# EXPERT REPORT OF DR. DAMIAN SHEA

## Reply Report to the Expert Response Report of:
## Donald F. Boesch and Stanley D. Rice

**In re: Oil Spill by the Oil Rig "Deepwater Horizon" in the Gulf of Mexico**

**MDL 2179**

**U.S. District Court for the Eastern District of Louisiana**

**September 26, 2014**

**CONFIDENTIAL PURSUANT TO PTO 13**

24 September 2014

DAMIAN SHEA, Ph.D. DATE

# I. OVERVIEW: THE UNITED STATES' ENVIRONMENTAL EXPERTS RELY UPON LIMITED DATA AND UNRELIABLE TOXICITY THRESHOLDS

Contrary to the assertions of the United States' environmental experts, my analysis contains a comprehensive discussion of the chemistry and toxicity data in this case as well as careful consideration of the peer-reviewed literature:

1. I considered all of the publicly available water and sediment chemistry data included on the Gulf Science Data website; Drs. Boesch and Rice emphasize only a tiny subset of the data even though all of data have been available to the United States for months.

2. I considered toxicity thresholds that were developed by the United States Environmental Protection Agency (EPA) over decades; Drs. Boesch and Rice rely upon a few deeply problematic laboratory studies that have never been verified or endorsed by any regulatory agency.

3. By utilizing the EPA toxicity benchmarks, I have incorporated over 170 toxicity studies (primarily from peer-reviewed publication), which address endpoints from 28 different species and over 30 of the studies tested toxicity to sensitive early life stages (embryos and larvae).[1] In addition, I reviewed and summarized over 900 toxicity tests conducted as part of the *Deepwater Horizon* investigation.[2] In contrast, Dr. Rice's proposed toxicity threshold of 0.5 parts per billion (ppb) was developed from a single toxicity test, the results of which have not been replicated nor confirmed by any other published study.

4. Using standard EPA methods (tried and true for over 30 years and utilized every day by the United States), I have considered the possibility of both lethal and sublethal, and both acute and chronic impacts from polycyclic aromatic hydrocarbons (PAHs) from the *Deepwater Horizon* oil. Drs. Boesch and Rice are wrong to claim otherwise.

5. I did not stop there. In addition to PAHs, I also considered potential impacts from BTEX (the volatile components of oil) as well as the constituents in dispersants (DOSS).

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[1] *See* US EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013, at Appendix C; and McGrath, J.A. and Di Toro, D.M. 2009. Validation of the target lipid model for toxicity assessment of residual petroleum constituents: Monocyclic and polycyclic aromatic hydrocarbons. Environ. Toxicol. Chem. 28:1130–1148.

[2] August 15, 2014 Report of Dr. Shea at 40-47.

6. For these reasons, my analysis is comprehensive, inclusive, and scientifically robust. The United States, on the contrary, selectively picks data and studies to greatly overstate the potential for environmental harm from the *Deepwater Horizon* incident.

| | Shea Reports | Boesch/Rice Reports |
|---|---|---|
| Number of PAH Water Samples Analyzed | **17,881** | **327*** |
| Source of Proposed PAH Toxicity Threshold | **U.S. EPA** | **Incardona Paper** (unverified and not replicated) |
| Studies Used to Develop PAH Toxicity Threshold | **173**** | **1** |
| Species Used to Develop PAH Toxicity Threshold | **28** | **1** |
| DWH-Specific Tests Used to Verify PAH Threshold | **Over 900** | **1** |
| Number of BTEX Water Samples Analyzed | **16,772** | **0** |
| Number of DOSS Water Samples Analyzed | **5,672** | **0** |
| Number of PAH Sediment Samples Analyzed | **8,181** | **0** |

*Note that 15,114 samples are summarized in Appendix A to the September 12, 2014 Boesch/Rice report. The actual text of that report discusses only plume samples from May 2010 (198 samples) and surface samples from May 2010 (129 samples). The August 15, 2014 Rice report similarly discusses only samples from May 2010 (650 samples). The numbers provided herein are based on the statistics provided in Appendix A of the September 12 Boesch/Rice report, which, as discussed elsewhere, I have been unable to replicate.
**An additional 29 datasets were used to develop the acute/chronic ratio.

***Table 1.*** *I relied upon the large volume of data available from the Gulf of Mexico and from several decades worth of toxicity data (primarily from the peer-reviewed literature), while Drs. Rice and Boesch selected a single flawed study as the basis of their entire analysis.*

## II. THE BOESCH AND RICE REPORTS LACK SCIENTIFIC RIGOR

1. In my initial report, I performed a rigorous analysis of both the quality and meaning of the chemistry and toxicity data collected as part of the *Deepwater Horizon* environmental investigation. For example, I performed a quality assurance review of the Field Sampling and Study Plans, the Laboratory Quality Assurance Project Plans, Standard Operating Procedures, and the quality control data used to assess data quality. Drs. Rice and Boesch undertook no such review.

2. The reports of Drs. Rice and Boesch lack basic scientific rigor. Their initial reports provided no data or independent analysis. In general, they simply inferred harm from either (a) other oil spills that were substantially different[3] or (b) a few recent scientific publications that largely discuss hypothetical impacts. Their second report did include some data (*see* Table 1 above), but for the reasons discussed herein, their analysis was incomplete and misleading.

3. For example, the maps shown in Appendix A to their joint report are not an appropriate way to display the complete chemistry data set. Drs. Rice and Boesch appear to have simply plotted individual samples that they selected on a map of the Gulf (Boesch/Rice Appendix A, Figs. 4 and 6) and then drawn lines to connect the dots (Boesch/Rice Appendix A, Fig. 6), creating crude polygons that help support their point. In reality, there was not continuous oiling from one sampling point to another. My analysis took the actual concentrations of all samples and used a scientifically-based statistical analysis method (called "kriging") to estimate the likely concentration at all locations, resulting in an unbiased presentation of the data. This is analogous to simply observing the height of a few points on a mountain and connecting those "dots" (the Boesch/Rice approach) compared to a detailed survey that provides a high resolution topographic map with contours of elevations (my approach).

4. Even so, although the maps shown by Drs. Rice and Boesch exaggerate exposure, they do not contradict my analysis and opinions. They show the highest concentrations of PAH that might (if the requisite exposures occurred) cause harm (above 5 or 10 ppb) are limited to very few samples and largely near the wellhead. Drs. Rice and Boesch also end their time series of maps in July, so they do not show how the higher concentrations of PAHs drop dramatically once the wellhead was capped.

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[3] In one recent study comparing the *Deepwater Horizon* to the *Exxon Valdez* spill, the authors found "considerably less" impact from the Deepwater Horizon with "faster recovery" and further concluded that "the fisheries at each location were affected differently by each oil spill, largely due to environmental differences between the areas." Nelson, J.R., Bauer, J.R., Rose, K. 2014. Assessment of Geographic Setting on Oil Spill Impact Severity in the United States – Insights from Two Key Spill Events in Support of Risk Assessment for Science-Based Decision Making. J. Sustainable Energy Eng. (accepted Sept. 5, 2014), DOI: 10.7569/JSEE.2014.629510.

| | Shea Reports | Boesch/Rice Reports |
|---|---|---|
| Reviewed field and laboratory quality assurance plans | YES | NO |
| Reviewed and performed quality assurance check on raw data | YES | NO |
| Took into account that different PAHs have different toxicities | YES | NO |
| Took into account that there are differences between PAH concentrations in lab studies versus field collected samples | YES | NO |
| Critically reviewed the lab exposure studies to determine how well they represent conditions in the Gulf of Mexico | YES | NO |
| Performed statistically-based mapping to estimate probabilities of exceeding benchmarks in the Gulf of Mexico | YES | NO |

***Table 2.*** *The reports of Drs. Rice and Boesch lacked the critical review and scientific rigor of my reports.*

# III. THE BOESCH AND RICE JOINT REPORT USES SELECTIVE DATA TO EXAGGERATE POTENTIAL HARM

1. In their joint report, Drs. Boesch and Rice discuss only the results of water column sampling from the month of May, 2010.[4] This focus on May sampling alone is misleading because May was the month during the spill in which the fewest number of samples were collected and sampling was targeted on areas with high concentrations of oil. The bias present in the May sampling data is immediately apparent when compared against data collected during the oil release in June and through July 15, 2010, when the wellhead was capped. After the wellhead was capped on July 15, the percentage of water column samples that exceeded the EPA chronic PAH benchmarks declined dramatically.

[4] September 12, 2014 Report of Drs. Boesch and Rice at 24-25.

| | Number of Water Samples Collected | % Exceeding EPA Chronic PAH Benchmark |
|---|---|---|
| **May 2010** | 777 | 10% |
| **June 2010** | 3,007 | 3% |
| **July 1-15, 2010** (wellhead capped July 15, 2010) | 1,628 | 2.6% |
| **July 15-30, 2010** | 1,265 | 1.3% |
| **August 2010** | 3,619 | 0.8% |

***Table 3.*** *Despite substantially similar conditions in May, June and the first half of July, the May sample concentrations are demonstrably biased high. This is likely due to the fact that relatively few samples were collected in May, and sampling activities were especially focused on finding oil. After the wellhead was capped on July 15, the percentage of samples that exceeded the EPA chronic PAH benchmark declined dramatically.*

2. In Appendix A to their joint report, Drs. Rice and Boesch include additional water sampling data, however they characterize the data in misleading ways, including by calculating an average concentration of total PAH using only slices of the data, generally from locations that contained the highest concentrations of oil, and comparing them against thresholds that have little, if any, support in the scientific literature. In contrast, I estimated a statistical probability of exposure and potential harm using all of the data. As explained in my initial report, by including samples that were specifically designed to find oil, my analysis is conservative and overstates potential harm.[5]

3. The Boesch/Rice bias in their Appendix A is evident in the bar charts in their Figure 5. In that Figure, they characterize data from only the top two meters of water and then end their time series in July 2010 so as not to show the dramatic decrease in PAHs after the wellhead was capped. Boesch and Rice then compare these samples against *total* PAH thresholds which, as discussed in my previous report, is not an appropriate means of

[5] August 15, 2014 Report of Dr. Shea at 15.

characterizing toxicity.[6] Even with this highly biased approach, the percentage of samples that exceeded 5 ppb of total PAH was only 12% in May, 3.5% in June and 2% in July.

4. Furthermore, I was unable to replicate the analysis conducted by Drs. Boesch and Rice. Appendix A to their joint report states that they obtained the data from "'BP Gulf Science Data (NRDA-publicly available),' file name 'WaterChemistry_W-01v02-01.csv' dated 5/23/2014." It further states that "[t]his resulted in 15,114 samples." However, when I download the exact same file, I find that there are 17,881 samples. One can obtain small differences in the sample count depending on how you treat replicate samples etc., but not a difference this large. It is therefore impossible for me to replicate the exact results reported by Drs. Boesch and Rice in Appendix A.

5. Finally, the Boesch/Rice joint report makes several accusations regarding my analyses of the water sampling data that are simply incorrect. They state with regard to the samples I considered that "[r]elatively few of the samples came from within a few meters of the surface and none of the samples came from intertidal zones and shallow water habitats."[7] In fact, as shown in the maps in Figures 1 and 2 of my initial report,[8] a substantial number of water samples were collected from nearshore intertidal zones and shallow water habitats, were analyzed for PAH concentrations, and were included in my summaries of the data. A substantial number of the PAH samples were taken from the top 10 meters of the water column [about 7250 or 40% of the samples] and were also included in my analysis and summaries of the data. As discussed in my initial report, my analysis and summaries also included samples collected at the locations where Drs. Boesch and Rice assert there was a "deep sea plume"[9] or at the seabed.[10] Their argument that the sampling did not incorporate oil from these areas is not true.

## IV. THE EPA WATER QUALITY BENCHMARKS INCLUDE SUBLETHAL AND CHRONIC TOXICITY ENDPOINTS

1. Drs. Rice and Boesch are critical of my approach, stating that my "[f]indings are derived from estimates of toxicity based on chemical measurements, not on direct measurement

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[6] Septemeber 12, 2014 Report of Dr. Shea at 4-5; 16-17.

[7] September 12, 2014 Report of Drs. Boesch and Rice at 23.

[8] August 15, 2014 Report of Dr. Shea at 13-14.

[9] August 15, 2014 Report of Dr. Shea at 26 (showing PAH sampling results by depth).

[10] August 15, 2014 Report of Dr. Shea at 37 (showing results of PAH sampling in the sediment).

of toxic effects,"[11] and that "[t]he actual toxicity derived from this methodology is underestimated because it is inferred from the measured concentrations..."[12]

2. Drs. Rice and Boesch fail to realize that I am following the most widely relied-upon standard practice, used by the EPA and others, to evaluate potential for harmful chemical exposures. For instance, the EPA states:

> Section 304(a)(1) of the Clean Water Act requires us to develop criteria for water quality that accurately reflects the latest scientific knowledge. These criteria are based solely on data and scientific judgments on pollutant concentrations and environmental or human health effects.[13]

3. In other words, the most common means of understanding harm from chemical exposure, and indeed the means that is required by regulation under the Clean Water Act, is to measure the concentration of the chemical and compare it against a benchmark (a water quality standard, criteria, guideline, etc.).

4. Benchmark comparisons that use reliable and validated methods are scientifically defensible. Here, the decades of research that went into the development of the EPA Water Quality Benchmark method meets these rigorous scientific requirements. The single paper published by Incardona a few months ago, and that provides the basis for the analysis of Drs. Rice and Boesch, does not meet these requirements.

5. Drs. Rice and Boesch are incorrect when they state that the EPA Water Quality Benchmark method considers only acute narcotic toxicity and does not consider chronic toxicity or other toxicity mechanisms.[14] While the EPA Water Quality Benchmark method was first developed with an extensive database on acute narcotic toxicity (usually based on 4 days of exposure), EPA has since developed a benchmark methodology that is

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[11] September 12, 2010 Joint Report of Drs. Boesch and Rice at 20.

[12] *Id.*

[13] http://water.epa.gov/scitech/swguidance/standards/criteria/index.cfm.

[14] September 12, 2014 Report of Drs. Boesch and Rice at p. 21 ("The primary flaw of using EPA benchmark methodology is that it is dependent only on narcotic response and does not consider other toxicity mechanisms."). *Id.* at p. 27 ("the EPA benchmark methodology relies primarily on acute toxicity narcosis as the toxicity mechanism, and dismisses many other toxicity mechanisms").

applied to chronic toxicity (usually based on 7-28 or even more days of exposure),[15] and others have concluded that this chronic benchmark methodology is protective of early life stage effects in fish embryos, including the very sensitive edema endpoints that Dr. Rice relies upon.[16] The EPA does not "dismiss" other toxicity mechanisms as Drs. Rice and Boesch claim, rather they have well-developed protocols and criteria for explicitly including toxicity data in their benchmarks

6. The EPA benchmark methodology with regard to chronic PAH toxicity was developed using 29 toxicity test results and is a well-accepted method employed by EPA for 30 years. [17] My initial report included an in-depth analysis of Gulf samples using this EPA chronic benchmark methodology.[18] As discussed by government scientists in the scientific literature,[19] and shown below in Figure 1, this method adequately protects against other chronic sublethal toxicity endpoints, including those for sensitive life stages of fish.

7. It is not possible to directly compare the EPA Water Quality Benchmark to the toxicity endpoints relied upon by Drs. Rice and Boesch (from Incardona) because Incardona incorrectly uses the gross measure of total PAH as the measure of exposure in his

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[15] McGrath, J.A. and Di Toro, D.M. 2009. Validation of the target lipid model for toxicity assessment of residual petroleum constituents: Monocyclic and polycyclic aromatic hydrocarbons. Environ. Toxicol. Chem. 28:1130–1148.

[16] Butler, J.D., Parkerton, T.F., Letinski, D.J., Bragin, G.E., Lampi, M.A., and Cooper, K.R. 2013. A novel passive dosing system for determining the toxicity of phenanthrene to early life stages of zebrafish. Sci. Total Environ. 463–464:952–958.

[17] U.S. EPA. 1985. Guidelines for Deriving Numerical National Water Quality Criteria for the Protection Of Aquatic Organisms and Their Uses. PB85- 227049. National Technical Information Service, Springfield, VA, 98 pp.. *See also*, U.S. EPA. 2010. Guidelines for Deriving Numerical National Water Quality Criteria for the Protection Of Aquatic Organisms and Their Uses. PB85-227049, 54 pp., and Burgess, R.M., Berry, W.J., Mount, D.R., and Di Toro, D.M. 2013. Mechanistic Sediment Quality Guidelines Based on Contaminant Bioavailability: Equilibrium Partitioning Sediment Benchmarks. Environ. Toxicol. Chem, 32:102-114.

[18] August 15, 2014 Report of Dr. Shea.

[19] Burgess, R.M., Berry, W.J., Mount, D.R., Di Toro, D.M. 2013. Mechanistic Sediment Quality Guidelines Based on Contaminant Bioavailability: Equilibrium Partitioning Sediment Benchmarks. Envrion. Toxicol. Chem, 32:102-114 at 106 ("PAHs demonstrate several modes of toxicity, including narcosis, carcinogenicity, mutagenicity, as well as photoenhanced toxicity. For benthic organisms and for some contaminants, the narcosis endpoint has been demonstrated to describe adequately observed sensitivity.").

experiments. As I described in detail in my previous report, and as discussed by others in the literature, the use of total PAH to characterize toxicity is incorrect.[20]

8. However, the scientific literature contains many examples of studies that measured both acute and chronic toxicity, including to the sensitive early life stages of fish (embryos and larvae) resulting from exposure to individual PAHs. In Figure 1, I have selected, as an example, studies that tested the toxicity of phenanthrene (a common 3-ring or tricyclic PAH in crude oil) because phenanthrene is a PAH that was present in MC252 oil and was identified by Incardona as a primary PAH that is most toxic to early life stages of fish.[21] The phenanthrene literature shows that the EPA Water Quality Benchmark is protective against chronic phenanthrene toxicities in fish embryos, including the very sensitive (but reversible) edema that is the focus of Dr. Rice's and Dr. Boesch's reports.[22] For example:

    a. Out of the 27 phenanthrene studies listed in Figure 1, only 2 found chronic toxicity thresholds below the EPA Benchmark value (19.13 ug/L).[23] One of these studies (1986 - Call-2) is not directly applicable to the *Deepwater Horizon*

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[20] September 12, 2014 Report of Dr. Shea at 4-5; 16-17.

[21] Incardona, J.P., Gardner, L.D., Linbo, T.L., Brown, T.L., Esbaugh, A.J., Mager, E.M., Stieglitz, J.D., French, B.L., Labenia, J.S., Laetz, C.A., Tagal, M., Sloan, C.A., Elizur, A., Benetti, D.D., Grosell, M., Block, B.A., and Scholz, N.L. 2014. Deepwater Horizon crude oil impacts the developing hearts of large predatory pelagic fish. PNAS 111(15):E1510-E1518.

[22] Butler, J.D., Parkerton, T.F., Letinski, D.J., Bragin, G.E., Lampi, M.A., Cooper, K.R. 2013. A novel passive dosing system for determining the toxicity of phenanthrene to early life stages of zebrafish. Sci. Total Environ. 463–464:952–958.

[23] US EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013, at p. 60. This EPA Benchmark value for phenanthrene should not be confused with the potency divisors that I used to calculate "Toxic Units" in my initial report. As described by EPA at http://epa.gov/bpspill/water-benchmarks.html, "Oil Related Organic Compounds are assessed jointly through a mixture approach because they all have the same type of effect on aquatic organisms. Potency divisors are not chemical-specific benchmarks, but are intermediates used in calculating the aggregate toxicity of the mixture. To assess the potential hazard to aquatic organisms, the sum of the calculated values is compared to a hazard index of 1. A value greater than 1 (>1) indicates that the sample has the potential to cause an acute or chronic effect on aquatic life like fish, crabs, and clams." Using the potency divisors for all the PAH and adding up their toxicities is the correct way to evaluate the toxicity of a PAH mixture. This is what I did in my initial report. EPA benchmarks for individual PAH are available, but are only useful for evaluating a single PAH at a time. I am using the EPA Benchmark for phenanthrene simply because phenanthrene is the example being used to illustrate how the EPA Benchmark method is protective of both acute and chronic toxicity.

incident because it involved exposure periods that were not likely to have occurred in the incident (90 days). The other study (2009 - EPA) is an unpublished 32-day study of toxicity to mysid shrimp with a toxicity threshold value at 5.5 ug/L. This species was extensively studied by both the United States and BP in laboratory toxicity tests discussed in my initial report[24] -- these tests indicated limited toxicity potential in Gulf of Mexico waters.

b. In addition to the 27 studies listed in Figure 1, in developing the EPA Benchmarks, the EPA considered an additional 30 studies that looked at the individual toxicity of phenanthrene.[25] Only one of these additional 30 studies found threshold values below the EPA phenanthrene Benchmark of 19.13 ug/L. This study tested toxicity to mysid shrimp, the same species discussed above that was extensively studied by both the US Government and BP in laboratory toxicity tests discussed in my initial report, and reported a threshold value of 17.7 ug/L. In other words, the EPA Benchmark value for phenanthrene is extremely conservative, even with regard to protecting against chronic toxicities.

c. A 2004 publication that looked at the toxicity of phenanthrene individually (rather than as part of a total PAH), found threshold toxicity values 260 times higher than the EPA Benchmark value. Interestingly, this 2004 study was authored by Dr. Incardona -- the same scientist relied upon by Drs. Rice and Boesch in their reports.

d. To put the EPA Benchmark value for phenanthrene (19.13 ug/L) in perspective, out of the nearly 18,000 water samples collected, **only 9** samples had concentrations of phenanthrene that exceeded the EPA Benchmark of 19.13 part per billion (ppb), 11 samples exceeded 10 ppb, 15 samples exceeded 5 ppb, and only 38 samples exceeded 1 ppb. While this comparison does not tell you enough to understand the potential toxicity in the samples because each sample is a mixture of PAHs, it does show that there were almost no instances of exposure to this particular PAH above levels of concern (for acute and chronic endpoints) despite the fact that this is one of the PAHs that the government scientists rely upon in their laboratory studies.

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[24] August 15, 2014 Report of Dr. Shea at 40-47.

[25] US EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013, Appendix C.

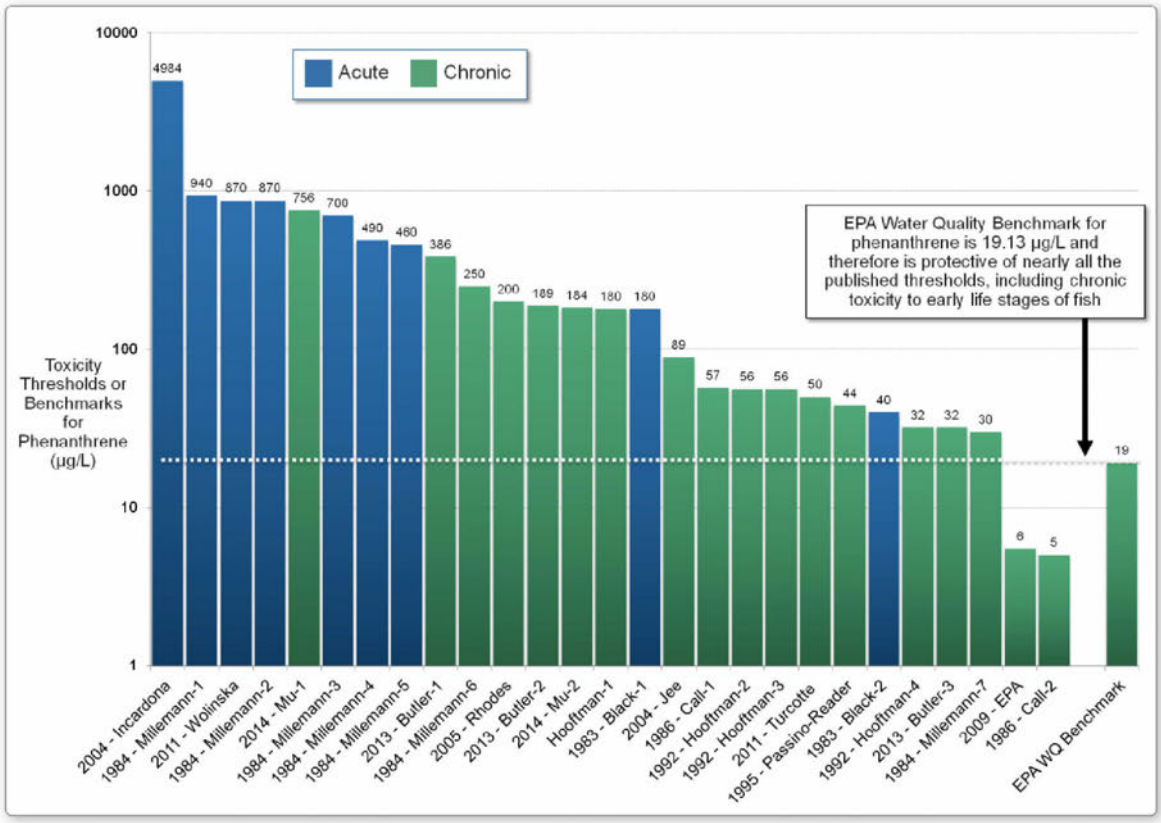


***Figure 1.*** *This chart summarizes 27 toxicity tests that looked at either the acute or chronic toxicity of phenanthrene. The EPA Water Quality Benchmark for phenanthrene is shown by the dotted line (19.13 ug/L). The chart shows that in all studies, other than two, the threshold at which toxic effects from phenanthrene were observed was higher than 19.13 ug/L (the EPA Benchmark). In other words, the EPA Benchmark is protective of both acute <u>and</u> chronic toxicities.*

# V. DRS. RICE AND BOESCH MISUNDERSTAND OR MISREPRESENT WHAT HAPPENS TO OIL WHEN IT WEATHERS AND OVERSTATE POTENTIAL HARM ASSOCIATED WITH WEATHERED OIL

1. Drs. Rice and Boesch do not correctly explain what happens to crude oil as it weathers and how this process affects toxicity.[26] It may be that Drs. Rice and Boesch have simply confused concentration (percentage) with quantity (amount) and/or have not adequately reviewed the PAH data from samples collected in the Gulf of Mexico. When saltwater is boiled, the *concentration* of salt increases as the water evaporates, but the *amount* of salt does not increase. Similarly, when oil weathers, the *concentration* of certain PAHs sometimes increases initially (as other oil components evaporate or dissipate more quickly) but the *amount* of those certain PAHs does not increase. And unlike in my analogy to saltwater where the salt itself does not degrade when boiled, these larger PAHs do degrade as the oil weathers, only at a slower rate compared to the smaller PAHs. This is explained in more detail below.

2. The amount of potentially harmful chemicals, including all BTEX and PAHs, decrease dramatically as oil weathers. Figure 2, below, shows the amount of total BTEX and PAH chemicals measured in MC252 oil when it is fresh compared to when it has weathered in the Gulf of Mexico. This decrease in the amount of BTEX and PAHs decreases the toxic potential of the oil – there are fewer potentially harmful chemicals in the environment.

3. When oil weathers, some larger PAHs decrease at a slower rate than the total mass of the oil, so the concentration of these larger PAHs can increase "per unit volume," but only during the *initial* stages of weathering. After two to three weeks in the Gulf of Mexico, both the amount and concentration of PAH decrease in the MC252 oil. This is shown in Figure 3, where the PAH concentrations in fresh MC252 oil are compared to weathered oil collected in the Gulf of Mexico. In the Gulf, both the total amount and the concentration of PAHs have decreased when the oil is weathered.

4. Even two co-authors of papers by Incardona (Drs. P.V. Hodson and T.K. Collier) have stated that "if weathering of spilled oil occurs without acute lethality to fish in the first 24

[26] September 12, 2014 Report of Drs. Boesch and Rice at 21-22.

hours, the overall impact of the oil will be greatly reduced."[27] In other words, the toxicity of oil decreases with weathering.

5. Furthermore, the residual PAHs in weathered oil are less bioavailable than in fresh oil. Physical and chemical changes to the oil make the PAHs less accessible to the water – the oil essentially "locks in" a large fraction of the PAHs and prevents them from escaping to the water. Dr. Rice admits this in his initial report stating "[f]or example, heavily weathered tar balls are nearly inert, with little exposure potential unless they have liquid oil centers that can release oil if punctured."[28]

6. On a related issue, Drs. Rice and Boesch misunderstand the behavior of PAHs in sediment, citing to a statement in the 2003 EPA benchmark document.[29] What EPA was referring to in this 2003 document was the potential for chemicals to not be in equilibrium with the sediment organic carbon when oil is present. However, it is well established that PAH exposure actually decreases in the presence of weathered oil.[30] This is a subject matter with which I am very familiar, having written the initial peer-reviewed publication in 1988 outlining the national sediment quality criteria[31] and having conducted research on this topic ever since.

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[27] Hodson, R.V., Collier, T.K., and Martin, J.D. 2011. Toxicity of Oil to Fish – Potential Effects of an Oil Spill into the Kitimat River from a Northern Gateway Pipeline Rupture. ENBRIDGE NORTHERN GATEWAY PROJECT. 113 pp.

[28] August 15, 2014 Report of Dr. Rice at 21.

[29] September 12, 2014 Report of Drs. Boesch and Rice at 21.

[30] Jonker, M.T.O., Sinke, A.J.C., Brils, J.M., and Koelmans, A.A. 2003. Sorption of polycyclic aromatic hydrocarbons to oil contaminated sediment: Unresolved complex? Environ. Sci. Technol. 37:5197–5203; Di Toro, D.M., McGrath, J.A., Stubblefield, W.A. 2007. Predicting the toxicity of neat and weathered crude oil: Toxic potential and the toxicity of saturated mixtures. Environ. Toxicol. Chem. 26:24–36.

[31] Shea, D. 1988. Developing National Sediment Quality Criteria. Environ. Sci. Technol. 22:1256-1261.

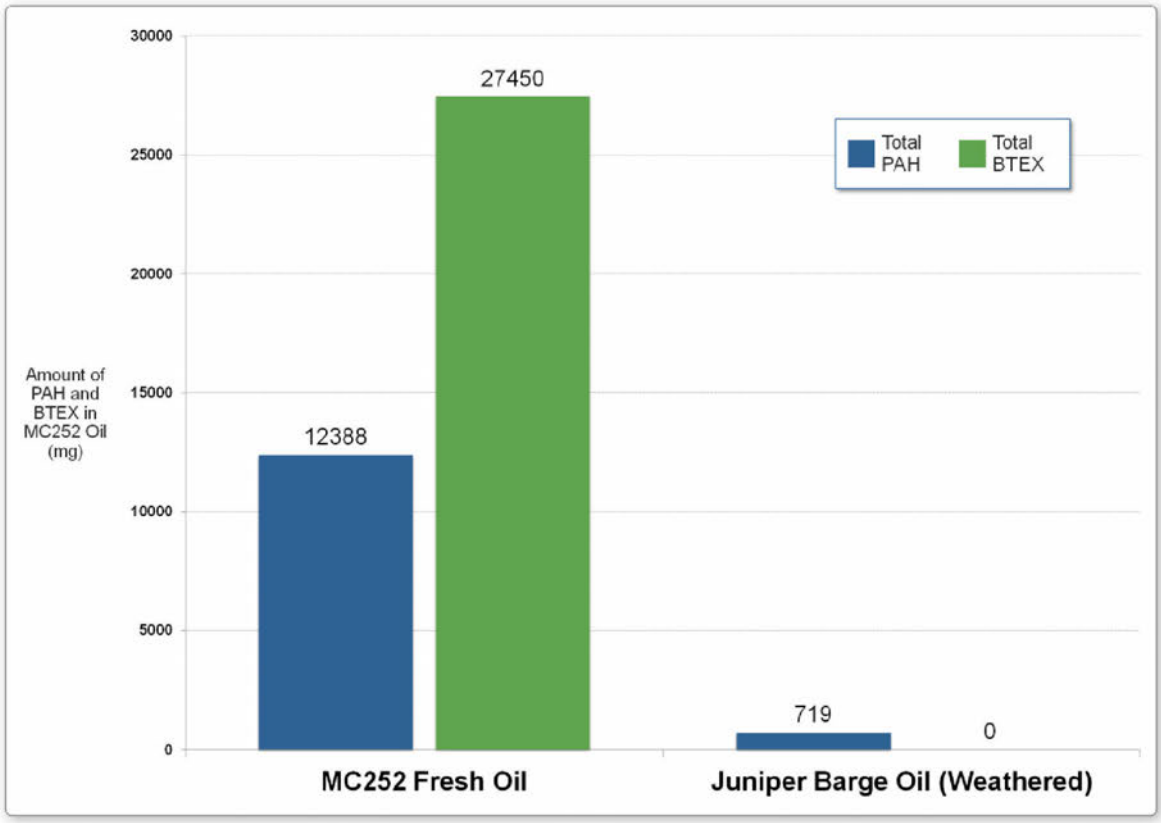


***Figure 2.*** *This graph shows the amount of total PAH (blue) and total BTEX (green) in fresh MC252 oil (on left) compared to what little remains after the oil has weathered in the Gulf of Mexico for 2-3 weeks. Weathering dramatically decreases the amount of potentially toxic chemicals. The Juniper Barge oil shown here is a weathered MC252 oil collected from the Gulf of Mexico that has been used extensively by the United States and BP to understand toxicity of the oil. Data are from file "OilChemistry O-04v01-03.xls" found at http://gulfsciencedata.bp.com. This chart does not speak to the actual toxicity of either fresh or weathered oil, it simply compares the amount of PAHs and BTEX present in each.*

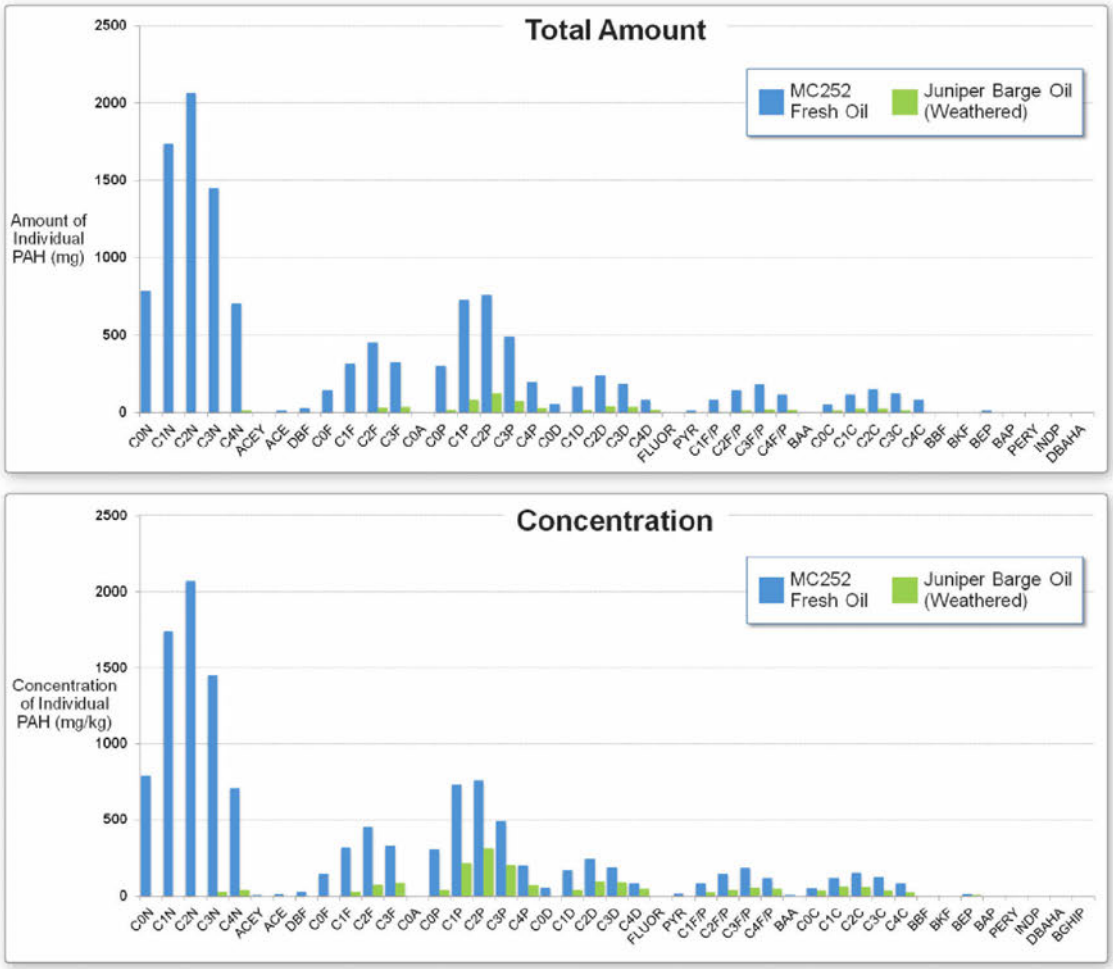


***Figure 3.*** *These graphs show the total amount (top graph) and the concentration (bottom graph) of individual PAH chemicals in one kilogram of fresh MC252 oil (blue) that was then weathered in the Gulf and collected on the Juniper Barge (green). The size of the PAH generally increases as you go from left to right in the graph. On the left, one can see that there is a dramatic decrease in these smaller PAH as the oil weathers. Starting in the middle of the graph, one can see that this decrease is not as rapid, but both the total amount (top graph) and the concentration per unit volume (bottom graph) decrease with weathering. The "concentration" does not decrease quite as fast as the total amount simply because other oil components are decreasing more quickly than these larger PAHs so these PAHs do not change as much "per unit volume" of oil. This is what Drs. Rice and Boesch refer to when they say the larger PAH "increase," but clearly these PAHs are actually decreasing. Data are from file "OilChemistry O-04v01-03.xls" found at http://gulfsciencedata.bp.com. These charts do not speak to the actual toxicity of either fresh or weathered oil, they simply compare the amount of PAHs and BTEX present in each.*

# VI. THE STUDIES RELIED UPON BY THE UNITED STATES' EXPERTS DO NOT CHANGE MY ANALYSIS

Drs. Rice and Boesch reference some recent papers in the scientific literature to support their claim of greater harm to the environment and are critical of my decision to not rely on those papers.[32] While all of these papers are peer-reviewed, that fact does not in and of itself mean the papers are sufficiently reliable or relevant such that they should inform our understanding of impacts. Rather, every publication must be reviewed critically. And, indeed, with regard to every publication Drs. Boesch and Rice list as studies that should have been included in my analysis, there are important reasons why I did not rely on the given study.

1. Regarding the papers by Incardona[33] and Mager,[34] I addressed the shortcomings of these papers in my previous reports, including: (1) the inappropriate use of a high energy water accommodated fraction (HEWAF) for the oil exposures; (2) the use of total PAH as their measure of exposure; and (3) that most of the endpoints were not quantitative and/or not actual physiological or developmental effects.[35]

2. The paper by Dubansky[36] received formal criticism in the journal where it was published[37] for many reasons, including inadequate study design, no calculation of

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[32] September 12, 2014 Report of Drs. Boesch and Rice at 26. ("including the experimental studies demonstrating not only the toxic effects of PAH at lower concentrations in fish embryos, larvae and juveniles, but also: bioaccumulation of PAHs and other hydrocarbons in plankton; mortality experienced by animals living in association with floating *Sargassum*; deposition of oily marine snow on sediment and coral habitats and PAH metabolites and lesions in bottom-dwelling fish") (internal references omitted).

[33] Incardona, J.P., Gardner, L.D., Linbo, T.L., Brown, T.L., Esbaugh, A.J., Mager, E.M., Stieglitz, J.D., French, B.L., Labenia, J.S., Laetz, C.A., Tagal, M., Sloan, C.A., Elizur, A., Benetti, D.D., Grosell, M., Block, B.A., and Scholz, N.L. 2014. Deepwater Horizon crude oil impacts the developing hearts of large predatory pelagic fish. PNAS USA 111:E1510-E1518.

[34] Mager, E.M., Esbaugh, A.J., Stieglitz, J.D., Hoenig, R., Bodinier, C., Incardona, J.P., Scholz, N.L., Benetti, D.D., and Grosell, M. 2014. Acute embryonic or juvenile exposure to Deepwater Horizon Crude oil impairs the swimming performance of Mahi-Mahi (*Coryphaena hippurus*). Environ. Sci. Technol. 48:7053-7061.

[35] September 12, 2014 Report of Dr. Damian Shea at 10-18.

[36] Dubansky, B., Whitehead, A., Miller, J.T., Rice, C.D., and Galvez, F. 2013. Multitissue Molecular, Genomic, and Developmental Effects of the Deepwater Horizon Oil Spill on Resident Gulf Killifish (Fundulus grandis). Environ. Sci. Technol. 47:5074-5082.

[37] Pearson, W. H. Comment on "Multitissue Molecular, Genomic, and Developmental Effects of the Deepwater Horizon Oil Spill on Resident Gulf Killifish (Fundulus grandis)." Environ. Sci. Technol. 2014, 48:7677-7678.

thresholds, inappropriate extrapolation of molecular/genomic "biomarkers" to population level effects, and failure to consider compensatory mechanisms that make fish populations resilient. In Dubansky's response, he conceded "[w]hether these impacts ultimately emerge as population-level effects…remains to be seen."[38]

3. As I discussed in my previous report,[39] the paper by Montagna[40] cannot be used to infer large or long-lasting impact in the seafloor due to the *Deepwater Horizon* incident because there is no chemical analysis matching or fingerprinting the MC252 oil to the degraded areas. Furthermore, even the impacts discussed in the Montagna paper are extremely limited.

4. The paper by Mitra[41] was intended to prove the hypothesis that the relative PAH distributions in zooplankton matched that of MC252 oil. However, the data had an insufficient number of individual PAHs to adequately "fingerprint" the PAH to test this hypothesis and in fact, Mitra never formally tested the hypothesis and concluded only that the "[s]pill may have contributed to contamination in the northern Gulf of Mexico ecosystem." In addition, the zooplankton were collected by towing a net for up to 60 minutes through the water and there is no way to distinguish PAH associated with the zooplankton from PAH associated with other material collected with the net.

5. The paper by Powers[42] presents research results on novel pathways for potential effects on floating mats of algae. There is no question that surface oil came into contact with floating algae, but Powers *et al.* states that their results only illustrate the

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[38] Dubansky, B., Whitehead, A., Miller, J.T., Rice, C.D., and Galvez, F. 2013. Multitissue molecular, genomic, and developmental effects of the Deepwater Horizon oil spill on resident Gulf killifish (*Fundulus grandis*). Environ. Sci. Technol. 47:5074-5082.

[39] September 12, 2014 Report of Dr. Shea at 27.

[40] Montagna, P.A., Baguley, J.G., Cooksey, C., Hartwell, I., Hyde, L.J., Hyland, J.L., Kalke, R.D., Kracker, L.M., Reuscher, M., Rhodes, A.C. 2013. Deep-sea benthic footprint of the deepwater horizon blowout. PLoS One 8:e70540; Fisher, C.R., Demopoulos, A.W.J., Cordes, E.E., Baums, I.B., White, H.K., and Bourque, J.R. 2014. Coral Communities as Indicators of Ecosystem-Level Impacts of the Deepwater Horizon Spill. BioScience 64:796-807.

[41] Mitra, S., Kimmel, D.G., Snyder, J., Scalise, K., McGlaughon, B.D., Roman, M.R., Jahn, G.L., Pierson, J.J., Brandt, S.B., Montoya, J.P., Rosenbauer, R.J., Lorenson, T.D., Wong, F.L., and Campbell, P.L. 2012. Macondo-1 well oil-derived polycyclic aromatic hydrocarbons in mesozooplankton from the northern Gulf of Mexico. Geophys. Res. Lett. 39:L01605.

[42] Powers, S.P., Hernandez, F.J., Condon, R.H., Drymon, J.M., and Free, C.M. 2013. Novel pathways for injury from offshore oil spills: direct, sublethal and indirect effects of the Deepwater Horizon oil spill on pelagic Sargassum communities. PLoS One 8:e74802.

potential for effects, provides no means to quantify any effects, and clearly acknowledges subsequent recovery of the algae. Thus, I do not question the potential for short-term effects on the algae that contacted the oil, but these would be the same locations where water samples were collected and analyzed. My analysis used the EPA Water Quality Benchmark method which is based on aquatic species more sensitive than algae.

6. The paper by Fisher[43] summarizes new data on deep-sea coral communities. The evidence for acute impacts is very limited, confounded in most cases by a lack of fingerprinting suspected oil as MC252 oil, and restricted to a far smaller area than I already showed could have been harmed in the deep sea in my initial report.[44] The authors also do not adequately consider injuries that could have been caused by natural oil seeps or baseline injuries that existed prior to the oil spill. Their suggestion of likely "invisible impacts" in other areas is highly speculative and not based on any actual data.

7. The paper by Murawski[45] looks at the prevalence of skin lesions in fish and tries to test the hypothesis that lesions were caused by PAHs originating from the MC252 oil. I reviewed their raw PAH data in detail and find that their conclusion the MC252 oil was the source of PAHs in fish tissue scientifically indefensible.[46] This invalidates any correlations with PAHs shown and alone makes this study minimally relevant. Furthermore, the PAH exposures were far below the levels known to cause lesions and PAH are more likely to cause liver lesions which were not observed,[47] the study

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[43] Fisher, C.R., Demopoulos, A.W.J., Cordes, E.E., Baums, I.B., White, H.K., and Bourque, J.R. 2014. Coral Communities as Indicators of Ecosystem-Level Impacts of the Deepwater Horizon Spill. BioScience 64:796-807.

[44] August 15, 2014 Report of Dr. Shea at 34.

[45] Murawski, S.A., Hogarth, W.T., Peebles, E.B., and Barbeiri, L. 2014. Prevalence of external skin lesions and polycyclic aromatic hydrocarbon concentrations in Gulf of Mexico fishes, post-Deepwater Horizon. Trans. Am. Fish. Soc. 143:1084-1097.

[46] The method blanks contained concentrations of naphthalenes at the same or at even higher levels than they measured in many of the actual fish samples. They also found concentrations of the PAH fluoranthene, which is nearly absent in MC252 oil, at high levels relative to most other PAH. The authors also do not consider how the PAH patterns are altered due to the well-known metabolism of PAHs by fish.

[47] *See* U.S. EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013, at Sec. 6.6; Collier, T.K., Anulacion, B.F., Arkoosh, M.R., Dietrich, J.P., Incardona, J.P., Johnson, L.L., Ylitalo, G.M., and Myers, M.S. 2014. Effects on fish of polycyclic aromatic hydrocarbon (PAH) and naphthenic acid

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did not follow US government scientists' recommendations on establishing a cause-effect relationship between PAH exposure and fish lesions,[48] and those data were not available to review. Finally, they were unable to prove that the lesions were not caused by something other than PAHs.

8. In addition to the papers listed above, there are other papers cited in the reports of Drs. Boesch and Rice that I did not specifically include in my analyses for various reasons. For example, Dr. Boesch cites a paper by Ortmann *et al.*[49] for the proposition that "[e]xperiments conducted in Alabama, however, indicated that, while chemically dispersed oil was assimilated by bacteria, dispersant-oil mixtures suppressed populations of planktonic protozoans, known as ciliates, that consume the bacteria."[50] That paper has been specifically criticized by two of the US' own witnesses on the issue of dispersants, both EPA scientists. Dr. Mace Barron determined that "the environmental relevance of the results were highly uncertain."[51] Dr. Robyn Conmy also had several criticisms of the Ortmann paper and stated that "even if these effects could represent the real world spill, the volume of the gulf impacted with high concentrations of dispersant is small compared to the unimpacted volume." [52]

In summary, while these papers cited by Drs. Rice and Boesch are peer-reviewed, they are not necessarily relevant or reliable. Drs. Rice and Boesch apparently performed no critical or independent review of these papers, accepting the opinions of the authors simply because the publications were "peer-reviewed." My analysis goes much further – I independently reviewed

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Footnote continued from previous page

exposures. In: Organic Chemical Toxicity of Fishes. Tierney, K.B. (ed). pp. 195-255. *See also*, Keke, I.R. 1989. Fin Erosion in Phractolaemus ansorgii (Boulenger) exposed to crude oil-in-water dispersions. Journal of Aquatic Sciences. 4:55-58 (discussing fin erosion).

[48] Myers, M.S., Johnson, L.L., Collier, T.K. 2003. Establishing the causal relationship between polycyclic aromatic hydrocarbon (PAH) exposure and hepatic neoplasms and neoplasia-related liver lesions in English sole (Pleuronectes vetulus). Human and Ecological Risk Assessment, 9:67-94.

[49] Ortmann, A.C., Anders, J., Shelton, N., Gong, L., Moss, A.G., and Condon, R.H. 2012. Dispersed oil disrupts microbial pathways in pelagic food webs. PLoS One 7:e42548, Deposition Ex. 12058.

[50] August 15, 2014 Report of Dr. Boesch at 22.

[51] Deposition of Dr. Mace Barron at 221-230; ORD Critical Review of Ortmann et al. (2012) Dispersed Oil Disrupts Microbial Pathways in Pelagic Food Webs. PLoS ONE 7:e42548, US_PP_EPA-7027, admitted in the Deposition of Dr. Mace Barron as Dep. Ex. 12057.

[52] Email from Robyn Conmy to Albert Venosa, Aug. 6, 2012, US_PP_EPA094257, admitted as Dep. Ex. 12074.

each of these papers and, when it was available, I analyzed the underlying raw data myself to confirm or disprove the theories set forth by the authors. Based upon my careful review of both these studies as well as the studies cited in my prior reports, my underlying opinion remains the same: the potential for environmental harm from the *Deepwater Horizon* incident was very limited in space and time.

# VII. APPENDIX A. REFERENCES USED TO OBTAIN THE PHENANTHRENE AQUATIC TOXICITY DATA FOR FIGURE 1.

McGrath, J.A. and Di Toro, D.M. 2009. Validation of the target lipid model for toxicity assessment of residual petroleum constituents: Monocyclic and polycyclic aromatic hydrocarbons. Environ. Toxicol. Chem. 28:1130–1148.

Butler, J.D., Parkerton, T.F., Letinski, D.J., Bragin, G.E., Lampi, M.A., and Cooper, K.R. 2013. A novel passive dosing system for determining the toxicity of phenanthrene to early life stages of zebrafish. Sci. Total Environ. 463–464:952–958.

Jee, H-J, Kim, S-G, and Kang, J-C. 2004. Effects of phenanthreneon growth and basic physiological functions of the olive flounder, Paralichthys olivaceus. J. Exp. Mar. Biol. Ecol. 304:123–36.

*Hooftman, R.N., Evers-de Ruiter, A. 1992. Investigations into the aquatic toxicity of phenanthrene (cover-report for reproduction tests with the waterflea Daphnia magna and an Early Life Stage (ELS) test with the zebra fish Brachydanio rerio). TNO-report R 92/290. Delft, the Netherlands: TNO Environmental and Energy Research, TNO Institute of Environmental Sciences.

Passino-Reader, D.R., Berlin, W.H., and Hickey, J.P. 1995. Chronic bioassays of rainbow trout fry with compounds representative of contaminants in great lakes fish. J. Great Lakes Res.21:373–83.

Rhodes, S.M., Farwell, A., Hewitt, L.M., MacKinnon, M.D., and Dixon, D.G. 2005. The effects of dimethylated and alkylated polycyclic aromatic hydrocarbons on the embryonic development of the Japanese medaka. Ecotoxicol. Environ. Saf. 60(3):247–58.

Turcotte, D., Akhtar, P., Bowerman, M., Kiparissis, Y., Brown, R., and Hodson, P.V. 2011. Measuring the toxicity of alkyl-phenanthrenes to early life stages of medaka (Oryzias latipes) using partition-controlled delivery. Environ. Toxicol. Chem. 30(2):487–95.

Wolińska, L., Brzuzan, P., Woźny, M., Góra, M., Łuczyński, M.K., Podlasz, P., et al. 2011. Preliminary study on adverse effects of phenanthrene and its methyl and phenyl derivatives in larval zebrafish, Danio rerio. Environ. Biotechnol. 7(1):26–33.

Millemann, R.E., Birge, W.J., Black, J.A., Cushman, R.M., Daniels, K.L., Franco, P.J., Giddings, J.M., McCarthy, J.F. and Steward, A.J. 1984. Comparative Acute Toxicity to Aquatic

Organisms of Components of Coal-Derived Synthetic Fuels. Trans. Am. Fish. Soc. 113(1):74-85.

*Hooftman, R.N. and Evers-de Ruiter, A. 1992. Early life stage tests with Brachydanio rerio and several polycyclic aromatic hydrocarbons using an intermittent flow-through system. TNO Report IMW-R 92/210. Institute for Inland Water Management and Waste Water Treatment (RIZA) and Ministry of Housing, The Netherlands.

*Call, D.J., Brooke, L.T., Harting, S.L., Poirer, S.H., McCauley, D.J. 1986. Toxicity of phenanthrene to several freshwater species. Final Report. U.S. Environmental Protection Agency, Washington, DC.

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

Black, J.A., Birge, W.J., Westerman, A.G., and Francis, P.C. 1983. Comparative aquatic toxicology of aromatic hydrocarbons. Fundam. Appl. Toxicol. 3:353–358.

Mu, J., Wang, J., Jin, F., Wang, X., and Hong, H. 2014. Comparative embryotoxicity of phenanthrene and alkyl-phenanthrene to marine medaka (Oryzias melastigma). Mar. Poll. Bull. 85:505–515.

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*I relied upon the data from these studies as reported in McGrath, J.A. and Di Toro, D.M. 2009. Validation of the target lipid model for toxicity assessment of residual petroleum constituents: Monocyclic and polycyclic aromatic hydrocarbons. Environ. Toxicol. Chem. 28:1130–1148.

# VIII. APPENDIX B. CONSIDERATION MATERIALS

| Doc Date | Document Title / Description |
|---|---|
| 1983-00-00 | Black, J.A., Birge, W.J., Westerman, A.G., and Francis, P.C. 1983. Comparative aquatic toxicology of aromatic hydrocarbons. Fundam. Appl. Toxicol. 3:353–358. |
| 2013-00-00 | Burgess, R.M., Berry, W.J., Mount, D.R., and Di Toro, D.M. 2013. Mechanistic Sediment Quality Guidelines Based on Contaminant Bioavailability: Equilibrium Partitioning Sediment Benchmarks. Envrion. Toxicol. Chem, 32:102-114. |
| 2013-10-00 | Butler, J.D., Parkerton, T.F., Letinski, D.J., Bragin, G.E., Lampi, M.A., Cooper, K.R. 2013. A novel passive dosing system for determining the toxicity of phenanthrene to early life stages of zebrafish. Sci. Total Environ. 463–464:952–958. |
| 1986-00-00 | Call, D.J., Brooke, L.T., Harting, S.L., Poirer, S.H., McCauley, D.J. 1986. Toxicity of phenanthrene to several freshwater species. Final Report. U.S. Environmental Protection Agency, Washington, DC. |
| 2014-00-00 | Collier, T.K., Anulacion, B.F., Arkoosh, M.R., Dietrich, J.P., Incardona, J.P., Johnson, L.L., Ylitalo, G.M., and Myers, M.S. 2014. Effects on fish of polycyclic aromatic hydrocarbon (PAH) and naphthenic acid exposures. In: Organic Chemical Toxicity of Fishes. Tierney, K.B. (ed). pp. 195-255. |
| 2014-06-24 | Deposition of Mace Barron. |
| 2007-01-00 | Di Toro, D.M., McGrath, J.A., Stubblefield, W.A. 2007. Predicting the toxicity of neat and weathered crude oil: Toxic potential and the toxicity of saturated mixtures. Environ. Toxicol. Chem. 26(1):24–36. |
| 2013-04-22 | Dubansky, B., Whitehead, A., Miller, J.T., Rice, C.D., and Galvez, F. 2013. Multitissue Molecular, Genomic, and Developmental Effects of the Deepwater Horizon Oil Spill on Resident Gulf Killifish (*Fundulus grandis*). Environ. Sci. Technol. 47:5074-5082. |
| 2014-06-19 | Dubansky, B., Whitehead, A., Miller, J.T., Rice, C.D., and Galvez, F. 2014. Response to Comment on "Multi-tissue Molecular, Genomic, and Developmental Effects of the Deepwater Horizon Oil Spill on Resident Gulf Killifish (*Fundulus grandis*)." Environ. Sci. Technol. 48:7679-7680. |
| 2012-08-06 | Email from R. Conmy (EPA) to A. Venosa re Assessment of Ortmann et al 2012 paper, admitted as Ex. 12074 in the Deposition of Robyn Conmy. |
| 2014-08-15 | Expert Report of Donald F. Boesch. |
| 2014-08-15 | Expert Report of Stanley Rice. |
| 2014-08-15 | Expert Report of Damian Shea. |

| Doc Date | Document Title / Description |
|---|---|
| 2014-09-12 | Rebuttal Expert Report of Donald F. Boesch and Stanley Rice. |
| 2014-09-12 | Rebuttal Expert Report of Damian Shea. |
| N/A | File OilChemistry_O-04v01-03.xls *available at* http://gulfsciencedata.bp.com. |
| 2014-09-00 | Fisher, C.R., Demopoulos, A.W.J., Cordes, E.E., Baums, I.B., White, H.K., Bourque, J.R. 2014. Coral Communities as Indicators of Ecosystem-Level Impacts of the Deepwater Horizon Spill. BioScience 64:796-807. |
| 2011-12-19 | Hodson, R.V., Collier, T.K., Martin, J.D. 2011. Toxicity of Oil to Fish – Potential Effects of an Oil Spill into the Kitimat River from a Northern Gateway Pipeline Rupture. ENBRIDGE NORTHERN GATEWAY PROJECT. 113 pp. |
| 2004-00-00 | Incardona, J.P., Collier, T.K., and Scholz, N.L. 2004. Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. Toxicol. Appl. Pharmacol. 196:191–205. |
| 2014-03-24 | Incardona, J.P., Gardner, L.D., Linbo, T.L., Brown, T.L., Esbaugh, A.J., Mager, E.M., Stieglitz, J.D., French, B.L., Labenia, J.S., Laetz, C.A., Tagal, M., Sloan, C.A., Elizur, A., Benetti, D.D., Grosell, M., Block, B.A., and Scholz, N.L. 2014. Deepwater Horizon crude oil impacts the developing hearts of large predatory pelagic fish. PNAS 111(15):E1510-E1518, doi: 10.1073/pnas.1320950111. |
| 2004-06-16 | Jee, H-J, Kim, S-G, and Kang, J-C. 2004. Effects of phenanthrene on growth and basic physiological functions of the olive flounder, *Paralichthys olivaceus*. J. Exp. Mar. Biol. Ecol. 304:123–36. |
| 2003-00-00 | Jonker, M.T.O., Sinke, A.J.C., Brils, J.M., and Koelmans, A.A. 2003. Sorption of polycyclic aromatic hydrocarbons to oil contaminated sediment: Unresolved complex? Environ. Sci. Technol. 37:5197–5203. |
| 1989-00-00 | Keke, I.R. 1989. Phractolaemus ansorgii (Boulenger) exposed to crude oil-in-water dispersions. Journal of Aquatic Sciences 4:55-58 (discussing fin erosion). |
| 2014-05-23 | Mager, E.M., Esbaugh, A.J., Stieglitz, J.D., Hoenig, R., Bodinier, C., Incardona, J.P., Scholz, N.L., Benetti, D.D., and Grosell, M. 2014. Acute Embryonic or Juvenile Exposure to Deepwater Horizon Crude Oil Impairs the Swimming Performance of Mahi-Mahi (*Coryphaena hippurus*). Environ. Sci. & Tech. 48(12):7053-7061. |
| 2009-00-00 | McGrath, J. and Di Toro, D.M. 2009. Validation of the Target Lipid Model for Toxicity Assessment of Residual Petroleum Constituents: Monocyclic and Polycyclic Aromatic Hydrocarbons. Environ. Tox. Chem. 28(6):1130-1148. |
| 1984-00-00 | Millemann, R.E., Birge, W.J., Black, J.A., Cushman, R.M., Daniels, K.L., Franco, P.J., Giddings, J.M., McCarthy, J.F. and Steward, A.J. 1984. Comparative Acute Toxicity to Aquatic Organisms of Components of Coal-Derived Synthetic Fuels. Trans. Am. Fish. Soc. 113(1):74-85. |

| Doc Date | Document Title / Description |
|---|---|
| 2012-01-14 | Mitra, S., Kimmel, D.G., Snyder, J., Scalise, K., McGlaughon, B.D., Roman, M.R., Jahn, G.L., Pierson, J.J., Brandt, S.B., Montoya, J.P., Rosenbauer, R.J., Lorenson, T.D., Wong, F.L., and Campbell, P.L. 2012. Macondo-1 well oil-derived polycyclic aromatic hydrocarbons in mesozooplankton from the northern Gulf of Mexico. Geophys. Res. Lett. 39:L01605. |
| 2013-08-07 | Montagna, P.A., Baguley, J.G., Cooksey, C., Hartwell, I., Hyde, L.J., Hyland, J.L., Kalke, R.D., Kracker, L.M., Reuscher, M., and Rhodes, A.C.E. 2013. Deep-Sea Benthic Footprint of the Deepwater Horizon Blowout. PLoS ONE 8(8):e70540. |
| 2014-02-18 | Mu, J., Wang, J., Jin, F., Wang, X., and Hong, H. 2014. Comparative embryotoxicity of phenanthrene and alkyl-phenanthrene to marine medaka (*Oryzias melastigma*). Mar. Poll. Bull. 85:505–515. |
| 2014-07-02 | Murawski, S.A., Hogarth, W.T., Peebles, E.B., Barbeiri, L. 2014. Prevalence of external skin lesions and polycyclic aromatic hydrocarbon concentrations in Gulf of Mexico fishes, post-Deepwater Horizon. Trans. Am. Fish Soc. 143:1084-1097. |
| 2003-01-00 | Myers, M.S., Johnson, L.L., and Collier, T.K. 2003. Establishing the causal relationship between polycyclic aromatic hydrocarbon (PAH) exposure and hepatic neoplasms and neoplasia-related liver lesions in English sole (*Pleuronectes vetulus*). Human and Ecological Risk Assessment, 9:67-94. |
| 2014-00-00 | Nelson, J.R., Bauer, J.R., and Rose, K. 2014. Assessment of Geographic Setting on Oil Spill Impact Severity in the United States – Insights from Two Key Spill Events in Support of Risk Assessment for Science-Based Decision Making. J. Sustainable Energy Eng. (accepted Sept. 5, 2014), DOI: 10.7569/JSEE.2014.629510. |
| 2012-00-00 | ORD Critical Review of Ortmann et al. (2012) Dispersed Oil Disrupts Microbial Pathways in Pelagic Food Webs. PloS ONE 7:e42548. US_PP_EPA070277, admitted as Ex. 12057 in the Deposition of Dr. Mace Barron. |
| 2012-07-00 | Ortmann, A.C., Anders, J., Shelton, N., Gong, L., Moss, A.G., and Condon, R.H. 2012. Dispersed Oil Disrupts Microbial Pathways in Pelagic Food Webs. PLoS One 7:e42548, admitted as Ex. 12058 in the Deposition of Mace Barron. |
| 1995-00-00 | Passino-Reader, D.R., Berlin, W.H., and Hickey, J.P. 1995. Chronic Bioassays of Rainbow Trout Fry with Compounds Representative of Contaminants in Great Lakes Fish. J. Great Lakes Res. 21:373–383. |
| 2014-06-19 | Pearson, W.H. 2014. Comment on "Multitissue Molecular, Genomic, and Developmental Effects of the Deepwater Horizon Oil Spill on Resident Gulf Killifish (*Fundulus grandis*)." Environ. Sci. Technol. 48:7677-7678. |
| 2013-09-00 | Powers, S.P., Hernandez, F.J., Condon, R.H., Drymon, J.M., Free, C.M. 2013. Novel Pathways for Injury from Offshore Oil Spills: Direct, Sublethal and Indirect Effects of the *Deepwater Horizon* Oil Spill on Pelagic *Sargassum* Communities. PLoS One 8(9):e74802. |

| Doc Date | Document Title / Description |
|---|---|
| 2005-03-00 | Rhodes, S.M., Farwell, A., Hewitt, L.M., MacKinnon, M.D., and Dixon, D.G. 2005. The effects of dimethylated and alkylated polycyclic aromatic hydrocarbons on the embryonic development of the Japanese medaka. Ecotoxicol. Environ. Saf. 60(3):247–258. |
| 1988-00-00 | Shea, D. 1988. Developing national sediment quality criteria. Environ. Sci. Technol. 22(11):1256-1261. |
| 2011-02-00 | Turcotte, D., Akhtar, P., Bowerman, M., Kiparissis, Y., Brown, R., and Hodson, P.V. 2011. Measuring the toxicity of alkyl-phenanthrenes to early life stages of medaka (*Oryzias latipes*) using partition-controlled delivery. Environ. Toxicol. Chem. 30(2):487–95. |
| 1985-00-00 | U.S. EPA. 1985. Guidelines for Deriving Numerical National Water Quality Criteria for the Protection Of Aquatic Organisms and Their Uses. PB85- 227049. National Technical Information Service, Springfield, VA, 98 pp. |
| 2003-11-00 | U.S. EPA. 2003. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: PAH mixtures. EPA-600-R-02-013, *available at* http://www.epa.gov/nheerl/download_files/publications/PAHESB.pdf. |
| 2010-12-00 | U.S. EPA. 2010. Guidelines for Deriving Numerical National Water Quality Criteria for the Protection Of Aquatic Organisms and Their Uses. PB85-227049, 54 pp. |
| N/A | U.S. EPA. Water Quality Criteria, *available at* http://water.epa.gov/scitech/swguidance/standards/criteria/index.cfm. |
| N/A | U.S. EPA. Water Quality Benchmarks for Aquatic Life, *available at* http://epa.gov/bpspill/water-benchmarks.html. |
| 2011-00-00 | Wolińska, L., Brzuzan, P., Woźny, M., Góra, M., Łuczyński, M.K., Podlasz, P., et al. 2011. Preliminary study on adverse effects of phenanthrene and its methyl and phenyl derivatives in larval zebrafish, *Danio rerio*. Environ. Biotechnol. 7(1):26–33. |