Boesch and I Rely upon Flawed Toxicity Methods is Unfounded

A. Dr. Shea and I Rely on Different Scientific Methods or "Paradigms" of Oil Toxicity: Dr. Shea's Analysis Relies on a Toxicity Unit Model, Whereas My Approach Relies on Peer-Reviewed Research

In his initial and Round 2 reports, Dr. Shea estimates potential for toxicity damage by comparing the measured polynuclear aromatic hydrocarbon (PAH) composition and concentration from 18,000 water samples from the Gulf following the Deepwater Horizon (DWH) spill event based on a "Toxicity Units" model that uses EPA-derived water quality and sediment benchmarks.[3] In his Round 2 report, Dr. Shea incorrectly suggests this is the "prescribed" approach to estimating potential harm from the spill.[4] In contrast, Dr. Boesch and I relied on an assessment of potential harm based upon a comprehensive body of peer-reviewed scientific literature that is founded and builds upon decades of research and widely-accepted methods for estimating toxicity.[5] Dr. Shea incorrectly asserts that our method "largely ignor[es] the actual chemistry data in the field" and attempts to dismiss the emerging developments in toxicological research that do not fit into his model as "misunderstandings."[6]

1. Difference between the Shea Toxic Unit (TU) Approach and the "Research" Approach to Predicting Toxicity Potential

As discussed on pages 20 and 21 of my Round 2 Report with Dr. Boesch, the toxicity unit (TU) approach is a limited interpretation of toxicity primarily based on acute bioassays of freshwater and shallow-coastal species utilizing narcosis as the primary end point. The TU approach calculates toxicity by adding up the total sum of individual PAH toxicity estimates.[7] In contrast, the "research" or damage assessment approach that has evolved over the last two decades when assessing oil spills, relies on direct measures of total PAH (lab or field), and comparing those

[3] Shea at 12-16, 20-30.
[4] Shea Round 2 Report at 4-5. Applies to Tunnell, Taylor, et al.
[5] Dr. Shea's Round 2 Report noted "nearly 18,000" water samples; his Round 1 Report reported 17,881 water chemistry samples in his reports (p 21), however neither report explains exactly how Dr. Shea arrived at these numbers, only the databases he used (Shea Round 1 Report, Appendixes C and D). These are the records marked "natural sample" in the BP's NRDA database WaterChemistry_W-01v02-01.csv. As Dr. Shea has not provided his exact dataset, we have done our best to adjust ours so that they match what BP uses. Our conclusions regarding the impacts from that data – namely that "samples from the plume and the upper surface to waters [were] harmful" (Round 2 Report at 24) – have not changed. See Appendix B.
[6] Shea at 3-6.
[7] The narcotic approach is too narrow to be predictive for all adverse biological responses.

results to literature of exposure tests that assess impacts to a wide swath of toxicity mechanisms (including acute narcosis), to get a better understanding of the possible impacts on different species and different life stages, in specific exposure environments. Thus, I have compared the measured toxicity in sensitive early life stages of fish embryos (from literature prior to DWH plus two studies in response to DWH), using total PAH as the descriptor of the dose levels, and compared those PAH values with those measured in the same 18,000 water samples. There are limitations with each approach.

2. The Primary Flaw with the Toxic Unit Approach Advocated by Dr. Shea is neither "highly conservative" nor "prescribed by EPA."

Dr. Shea claims that his findings of potential harm were "highly conservative" because it relies on "very protective assumptions" (i.e., the TU approach).[8] However, the TU approach relies on the flawed assumption that acute narcosis toxicity is the primary toxicity mechanism responsible for death and other effects, which has the effect of making it under protective.[9] As pointed out on page 21 of the Boesch-Rice Round 2 Report, this flaw was recognized internally by EPA. Exchanges between toxicologists at EPA discussing the benchmark noted that "[s]ome literature data indicate that toxicity to certain organisms, such as early life stages of fish, occurs through other toxic mechanisms, resulting in an underestimation of toxicity by the current narcosis-based approach."[10]

Each approach has merit if used appropriately under the right set of circumstances. If the question is a screening issue (as was EPA's stated purpose in using it), the TU approach's assumption of acute narcosis as the primary toxicity mechanism is probably adequate. If the question is more of a damage assessment issue, particularly if also accounting for sensitive life stages and processes, then using measured toxicities with relevant species and life stages in the spill area will likely be more informative.[11] This is no doubt why EPA expressly stated when setting the benchmarks that they were not intended to be used for natural resource damage assessments:[12] Dr. Shea's unsupported assertion that EPA "prescribes" adherence to this method is patently false.[13],[14] Acute narcosis toxicity tests can define a harmful dose, but seldom defines a safe dose. The research approach examines effects other than acute mortality, other toxicity mechanisms and end points, and has a higher probability of finding exposure levels that are safe in addition to those that are harmful. This is a developing science, and as such is imperfect,

[8] Shea Round 2 at 3.
[9] Boesch-Rice Report at 20.
[10] US_PP_EPA045086 - US_PP_045086.
[11] As noted above, the TU method is not based on many of the species at issue in the Gulf. See also, Boesch-Rice Round 2 Report at 21.
[12] http://www.epa.gov/bpspill/water-benchmarks.html#ganda ("Benchmarks are meant to be used **for screening purposes only**; they are **not** regulatory standards, site-specific cleanup levels, or remediation goals" (emphasis in original)).
[13] Shea Round 2 at 4.
[14] Boesch-Rice Report at 20-21.

scientists continue to look for more sensitive tools (analytical, chemical, and biological) to detect increasingly tiny amounts of harmful chemicals and help scientists evaluate sensitive processes (including genetics, development, physiology, behavior) and impacts to sensitive early life stages. The TU approach chosen by Dr. Shea relies heavily on the chemistry of the toxic solutions, and tends to undervalue the varied biology of sensitive processes and life stages.

a. BP's Own Toxicity Test Demonstrate That a TU of 1 is Not Safe

Although in his Round 2 report Dr. Shea attempts to dismiss the studies I relied upon demonstrating toxic effects at levels below the supposedly "safe" TU of 1 as "novel, unreliable, and unrealistic"[15] (for reasons I will address further below), Dr. Shea fails to note in either of his reports that even the presumably non-objectionable and "comprehensive investigation into the potential toxicity of the oil and oil-dispersant mixtures"[16] conducted by BP actually demonstrate toxic effects below the TU of 1.

For example, bioassays completed by BP on Pompano (*Trachinotus carolinus*) and amphipods (*Leptocheirus plumulosus*) demonstrate adverse effects below the acute TU of 1. As explained further in Appendix A, BP's water bioassay of pompano embryos demonstrated significant mortality (30%) at a chronic TU of 0.82; in acute tests, significant mortality (30%) was seen at an acute TU of 0.20 and 90% mortality was seen at a level of 0.4. BP's sediment bioassay of benthic amphipods demonstrated significant mortality at a chronic TU of 0.12 (see Appendix A).

This refutes Dr. Shea's assertion that the toxicity bioassays being conducted under NRDA are "consistent with the [TU] analysis" as well as his latest assertion that TU's are "very protective assumptions" that are "highly conservative."[17] These results further demonstrate my point that the TU model upon which Dr. Shea bases his criticisms is inadequate for determining potential toxicological harm.

B. Dr. Shea's Criticism of Methods Except Those Used to Study Acute Narcosis Toxicity is Inconsistent with the Current State of Science

1. Dr Shea is Unjustifiably Critical of Methods That Were Not Designed to Study Acute Narcosis Toxicity

Dr. Shea criticizes many published studies because their methods do not conform to old standards and rejects state-of-art studies that have developed new methodology more applicable to dispersed oil spills such as the DWH. For example, studies by Incardona et al (2014), and Mager et al. (2014) measured the toxicity of DWH oil to embryos, using techniques accepted in

[15] Shea Round 2 at 10.
[16] Shea Round 1 at 43.
[17] Shea Round 1 at 45; Shea Round 2 at 3.

the literature for the last decade[18] and have used state of the art chemistries (GC-MS[19] for example) to characterize the oil PAH composition, concentration, weathering, and concentration change.

Dr. Shea appears to advocate for more primitive bioassays by measuring acute narcosis toxicity primarily in adult animals, rather than the toxic responses during the development process to a sensitive life stage. Researchers in the last 25 years have gone beyond acute narcosis toxicity in their evaluation of toxic spills, and examine the organism response with many different end points (death, but also growth, reproduction, biomarkers such as cytochrome P450s,[20] DNA damage, tissue concentrations, lesions, malformations of tissues, or embryos, etc. (see Rice report). Dr. Shea's model lumps embryos in with adult/juvenile tests, and does not consider the vulnerability of developing embryos to chemical toxins as any different than challenges to adults/juveniles, even though Dr. Shea himself admits that embryos and newly hatched eggs "are usually most sensitive to oil chemicals such as PAHs".[21] Additionally, although Dr. Shea admits on page 44 of his initial report that UV light has been reported to increase the toxicity of some PAHs (also known as phototoxicity), he fails to note that the TU method does not account for phototoxic effects (again, phototoxic mechanisms are not the same as the generalized narcosis mechanism).[22]

2.Dr. Shea's Critique of the Research Approach is Overstated.

As mentioned above, there is some validity to both the TU and Research approaches, due in large part to a shared set of bioassays between the TU and the research approaches. But while the research approach includes the narcotic effect bioassays of the TU approach, the TU approach cannot account for the toxicity levels found by the research approach, because it does not include all of the data from those additional bioassays. Rather than acknowledge this difference, Dr.

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[18] Barron, M. G., M. G. Carls, et al. (2003). "Photoenhanced toxicity of aqueous phase and chemically dispersed weathered Alaska North Slope crude oil to Pacific herring eggs and larvae." Environmental Toxicology and Chemistry 22(3): 650-660; Hodson, P. V., C. W. Khan, et al. (2007). Alkyl PAH in crude oil cause chronic toxicity to early life stages of fish. Proceedings, 28th Arctic and Marine Oilspill (AMOP) program, Technical Seminar, Edmonto, AB, Canada June 4-7, pp 291-300; Carls, M. G., L. Holland, et al. (2008). "Fish embryos are damaged by dissolved PAHs, not oil particles." Aquatic Toxicology 88: 121-127; Adams, J., M. Sweezey, et al. (2013). "Oil and oil dispersant do not cause synergistic toxicity to fish embryos." Environmental Toxicology and Chemistry 33: 107-114; Adams, J., J. M. Bornstein, et al. (2014). "Identification of compounds in heavy fuel oil that are chronically toxic to rainbow trout embryos by effects-driven chemical fractionation." Environmental Toxicology and Chemistry 33(4): 825-835; Redman, A. D., T. F. Parkerton, et al. (2014). "Evaluating Toxicity of Heavy Fuel Oil Fractions Using Complementary Modeling and Biomimetric Extraction Methods," Environmental Toxicology and Chemistry 33(9): 2094-2104.

[19] GC-MS; gas chromatography with a mass spectrometer detector is used to separate and identify individual compounds such as PAH, so that both composition and concentration of individual PAH can be measured.

[20] P450s are oxidase enzymes. In fish and other vertebrates, PAH exposure stimulates specific enzyme systems to degrade PAH, particularly in the liver and gill tissues.

[21] Shea Round 1 at 44.

[22] The TU method also fails to account for changes in oil toxicity due to weathering.

Shea mischaracterizes the research approach (pp 4, 6-7, 9) as "novel" and ignorant of "the very basic premise of technology" for using total PAH instead of individual PAH concentrations.

Dr. Shea's critique of methods based on total PAH concentrations is overstated. In fact, if you translate Toxic Units to total PAHs, you will see that the vast majority of the studies Dr. Shea seeks to discredit are often in line with toxicity estimates based on TU calculations (the sum of all individual PAHs estimated to be toxic), as is not surprising since they share a partial set of data. . When one examines the range of water samples defined as toxic by Dr. Shea (a TU of 1 or below) and calculates what those toxic samples would be in terms of total PAH, one finds that total PAH concentrations as low as 0.51 µg/L resulted in a toxic unit of 1. Thus nearly all minimum toxic concentrations published by researchers at the Auke Bay Laboratory fits within the range defined as toxic by Dr. Shea, invalidating his multiple arguments to discount these "controversial" studies.[23] In addition, minimum toxic concentration estimates in the Incardona et al. (2014) study Dr. Shea discounts as "non-standard" also fall almost entirely within the toxic range defined by Dr. Shea. Other investigators also report negative biological responses in the 1 – 10 µg/L total PAH range.[24] Thus, Dr. Shea's wholesale rejection of these studies is entirely unwarranted.

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[23]Marty, G. D., J. W. Short, et al. (1997). "Ascites, premature emergence, increased gonadal cell apoptosis, and cytochrome P4501A induction in pink salmon larvae continuously exposed to oil-contaminated gravel during development." Canadian Journal of Zoology 75(6): 989-1007; Carls, M. G., S. D. Rice, et al. (1999). "Sensitivity of fish embryos to weathered crude oil: Part I. Low-level exposure during incubation causes malformations, genetic damage, and mortality in larval Pacific herring (*Clupea pallasi*)." Environmental Toxicology and Chemistry 18(3): 481-493; Carls, M. G., R. A. Heintz, et al. (2005). "Cytochrome P4501A induction in oil-exposed pink salmon *Oncorhynchus gorbuscha* embryos predicts reduced survival potential." Marine Ecology-Progress Series 301: 253-265; Carls, M. G., L. Holland, et al. (2008). "Fish embryos are damaged by dissolved PAHs, not oil particles." Aquatic Toxicology 88: 121-127; Heintz, R. A., J. W. Short, et al. (1999). "Sensitivity of fish embryos to weathered crude oil: Part II. Increased mortality of pink salmon (*Oncorhynchus gorbuscha*) embryos incubating downstream from weathered *Exxon Valdez* crude oil." Environmental Toxicology and Chemistry 18(3): 494-503; Heintz, R. A., S. D. Rice, et al. (2000). "Delayed effects on growth and marine survival of pink salmon *Oncorhynchus gorbuscha* after exposure to crude oil during embryonic development." Marine Ecology-Progress Series 208: 205-216.

[24] Johannessen, K. I. (1976). "Effects of seawater extract of Ekofisk oil on hatching success of Barents Sea capelin." International Council for the Exploration of the Sea E:29; Pearson, W. H., D. L. Woodruff, et al. (1985). Oil effects on spawning behavior and reproduction in Pacific herring (*Clupea harengus pallasi*). Washington, DC, Final report to the American Petroleum Institute, Environmental Affairs Department; Brannon, E. L., K. M. Collins, et al. (2006). "Toxicity of weathered *Exxon Valdez* crude oil to pink salmon embryos." Environmental Toxicology and Chemistry 25(4): 962-972; Farwell, A., V. Nero, et al. (2006). "Modified Japanese medaka embryo-larval bioassay for rapid determination of developmental abnormalities." Archives of Environmental Contamination and Toxicology 51(4): 600-607; Olsvik, P. A., B. H. Hansen, et al. (2011). "Transcriptional evidence for low contribution of oil droplets to acute toxicity from dispersed oil in first feeding Atlantic cod (*Gadus morhua*) larvae." Comparative Biochemistry and Physiology C-Pharmacology Toxicology & Endocrinology 154: 333-345; Olsvik, P. A., K. K. Lie, et al. (2012). "Is chemically dispersed oil more toxic to Atlantic cod (*Gadus morhua*) larvae than mechanically dispersed oil? A transcriptional evaluation." BMC Genomics 13: 702.

3. The Oil Spill Research Community Does Not Use the EPA Toxic Unit Approach, but Instead Measures Impact in the Field, or in the Labs, and Measures total PAH.

While acute toxicity, including narcosis, has been a subject of extensive oil spill scientific literature, the use of TUs is virtually absent. The adoption of this approach by researchers can be discerned in the literature. Using a professional journal browser (Web of Science), we find 22,604 papers that refer to PAH (from 1965 to 2014). A search for PAH and oil reduces this to 2251 papers. The subset of papers that includes ESB and PAH is 4, the subset with EPA and threshold and PAH is 16, the subset with "equilibrium partitioning sediment benchmarks" is 8 (spanning 2007 to 2014), and the subset with TU and PAH is 21. In other words, less than 1% of published papers include the TU approach. This is because it is logical to assume that oil affects many different parts of cells, tissues, organs, etc, and researchers are attempting to sort out the toxic mechanisms that matter from those that do not. Narcosis matters if the organism is "swamped" with a toxic exposure; but if it is not, many different toxicity mechanisms determine whether organisms, including sensitive life stages, can survive the lower exposure concentrations that do not cause acute narcosis toxicity but can still affect their fitness for survival over the long term, and which can still have impacts at the population level. For these scenarios, the questions become: which of the many other toxicity mechansims can affect the organism, and subsequently the population? Is it energy acquisition or utilization? Is it cellular function, or organ function? Is it reproduction? All of the above? For fish embryos, we and others have documented that the net result of low-level exposures to sensitive life stages (delayed mortality, abnormalities such as cardiac edema, slow growth, etc) result in lower fitness levels that impair survival, but these effects are sometimes delayed before they can be seen and measured.

The TU approach, as complex as it is, is still too simplistic to account for such questions, as it relies basically on one toxicity mechanism, which is why the vast majority of researchers do not accept the relatively simplistic TU approach to account for the effects to early life stages.

Dr. Shea inappropriately attacks use of "total PAH" as though we and others consider the toxicity of all PAHs to be the same. This is patently untrue. My Round 1 Report discusses PAH structure and toxicity, as this has been a known fact for decades. The peer-reviewed literature on toxic effects recognizes that PAH toxicity is based on structure,that different compounds have different toxicities, and that composition as well as concentration is critical to the understanding of oil toxicity effects. Hence increasingly sophisticated chemical analytical procedures have developed to identify both PAH composition and concentration. However, when multiple mechanisms of toxic action occur due to a mixture of toxicants, the observed response can be additive, more than additive, or less than additive,[25] thus models (such as the TU model) cannot simply replace empirical evidence.

4. Dr. Shea Inappropriately Criticizes HEWAF Mixing Methods

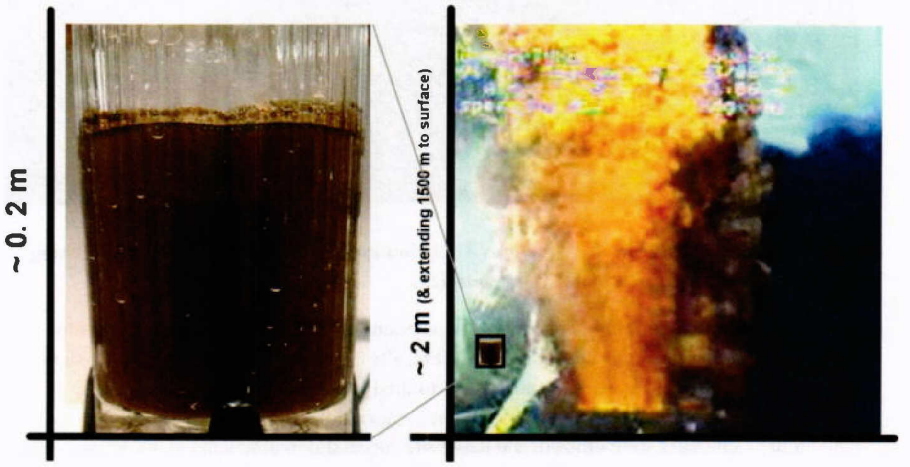
[25] Carls, M. G. and J. P. Meador (2010). "A perspective on the toxicity of petrogenic PAHs to developing fish embryos related to environmental chemistry." Human and Ecological Risk Assessment 15(1084-1098).

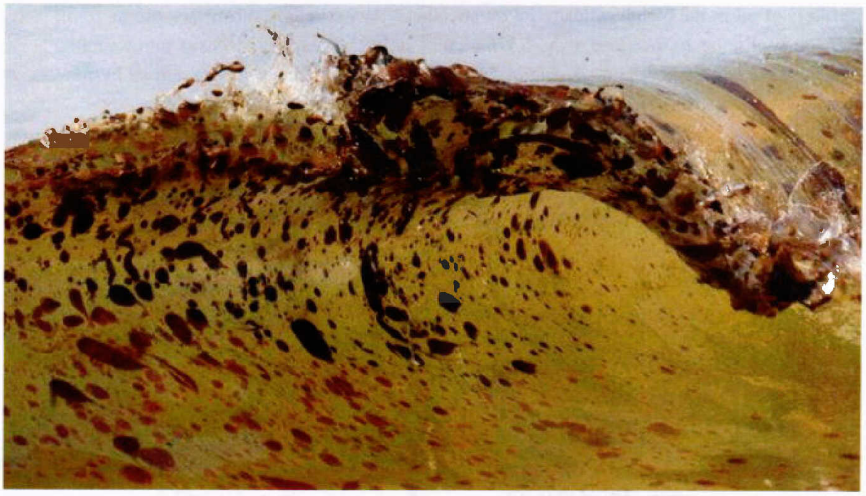
Dr. Shea criticizes the high energy water accommodated fraction of oil (HEWAF) mixing methods used in some studies, favoring instead the low energy standardized mixing in the EPA benchmark tests that underlie the TU approach. As shown above, and indicated by Dr. Shea, composition of PAHs is key to understanding the toxicity of oil, because of the differential toxicity by different PAHs. This is an important concept, whether using toxicity units approach or the research approach that uses total PAH as the number along with descriptions of the changing PAH composition during a test or during and environmental event. Dr. Shea's emphasis on standardized mixing methods is not warranted, because the composition and concentration of individual PAH can be determined with high quality analytical chemistries (not available in the past). PAHs are not stable in water solutions, hence there is the need for accurate measurement of PAH composition (field or lab exposures), and how it changes in bioassays over time, which allows better comparison of data results across different tests (different species, life stages, field or test conditions, different oils, different weathering states, with or without dispersants, etc.). Different mixing methods, with strong supporting chemistries, can be designed to meet the needs of the test, to emulate specific conditions of a spill situations, for adults or for embryos. Standardized oil mixing methods were very important 3 decades ago when test results were published with poor supporting chemistries (doses were often quantified as "oil added"); yet even with standardized mixing there was difficulty reproducing the exact composition and concentration from one test to the next, even within the same lab. Changes in the oil viscosity (or changes to the oil, the evaporative loss, or the temperature) and standardized mixing will not reproduce the same dose of PAHs- hence the need for accurate PAH measurements of the testing solution itself, both in quantity and composition.

The low energy mixing, favored by Dr. Shea and the TU benchmark tests, biases composition toward the smallest, least toxic aromatics, while high energy mixing has the energy to entrain dispersed small dispersed droplets into the water which promotes some of the heavier (and more toxic compounds) into solution. These methods certainly affect the composition, significantly, but the need or reason to restrict science to one standard method does not satisfy the needs of tests with different objectives and restrictions. For example, the HEWAF method attempts to mimic the high energy mixing of DWH oil at the riser pipe (remember the video of the turbulent violent mixing; Fig. 1) as well as energetic mixing by wind and waves of the surface slicks. Similarly, CEWAF, using dispersants, creates a different PAH composition, to meet the needs of dispersant tests. The objectives of the test are important, and the mixing methods will need to be appropriate to the objectives, species, and life stages being tested.

Fig. 1. Visual Comparison of the HEWAF blender illustrated by Dr. Shea with the high-energy oil dispersion continuously created by the Deepwater Horizon blowout for 3 months. Significant amounts of liquid oil were dispersed near the wellhead and some oil droplets were < 100 microns in diameter (Camilli et al. 2010; Lehr et al. 2010). (Incardona et al. 2014) demonstrated close correspondence between PAH composition in HEWAF and in Gulf of Mexico water. Daily DWH oil release ranged from 7.9 to 11 million liters (Spier et al. 2013), rather more than the few

milliliters of oil in the blender illustrated by Dr. Shea. A continuous subsurface plume of oil extended continuously for more than 35 kilometers and persisted for months (Camilli et al. 2010). Wave and wind action also created dispersions at the surface slick as shown in the second photo of waves in Orange Beach, Alabama during the DWH spill. (Source: http://www.nola.com/business/index.ssf/2014/09/halliburton_energy_services_ag.html). It is also important to note that Dr. Shea's image appears to only reflect oil dispersion immediately after blending; HEWAF methods require an hour of settling time prior to use in experiments and a dilution of 1:1,000 (oil to water); water used from a HEWAF for testing does not generally contain visible oil.





5. Dr. Shea's Assertions That Embryo Fish Toxicity Studies Were Using Stressed Fish Are Unfounded.

Dr. Shea cited the high mortality in controls populations of bluefin tuna (40%) as a basis to discount the tests of Incardona et al., but the test results indicate otherwise. Testing off-shore species is difficult; these species are not easily cultured in confining tanks, and it is often difficult to stimulate spawning in a restricted environment. Nevertheless, specialized mariculture techniques were utilized, and they were successful in producing viable spawn. The toxicity results were dose related, and the effects were independent of the mariculture process, as accounted for by the mortality rates. In the yellowfin and amberjack tests, the mortality rates for controls were less (28% and 7% respectively, in the same paper), and yielded the same results: PAH dose-related impacts, and identical cardiac response were found in all three species. These findings of edema and cardio effects at part per billion levels of PAH are consistent with embryo toxicity studies published from other labs and species.

The bottom line is the methods in the research approach (mixing methods, embryo toxicity methods) rejected by Dr. Shea are more appropriate than the one he favors because they successfully emulated conditions caused by the DWH blowout with species and life stages that were present in the off-shore waters of the Gulf (Incardona et al. 2014).

6. Dr. Shea's Attack on the Use of Peer-Reviewed Studies is Unsupported

Dr. Shea's attack on peer-reviewed literature is egregious, for many reasons. Scientific advancement has been based historically on the debates in the peer reviewed literature. It is

sometimes a slow process, but the point of having detailed methods is so that those who follow can either use or adapt your methods to advance the science. It is not a perfect system, as mistakes, imperfections, and even deceptive publications can be introduced into the literature, even peer reviewed literature. Over time, new studies will add to and replace older studies, and science will eventually arrive at a truth that has wide acceptance.

Dr. Shea uses a series of papers attacking some of the papers that I and co-authors published while investigating the Exxon Valdez spill. These were all papers from Exxon supported contractors, who have long attacked the low level toxicity work done by me, my co-authors, and anyone else using the research approach. Usually it is a re-analysis of the work, rather than new work with new oil exposures and new chemistries. Like Dr. Shea, they have attacked methods, the findings of oil persistence in the environment, and the low level effects to fish embryos. They are fine with the old literature that relied on acute toxicity narcosis assays, and object to long-term low-level studies of embryo toxicity. Over time, our research has been put to the test, via corroborating studies with other species, other spills, and other labs, which have established that fish embryos are more sensitive that older life stages, and that concentrations of total PAHs that affect embryos can be very low, in the parts per billion of PAH.[26]

In contrast, Dr. Shea relies on his own analysis of the 18,000 water samples, and offers conclusions and assertions that we dispute, has no peer-review of the analysis that he completed, and uses very few peer-reviewed publications to compare his finding to.

C. Dr. Shea Attempts to Trivialize Evidence of Potential Harm by Minimizing the Significance of Oil in Water Samples and Focusing on Where Oil Was Not Rather Than Where It Was

As I pointed out in my Round 1 Report, toxicity potential is the net result of concentration, composition, and length of exposure.[27] Dr. Shea's Round 2 Report reiterates his assertion that we overstate the presence of oil in the Gulf based on water chemistry samples, but once again ignores that, as we pointed out in our Round 2 Report, he analyzed thousands of water samples that were either zero, or near zero in PAH, but were also in locations that were not expected to contain oil.[28] His analysis once again seeks to hide the significant levels of oil that was in the Gulf by inappropriately averaging the tests across time, space, or specific location. We can identify a number of samples with toxicity potential (deep water plume samples, near surface samples, nearshore samples), leading to the conclusion that there are at least four habitat areas at considerable risk of toxic harm following the DWH spill.

1. The deep water environment where a dispersed oil plume was found, extending from the well head, and involving small dispersed droplets of oil, that could introduce unusual amounts of

[26] See Appendix C.
[27] Rice Round 1 Report at 9-12.
[28] Boesch-Rice Round 2 Report at 5, 23, 28.

dissolved PAH into the water, and the droplets themselves could be extracted from the environment by filter feeding organisms (like deep corals).

2. The upper surface layers, just below the slick, that were subject to rising oil as well as surface oil driven back into the surface water layer by wave action and possibly by dispersants, where early life stages may be vulnerable to patchy oil concentrations.

3. The surface interface, where slicks were observed for a period of time, making birds, turtles, and marine mammals (air breathers) vulnerable to high concentrations of oil.

4. The nearshore, where water is very shallow, and shoreline contamination from surface slicks, which can also re-introduce the oil to the shallow water and extend the exposure time for selected species (wetland habitat for example).

III. Dr. Shea's Assertion That the Environmental Impact from Surface Oil Was Far Less Than the United States Claims Relies on Incorrect Analysis

Dr. Shea claims Dr. Boesch and I have overestimated the oiling of the surface, and thus have overestimated the potential harm from the surface oil. The opposite is more likely. Dr. Shea quibbles about the area being covered (exposure potential), but fails to acknowledge the likely biological impacts to the species using the surface. Surface oil, which presents both toxicity and fouling hazards, is particularly harmful to the air-breathing birds, marine mammals, and turtles which need to break through the film of oil to either breathe or to feed. These surface oil slicks also become part of the source oil exposure to organisms just below the surface when oil dispersions are entrained into the water column (as was occurring through physical and extensive chemical dispersion).

Dr. Shea spends considerable time challenging use of SAR data to estimate slick extent.[29] Estimating the area of the open ocean contaminated with surface oil is a challenging task, particularly when the oil is off-shore, moving daily with wind and currents, and weather can prevent visual coverage on some days. Dr. Shea points out that over flights will yield fewer false positives compared to satellite coverage (e.g. anomalies from seaweeds rather than oil but the ability to search and document oil in large areas is not practical on a daily basis using over flights. Given the length of the spill (approximately 3 months), and the thousands of square miles that needed to be assessed, the satellite imaging is more likely to provide unbiased coverage from beginning to end of the spill. Neither method has yet provided an assessment of the thickness of oil, so both methods have limitations. Further, confirmation by boats, including sampling, confirm the oil slicks were extensive. Measured PAH concentrations were consistently elevated in water in the general area where satellite imagery suggested oil, thus confirming the remote sensing method. It is well known that organisms bioaccumulate hydrocarbons from non-uniform

[29] Shea Round 2 at 19-23.

concentration distributions in water.[30] Dr. Shea's detailed criticisms about false positives are without merit and grossly underestimate the obvious risk to the surface species.

A. Assessing the Likely Impacts Is Even More Challenging Than Assessing Exposure, But Is the Primary Question

Birds are vulnerable to surface oil (toxicity and fouling), but assessing impacts to the off-shore birds is problematic, even though there were likely hundreds of thousands of mortalities. While Dr. Shea speaks to the issue of surface oiling (but not on direct impacts to birds), Dr. Tunnel's report minimizes the impact to birds by discussing the lack of impact to coastal birds, and uses data like the Christmas bird counts (on-shore birds) and the low numbers of carcasses collected on the shoreline. In contrast, two recent papers by Haney et al. (in press) model off shore bird mortalities, based on two independent models. One from modeling the surface oiling during the spill event (the encounter rate based on mapped oil, on a daily basis) that is somewhat analogous to Dr. Shea's approach (analyzing the risk of exposure). The second model is more analogous to Dr. Tunnel's approach, by modeling population numbers from extrapolated bird carcasses. Both models used by Haney et al. (in press) estimate hundreds of thousands of off shore sea bird died.

The Haney et al. modeling approach was driven in part by the lack of carcasses collected off-shore, a major problem when attempting to assess surface oil impacts to the off-shore species. However, Haney et al. explains that the probability of a bird carcass transporting across the shelf via currents and wind to a shoreline that was assessed for bird carcasses was very low. During the weeks of transport (about the same length of time for oil to come ashore from the well head), there would be ample time for decomposition (degassing and then sinking). Given this problem of low carcass recovery, Haney et al. also rely then on an independent model based on the observations of oil slicks and the likelihood of contamination of off-shore birds. The two different approaches both arrive at similar convergence of bird mortalities (600,000 and 800,000 estimates of bird mortalities, with a wide range of confidence limits). Unfortunately, there does not appear to be a better estimate of off-shore bird mortalities than these two model estimates. We will likely never have a precise estimate of bird mortalities, but it is considerably larger than the few thousand carcasses that were recovered at the Gulf shoreline.

Significance of bird mortalities: These off shore birds are all predators, and because the numbers are large (hundreds of thousands killed), it is easy to speculate that the predator load

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[30] Huckins, J. N., M. W. Tubergen, et al. (1990). "Semipermeable membrane devices containing model lipid: a new approach to monitoring the bioavailability of lipophilic contaminants and estimating their bioconcentration potential." Chemosphere **20**: 533-552. Prest, H. F., J. N. Huckins, et al. (1995). "A survey of recent results in passive sampling of water and air by semipermeable membrane devices." Marine Pollution Bulletin **31**: 306-312. Echols, K. R., R. W. Gale, et al. (2000). "Comparing polychlorinated biphenyl concentrations and patterns in the Saginaw River using sediment, caged fish, and semipermeable membrane devices." Environmental Science & Technology **34**(19): 4095-4102.

was considerably reduced in the northern Gulf of Mexico, possibly having a significant ecosystem effect.

1. Impacts to Herring Exemplify the Challenges in Determining Population Impacts

Dr. Tunnel says (page 9) of his rebuttal report, that we (Rice and Boesch expert reports) "misconstrue the lessons from the Exxon Valdez spill", and uses the confused and complex herring story as his example. The facts are: the 1989 herring year class had the lowest recruitment on record, there was a population crash detected in 1993 (four years after the spill), there was no population crash in other Alaska herring populations that year. Although there have been many years of studies on herring, there is no satisfactory explanation of their crash, whether it was oil related, or not. With the information collected during those years, it is not possible to prove that the oil spill caused the crash, nor does that prove that oil was not a contributing factor- we simply do NOT know the cause, even though herring populations have continued to be studied. Herring are an important forage species in Prince William Sound, capturing energy from zoo plankton, and are a dominant prey source for many species of fish, birds and marine mammals, thus transferring that energy up the food chain. Their value in the ecosystem is the justification for the continued study by the Exxxon Valdez Oil Spill Trustee Council, who devote a large proportion of their restoration funds in attempting to understand the factors that affect the herring population, and their continued lack of recovery.

Dr. Tunnel discusses herring as an example of how "we misconstrue" an enigmatic species, but fails to cite or discuss the compelling evidence of long term impacts that have been the basis for much of the valuable Exxon Valdez spill literature, such as: field and laboratory embryo toxicity studies that make a compelling case for four years of oil related effects to pink salmon in Prince William Sound; field studies that document 40% decrease in two pods of killer whales (impacts that will last for decades); contamination in the intertidal zone that has lasted for decades, and will continue to do so; and two decades of effects to sea otters that forage in the intertidal zone. These issues likely affected the ecosystem in Prince William Sound.

Dr. Tunnel is correct that Prince William Sound is different than the Gulf of Mexico, hence we cannot extrapolate the negative impacts measured in Prince William Sound directly to the Gulf spill, but we can infer that there can be long term effects, that toxicity can certainly play a role as it did in Prince William Sound, and surprises may be expected. The initial damage assessment studies with Exxon Valdez were very species-centric for the first few years; several years were required before different lines of evidence by different researchers were combined to explain some of the long-term effects that were quite evident by the 10 year anniversary. Every spill is different in many ways (volume and type of oil spilled, temperature of the environment, etc), yet the processes are similar, niches are similar, and it is reasonable to assume that the direction of effect will be similar from one spill to the next.

IV. The Analysis Dr. Boesch and I Presented Regarding the Toxicity of Dispersants is Well-Founded

Dr. Shea asserts on page 26 of his report that "at the request of NOAA," over 50 scientists and other spill professionals met to provide input to the government on the use of dispersants during the DWH spill. I was one of the scientists that met in Baton Rouge in late May 2010 to discuss the pros and cons of continuing dispersant application. After much angst, it was generally agreed that dispersant application should continue, that we were trading reduced risk and harm to the shoreline and marshes for greater risk and harm to organisms in the water column and the benthic ocean, though what the exact extent of that harm would be was unknown. Continued dispersant application was considered to be the lesser of two evils.

Dr. Shea is wrong in his implication[31] that dispersants do not increase the toxicity of oil, but simply accelerate dilution and degradation. In the long term, that may be correct (depending on mixing effectiveness), but in the short term, the toxicity risk is shifted from harm to species at the surface (birds, marine mammals) and the shoreline (crabs, some fish) to the marine organisms that live in the water column (pelagic fish, invertebrates, embryos, larvae,). If the oil is on the surface, and effective dispersants are applied, the first step is the movement of dispersed oil into the water column; that immediately moves the risk from the surface to those species and life stages in the water column. In the short term, oil concentrations rise, and subsequently will be diluted and degraded, but initially, if the dispersant is effective there is an increase in PAH and toxicity risk in the waters immediately below the surface application. For small organisms (2mm and less), particularly embryos and larvae, this transient exposure period can be lethal or damaging. These organisms and early life stages can absorb PAH loads rapidly across membranes, trapping PAH molecules into membranes, lipids, and yolks; with effects to follow.

Because of the dispersion of oil droplets, this spill had more exposure potential for organisms in the water column than probably any previous oil spill in history. The primary dispersive mechanism was the high pressure injection of oil into water at the broken riser pipe. Injection of dispersant at depth may have further dispersed the oil but their effectiveness was very difficult to assess. The effectiveness of the unprecedented dispersant application at the surface is also difficult to assess and requires the right combination of wind and wave energy, and the sampling resources at the time of the application of dispersants. I have yet to see the peer-reviewed quantitative data that defines the effectiveness of dispersant application, at the surface or at depth. For future decisions, this is an important task to complete; for the evaluation of toxicity potential, it is not. Monitoring data demonstrate that dispersions of oil were present, at depth and at the surface, and though we do not know the relative contribution of physical and chemical processes, the important fact is that dispersed oil droplets have a very significant impact on the

[31] Shea Round 2 Report at 23-26.

surface area of oil exposed to water and the subsequent loss of PAHs into water, thus increasing bioavailablity and increasing toxic risk.

V. Dr. Shea's Dismissal of the "Dirty Blizzard" Concept Ignores Key-Peer-Reviewed Evidence

Dr. Shea dismisses the "dirty blizzard" concept as theoretical, and finds little evidence of exposure in his analyses of deep sediment samples, and concludes (page 28) "There is no conclusive evidence showing that oil from the spill resulted in these impacts to deepwater coral communities". There are several lines of evidence that Dr. Shea ignores in coming to his conclusion. Again, he relies heavily on sediment samples as representative of exposure, rather than relying on evidence of impacts.

Two critical papers found harm to three deep coral communities, ranging from 6 to 11 to 22 km from the well head (White et al. 2012; Fisher et al. 2014). These communities were in the area of the detected plume (White et al. 2012; Fisher et al. 2014). The plume was drifting slowly with deep currents, consisting of fine dispersed droplets of oil, offering time and surface area for microbial action to degrade the oil and change the buoyancy of the dispersed oil droplets, and initiate the slow falling out of PAH contaminated particles (i.e. "dirty blizzard"). Processes at depth are slow (temperatures are near 4°C), and detection of PAH will be difficult given the degradation and spreading (dilution) by current.

Most importantly, the initial paper by White et al. (2012), makes the chemical connection to DWH oil by detecting PAH on impacted corals found at 1370 m about 13 km from the well head. This first study was conducted several months after the well was capped, and included state of the art chemical analyses of sediments and brown "floc" on damaged corals (conducted via GCMS and GCGC analyses of the brown floc on the surface of the impacted corals). A perfect match with DWH oil was not likely with highly weathered brown floc (degraded oil and microbial biomass), yet the authors found hopanoid biomarkers[32] that matched the spilled oil, yielding forensic evidence of the DWH oil as the source (the hopanoid biomarkers are very resistant to microbial degradation, hence their persistence and detection). Given the volume of oil spilled, over a lengthy period of time, low temperatures, within the documented path of the plume of dispersed oil, these findings are consistent with the spill event. Fisher et al. (2014), expanded the survey a year later (November 2011), and found two other coral communities impacted by oil. The closest community, 6 km from the well head, had impact to 90% of the community. The second community had less impact, but doubled the distance from the well head from the initial site discovered in 2010 (11 km to 22 km). These three communities were within the path of the plume, while communities outside of the path were not affected, further suggesting that these impacts are tied to the spill event.

[32] "Biomarkers" are chemical compounds highly resistant to degradation, hence their proportions can be used to trace the source oil in highly degraded oils.

This habitat, 1300 to 1900 meters deep is the most challenging area to study impacts, requiring state of the art technology to get even a photographic glimpse of the fauna. As discussed by Fisher et al. (2014), "we know relatively little about deep-sea fauna and communities, and therefore the full spectrum of ecosystem services derived from deep-sea biota and habitats is largely unknown." Given the age of deep corals (hundreds of years old), and slow growth, impacts to these colonies could persist for centuries before recovery is fully achieved. In the meantime, valuable cover and spawning habitat is diminished in the impacted area for an equally long time.

VI. Other Responses to Dr. Shea

B. Dr. Shea discounts edema as "quickly reversible."[33] In our experience this does not happen. Edema is coincident with damaged circulation, creating multiple problems throughout the embryos and it is never trivial. Embryos with edema do not survive as larvae. Damage can happen quickly and despite Dr. Shea's claims, is typically not reversible. For example, Pacific herring exposed to 0.7 ppb TPAH for 4 days (at a few degrees C) and then incubated in clean water were damaged - and this was observed about 20 days later (Carls et al. 1999). In rapidly developing tropical fish, time to damage is considerably more rapid (Mager et al. 2014). Swimming speed of juvenile mahi-mahi exposed for 48 h as embryos was reduced (Mager et al. 2014).

C. Dr. Shea claims (page 5) that I am incorrect in claiming that weathered oil is more toxic than fresh oil. Dr. Boesch and I explained at pages 21-22 of our Round 2 Report the flaws in Dr. Shea's logic, and I stand behind my opinion that weathered oil, on a volumetric basis, is generally more toxic than fresh oil. Tellingly, Dr. Shea fails to cite to any of the toxicity tests performed by BP or the United States for support because those data contradict his position.

D. Dr. Shea says (page 17-18) that laboratory tests have limited utility, as detection of effects at the organism level do not translate to population or ecosystem effects. He cites Fodrie et al. (2014), as evidence for the lack of significant population responses following the spill. Dr. Shea presents only half the story as he fails to note the complications of linking organism effects with population responses.

Science has great tools to detect effects at the organism level (from acute narcosis death, edema in embryos, poor growth, enzymes that respond to PAH such as P450s, and new tools such as genomic responses to exposure). In contrast, our tools at the population and ecosystem level are far less precise. First, population data is limited to a few species, usually commercial species. Even here, populations can be tracked,

[33] Shea Round 2 Report at 16.

but changes in the population are difficult to predict when they vary from trends, because we often lack the necessary information on the multitude of factors that affect populations. Peterson et al. (2003) concluded there were population and ecosystem effects following the Exxon Valdez spill, but it required a decade of information, often from multiple research efforts that needed to be combined and synthesized. Killer whale population effects were not detected for nearly 5 years; mechanism for pink salmon effects was not understood for a decade; continued oil impacts to sea otters were not figured out until oil was found in their foraging habitat, a decade after the spill. The point is, population and ecosystem effects are complex, with multiple factors including compensatory mechanisms, will be difficult to detect, and even more difficult to identify the primary cause. It is premature to expect these impacts following the DWH spill until there are more data synthesis, more time has passed, and more peer reviewed publications have matured.

E. The point (Fodrie et al. 2014) makes is that population level problems are difficult to discern in real world situations despite known damage on the individual level. We seldom have accurate population data, over time, and we seldom have a quantitative understanding of the factors that affect populations. Consequently, failure to measure population effects in the context of natural variability is not the same as no negative impact.

VII. Conclusion

I stand by my conclusion that serious potential and actual harm occurred in the Gulf of Mexico as a result of the Deepwater Horizon oil spill. This is based on both the chemical analyses of PAH concentrations in tens of thousands of water samples, but also takes into consideration the biological impacts measured in previous spills as well as the observations taken in support of the DWH spill. In contrast, Dr. Shea relies heavily on his analyses of the PAH concentration data based on a less protective "toxic units" approach, and ignores the evolution of literature on impacts from previous spills as well as the literature from the DWH spill.

The rebuttal by Shea is riddled with misinformation deliberately placed in scientific literature by industry researchers to brand legitimate research as 'controversial,' it relies on insensitive mathematical estimates of toxicity, and ignores all research that fails to support the simplistic conclusion that oil was not harmful in the vast majority of areas investigated. Each of the choices[34] Dr. Shea makes leads to less sensitivity and a lower probability of toxic impacts. Dr. Shea's position is industry advocacy and has little to do with careful science and discernment of environmental damage.

[34] I.e., choosing TUs over TPAH to estimate potential toxicity, rejecting embryo studies, and rejecting the HEWAF method

We continue to conclude that there were four habitats that had the greatest risk to oil exposure: 1) the deep area in and below the plume of dispersed oil were impacted deep water corals were observed; 2) the upper two meters of surface waters, where PAH concentratons were patchy but high enough to harm embryos, larvae, and other plankton ; 3) the surface slick where hundreds of thousands of birds and marine mammals were likely exposed during the nearly three months of the spill; and 4) the shallow nearshore and shoreline including marshes that were contaminated by oil, and can have extended exposures to oil because of the stranded oil.

It is difficult to translate the toxic effects to individuals into population effects, and subsequently into ecosystem effects, and if they occur, they will be difficult to detect. Impacts to embryos and larvae could lead to impacts on prey species, while the surface slicks affecting hundreds of thousands of birds may have significant impacts on predator loads. Long term population impacts were detected in several species long after the Exxon Valdez spill (pink salmon, sea otters, killer whales) , which led Peterson et al. (2003) to conclude that ecosystem effects were indeed possible following a large oil spill.

The bottom line is that the DWH spill, with protracted dispersive release at depth and affecting wide swaths of the water column for months, had an unusually high probability of causing serious toxic damage to species and populations, and ecosystem harm with oil fouling and toxicity.

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Appendix A

Appendix A

BP *Trachinotus carolinus* (Florida pompano) bioassay

Summary

Significant pompano mortality occurred at toxic units chronic (TUs) as low as 0.82, corresponding to and acute TU of 0.20, below the presumptive protective value of 1.0. Toxicity is **underestimated** because estimates are based on initial concentrations; endpoint concentrations were 37% of initial concentrations in this 4 day aqueous bioassay. Mortality increased with total polynuclear aromatic hydrocarbon (TPAH) concentration and with TU.

Details

Pompano broadcast buoyant pelagic eggs. Assays were completed on 10 d old organisms, presumably larvae.

Method: low energy WAF ("non-vortex")

96 hour aqueous bioassay with six treatment levels and 4 replicates per level.

Initial TPAH concentrations were reported for control, lowest and highest treatment. Other concentration levels were estimated as percentages of the highest WAF. Final concentrations were reported for the control and uppermost treatment. The measured low dose, 0.66 µg/L was somewhat lower than predicted from the high WAF (0.94 µg/L = 0.0625 * 15.07 µg/L).

Initial TPAH concentrations ranged from 0.03 (control) to 15.07 µg/L. Final concentrations were 0.04 to 0.07 µg/L (control) and 4.71 to 5.51 µg/L (high treatment).

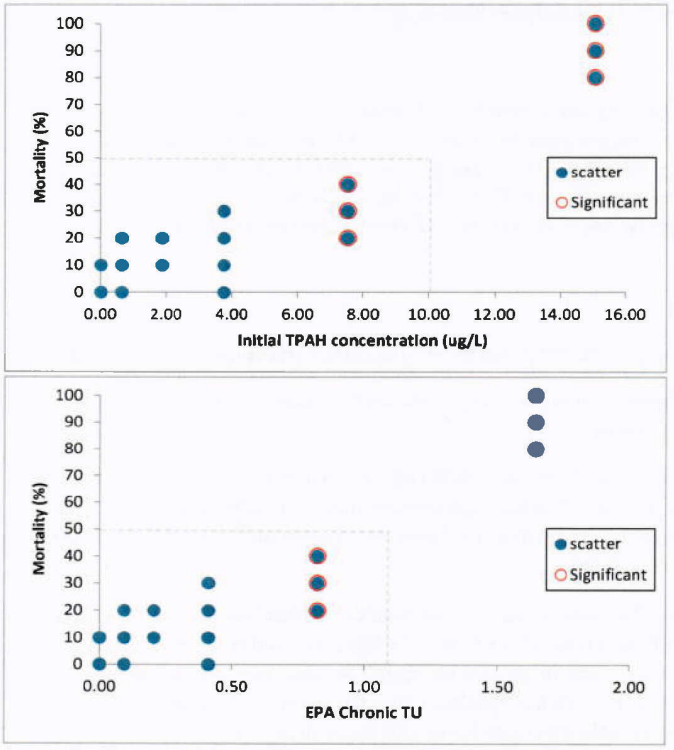
Reported animal response was percent survival. Percent mortality was calculated as 100 – percent survival.

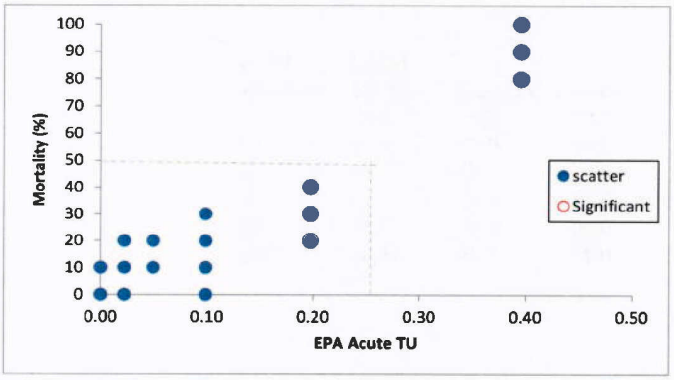
Mortality increased with dose and was significant in the upper two treatments (Fig. 1).

The lowest observed effective concentration was 7.54 µg/L, corresponding to an acute TU of 0.20 and a chronic TU of 0.82. Increased mortality was evident but not significant at the next lower dose (3.77 µg/L).

TU_{acute}	$TU_{chronic}$	Initial TPAH	Mean mortality
0.00	0.00	0.03	5
0.02	0.09	0.66	10
0.05	0.21	1.88	15
0.10	0.41	3.77	15
0.20	0.82	7.54	30
0.40	1.65	15.07	90

Fig. 1. Pompano mortality.





BP *Leptocheirus plumulosus* (amphipod) bioassays.

Summary

Significant amphipod mortality occurred at toxic units (TUs) as low as 0.07, or more likely at 0.12, well below the presumptive protective value of 1.0. Mortality increased with total polynuclear aromatic hydrocarbon (TPAH) concentration and with TU. The oil was weathered; percent naphthalenes ranged from 4.4 to 22.2% (Fig. 1). Conversely, percent chrysenes ranged from 5.9 to 46.0%. Naphthalene content in fresh DWH oil was substantially greater (Liu et al. 2012).

Details

A series of paired bioassays were completed by BP with control and oil-contaminated sediment.

Based on a personal communication, we assumed that total organic carbon was reported as a percentage, not mg/kg as stated.

The TPAH concentration ranged from about 74000 µg/kg to control (presumably ~ 0 but illustrated as 1 µg/kg in the figure to allow logarithmic scaling). Corresponding chronic TUs, estimated by the EPA method, ranged from 4.48 to control (presumably ~0 but illustrated as 0.01 units to allow logarithmic scaling).

Significant responses are illustrated in red as indicated in the BP database (Fig. 2). Comment: there must be some reporting errors in the database; the three red points near the x-axis are unlikely significant. If the 3 points in question are significant, then there was significant mortality at a TU = 0.07. If they are not significant, then the minimum damaging TU was 0.12. In either case, damaging TU values were far below the presumptively protective TU = 1.0 value.

Fig. 1. Example PAH composition in amphipod sediment assays. The top panel is the sample with the probable minimum chronic TU (0.27 units); percent chrysenes = 31. Total PAH in the bottom example was more than 20 times greater; TU = 4.38 and percent chrysenes = 9.9.

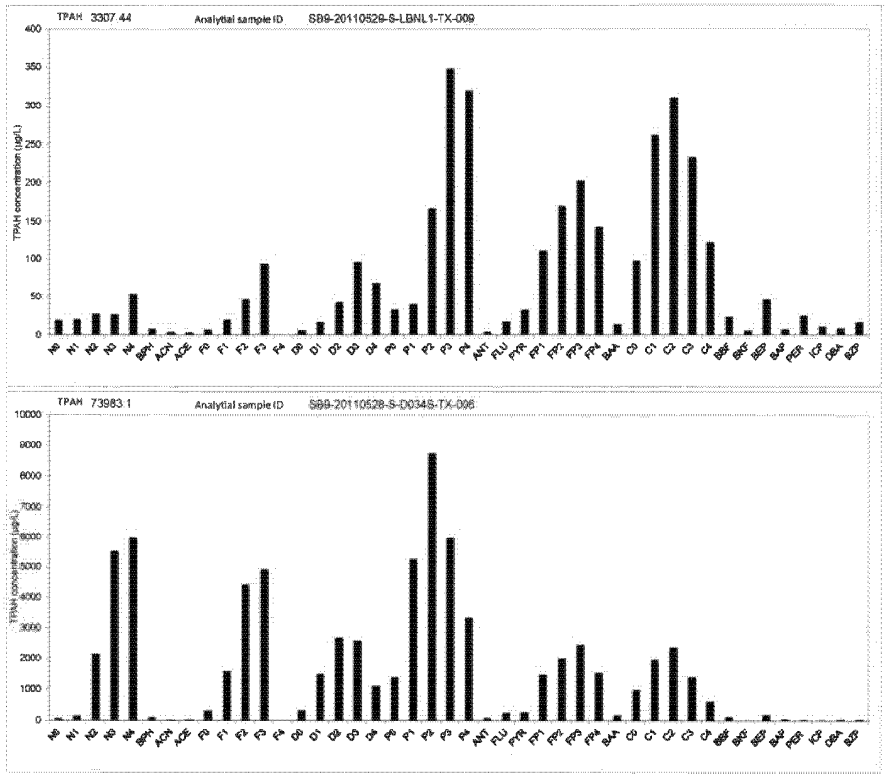
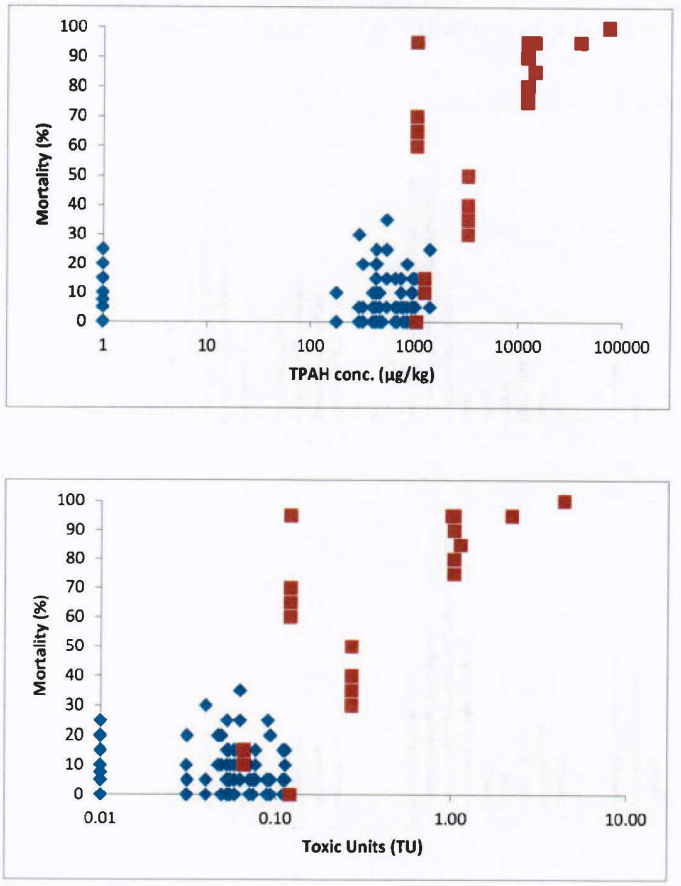


Fig. 2. Amphipod mortality as a function of TPAH (top panel) and chronic TUs (bottom panel).



Liu, Z., J. Liu, et al. (2012). "The weathering of oil after the *Deepwater Horizon* oil spill: insights from the chemical composition of the oil from the sea surface, salt marshes and sediments." Environmental Research Letters 7(3).

Appendix B

APPENDIX B

Source Information

Data file:

WaterChemistry_W-01v02-01.csv

..\analysis\WaterChemistryPAH.mdb

..\analysis\HC Gulf Mexico WATER 2010 combined v4.xlsx

..\analysis\EPA tox estimates WATER.xlsx

..\analysis\TPAH by month place depth.xlsx

..\reports\ 9-12-14 Estimation of toxic potential in DWH water samples version 4-doj cmts mgc 9-24-14 update.docx

..\analysis\maps\Water TPAH surface May Jun Jul.mxd

..\analysis\maps\Water TPAH surface CHECK May Jun Jul.mxd

Estimation of toxic potential in DWH water samples.

1. Samples were obtained from "BP Gulf Science Data (NRDA-publicly available)," file name "WaterChemistry_W-01v02-01.csv" dated 5/23/2014.
2. The CSV file was too long to read directly with Excel, thus was subdivided into several files.
3. All 2010 data were extracted into Excel.
4. PAH data were assembled to yield one record per sample. Samples were identified by "Laboratory sample ID." This resulted in 16,167"natural samples" in 2010, excluding replicate samples. (There were 138 replicates, labeled "field duplicate.") For comparison with Dr. Shea's analysis, there were 17,881 "natural samples" in the NRDA database, thus 1714 samples were collected after 2010; these were not analyzed here.
5. Polynuclear aromatic hydrocarbon (PAH) analytes are listed below. Not all of these were measured in every sample. ND concentrations were 0.

N0	D0	PYR	BBF
N1	D1	FP1	BKF
N2	D2	FP2	BEP
N3	D3	FP3	BAP
N4	P0	FP4	PER
ACN	P1	BAA	ICP
ACE	P2	C0	DBA
F0	P3	C1	BZP
F1	P4	C2	
F2	ANT	C3	
F3	FLU	C4	

6. Concentrations were summed to yield total PAH (TPAH). Data were analyzed by month, depth, and location (Fig. 1).
7. The EPA threshold method was applied to each water sample. The acute version was corrected per discussion with Dr. David Mount, one of the original EPA authors. All alkylated PAHs were included in the model, thus no alkyl-adjustment multipliers were required (Fig. 2).
8. Alternative estimations of toxicity were based on Gulf of Mexico larval fish assays: (Incardona et al. 2014) reported threshold TPAH concentrations as low as 0.3 µg/L. Numbers of samples above this threshold (and several other comparison values, 0.5, 1, and 2 µg/L) were summed by month to calculate the fraction toxic (per month, depth, and location) (Fig. 3a-d).
9. To estimate fractions toxic within the slick area only, data were plotted by month with ArcMap along with satellite slick information (SAR). Offshore samples within polygons bounding the slick area were identified with ArcMap (Fig. 4).
10. The toxic fraction in the offshore surface water (0 –2 m) within slick boundaries was estimated for May through July with the TPAH concentration method described in step 8 (Figs. 5 - 6).
11. It should be noted that this analysis is not intended to be a quantitative assessment of the extent of oil contamination in the Gulf. That is more properly a part of the NRD Assessment, which is still ongoing and may employ additional data and methods of analysis. The purpose of

this exercise is simply to point out that Dr. Shea's opinion regarding the extent of toxic concentrations of PAHs in the Gulf is misleading because it fails to employ the appropriate toxicological thresholds and fails to focus on the areas and times when high concentrations of PAHs were likely to occur.

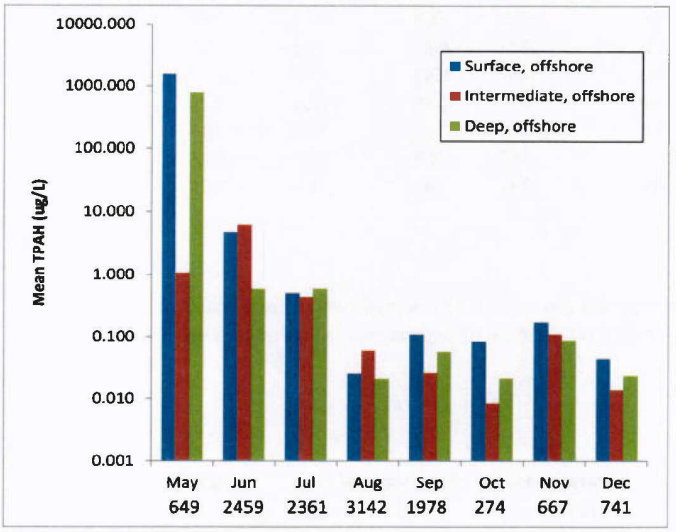
Roughly one quarter of the samples were collected nearshore; the remainder were collected offshore.

	n offshore	*n* Total	%nearshore	%offshore
May	646	970	33.4	66.6
Jun	2452	3093	20.7	79.3
Jul	2334	2962	21.2	78.8
Aug	3087	3643	15.3	84.7
Sep	1943	2525	23.0	77.0
Oct	270	920	70.7	29.3
Nov	667	1067	37.5	62.5
Dec	741	987	24.9	75.1

Within the offshore data set, about one quarter of the samples were from the surface (upper 2 m), one quarter were from plume depths (≥1000 m), and the remaining half were from elsewhere in the water column.

	n Surface	n Plume	%Surface	%Plume	%Other
May	129	197	20.0	30.5	49.5
Jun	739	619	30.1	25.2	44.6
Jul	739	715	31.7	30.6	37.7
Aug	723	1043	23.4	33.8	42.8
Sep	371	711	19.1	36.6	44.3
Oct	98	23	36.3	8.5	55.2
Nov	163	219	24.4	32.8	42.7
Dec	135	377	18.2	50.9	30.9

Fig. 1. Mean total aqueous PAH concentration by month. Surface is ≤ 2 m, deep is ≥ 1000 m and intermediate is all depths between. The total number of samples analyzed each month for offshore and nearshore sets is listed along the x-axis.



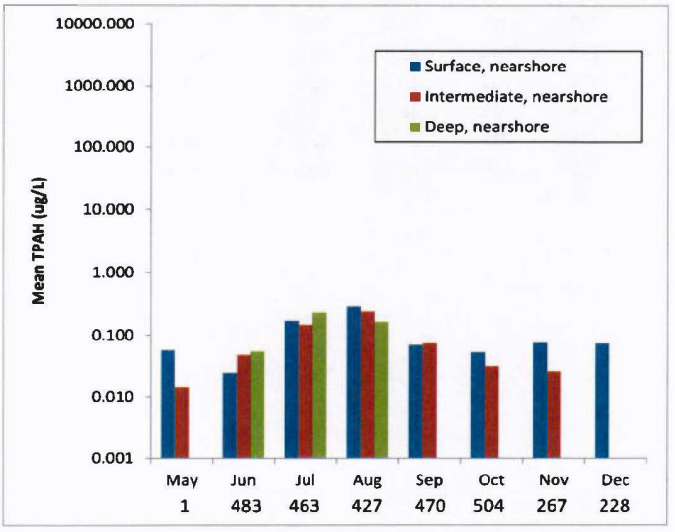
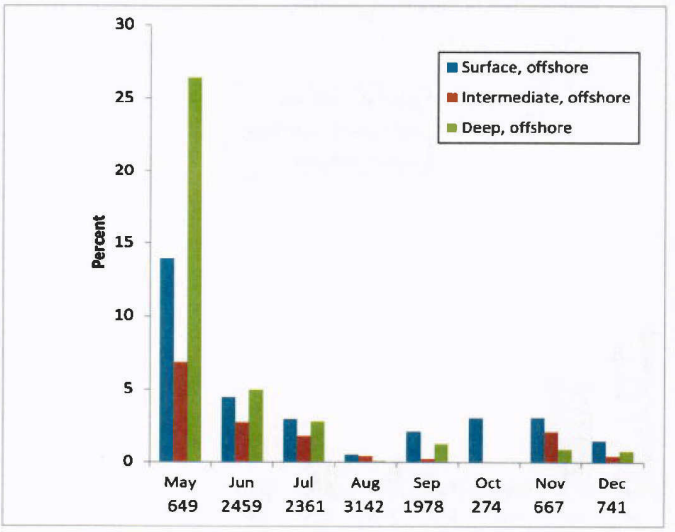


Fig. 2. Percent of samples exceeding EPA toxicity threshold for water samples as a function of time using the EPA threshold method (chronic toxicity).



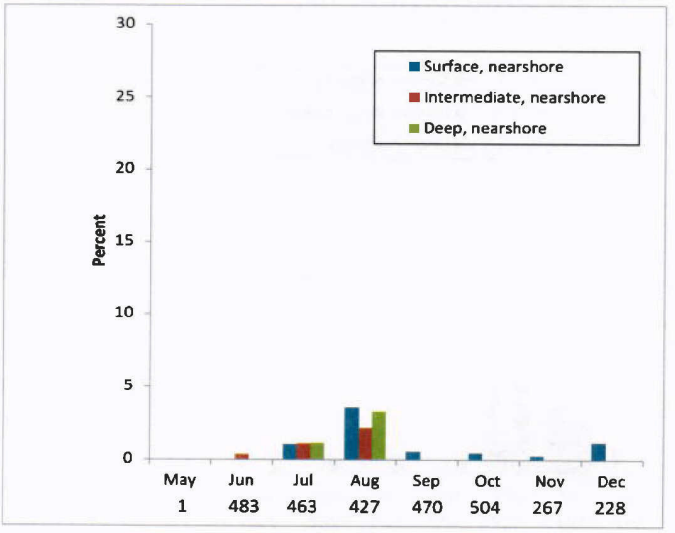


Fig. 3a. Percent of water samples as a function of time that exceed toxicity threshold using embryo sensitivity estimates: 0.3 µg/L. Estimated embryo toxicity thresholds were as low as 0.3 for bluefin tuna and were between 1 and 6 µg/L for amberjack (Incardona et al. 2014).

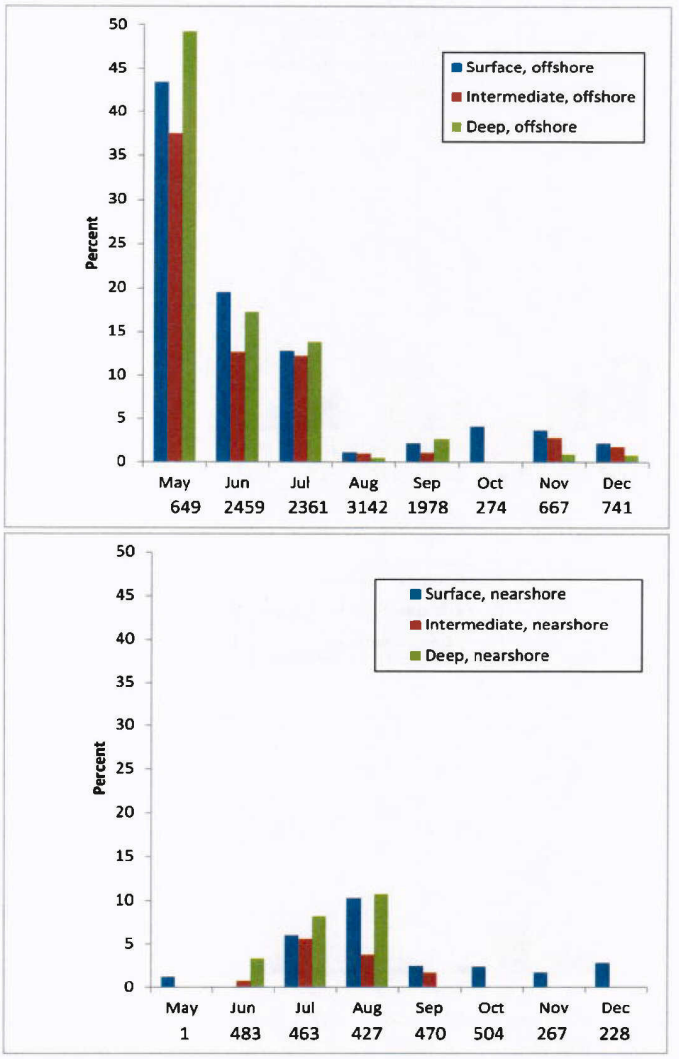
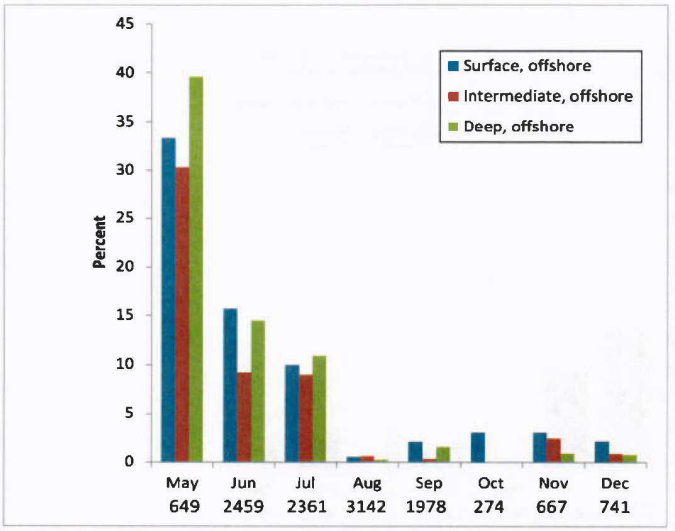


Fig. 3b. Percent of water samples as a function of time that exceed toxicity threshold using embryo sensitivity estimates: 0.5 µg/L. Estimated embryo toxicity thresholds were as low as 0.3 for bluefin tuna and were between 1 and 6 µg/L for amberjack (Incardona et al. 2014).



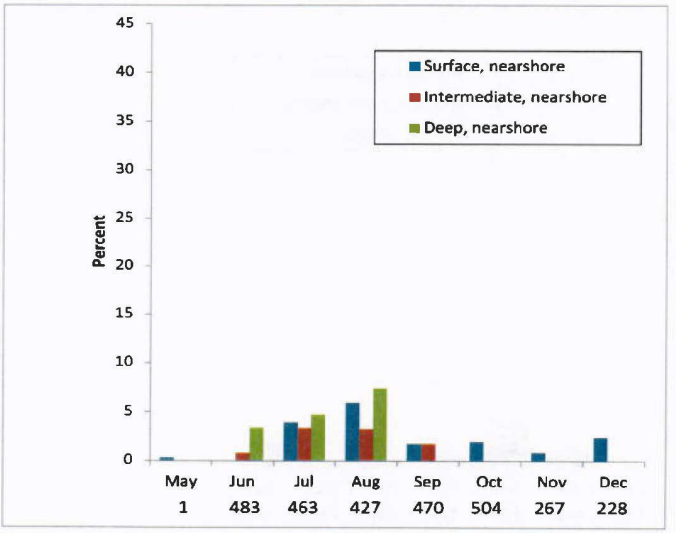
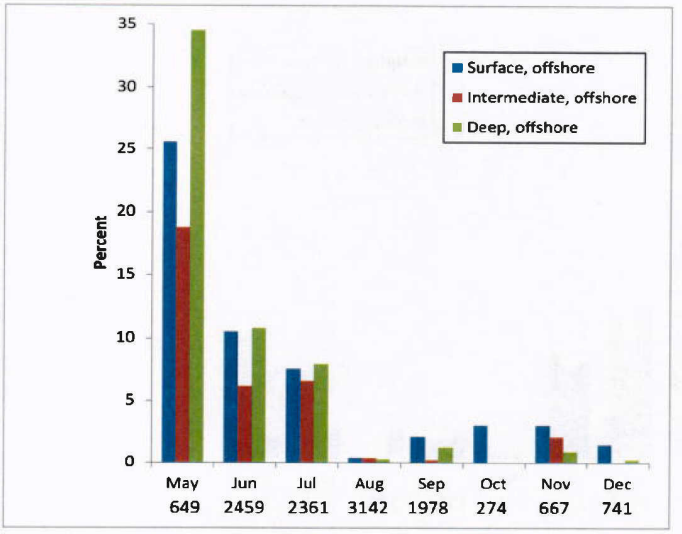


Fig. 3c. Percent of water samples as a function of time that exceed toxicity threshold using embryo sensitivity estimates: 1.0 µg/L. Estimated embryo toxicity thresholds were as low as 0.3 for bluefin tuna and were between 1 and 6 µg/L for amberjack (Incardona et al. 2014).



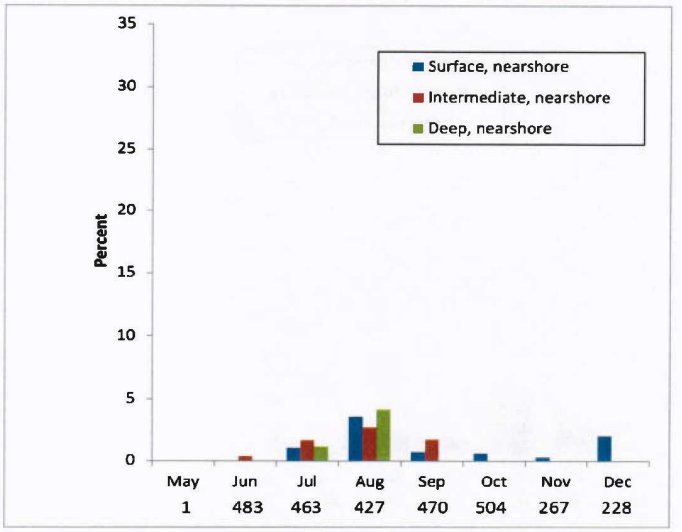


Fig. 3d. Percent of water samples as a function of time that exceed toxicity threshold using embryo sensitivity estimates: 2.0 µg/L. Estimated embryo toxicity thresholds were as low as 0.3 for bluefin tuna and were between 1 and 6 µg/L for amberjack (Incardona et al. 2014).

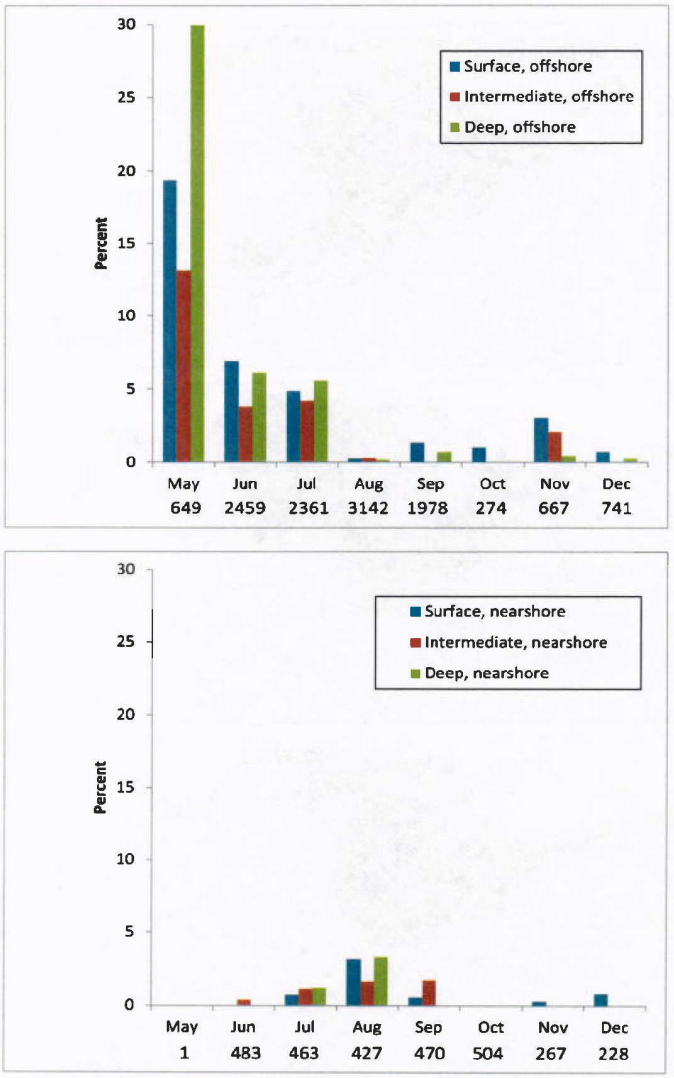


Fig. 4. Offshore samples within slick areas were defined as those within the slick boundaries identified by satellite (dark grey). Samples to the west of the primary slick boundary in June were not included as "within."

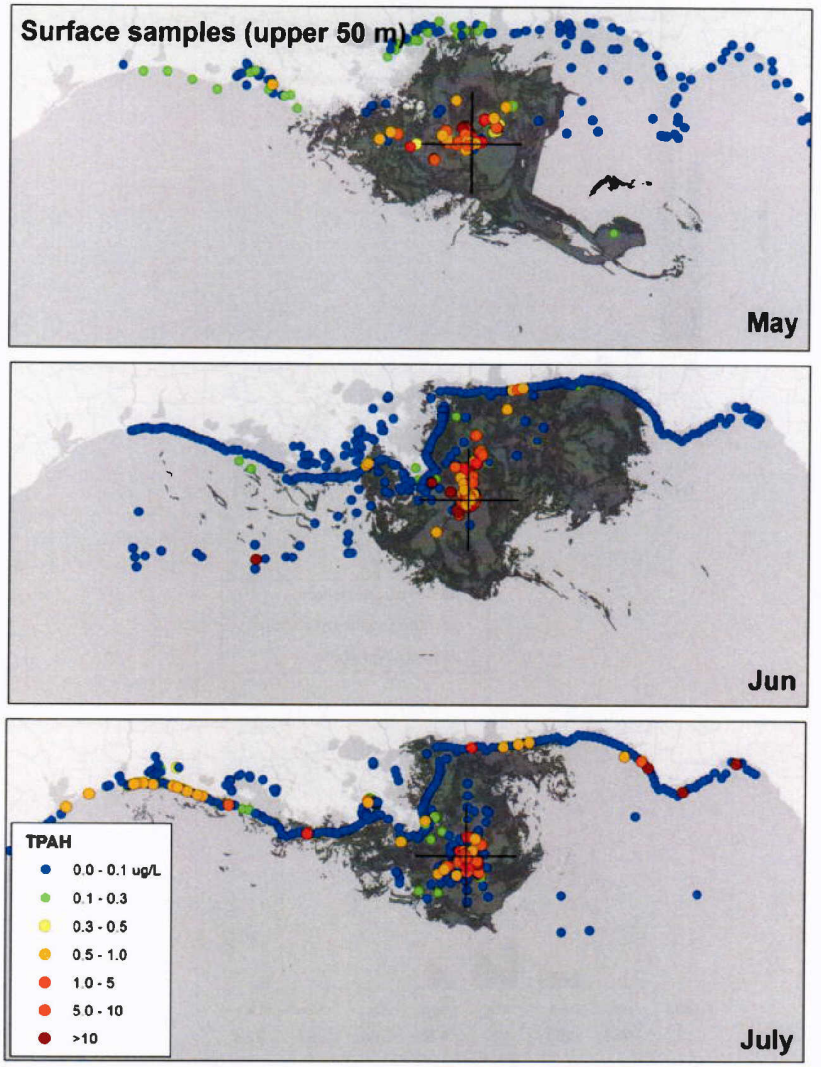


Fig. 5. Toxicity estimate in surface water (0 – 2 m) within the slick area only during the time the slick was present (May – July). Total slick area was determined by satellite and composited by month.

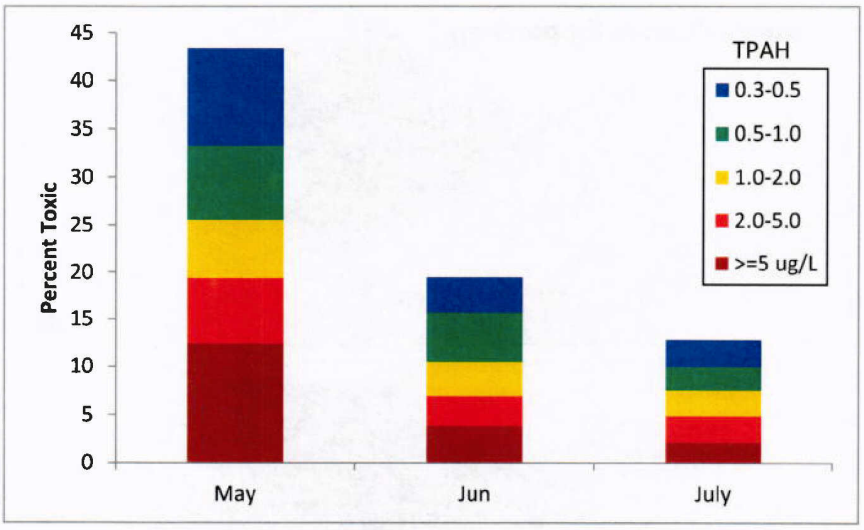
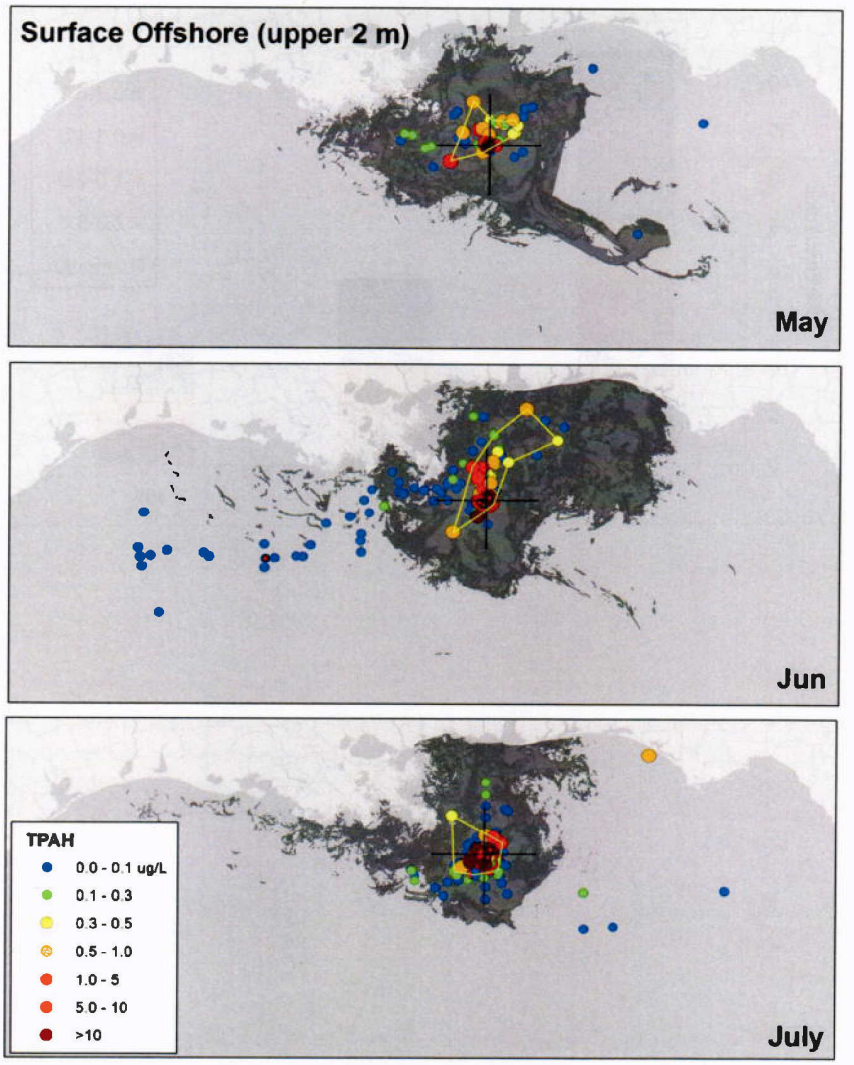


Fig. 6. Offshore surface water samples only. Surface is defined as the upper 2 meters. Yellow polygons bound observed area with toxic concentrations (at 0.3 µg/L). These areas are 5460, 12131, and 6225 km^2.



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Incardona, J. P., L. D. Gardner, et al. (2014). "Deepwater Horizon crude oil impacts the developing hearts of large predatory pelagic fish." Proceedings of the National Academy of Sciences of the United States of America **www.pnas.org/cgi/doi/10.1073/pnas.1320950111**: E1510–E1518.

Appendix C

Toxicity research demonstrating embryo responses at concentrations ≤ 20 µg/L.

Species	Life stage	Response	Toxin	Conc. (µg/L)	Exposure time (d)	Method	Author
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	Pericardial & yolk-sac edema, threshold	DWH oil: TPAH	0.3	Incubation period	HEWAF (high energy water-accommodated fraction)	Incardona et al. 2014
Pacific herring (*Clupea pallasii*)	Embryo	Abnormalities, genetic damage, growth, mortality	aqueous TPAH, more weathered Alaska North Slope crude oil	0.4	16	ORC	Carls et al. 1999
Yellowfin tuna (*Thunnus albacares*)	Embryo	Pericardial & yolk-sac edema, threshold	DWH oil: TPAH	0.5 - 1.3	Incubation period	HEWAF	Incardona et al. 2014
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	EC50, edema	DWH oil: TPAH	0.8	Incubation period	HEWAF	Incardona et al. 2014
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	Pericardial & yolk-sac edema, EC50	DWH oil: TPAH	0.8	Incubation period	HEWAF	Incardona et al. 2014
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Post-emergent growth	aqueous TPAH, weathered Alaska North Slope crude oil	0.94	~198	ORC	Carls et al. 2005
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Mortality	aqueous TPAH, very weathered Alaska North Slope crude oil	1	~240	ORC	Heintz et al. 1999

Yellowfin tuna (*Thunnus albacares*)	Embryo	heart rate bradycardia threshold	DWH oil: TPAH	1.0 - 2.6	Incubation period	HEWAF	Incardona et al. 2014
Greater amberjack (*Seriola dumerili*)	Embryo	Pericardial & yolk-sac edema, threshold	DWH oil: TPAH	1.0 - 6.0	Incubation period	HEWAF	Incardona et al. 2014
Atlantic cod (*Gadus morhua*)	Larvae	CYP3A induction	unspecified crude oil, possibly North Sea	1.2	4	Mechanical dispersion	Olsvik et al 2011
Mahi-Mahi (*Coryphaena hippurus*)	embryo	Edema	DWH oil: TPAH	1.2	2	HEWAF	Mager et al 2014
Mahi-Mahi (*Coryphaena hippurus*)	embryo	Reduced swimming speed	DWH oil: TPAH	1.2	2		Mager et al 2014
Atlantic cod (*Gadus morhua*)	Larvae	Reduced survival	unspecified crude oil, possibly North Sea	<2	4	Mechanical dispersion	Olsvik et al 2012
Atlantic cod (*Gadus morhua*)	Larvae	CYP3A induction	unspecified crude oil, possibly North Sea	2.1	4	WSF isolated from Mechanical dispersion	Olsvik et al 2011
Yellowfin tuna (*Thunnus albacares*)	Embryo	EC50, edema	DWH oil: TPAH	2.3	Incubation period	HEWAF	Incardona et al. 2014
Yellowfin tuna (*Thunnus albacares*)	Embryo	Pericardial & yolk-sac edema, EC50	DWH oil: TPAH	2.3	Incubation period	HEWAF	Incardona et al. 2014
Yellowfin tuna (*Thunnus albacares*)	Embryo	EC50 for prolongation of systole	DWH oil: TPAH	2.6	Incubation period	HEWAF	Incardona et al. 2014

Greater amberjack (*Seriola dumerili*)	Embryo	heart rate bradycardia threshold	DWH oil: TPAH	2.2 - 6.5	Incubation period	HEWAF	Incardona et al. 2014
Yellowfin tuna (*Thunnus albacares*)	Embryo	Extracardiac defects	DWH oil: TPAH	3.4	Incubation period	HEWAF	Incardona et al. 2014
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	CYP1A induction	aqueous TPAH, weathered Alaska North Slope crude oil	3.7	~198	ORC	Carls et al. 2005
Pacific herring (*Clupea pallasii*)	Embryo	Abnormalities	Whole oil	4	13	Flowing oil-water contact	Pearson et al. 1985
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	heart rate bradycardia threshold	DWH oil: TPAH	4	Incubation period	HEWAF	Incardona et al. 2014
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Ascites, premature emergence, gonadal cell apoptosis, induction of CYP1A	aqueous TPAH, weathered Alaska North Slope crude oil	4.4	177	ORC	Marty et al. 1997
Greater amberjack (*Seriola dumerili*)	Embryo	heart rhythm irregularities, minimum influential exposure conc.	DWH oil: TPAH	4.5	Incubation period	HEWAF	Incardona et al. 2014
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Marine survival	aqueous TPAH, weathered Alaska North Slope crude oil	5.4	~240	ORC	Heintz et al. 2000

Yellowfin tuna (*Thunnus albacares*)	Embryo	heart rate IC50 (half max inhibitory conc)	DWH oil: TPAH	6.1	Incubation period	HEWAF	Incardona et al. 2014
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	heart rate IC50 (half max inhibitory conc)	DWH oil: TPAH	7.7	Incubation period	HEWAF	Incardona et al. 2014
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Blue sac disease (ascites)	aqueous TPAH, weathered Alaska North Slope crude oil	7.8	83	ORC	Brannon et al. 2006
Atlantic bluefin tuna (*Thunnus thynnus*)	Embryo	Extracardiac defects	DWH oil: TPAH	8.5	Incubation period	HEWAF	Incardona et al. 2014
Greater amberjack (*Seriola dumerili*)	Embryo	EC50 for prolongation of systole	DWH oil: TPAH	8.6	Incubation period	HEWAF	Incardona et al. 2014
Pacific herring (*Clupea pallasii*)	Embryo	Abnormalities, growth, mortality	aqueous TPAH, weathered Alaska North Slope crude oil	9.1	16	ORC	Carls et al. 1999
Capelin (*Mallotus villosus*)	Embryo	Reduced hatching success	WSF, Ekofisk crude oil	<10	42-49	Flowing oil-water contact	Johannessen 1976
Japanese medaka (*Oryzias latipes*)	Embryo	LOEC, hatch length	PAHs	11	18	static renewal	Farwell et al. 2006
Greater amberjack (*Seriola dumerili*)	Embryo	EC50, edema	DWH oil: TPAH	12.4	Incubation period	HEWAF	Incardona et al. 2014
Greater amberjack (*Seriola dumerili*)	Embryo	Pericardial & yolk-sac edema, EC50	DWH oil: TPAH	12.4	Incubation period	HEWAF	Incardona et al. 2014

Greater amberjack (*Seriola dumerili*)	Embryo	Extracardiac defects	DWH oil: TPAH	13.8	Incubation period	HEWAF	Incardona et al. 2014
Greater amberjack (*Seriola dumerili*)	Embryo	heart rhythm irregularities, significant	DWH oil: TPAH	13.8	Incubation period	HEWAF	Incardona et al. 2014
Zebrafish (*Danio rerio*)	Embryo	Cardiac abnormalities	Aqueous TPAH, Alaska North Slope crude oil	15	2	Mechanical dispersion	Carls et al. 2008
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Mortality	aqueous TPAH, weathered Alaska North Slope crude oil	16.4	83	ORC	Brannon et al. 2006
Pacific herring (*Clupea pallasii*)	Embryo	Edema	aqueous TPAH, more weathered Alaska North Slope crude oil	17.3	4	ORC	Carls et al. 1999
Pink salmon (*Oncorhynchus gorbuscha*)	Embryo-larvae	Post-emergent growth	aqueous TPAH, weathered Alaska North Slope crude oil	18	~240	ORC	Heintz et al. 2000
Greater amberjack (*Seriola dumerili*)	Embryo	heart rate IC50 (half max inhibitory conc)	DWH oil: TPAH	18.2	Incubation period	HEWAF	Incardona et al. 2014

References, embryo toxicity table

See "Toxicity table.xlsx"

Barron, M. G., M. G. Carls, et al. (2003). "Photoenhanced toxicity of aqueous phase and chemically dispersed weathered Alaska North Slope crude oil to Pacific herring eggs and larvae." Environmental Toxicology and Chemistry **22**(3): 650-660.

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Carls, M. G., L. Holland, et al. (2008). "Fish embryos are damaged by dissolved PAHs, not oil particles." Aquatic Toxicology **88**: 121-127.

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Farwell, A., V. Nero, et al. (2006). "Modified Japanese medaka embryo-larval bioassay for rapid determination of developmental abnormalities." Archives of Environmental Contamination and Toxicology **51**(4): 600-607.

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Pearson, W. H., D. L. Woodruff, et al. (1985). Oil effects on spawning behavior and reproduction in Pacific herring (*Clupea harengus pallasi*). Washington, DC, Final report to the American Petroleum Institute, Environmental Affairs Department.

(Olsvik, Lie et al. 2012)
(Olsvik, Hansen et al. 2011)
(Johannessen 1976)
(Farwell, Nero et al. 2006)
(Pearson, Woodruff et al. 1985)
(Carls, Rice et al. 1999)
(Barron, Carls et al. 2003)
(Incardona, Gardner et al. 2014)
(Brannon, Collins et al. 2006)
(Marty, Short et al. 1997)
(Heintz, Short et al. 1999)
(Heintz, Rice et al. 2000)
(Carls, Heintz et al. 2005)
(Carls, Holland et al. 2008)

Appendix D

Sources Considered
(In addition to the documents cited in my Round 1 and Round 2 and Round 3 reports and my Round 1 and Round
2 lists of documents considered)

Bates, Exhibit, TREX, or Other Description
BP-HZN-2179MDL02042412-BP-HZN-2179MDL02042420
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BP-HZN-2179MDL09309873-BP-HZN-2179MDL09309886
C1B004-000344-C1B004-000353
Deposition Exhibit 9182
Expert Report of Dr. Damian Shea
Expert Report of Dr. John W. Tunnell, Jr.
Expert Report of Joseph R. Geraci
Rebuttal Report of Elliott Taylor
US_PP_DBO003442-US_PP_DBO003449
US_PP_DBO004015-US_PP_DBO004022
US_PP_DBO005004-US_PP_DBO005012
US_PP_DBO006193-US_PP_DBO006258
US_PP_DBO007388-US_PP_DBO007398
US_PP_EPA050352-US_PP_EPA050366
US_PP_NOAA2_0141865-US_PP_NOAA2_0141873
US_PP_RICE000372-US_PP_RICE000380

Sources Considered
(In addition to the documents cited in my Round 1 and Round 2 and Round 3 reports and my Round 1 and Round
2 lists of documents considered)

Bates, Exhibit, TREX, or Other Description
US_PP_RICE002728-US_PP_RICE002738
US_PP_RICE003111-US_PP_RICE003122
US_PP_RICE003200-US_PP_RICE003204
US_PP_RICE003268-US_PP_RICE003277
US_PP_RICE003305-US_PP_RICE003317
US_PP_RICE003324-US_PP_RICE003340
US_PP_RICE003377-US_PP_RICE003389
US_PP_RICE004505-US_PP_RICE004523
US_PP_RICE005057-US_PP_RICE005192
US_PP_RICE005193-US_PP_RICE005201
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US_PP_RICE005228-US_PP_RICE005229
US_PP_RICE005230-US_PP_RICE005242
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