Hearing Order OH-001-2014
Trans Mountain Pipeline ULC (Trans Mountain)
Application for the Trans Mountain Expansion Project (Project)

Written evidence of Dr. Riki Ott

Name of intervenor: **North Shore No Pipeline Expansion ("NSNOPE")**

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1.0 Introduction

1.1 Scope of Work: Supplement with NEW Evidence

1. This is the written evidence of Dr. Riki Ott filed on behalf of the intervener North Shore No Pipeline Expansion (NSNOPE) to address human health impacts and risks of oil spills.

2. In my May 2015 testimony, I established that oil spills and the Corexit dispersants preferentially used in the United States during oil spill response contain dangerous chemicals. These commonly used dispersants are Corexit 9500A, 9527A, and to a more limited extent, 9580A. I also discussed acute effects to human health and ecosystem impacts of the BP Deepwater Horizon Oil Spill (DHOS) disaster and to "dilbit" spills of blended Alberta tar sands and diluent: the July 2010 the Enbridge Lakehead Pipeline 6B and the April 2013 the ExxonMobil's Pegasus Pipeline.

3. This supplement focuses on NEW evidence, primarily from the BP Deepwater Horizon Oil Spill (DHOS) disaster, that people – both response workers and the general public – were exposed to dangerous levels of dangerous chemicals and the resulting long-term health impacts from these exposures. NEW collaborating evidence is presented from studies on dolphins and other wildlife from the BP DHOS disaster, studies on dangerous levels of PAHs from the dilbit disasters (although long-term follow up studies on human health were not conducted for the two spills discussed previously), and impact studies on freshwater ecosystems from diluted bitumen.

4. This supplement also includes a NEW subsection to specifically address oil spill disaster preparedness to minimize health risks and exposures to response workers, the general public, and ecosystems, based on lessons learned from the BP DHOS and other disasters.

5. Finally, an updated curriculum vita supplement is included that highlights my new and current activities, relating to my expertise and experiences on these issues.

1.2 Statement of Qualifications: Updates for This Supplement

1.2.1 Education

6. One NEW addition in curriculum vita supplement.

1.2.2 UPDATES to Experience

7. I updated my experiences to include work with First Nations in Canada and a current focus, since 2015, on oil spill response regimes, rulemakings on dispersant or spill-treating agents, and opportunities for citizen engagement in the United States and Canada.

1.2.3 AFFIRMED: Qualification of Opinions

8. As before, my opinions are based on my education, professional experience, and information and data available in the scientific literature, court records, government reports, investigative and other media, graduate student research and studies, my own research and other writings, and my personal experience. My UPDATED curriculum vita supplement is attached as Appendix A.

2.0 Impacts to Worker Health & Wildlife from Oil Spills

2.1 State of Knowledge before the BP Deepwater Horizon Oil Disaster

9. Summarized in previous testimony.

2.2 State of Knowledge after the BP Deepwater Horizon Oil Disaster

2.2.1 Airborne Levels of Dangerous Chemicals during the BP DHOS Disaster

10. In this subsection, I focus on air emissions, because inhalation was the primary route of exposure for every person – responders and the general public – in the oil spill-impacted region. Estimates of BP DHOS responders range from 110,000 to 140,000.[1] Based on U.S. 2010 census data, I calculated the population in the counties and parishes directly impacted by the BP DHOS from Louisiana to the western Florida panhandle, to be, conservatively, 6 million. No attempt was made to account for millions of Gulf coast visitors, myself included, who would also have been exposed to oil spill contaminants.

11. During the first 30 days of the BP DHOS disaster, National Oceanic and Atmospheric Administration scientists estimated, based on aircraft measurements, that over 1,000 metric tons of soot particles were emitted from controlled burns

[1] Kwok RK, Engel LS, Miller AK, et al. The GuLF STUDY: A prospective study of persons involved in the BP DHOS response and clean-up. *Environ Health Perspect*. **2017**;125(4):570-578.

and over 10,000 metric tons of secondary aerosol particles were created from evaporating hydrocarbons.[2]

12. These oil contaminants became part of the Gulf hydrologic cycle and were measured during NASA's airborne remote sensing project. According to an interview with the chief mission coordinating scientist for the Airborne Visible/Infrared Imaging Spectrometer (AVIRIS),[3] clouds and air over the oil-impacted ocean were found to have "a very high hydrocarbon load" at levels "suggesting that there are reasons for concern" for human health. [4]

13. Of the several human health studies conducted after the BP DHOS disaster, only two studies analyzed actual ambient air concentrations of dangerous chemicals. These two studies are discussed and compared to determine: 1) whether the concentrations of oil contaminants at the sea surface and over land masses adjacent to oil-impacted seas were at dangerous levels; 2) what standards are appropriate to assess worker and public health; and 3) what was learned about monitoring ambient air concentrations of oil contaminants that might benefit planning and response efforts for future oil disasters.

14. The Southeast Louisiana (SELA) air monitoring study, conducted through Dillard University and Texas Southern University, has several features that make it both unique among, and extremely relevant to, all the human health studies conducted after the BP DHOS disaster – as well as future oil disaster planning and response.[5]

15. First, the study area was the 4,138-square mile land mass located closest to the offshore oil spill, and it had the largest potentially exposed population.[6] Further, the area along the coast is populated with socioeconomically disadvantaged groups more likely to be at risk of environmental hazards,[7] as well as representative of residents who worked on BP's oil spill response. This population makes this study

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[2] Middlebrook AM, Murphy DM, Ahmadov R, et al. Air quality implications of the BP DHOS. *Proc. Natl. Acad. Sci. U.S.A.* **2012**, 109:20280–20285. doi:10.1073/pnas.1110052108

[3] Foulsham, G. UCSB scientist has key role in Gulf oil spill studies. *UC Santa Barbara, The Current. Sci. & Technol.* **2010**, May 7. www.news.ucsb.edu/2010/012843/ucsb-scientist-has-key-role-gulf-oil-spill-studies

[4] Cope J. NASA data strengthens reports of toxic rain on the Gulf coast from the BP spill. *Huffington Post,* **2011**, Mar 7. www.huffingtonpost.com/jerry-cope/nasa-data-toxic-rain_b_830481.html

[5] Nance E, King D, Wright B, Bullard RD. Ambient air concentrations exceeded health-based standards for fine particulate matter and benzene during the BP DHOS. *J. Air Waste Manag. Assoc.* **2016**, Feb, 66(2):224-36. doi: 10.1080/10962247.2015.1114044.

[6] Id., Nance et al. 2016, Ambient air concentrations, Footnote (FN) 5.

[7] Bullard RD, Mohai P, Saha R, Wright B. *Toxic wastes and race at twenty 1987–2007.* A report prepared for the United Church of Christ Justice & Witness Ministeries. **2007**. www.ucc.org/environmental-ministries_toxic-waste-20

relevant to interpreting human health effects of responders and residents across the Gulf Coast, not just in Southeast Louisiana.

16. Second, the time frame for the study was May 1, 2010, to September 30, 2010.[8] This narrow window coincided with the core period of oil spill emissions and burning and the residual emissions after the well was temporarily capped in July, then permanently capped in early September. This focus makes the resulting dataset extremely relevant to interpreting human health effects of the exposed population.

17. Third, the area was well-sampled during – and to some extent before, the study time frame.[9] Over 1 million ambient air measurements were gathered during the oil spill. This included data from permanent regulatory monitors in the urban areas, which existed before the disaster, and emergency stationary monitors installed by the Environmental Protection Agency (EPA) along the coast – the area most likely impacted by oil spill emissions. In addition, emergency mobile monitors were installed on vehicles that traversed the region (coastal and urban) during the study period. Only results from the permanent regulatory monitors in urban areas have to be reported to the public, because only these monitors meet Clean Air Act regulatory criteria. The SELA air monitoring study compared and reported data gathered by all three types of monitors in urban, coastal, and regional areas to assess potential public health impacts. This approach makes this study unique – and relevant, because other studies only reported the more limited required data.

18. Fourth, the study assessed ambient air concentrations of benzene and fine particulate matter ($PM_{2.5}$) "because of the potential for significant ambient air emissions, the availability of data across the study area, and the clear health-based guidelines/standards."[10] This is the only study to analyze actual data for both benzene and $PM_{2.5}$, two contaminants of primary concern during the BP DHOS disaster response. This makes the SELA air monitoring study unique and extremely relevant – a model, against which to interpret human health effects in all other BP DHOS studies, and a model for ways to mitigate harm in real time during future oil spill disasters.

19. Significantly, the State of Louisiana reported, as required, data collected from only the permanent regulatory monitors in the urban areas. These data showed no exceedances of Clean Air Act standards during the oil spill. According to Nance et al. (2016), "this finding implies there were no public health impacts of concern."[11]

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[8] Nance et al. 2016, Ambient air concentrations, FN 5.
[9] Id., Nance et al. 2016, Ambient air concentrations, FN 5.
[10] Id., Nance et al. 2016, Ambient air concentrations, FN 5.
[11] Id., Nance et al. 2016, Ambient air concentrations, pp. 225–226, FN 5.

20. The EPA established a 1-year screening level for benzene during the BP DHOS disaster of 20 part per billion (ppb). This defined the level above which action would be taken to protect public health. However, this screening level was higher than all other relevant state, national, and international benzene standards, as shown in Table 1, including the Clean Air Act Unacceptable Cancer Risk Level of 13 ppb and the state of Louisiana's ambient standard of 12 ppb.[12] The SELA air monitoring study found regional and coastal mean benzene concentrations of 4.83 and 2.96 ppb, respectively, as shown in Table 2A.[13] These levels were lower than the three health standards, implying that the mean airborne concentrations of benzene were not of a concern for public health.

21. However, the EPA's oil spill screening level and Louisiana's ambient benzene standard were not health protective. Nance et al. (2016) decided to use the Clean Air Act's Low Cancer Risk Level for benzene of 0.13 ppb to assess potential health impacts. Against this standard, Nance et al. (2016) found that ambient levels of benzene during the 5-month time period were high enough to cause health impacts. Specifically, they found benzene levels went from the low end of the cancer risk range (1–10 in-a-million-cancer-risk) to the high end of that range (10–100 in-a-million-cancer-risk) in each of the six subareas (parishes) that were monitored. Risk of exposures to, and ambient air levels of, hazardous pollutants were higher along the coast compared to urban centers.

22. Nance et al. (2016) found the mean concentrations of fine particulate matter ($PM_{2.5}$) of 22.30 and 17.33 $\mu g/m^3$ from regional and coastal monitoring stations, respectively, exceeded the annual $PM_{2.5}$ standard of 12 $\mu g/m^3$ used to enforce the Clean Air Act in all six subareas, as shown in Table 2B. When Air Quality Index (AQI) exceedances for $PM_{2.5}$ during the spill were compared against background level exceedances in 2009, a sharp increase in PM was found, indicating an escalation of exposure to ambient fine particulate matter consistent with oil spill emissions. The study found long-term $PM_{2.5}$ concentrations during the oil spill were approximately 2–3 times higher than concentrations observed prior to the disaster, indicating possible public health impacts.

[12] Id., Nance et al. 2016, Ambient air concentrations, FN 5.
[13] Id., Nance et al. 2016, Ambient air concentrations, FN 5.

Table 1. Comparison of benzene health risk standards (adapted from Nance et al. 2016)

Source	Standard	Limit (ppb)
US EPA	BP DHOS 1-year screening level	20 [a]
Clean Air Act	Unacceptable Cancer Risk Level [b]	13
Louisiana	ambient	12
Texas	1-year screening level	4.5
CDC ATSDR, MRLs [c]	chronic, > 365 days	3
Clean Air Act	High Cancer Risk Level [d]	1.3
US EPA	regional cancer screen	0.312
Clean Air Act	Low Cancer Risk Level [e]	0.13 [f]
World Health Org.	ambient guideline	0.02

[a] EPA's standard above which action would be taken to protect public health
[b] Correlates to 100-in-a-million cancer risk
[c] CDC ATSDR, Minimum Risk Levels (MRLs): www.atsdr.cdc.gov/mrls/mrllist.asp
[d] Correlates to 10-in-a-million cancer risk
[e] Correlates to 1-in-a-million cancer risk
[f] Standard used to assess potential public health impacts in Nance et al. 2016

Table 2. Ambient air concentrations in the Southeast Louisiana air monitoring study (adapted from Nance et al. 2016)

A. Benzene (in ppb) [a]

Descriptive statistics	Mean +/- Std. Dev.	Minimum	Maximum	Number
Regional	4.83 +/- 6.76	0.12	82	2,791
Coastal	2.96 +/- 22.95	0.14	290	160
Urban	0.86 +/- –	0.51	2.33	936

B. Fine particulate matter ($PM_{2.5}$) [b]

Descriptive statistics	Mean +/- Std. Dev.	Minimum	Maximum	Number
Regional	22.29 +/- 10.65	3.7	89.16	3,133
Coastal	17.32 +/- 7.03	6.35	37.82	51
Urban	9.68 +/- 3.86	2.0	21.40	277

Potential public health impacts	Standard	Before	After
EPA AQI exceedance days [c]	100 [d]	0–93 days (with SO_2)	24–45 days (without SO_2)
NAAQS annual mean [e]	12 µg/m^3	8.26–10.98 µg/m^3	19.79–23.84 µg/m^3

[a] Only EPA ambient benzene concentration data were used, because BP's benzene data did not meet instrument sensitivity criteria. Total raw sample size for benzene was 3,887 data points.
[b] EPA and BP data were used for a total raw sample size for $PM_{2.5}$ of 102,682 data points.
[c] EPA AQI, Air Quality Index, for $PM_{2.5}$
[d] When an EPA AQI value exceeds 100, air quality is considered unhealthy for certain sensitive groups.
[e] National Ambient Air Quality Standards, for enforcement of Clean Air Act for $PM_{2.5}$ based on annual means

23. Based on their evaluation of a total of 106,569 measurements of fine particulate matter and benzene, Nance et al. (2016) conclude their findings indicate that the ambient air concentrations for these pollutants was a likely threat to public health during the oil spill and should have been a cause for concern – and preventative action. For example, they found that the "geographic exposure disparities were measurable in real time and therefore could have been used to issue region-specific preventive health announcements and precautions" (p. 233). Nance et al. recommend adopting health-based disaster thresholds to "facilitate decision-making, enhance public awareness, and reduce potential public health impact during an environmental crisis" (p. 234). The implications of this study for future oil spill response planning and preparation are discussed in Section 4.3.

24. The Gulf Long-Term Follow Up (GuLF) study also analyzed actual ambient air concentrations of dangerous chemicals.[14] This is a cohort study investigating the health of workers involved in the BP DHOS disaster response. The health findings are discussed in the next subsection. Here, I focus on the methodology for data collection and analysis). Like the SELA air monitoring study, the GuLF study also has implications for oil spill response planning and preparation.

25. The goal of the GuLF study was to develop and assign exposure estimates of Total Hydrocarbons (THC), dispersants and particulates, using available air samples collected by government agencies and the spiller BP. Ordinal estimates of exposures to these pollutants would create an ordered ranking of exposure levels, against which to assess and interpret potential health impacts of the cohort.

26. The available samples proved problematic. "The number of THC measurements taken by any single government agency was small (<100), of short-duration (≤ 4 hours), and generally reported as non-detectable and, therefore, too limited to support the development of the [GuLF] study's exposure estimates" (p. 3).

27. BP's THC samples also proved problematic. Some 28,000 full-shift airborne THC were collected from passive dosimeters on workers from late April 2010 to June 2011. Although the number of samples collected was large, the labs analyzing the data reported a limit of detection of about 0.4 parts per million (ppm) – i.e., 400 ppb – for a 12-hour sample and provided a dataset with 82% left-censored data or non-detects.

28. As shown in Table 1, the analytic laboratories' detection limit of 400 ppb is over 1 order of magnitude higher than the EPA's screening level for benzene of 20 ppb

[14] Stewart PA, Stenzel MR, Ramachandran G, et al. Development of a total hydrocarbon ordinal job-exposure matrix for workers responding to the BP DHOS: The GuLF STUDY. *J. Expo Sci Environ Epidemiol.* 2018 May, 28(3):223–230. doi: [10.1038/jes.2017.16] Epub18 Oct **2017**.

and the Clean Air Act's Unacceptable Cancer Risk Level for benzene of 13 ppb. It is 3 orders of magnitude higher than the Clean Air Act's Low Cancer Risk Level for benzene of 0.13 ppb. Significantly, the limit of detection is higher than the highest recorded/reported airborne benzene level of 290 ppb, collected at a coastal station in the SELA air monitoring study (Table 2A) and 2 orders of magnitude above the levels found to be a cause of concern for public health in this study. In other words, a detection limit of 400 ppb would not support findings of any chemical exposure-related health effects that occurred below 400 ppb. This detection limit is simply too high for this type of study.

29. The need to include data below the limits of detection of the reporting analytic laboratory in data analysis is an important part of the exposure assessment strategy, as harmful health effects are known to occur at air concentrations below established the analytical limits of detection. The National Institute for Environmental Health Sciences (NIEHS), the entity sponsoring the GuLF study, sponsored a simulation study to evaluate three established methods for analyzing datasets with censored observations to estimate arithmetic mean, geometric mean and standard deviation, and the 95th percentile of the exposure distribute – to determine the method that would accurately reveal as much of what was censored as possible.[15] Against evaluation metrics, the ß-substitution method was found to outperform the other two methods.[16] The ß-substitution method was then compared against a Bayesian method. The latter generally provided accurate estimates and, significantly, distributions of all of the parameters, while the ß-substitution method only provided estimates of uncertainty for the arithmetic mean.[17]

30. In the GuLF study, Stewart et al. (2017) obtained BP's data from the analyzing labs and recalculated the measurement results to reflect the analytical sensitivity, as revealed using the Bayesian method for analyzing left-censored datasets. This lowered the limit of detection, based on a 12-hour sample, to 0.01–0.1 ppm "depending on the lab" (p. 3) – i.e., 10–100 ppb – and reduced the censored data, the non-detects, to 19% of the database. The GuLF study was left with 26,588 THC data points – a large enough database to develop ordinal exposure categories for airborne THC.

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[15] Huynh T, Ramachandran G, Banerjee S, et al. Comparison of methods for analyzing left-censored occupational exposure data. *Annals of Occupational Hygiene*. **2014** Nov, 58(9):1126-1142. Epub 26 Sep 2014.

[16] Huynh T, Quick H, Ramachandran G, et al. A Comparison of the ß-substitution method and a Bayesian method for analyzing left-censored data. *Ann Occup Hyg*. 2016 Jan, 60(1): 56-73. Epub Jul 24 **2015**.
Quick H, Groth C, Banerjee S, et al. Exploration of the use of Bayesian modeling of gradients for censored spatiotemporal data from the BP DHOS. *Spatial Statistics.* **2014** Aug, 9:166-179. Epub 19 March 2014.

[17] Id., Huynh et al. 2015, Comparison of methods, FN 16.

31. In contrast, the GuLF study found "there were less than 1,000 personal measurements of dispersant vapor concentrations, and one of the major sources of dispersant applications (aerial application to the water surface) was not directly measured at all. There were no particulate measurements of the two primary sources of particulate (oil/gas burning at the wellhead and in situ burning offshore)" (p. 5). For this reason, the GuLF study had to base ordinal "estimates of dispersants and particulate exposure on the questionnaire responses alone" (p. 5) – in other words, to use qualitative estimates for these two pollutants of concern, instead of air monitoring data.

32. Despite the revealing shortcomings in BP's database, the GuLF study contains revealing statements of ambient air concentrations for THCs. For example, four "vessels sprayed Corexit 9500A on the water surface in the hot zone/ source area from May 15 to about July 7 when volatile organic chemicals (**VOC**) levels **exceeded 50 ppm**" (p. 6). "Also, BP had direct-reading instruments on many of the vessels in the hot zone/ source areas where the oil was reaching the surface within hours of release. When air concentrations [of **VOCs**[18]] **exceeded 20 ppm**, as measured by the direct-reading instruments, water was sprayed, and **above 70 ppm**, dispersant was sprayed, to disperse the sheens" (p. 9). "Only 15 of the 28,000 **THC** personal samples were ≥ **100 ppm**, with only **3 exceeding 200 ppm**" (p. 9).

33. Even against the backdrop of other oil spills, these levels are extraordinarily high. The maximum air concentrations during the *Exxon Valdez* 5-month oil spill response, reported by Exxon, exceeded OSHA's Permissible Exposure Limits (or proxies**) for **benzene** (**7.8 ppm**, geometric mean 0.069 ± 0.596 ppm CI_{95}), **oil mist**** (**20 mg/m3**, GM 0.615 ± 4.0 mg/m^3 CI_{95}), **PAH aerosol**** (**8.6 mg/m3**, GM 2.297 ± 1.15 mg/m^3 CI_{95}), and **2-butoxyethanol** (**99 ppm**, GM 1.66 ± 19.2 ppm CI_{95}).[19] As reported in Stewart et al. (2017), airborne **THC** concentrations at a dwelling nearest the wreck of the tanker *Braer* were **6.3** and **0.7 ppm** the first two

[18] Stewart et al. 2017, GuLF Study job-exposure matrix, p. 9, FN 14. It is unclear if these measurements refer to VOCs, a subset of THCs, or THCs.

[19] Med-Tox. Air monitoring results for oil mist: VOCs master by task and VOCs master by date. **1989**. See Table A.1. in Ott R, *Sound Truth and Corporate Myths: The Legacy of the Exxon Valdez Oil Spill*, 2004 (Dragonfly Sisters Press: Cordova, Alaska); data obtained from *Stubblefield v. Exxon* 1994 before case was sealed until 2024. **Exxon used mineral oil as a proxy for crude oil mist as OSHA has not established PELs for crude oil mist.

Med-Tox. Results of air sampling for PAHs. **1989**. See Table A.1. in Ott R, *Sound Truth and Corporate Myths: The Legacy of the Exxon Valdez Oil Spill*, 2004 (Dragonfly Sisters Press: Cordova, Alaska); data obtained from *Stubblefield v. Exxon* 1994 before case was sealed until 2024. **Exxon used nuisance dust as a proxy for PAH aerosols as OSHA has not established PELs for PAH aerosols.

Garry Stubblefield and Melissa Stubblefield v. Exxon Shipping Company, Exxon Corporation, VECO, Inc., and Norcon, Inc. 3AN–91–6261 CV (HBS), Alaska Superior Court, Third Judicial District at Anchorage (**1994**).

days of the spill. Air concentrations over the first 20 days near where the tanker *Nakhodka* wrecked averaged **0.04–0.56 ppm THC**.

34. Since there are no specific occupational guidelines for THC, Stewart et al. (2017) chose petroleum naphtha as a proxy. OSHA's Permissible Exposure Limit for naphtha is 500 ppm, based on an 8-hour total weighted average. Against this guideline, Steward et al. (2017) declared the airborne "THC levels were low compared to the occupational standards" (emphasis added, p. 9). But OSHA guidelines assume workers are wearing recommended Personal Protective Equipment and working a 40-hour week in five 8-hour days with off-work hours and weekends of zero exposure.

35. Neither of these assumptions were true for the majority of contract (and/or non-governmental) workers. In fact, BP DHOS Vessel of Opportunity contract workers were repeatedly and consistently told if they wore respirators, for example, their jobs would be terminated.[20] The GuLF study acknowledges "sources of exposure misclassification include the lack of exposure estimates at night for participants who spent the night on vessels that remained in the Gulf; dermal exposures; and the long-working hours, few days off, and high temperatures and humidity" (p. 10).

36. *Most importantly, exposures of 24/7 (24 hours, 7 days a week) with no (or inadequate) protection argue for using standards for public health, not occupational guidelines.* Relative to the public health standards for benzene in Table 1, the reported high-end VOC (or THC) concentrations were extremely high, not low. The levels are literally off the chart, as they are 4–5 orders of magnitude above the Clean Air Act's Unacceptable Cancer Risk Level for benzene. In the GuLF study, Stewart et al. (2017) created 7 ordinal THC levels, ranging from a category 1 of less than 30 ppb to a category 7 of 10,000+ ppb. Even the lowest level is double the Unacceptable Cancer Risk level and above EPA's screening threshold to trigger action to protect public health in Table 1. If human health effects could be anticipated in all of the THC ordinal exposure categories, it could make human health effects difficult to assess, as one is essentially comparing exposed individuals with exposed individuals.

37. Dispersant and particulate ordinal categories in the GuLF study also contain other sources of exposure misclassification, based on my experience of living in oil spill-impacted Gulf Coast communities and working daily with residents from May 4, 2010 through late April 2011. For example, Stewart et al. (2017) notes that for "both dispersants and particulates, although participants may have reported dates

[20] Government Accountability Project, Shanna Devine and Tom Devine. Deadly dispersants in the Gulf: Are public health and environmental tragedies the new norm for oil spill cleanups? **2013**. http://www.whistleblower.org/gulftruth

outside of the event dates (e.g., April 28 and July 19 for *in situ* burns), exposure was only considered in the time period within the date boundaries" (p. 7). I wrote an open letter to EPA with documentation of aerial and on-the-water spraying of dispersants through August in *state waters*, nearshore, near populated areas.[21]

38. Photo-documentation of dispersant-spraying and use in state waters, outside of the official time frames provided by US Coast Guard Incident Command, are included in Appendix B.[22] For example, Appendix B, p. 5, photos taken by local residents document dispersant spraying on August 10, 2010, in coastal seas near Pass Christian, Mississippi (top photos). The staging area was near their home. Another staging area for spraying Corexit 9500A in nearshore waters was on August 21, 2010, on Dauphin Island, Alabama (bottom photos).

39. In October 2010, I also met privately with *in situ* burn crews in south Louisiana, including the crews who had been medevacked off their boats on May 26, 2010, and treated for acute respiratory failure and other work-related exposure symptoms, according to crew members. Some individuals were disabled from their chemical exposures and had filed work-related chemical injury lawsuits. In my opinion, the *in situ* burn crews were exposed to some of the very highest particulate exposures, because of the crews' close proximity to the source of particulates and to the source of fresh oil upwelling daily from depth. Yet in the GuLF study, Stewart et al. (2017) "assigned a "low" particulate exposure category to participants… because [they] were positioned upwind from the burn and because of the low number of burns compared to participants." However, photo documentation from a *Los Angeles Times* photographer provides evidence that workers were not always positioned upwind of a burn (Appendix B, p. 1). Also, anyone who has sat upwind of a campfire can attest that their clothes still smell of wood smoke (particulates).

40. Photo-documentation of the particulate exposure hazard associated with *in situ* burns was provided by one of the fishermen captains who was medivacked off his boat on May 26, 2010, for acute respiratory failure (Appendix B, p. 2).[23] *In situ* burn teams were told not to wear respirators and were not initially provided with respirators. During the BP-contracted safety training program, trainers presented the "respirator statement" slide (Appendix B, p. 2), documenting that workers were told during their required safety training, not to wear respirators.

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[21] Ott R. An open letter to US EPA, Region 6. *Huffington Post,* Aug. 27, **2010**. www.huffingtonpost.com/riki-ott/an-open-letter-to-us-epa_b_697376.html

[22] Ott R. Public comments on behalf of Ultimate Civics and a signatory coalition on the U.S. Environmental Protection Agency rulemaking on the National Oil and Hazardous Substances Pollution Contingency Plan, Subpart J, dispersant use. 40 CFR Parts 110 and 3002015, [Document ID No. EPA–HQ–OPA–2006–0090; FRL–9689–9– OSWER] RIN 2050–AE87. Apr. 22, **2015**.

[23] Id., Ott, 2015, Public comment to EPA, FN 22.

41. Another critical factor to keep in mind is the role of dispersants and the *form* of the chemically-enhanced oil, whether in the air or water. Dispersants break up surface oil into small droplets and envelop the oil droplets in bubble-like structures.[24] Chemically-enhanced (or in this case, Corexit-enhanced) (CE) oil droplets are released into the air and the water column through wave action. Because of the widespread and unprecedented aerial (and subsurface) application of dispersants during the BP DHOS disaster, the most likely form of oil encountered by people was as CE-oil mists or droplets (particulates).

42. As Dr. Daniel Teitelbaum explained in his expert witness testimony in *Stubblefield v. Exxon* (1994), it is important to look at the high-end exposures.[25] "The concentration of oil within the droplet is extremely high, although the concentration in any cubic meter may be low, because the droplets may be widely dispersed. Those droplets, when they hit, are usually very highly contaminated. It's part of the problem of trying to look at a particulate or an aerosol. Aerosols tend to be very concentrated" (p. 174). In other words, the risk of inhaling droplets is usually low, but the risk of health impacts is high when one inhales or has dermal contact with contaminated mists or aerosols. During the BP DHOS response, during active spraying of dispersants or decontamination degreasers, even the risk of inhaling droplets was very high, given recorded THC air concentrations of over 100 ppm.[26] Absolute measurements of THC in airborne mists or PAHs in particulates reflect concentration in the volume of the air sampled and, therefore, would understate the potential risk to human health from CE-oil mists and aerosols.

43. In summary, the main findings of this subsection are:

- Ambient air levels of benzene and fine particulate matter in Southeast Louisiana during at least May 1 through September 30, 2010, the core or peak emission period, were at concentrations that were likely a threat to public health and should have been a cause for concern and preventative action, based on health-protective federal standards.
- Ambient air levels of total hydrocarbons during this same timeframe were also at concentrations that were likely a threat to worker health and should have been a cause for concern and preventative action.
- The most likely form of oil encountered by people was as CE-oil mists or droplets (particulates), and measurements of THC in airborne mists or PAHs in particulates would understate the potential risk to human health from CE-oil mists and aerosols.

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[24] K. Burns and M.R. Harbut, Gulf Oil Spill Hazards, Sciencecorps, Lexington, MA, June 14, **2010**. www.Sciencecorps.org/crudeoilhazards.htm

[25] Teitelbaum DT, MD. Deposition. Oct. 12, **1993**. In *Stubblefield v. Exxon* 1994, FN 19.

[26] Steward et al. 2017, GuLF Study job-exposure matrix, FN 14.

- Standards for public health should be used to assess potential occupational health impacts, especially for contract workers, because these responders were told not to wear respirators, and many were exposed 24/7 to oil contaminants.
- Requirements for reporting federal air monitoring data were insufficient to detect public health concerns and failed to accurately communicate health threats in real-time.
- Industry datasets cannot be relied on to sample exposures commensurate with the anticipated risk of exposures, or to analyze samples with sufficient analytical sensitivity to detect compounds of concern to human health.

2.2.2 Waterborne Levels of Dangerous Chemicals during the BP DHOS Disaster

44. In this subsection, I focus on waterborne levels of petroleum hydrocarbons and other pollutants associated with oil spill response, because dermal contact was another important route of exposure for responders, fishermen, field scientists, SCUBA divers, beach-goers who walked on seawater-rinsed sand, waded, or swam in the ocean, and wildlife, in the oil spill-impacted region. The science now supports an understanding of oil distribution from sea surface to ocean floor and of the role of surface and subsurface dispersant use in oil distribution and, critically, persistence.

45. An estimated 60% of the 210,000,000 gallons of crude oil, mostly released at depth from the blowout, reached the sea surface.[27] But between the ocean floor and sea surface, much happened with ramifications for human (and ecosystem) health. In 2016, BP released its comprehensive Gulf Science Dataset with more than 24,500 water samples, collected from the sea surface to a maximum depth of 2,850 m from May 5 to December 31, 2010.[28] The dataset allowed scientists to qualitatively examine the partitioning and distribution of various crude oil fractions (i.e., light and heavy hydrocarbons) – and the impact of 771,000 gallons of Corexit 9500A dispersant, injected directly at the wellhead, on petitioning and distribution of crude oil fractions.

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[27] Reddy CM, Arey JS, Seewald JS, et al. Composition and fate of gas and oil released to the water column during the BP DHOS. *Proc. Natl. Acad. Sci. USA.* **2012**, 109:20229–20234. doi: 10.1073/pnas.1101242108

[28] BP Gulf Science Data1. Chemical analysis of oil samples from the Gulf of Mexico and adjoining states from May 2010 to March 2014. **2016.** Filename: WaterChemistry_W-01v02-01_xTab. Available at: https://data.gulfresearchinitiative.org/data/BP.x750.000:0005

BP Gulf Science Data2. Subseadispersant application records collected during the BP DHOS accident near the Mississippi Canyon block 252 wellhead from April 30 to July 22, 2010. **2016.** Filename: DispersantApplication_OTH-02v01-01. Available at: https://data.gulfresearchinitiative.org/data/BP.x750.000:0018#

46. Only one study to-date has analyzed BP's Gulf Science Dataset to determine: 1) the distribution of oil contaminants within the water column; 2) whether the concentrations of waterborne oil contaminants occurred at dangerous levels; and, 3) how subsea dispersant injection influenced distribution and concentration of oil contaminants.

47. This study classified the water chemistry results into two hydrocarbon fractions, based on molecular weight: a light fraction between 5–12 carbons and a heavier fraction with 13 or more carbons that contained primarily PAHs.[29] Of the 13,218 water sample oil concentrations reported in 2010, 59% and 26% were collected within a radial distance of 100 km and 10 km from Macondo, respectively. Water column samples within 100 km of the wellhead were used to analyze distribution of the hydrocarbon fractions. Concentrations within 100 km of the wellhead source ranged **0.0008–58,730 ppb** for the **light oil fraction** and **0.0004–101,768 ppb** for the **heavier fraction**. Water column samples within the 10 km subset were used to specifically determine whether subsea dispersant injection would reduce the amount of oil rising directly at the response site, as intended.

48. The understanding that emerged from this study found that oil distribution was controlled by temperature- and pressure-dependent processes, rather than dispersants.[30] The pressurized jet of oil that blew out of the wellhead led to rapid expansion of the dissolved gases, which atomized the gas-saturated oil into micro-droplets. This shifted the droplet size distribution to smaller droplets that remained suspended thousands of meters below the surface. High concentrations of both the light and heavier fractions formed a deep plume, centered between 1,000–1,300 m until it started to break down after the discharge stopped in July. Efforts to control the Macondo blowout and repair the riser increased the turbulent energy and increased the flow rate, which also mechanically dispersed the oil into micro-droplets that remained suspended at depth, as evidenced by the data. The timing of these operations coincided with increased subsea dispersant injection and oil collection at the wellhead. Disaster responders at the surface attributed the decrease in benzene and other light hydrocarbons upwelling from depth to successful use of dispersants, rather than mechanical dispersion, as supported by the data.

49. As further support against dispersant use during deep-sea blowouts, the study also found that water depth and time from the blowout controlled much of the distribution of PAHs in the water column, not volume of chemical dispersants

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[29] Paris CB, Berenshtein I, Trillo ML, et al. BP Gulf Science Data reveals ineffectual subsea dispersant injection for the Macondo blowout. *Front. Mar. Sci.*, 30 October **2018**. doi.org/10.3389/fmars.2018.00389

[30] Paris et al. 2018, BP Gulf Science Data, FN 29.

injected at the wellhead.[31] Besides the deep plume, high concentrations of the heavier fraction were found in the upper water column, above 200 m and mostly near the BP DH response site, to about 80 km downstream from May through December 2010. Unlike the light fraction, concentrations of PAHs above the 90 percentile were found after the well was capped, from August through September. Concentrations above the median were also observed later in the year.

50. Citing evidence that chemical dispersants may have inhibited biodegradation of PAHs in the Gulf of Mexico,[32] the scientists postulated that dispersants may have increased the residence time of these persistent and mobile pollutants. The authors concluded, "it is clear that oil degradation is not necessarily accelerated after dispersant addition, potentially undermining the utility of subsea dispersant injection" (p. 5). For the purposes of health impacts to humans and the Gulf ecosystem, it is important to understand that high concentrations of PAHs persisted in the upper water column and at the sea surface, at least within 100 km of the spill source, through the end of 2010.

51. Another distinctive feature of the BP DHOS disaster was the finding of accumulation of oil on the seafloor as a previously unaccounted for, and significant, sink. The amount of oil that reached the seafloor was estimated, at the lower limit, to be up to 14 percent.[33] The mechanism was found to be naturally-occurring marine snow, a fluffy aggregation of marine organic detritus and various microbes (bacteria, diatoms, eukaryotic phytoplankton) glued together with mucus biopolymers secreted by the microbes.[34] The mucus biopolymers were found to have amphiphilic properties, among other characteristics, that allow them to attract and interact with hydrophobic organic compounds like oil hydrocarbons.[35] In the presence of oil, the community composition of the marine snow rapidly shifts to a dominance of naturally-occurring oil-degrading microbes and oil-centric aggregates rapidly form.[36] The marine oil snow aggregate continues to captures oil

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[31] Paris et al. 2018, BP Gulf Science Data, FN 29.

[32] Kleindienst S, Seidel M, Ziervogel M, et al. Chemical dispersants can suppress the activity of natural oil-degrading microorganisms. *Proc. Nat. Acad. Sci. USA.* **2015**, 48:14900–14905. doi: 10.1073/pnas.1507380112

[33] Passow U, Sweet J, Quigg A. How the dispersant Corexit impacts the formation of sinking marine oil snow. *Mar Pollut Bull.* **2017** Dec 15, 125(1–2):139–145. doi: 10.1016/j.marpolbul.2017.08.015. Epub 12 Aug 2017.

[34] Suja LD, Summers S, Gutierrez T. Role of EPS, dispersant and nutrients on the microbial response and MOS formation in the subarctic northeast Atlantic. *Front Microbiol.* **2017**, 8:676. Epub 21 Apr 2017. doi:10.3389/fmicb.2017.00676

[35] Id., Suja et al. 2017, Role of EPS, FN 34.

[36] Doyle SM, Whitaker EA, De Pascuale V, et al. Rapid formation of microbe-oil aggregates and changes in community composition in coastal surface water following exposure to oil and the dispersant Corexit. *Front Microbiol.* **2018** Apr 11, 9:689. doi: 10.3389/fmicb.2018.00689.

droplets from the sea surface and upper water column, slowly increasing in mass and sinking, and, in the process, transporting large quantities of oil to the sea floor.

52. Dispersants were found to interact with this naturally-occurring phenomena in ways that can directly impact human health. For example, surface spraying of Corexit dispersants increased the amount of oil, suspended as micro-droplets, within the water column. In lab studies, the highest abundances of oil-centric microbe aggregates occurred in the presence of the emulsifying agent, Corexit, at oil and dispersant concentrations consistent with measurements from within the subsurface plume during the BP DHOS disaster.[37] (Higher concentrations of Corexit were found to dissolve the mucus biopolymers and/or kill certain species of the oil-degrading microbes.)

53. The CE-marine oil snow formed visible reddish-orange stringers on the sea surface that could be avoided by beach-goers who swam or waded in coastal seas. Encounters with CE-marine oil snow entrained in the water column were avoidable only by avoiding contact with the water. As documented in my previous testimony, PAH concentrations in the aggregates and tarry masses sampled nearshore were "consistently in excess of the IDLH (Immediately Dangerous to Life or Health) limits (80 mg/m3)," [38] as stated by NIOSH and OSHA.[39] What I did not document previously were my observations of the deadly consequences of exposure to CE-marine oil snow, now described in subsection 3.1.

54. Another human health risk from dermal contact with CE-marine oil snow is related to shifts in community composition of oil-degraders, which occurred rapidly in the presence of oil and CE-oil,[40] including an enrichment of *Vibrio* bacteria in the presence of dispersant.[41] One study noted: [42]

> *"The enrichment of these organisms is not frequently observed at contaminated sites in the marine environment, although there are snippets in the literature reporting on the enrichment of these organisms by crude oil. For example, members of the Vibrionales were found enriched in beach sands of the Gulf coast that had become contaminated with Macondo oil from the DWH spill, and several oil-degrading Vibrio spp. were isolated and found to*

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[37] Id., Doyle et al. 2018, Rapid formation of aggregates, FN 36.
[38] James "Rip" Kirby, III, "Findings of Persistency of Polycyclic Aromatic Hydrocarbons in Residual Tar Product Sourced from Crude Oil Released during the BP DHOS MC252 Spill of National Significance," supported by the Surfrider Foundation, April 14, **2012**. http://surfrider.org/images/uploads/publications/Corexit_Connections.pdf
[39] NIOSH Pocket Guide to Chemical Hazards; DHHS (NIOSH) Publication No. 2005-149; Sept **2007**.
[40] Doyle et al. 2018, Rapid formation of aggregates, FN 36.
Suja et al. 2017, Role of EPS, FN 34.
[41] Id., Suja et al. 2017, Role of EPS, FN 34.
[42] Id., Suja et al. 2017, Role of EPS, FN 34.

degrade hydrocarbons.[43] *Also, a 91-fold increase in the relative abundance of Vibrionales was detected in oil contaminated sea surface oil-slick water samples from DWH when incubated to develop anaerobically.*[44] *An analysis of the genomes of several Vibrio species found these organisms capable of metabolizing hydrocarbons, including PAHs.*[45]"

55. One of the *Vibrio* species that was found in high numbers in tar balls collected from areas impacted by the BP DHOS was *Vibrio vulnificus*, a human flesh-eating pathogen, capable of causing severe wound infections.[46] My observations of the consequences of exposure CE-oil sand are described in subsection 3.1.

56. Finally, another human and environmental health hazard linked with use of Corexit dispersants is that of harmful dinoflagellate blooms or "red tides." While formation of red tides after oil spills has been observed in spills where chemical dispersants were applied, including the BP DHOS, a new study found exponential growth in bloom-forming dinoflagellates exposed to cultures of crude oil alone (1 ppb), Corexit 9500A-treated oil (1 ppb), and dispersant alone (0.05 ppb).[47] Field tests with natural plankton assemblages from coastal and offshore waters in the northern Gulf of Mexico, also exposed to different concentrations of crude oil (1–25 ppb), Corexit 9500A (0.05–1.25 ppb), and Corexit-treated oil (1–25 ppb), confirmed that certain bloom-forming dinoflagellates have a tolerance to these pollutants higher than their main grazer predators (ciliates and heterotrophic dinoflagellates). The study concluded that harmful algae blooms after oil spills can be the result of a disruption in microzooplankton grazing pressure on bloom-forming dinoflagellates, and that the Corexit dispersant is more toxic than previously assumed, especially for small planktonic organisms.[48]

57. In summary, the main findings of this subsection are:

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[43] Kostka JE, Prakash O, Overholt WA, et al. Hydrocarbon-degrading bacteria and the bacterial community response in Gulf of Mexico beach sands impacted by the BP DHOS. *Appl. Environ. Microbiol.* **2011**, 77:7962–7974. doi: 10.1128/AEM.05402-11

[44] Gutierrez T, Berry D, Teske A, et al. Enrichment of *Fusobacteria* in sea surface oil slicks from the BP DHOS. *Microorganisms*, **2016**, 4:24. doi: 10.3390/microorganisms4030024

[45] Grimes DJ, Johnson CN, Dillon KS, et al. What genomic sequence information has revealed about *Vibrio* ecology in the ocean – a review. *Microb. Ecol.* **2009**, 58:447–460. doi:10.1007/s00248-009-9578-9

[46] Tao Z, Bullard S, Arias C. High numbers of Vibrio vulnificus in tar balls collected from oiled areas of the north-central Gulf of Mexico following the 2010 BP DHOS. *Ecohealth,* **2011**, 8(4):507–511. doi: 10.1007/s10393-011-0720-z.

[47] Almeda, Rodrigo & Cosgrove, Sarah & Buskey, Edward. Oil spills and dispersants can cause the initiation of potentially harmful dinoflagellate blooms ("red tides"). *Environ Sci & Technol*. **2018**, 52:5718–5724. doi: 10.1021/acs.est.8b00335.

[48] Almeda et al. 2018, Dispersants can cause red tides, FN 47.

- Partitioning and distribution of oil released at the subsea source was controlled by temperature- and pressure-dependent processes, rather than subsea dispersant injection.
- High concentrations of PAHs persisted in the upper water column and at the sea surface, at least within 100 km of the spill source, through the end of 2010, and coincided with high ambient air levels of oil contaminants.
- Dispersants may have increased the residence time of the toxic PAHs, but they did not accelerate oil degradation; these first three points undermine the utility of subsea dispersant injection.
- As further evidence against use of subsea dispersant injection (and surface spraying of dispersant), dispersants alone and with CE-oil were found to: 1) enhance formation of naturally-occurring marine snow and indirectly increase sedimentation of surface oil to the sea floor; 2) increase risk of human health encounters with CE-marine oil snow; 3) enrich presence of *Vibrio* bacteria, a dangerous human pathogen, in CE-marine oil snow and tar balls/mats; and 4) disrupt microzooplankton grazing pressures in a way that initiates red tide blooms.

58. In conclusion of these subsections on dangerous levels of dangerous chemicals in air and water, in my opinion, exposure to the BP DHOS disaster is like exposure to Agent Orange in Vietnam, in the sense that, if you were there, you were exposed. When Congress finally became convinced of the human health consequences of aerial spraying of Agent Orange, it passed the Vietnam Veterans Agent Orange Relief Act in 1984.[49] The act established a service-connected entitlement to disability compensation for veterans who suffered from specific disorders, but instead of having to prove exposure, the US Department of Veterans Affairs accepted "presumptive diseases." The VA recognizes 14 cancers and other health problems as indicative of exposure to Agent Orange. The VA also presumes certain birth defects in children of Vietnam (and Korea) veterans are associated with veterans' qualifying military service.[50] In other words, if you were in Vietnam during the war, you were exposed – and so might be your children conceived after your tour of duty.

59. I believe the same is likely true of the BP DHOS disaster.[51] If you were there, living, working or visiting, Gulf coast communities impacted by the BP DHOS disaster from April 20, 2010 to September 30, 2010, you were exposed to potentially dangerous levels of dangerous chemicals – oil, dispersants, and particulates. It

[49] U.S. House of Representatives, Office of History, Art & Archives. Agent Orange Relief Act. https://history.house.gov/HouseRecord/Detail/15032436192 Visited Nov. 23, **2018**.

[50] U.S. Dept. of Veterans Affairs. Public Health, Veterans' diseases associated with Agent Orange. www.publichealth.va.gov/exposures/agentorange/conditions/ Visited on Nov. 23, **2018**.

[51] Sneath S. 8 years after BP oil spill, thousands of medical claims still not paid. *The Times-Picayune*, April 20, **2018**. www.nola.com/environment/index.ssf/2018/04/eight_years_after_bp_oil_spill.html

didn't matter if it was a week or five or more months, because dose plus host (susceptibility) makes the poison.[52] This observation is supported with evidence in the next subsection on BTEX levels found in human blood.

2.2.3 Human blood levels of dangerous chemicals from the BP DHOS Disaster

60. In this subsection, I focus on levels of BTEX, alkanes, and/or PAHs found in human blood after the BP DHOS disaster. Blood levels of the crude oil compounds, especially from samples collected close to real-time exposures, can provide strong evidence of exposure and can be a critical link between exposures to airborne and waterborne oil spill contaminants and human health effects.

61. As documented in my previous testimony, after the BP DHOS disaster, National Aeronautics and Space Administration "scientists found oil and oil-dispersant droplets aerosolized daily and became part of the Gulf hydrologic cycle.[53] What I did not share in my previous testimony was my personal experience, observations, and actions, based on this knowledge and which are now relative to this testimony.

62. On May 4, 2010, I arrived in Venice, Louisiana, the closest community to the offshore blowout and staging center for media and local fishermen responders involved with *in situ* burn teams. I gave my first community talk that afternoon, organized by the Louisiana Shrimp Association. During that first talk, people shared health symptoms (described in previous testimony), relayed from family or friends working offshore on oil spill response. I also walked the local beach and observed a healthy beach ecosystem and residents wading in the water and walking and riding horses along the beach. Small globs of oil had washed ashore on April 30, but in early May, beaches were still relatively oil-free. Aerial spraying of Corexit dispersants had already started offshore (Corexit 9527A on April 22; Corexit 9500A on April 27),[54] but the air still seemed relatively fresh (minimal hydrocarbons).

63. That changed rapidly. About mid-May, the oil hit beaches across the Gulf coast from Louisiana to western Florida. Aerial spraying of dispersants in coastal waters was consistently reported by residents. (All oil-impacted coastal states had signed letters that pre-authorized dispersant use in state waters from the coast to 3 miles offshore in EPA Regions 4 and 6.) During every community talk in every state, people shared health symptoms, described previously and documented in

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[52] Ott R. BP, governments downplay public health risk from oil and dispersants. *Huffington Post,* posted July 7, **2010**, updated May 25, 2011. www.huffingtonpost.com/riki-ott/the-big-lie-bp-government_b_638369.html

[53] Foulsham 2010, UCSB scientist has key role, FN 3.
Cope 2011, NASA data strengthens reports, FN 4.

[54] Steward et al. 2017, GuLF Study job-exposure matrix, FN 14.

Huffington Post blogs, that either they were experiencing or their family or friends who were working on the disaster response were experiencing. By early to mid-June, I realized people onshore were experiencing the same symptoms as the offshore responders.

64. At community meetings, people across the four oil-impacted Gulf coast states began to ask me how they could prove that oil spill exposures were making them sick. I advised people to ask their health care providers for Metametrix Volatile Solvent Whole Blood Profiles.[55] I knew the people who developed this test, and I had high confidence in its ability to detect BTEX compounds and key alkanes, well after the elusive and highly volatile benzene had been flushed from the body. If concentrations for some of these other compounds were high, it would indicate a high exposure to benzene.

65. At the time (June through October 2010), I felt there was little risk of false negatives – profiles with no detects, because of the daily presence of oil in the air *at levels that caused immediate acute symptoms like headaches, burning or teary eyes, raw throats, and a persistent cough*. During this timeframe, I observed oil coatings on car windshields, windward and outside-facing windows of houses, and hotel beach furniture and outdoor cloth umbrellas and poles, and – significantly – continued aerial spraying of Corexit dispersants and use of dispersants in decontamination staging areas. I felt the potential gain for Gulf coast residents, in terms of establishing exposure to BP DHOS pollutants, outweighed the risk. BTEX, alkanes, and/or PAHs are important predictors of potential human health effects. Early biomonitoring yields critical metrics of personal exposure that are more relevant than estimates obtained through ambient air and water monitoring, because biomonitoring provides an internal dose of the chemicals, capturing exposure from different routes (e.g., inhalation, adsorption, ingestion) and potentially different sources.

66. Hundreds of people from age 2 to over 80 took the Metametrix test through their licensed health care provider from June 2010 into early 2011 (2–10 months post disaster) when hydrocarbon levels in the blood work started to drop out of the upper 95th percentiles. No published synthesis of these data is available – yet. However, people blogged about their test results and medical doctors and public health experts commented on them. For example, Michael Harbut, MD, director of the Environmental Cancer Program at Wayne State University's Karmanos Cancer Institute in Michigan, stated in an interview that hexane, one of the alkanes measured by Metametrix, causes "dying back neuropathy, meaning the nerve cells in the arms and legs die back from the distal tips to the proximal end, [causing]

[55] Genova Diagnostics (formerly Metametrix). Volatile Solvents Profile – Whole Blood. www.gdx.net/product/volatile-solvents-test-blood Visited on November 26, **2018**.

numbness, pain, all sorts of things. Hexane is a direct petroleum product, so where you see hexane, you would expect to see benzene." [56] Tingling appendages was a commonly reported exposure symptom during 2010. Harbut told the interviewer, a frequent visitor to the Gulf coast after the BP DHOS disaster, that the blood work indicated the presence of "biomarkers" – "evidence that you inhaled it, because it's in your blood." Harbut stressed that it was important for the medical community "to look for end organ damage rather than the presence of a solvent, because the solvent could have evaporated after it has already whacked the brain or whacked the liver."

67. Media reports also described high levels of BTEX chemicals in blood taken from Gulf coast residents during this time.[57] For example, Wilma Subra, a MacArthur Fellow and chemist in Louisiana, told *Al Jazeera*, "ethylbenzene, m,p-xylene, and hexane are volatile organic chemicals that are present in the BP crude oil. We're finding these in excess of the 95th percentile [profile of the US National Health and Nutritional Examination Survey (NHANES)], which is [based on] the average for the entire nation. Sometimes we're finding amounts 5 to 10 times in excess of the 95th percentile." [58] Levels in or upwards of the 95th percentile indicate extremely high levels of contaminants. Subra added, "The presence of these chemicals in the blood indicates exposure." She explained, there has been long enough exposures so as to create chronic impacts including "liver damage, kidney damage, and damage to the nervous system." [59]

68. For comparison purposes, the NHANES 95th percentile for **benzene** is **0.056 ppb** in the NHANES 2005–2008 study cohort (n = 4,442).[60] The NHANES level – and the levels observed in Metametrix blood profiles of Gulf residents described above – fall within the range of biological equivalents for benzene in blood from 0.04 to 1.29 ppb considered to harm production of all types of blood cells.[61]

69. Three subsequent studies were published on BTEX concentrations in human blood at separate intervals after the BP DHOS disaster: 8–22 months post disaster, 12–36 months post disaster, and 29–36 months post disaster. Of these, only the first

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[56] Cope J. No safe harbor on the Gulf coast: Human blood tests show dangerous levels of toxic exposures. *Huffington Post,* Sept. 2, **2010**. www.huffingtonpost.com/jerry-cope/no-safe-harbor-on-gulf-co_b_698338.html

[57] Jamail D. BP dispersants 'causing sickness.' *Al Jazerra,* Oct. 29, **2010**. www.aljazeera.com/indepth/features/2010/10/201010271321362203 70.html

[58] Id., Jamail 2011, Gulf spill sickness, FN 57.

[59] Id., Jamail 2011, Gulf spill sickness, FN 57.

[60] Doherty BT, Kwok RK, Curry MD, et al. Associations between blood BTEXS concentrations and hematological parameters among adult residents of the U.S. Gulf states, Table 2. *Environ Res*. **2017** Apr 26;156:579-587. doi:10.1016/j.envres.2017.03.048

[61] Hays SM, Pyatt DW, Kirman CR, et al. Biomonitoring equivalents for benzene. *Regul Toxicol Pharmacol.* **2012**, 62(1):62–73. [PubMed:22178585]

confirmed levels of BTEX compounds in human blood at the levels anywhere near what I had observed in dozens of blood profiles before the end of 2010.[62]

70. In this first study, the cohort was comprised of 69 adults and 8 children.[63] All subjects had reported medical problems that they related to exposure through inhalation and/or dermal contact to crude oil and/or dispersants from the BP DHOS. Genova Diagnostics (formerly Metametrix) performed the analyses. Data were pooled for all subjects to focus on overall blood concentrations. The average blood concentrations of the study group for four aromatic compounds – m,p-xylene, toluene, ethylbenzene, and benzene – were similar to the NHANES 95th percentile, indicating concentrations significantly higher than normal. The average blood concentrations of the alkanes, although high, were not ranked among the NHANES highest percentiles.

71. Sammarco et al. (2016) interpreted this to mean the body "may have a mechanism by which to clear or metabolize alkanes more readily than aromatic hydrocarbons." [64] Or, alternatively, "it is also possible that the alkanes may have been accumulated elsewhere in the body and may no longer be evident in the blood" (p. 835). It is well established that these organic compounds are lipophilic and easily taken up by human tissues, such as fat, kidneys, liver, blood, and more. As for the confounding issue of smokers, which was not accounted for in this study because data were pooled, the authors felt it "unlikely… that on the average, most of the [the cohort] would exhibit petroleum hydrocarbons concentrations above the national 95% confidence limits… What they do have in common is some exposure to the BP DHOS" (p. 835).[65]

72. The scientists suggested that use of dispersants prolonged retention of crude oil in the water column by drawing the oil down into the water column. The high concentrations of aromatic hydrocarbons or PAHs were not anticipated to persist so long after the disaster event and/or capping of the well.[66] If CE-oil was retained in the water column, as this study postulated, then it would also have been available in the air, because of the unique hydrologic features of the Gulf of Mexico; i.e., the high daily evaporation from the sea surface. As discussed in subsection 2.2.2, analysis of empirical data found that high concentrations of PAHs persisted in the

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[62] Sammarco PW, Kolian SR, Warby RA, et al. Concentrations in human blood of petroleum hydrocarbons associated with the BP DHOS, Gulf of Mexico. *Arch Toxicol.* **2016** Apr, 90(4):829-37. doi: 10.1007/s00204-015-1526-5. Epub 22 May 2015.

[63] Sammarco et al. 2016, Concentrations in human blood, FN 62.

[64] Id., Sammarco et al. 2016, Concentrations in human blood, FN 62.

[65] Id., Sammarco et al. 2016, Concentrations in human blood, FN 62.

[66] Id., Sammarco et al. 2016, Concentrations in human blood, FN 62.

upper water column and at the sea surface, at least within 100 km of the BP DHOS source, through the end of 2010.[67]

73. The GuLF study also looked for associations between blood BTEX concentrations and blood disorders in two separate sets of samples collected "approximately 3 years" post disaster (~3-year blood study)[68] and 2.5–3 years post disaster (2.5-year blood study).[69] Taken together with the earlier Metametrix data, these studies provide a time sequence of blood BTEX measurements from 2 months to 36 months post disaster.

74. Well-aware that BTEX compounds are rapidly metabolized and excreted, the GuLF studies were intended to look at contemporary exposures, such as from the petrochemical industry or smoking, not exposures related to the BP DHOS disaster. In addition, the 2.5-year blood study was intended to alleviate "heightened concern among Gulf coast residents about possible ongoing exposure to BTEX and associated health effects" from the media reports that had described high levels of BTEX chemicals in blood taken from Gulf coast residents.[70]

75. Results in both studies were analyzed and discussed separately for tobacco smoke unexposed and tobacco smoke exposed, as determined by blood levels of the biomarker 2,5-dimethylfuran.[71] Tobacco smoke exposure was the primary source of blood BTEX levels. Only results for non-smokers are discussed below in both studies (unless noted), as this is of primary interest in this testimony.

76. In the ~3-year blood study, the study cohort consisted of 406 racially diverse (52% non-white), mostly male (75%) individuals of whom 95% lived in a county adjacent to the oil-impacted Gulf coast, 89% had worked on BP DHOS disaster response, 68% reported tobacco smoke exposure (n = 146 for tobacco smoke unexposed), and nearly half (48%) were obese.[72]

77. Of interest are three findings. First, blood BTEX concentrations were approximately log-normally distributed with long right tails, indicating a disproportionate number of high levels. Indeed, geometric means of blood BTEX concentrations for the GuLF study cohort were comparable to the NHANES participants for the five BTEXs also

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[67] Paris et al. 2018, BP Gulf Science Data, FN 29.
[68] Doherty et al., 2017, Associations between blood BTEXS, FN 60.
[69] Werder EJ*, Gam KB*, Engel LS, et al. Predictors of blood volatile organic compound levels in Gulf coast residents. *J Expo Sci Environ Epidemiol.* **2018** June;28(4):358-370 *equal contribution.
[70] Id., Werder EJ, et al. 2018, Predictors of blood volatile organic compound levels, FN 69.
[71] A blood concentration of ≤ 0.014 ppb 2,5-dimethylfuran was identified to discriminate between less-than-daily smokers and daily smokers (> 0.014 ppb 2,5-dimethylfuran). Chambers DM, Ocariz JM, McGuirk MF, et al. Impact of cigarette smoking on volatile organic compound (VOC) blood levels in the U.S. population: NHANES 2003–2004. *Environ Int.* **2011**, 37:1321-1328. [PubMed: 21703688]
[72] Doherty et al., 2017, Associations between blood BTEXS, FN 60.

analyzed by Metrmetrix (benzene, toluene, ethylbenzene, o-xylene, and m,p-xylene). However, the 95th percentile of blood BTEX levels were higher in 4 of 5 VOCs among GuLF study participants than NHANES values (Doherty et al. 2017, Table 2). Toluene was only slightly lower, but of the same order of magnitude (0.314 versus 0.329 ppb for GuLF study and NHANES, respectively). This may well indicate a lingering, yet retreating, shadow of presumably much higher blood levels in the post disaster months of 2010 for reasons described below.

78. The second finding of interest is a brief statement: "In a supplementary analysis, we observed no apparent differences in blood BTEX concentrations or hematological parameters between those who participated in oil spill work and those who did not." [73] Of this, without seeing the data, I would issue three cautions. First, the finding is based on the full cohort (tobacco smoke exposed and unexposed participants), which likely confounded the analysis. Second, the distinction (such as it was) between workers and nonworkers was also confounded by comparing participants in Non-Worker, Very Low, and Low job-exposure categories with participants in Medium and High categories, developed in the ordinal job-exposure matrix. As discussed earlier, these categories are prone to misclassification that would tend to obfuscate comparisons like the one in this ~3-year blood study.

79. The third finding of interest is the conclusion that current ambient exposure to these BTEX compounds, particularly benzene, may be associated with hematological effects, including reduced red blood cell count and concentration, and increased red cell distribution width – all indicators of anemia.[74] It is currently understood that benzene metabolized in the liver and bone marrow forms multiple reactive metabolites that harms and kills blood-forming cells and mature blood cells, as described in the ~3-year blood study. My concern lies with "ambient exposure."

80. In my opinion, Doherty et al. (2017) incorrectly assumes that historic BTEX levels do not influence present (current) physical health associations, noted in the ~3-year blood study: "while it is possible this population's experience with the oil spill influenced the associations we observed, this is unlikely given the biological half-lives of VOCs and the recovery period for benzene-induced hematoxicity." As observed by Dr. Harbut,[75] unusually high blood BTEX levels, especially for prolonged periods – which this entire study cohort almost certainly had during the months post disaster in 2010 – <u>are</u> predictors of long-term to blood and other organs. High background levels of these same compound could also contribute to health issues.

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[73] Doherty et al. 2017, Associations between blood BTEXS, FN 60.
[74] Doherty et al. 2017, Associations between blood BTEXS, FN 60.
[75] Cope, 2010, No safe harbor, FN 56.

81. In further support of my opinion are the high percentage of over-weight (31%) and obese (56%) participants in the tobacco smoke unexposed group. When VOCs "disappear" out of the blood, they do not necessarily "disappear" out of the body. Oil contaminates, including PAHs, are lipophilic and known to sequester in fat-rich body tissues. The contaminants can mobilize back into the blood during times of physical exertion, with resulting acute symptoms similar to initial exposures. For example, I frequently heard from "oil sick" Gulf residents in the years following the disaster of instances in which a person felt better and so mowed the lawn or played with the grandkids, only to become "oil sick" all over again and bed-ridden. Controlled and physician-supervised mobilization of sequestered toxins is one of the methods used by health care providers to detox patients of environmental pollutants.

82. Given the finding in the ~3-year blood study of a high (and unexplained) prevalence among the (tobacco smoke unexposed) cohort of blood BTEX levels in the 95th percentile of NHANES distribution and the prevalence of over-weight participants, I would conclude that the group's oil spill experience cannot be dismissed as a possible (and likely) influence on the findings of adverse hematologic effects.

83. In the 2.5-year blood study, the study cohort (n = 718) consisted of population with similar characteristics, as described above. The tobacco smoke unexposed group is (n = 146) the focus of discussion. There are two findings of interest.

84. First, blood BTEX levels in this group were found to be similar to the NHANES study of 2005–2008.[76] Compared to the Metametrix blood profiles taken in the post disaster months of 2010, this may indicate that somewhere between 2 to 3 years post disaster, blood BTEX levels may have returned to baseline for exposed workers and the general population.

85. Second, in addition to evaluating a suite of predictors, the study also evaluated three exposure sources of concern to the community: previous work on the BP DHOS disaster response, seafood consumption, and well water consumption. None of the community concerns were predictive of BETX exposure, which the study found "may provide some reassurances for oil spill response workers and community members concerned about lasting exposure to BTEX and related health effects." [77] However, as I previously explained, this is simply not the case. High blood BTEX levels, especially for prolonged periods, are predictors of potential long-term adverse health effects that may show up well after blood BETX levels

[76] Werder EJ, et al. 2018, Predictors of blood volatile organic compound levels, FN 69.
[77] Id., Werder EJ, et al. 2018, Predictors of blood volatile organic compound levels, FN 69.

have returned to baseline levels. This study overstated its results: the current low blood BETX levels are not predictors of BP DHOS exposure-related health effects from past high exposures to BETX and other oil contaminants.

86. In summary, the main findings of this subsection are:

- Reports and an early study of human blood BTEX levels showed dangerously high levels, likely mirroring high ambient air and water levels of oil contaminants in the first year following the BP DHOS.
- Somewhere between 2 to 3 years post disaster, human blood BTEX levels may have returned to a pre-spill baseline for exposed workers and the general population, as evidenced by the later two GuLF blood BTEX studies.
- The GuLF studies are overstating their results, as findings of low blood BETX levels, 2.5–3 years post disaster, are not predictive of BP DHOS exposure-related health effects from past high exposures to BETX and other oil contaminants.

87. This means that the large-scale Gulf studies that were initiated 1 to 3 years post disaster to investigate human health effects of the BP DHOS disaster would do well, as Dr. Harbut warned, "to look for end organ damage rather than the presence of a solvent [i.e., BTEX compounds], because the solvent could have evaporated after it has already whacked the brain or whacked the liver." [78] The next subsection examines these studies.

2.2.4 Human Health Effects of the BP DHOS Disaster & Collaborating Evidence

88. In this subsection, I focus on human health effects, primarily on worker health here and public health in section 3.1. Ideally, a worker safety program would establish health baselines of job candidates pre-hire. For oil spill work, collection of blood BTEX samples could be part of a safety training program. Ideally, a long-term follow up study of occupational exposures from oil spill work would assess the parameters that had already been established in a baseline health examine – blood and liver parameters, as well as pulmonary, cardiac, and neurological functions – close to the time of the exposure or termination of work. These same parameters would then be evaluated and compared over time. Health baselines were not conducted, making it difficult to infer causality with any study design.

89. However, the weight of evidence is also important, and a growing body of literature has identified a consistent set of health symptoms associated with oil spill exposures – significantly, with no or minimal dispersant use. A smaller set of longitudinal studies, mostly from researchers following the 2002 *Prestige* spill in

[78] Cope 2010, No safe harbor, FN 56.

Spain and the 2007 *Hebei Spirit* oil spill in South Korea, have identified long-term diseases and illnesses, stemming from acute exposures, as was presented in my earlier testimony. Significantly, the BP DHOS disaster is the first to contribute findings on effects of CE-oil exposures and dispersants. NEW findings in human health, related to oil spill exposures, are discussed below.

90. While several long-term studies have been initiated, only one longitudinal study to-date has been published on the BP DHOS disaster. As discussed in my previous testimony, D'Andrea and Reddy reported that BP response workers had significantly altered blood profiles and liver enzymes, indicating higher risk for blood-related disorders, and a high prevalence of somatic symptoms consistent with previously reported studies on major spills.[79] The study cohort was not previously explained, nor the significance of the findings relative to long-term illnesses. This is now presented to help interpret the 7-year follow-up study.

91. The initial study consisted of spill workers from south Louisiana who had worked for over 3 months on the BP DHOS disaster and were exposed to oil and dispersants.[80] Exposed subjects were referred to the clinic for medical evaluation by the subject's legal representative (n = 117). Unexposed subjects lived at least 100 miles away from the Gulf coast of Louisiana (n = 130), had visited the clinic for a wellness check-up, and were randomly selected for the study by their primary care physicians. The unexposed group differed significantly from the exposed group, having 67% females (v. 89% males in exposed group) and a mean age of 50 (v. 36 in exposed group). Differences in demographic characteristics were found not to affect results.

92. The results of this initial study indicate that oil spill exposure can have a significant, adverse, long-term effects on worker health. As explained in the study, serum levels of specific enzyme (ALP, AST, and ALT, described below) are considered biomarkers of liver damage. Specifically, phosphatases, amino transferases, and dehydrogenases play critical roles in biological processes, specifically, in detoxification, metabolism, and biosynthesis of energetic macromolecules that are important for different essential functions. Alterations in the levels of these enzymes, such as reported in the initial medical evaluations, can result in biochemical impairment, lesions in the tissue, and cellular dysfunction.[81]

93. The excretion of phenol in urine is a biomarker of benzene exposure (BUN, below). The initial study also found significant amounts of phenol in urine of

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[79] D'Andrea MA, Reddy GK. Health consequences among subjects involved in Gulf oil spill cleanup activities, *The American Journal of Medicine*, **2013**, 126(11):966–974. doi: 10.3389/fpubh.2018.00117

[80] Id., D'Andrea and Reddy 2013, Health consequences, FN 79.

[81] Id., D'Andrea and Reddy 2013, Health consequences, FN 79.

exposed subjects but not unexposed subjects, indicating "that subjects involved in the oil spill clean-up activity were inherently exposed to benzene." [82]

94. The researchers noted that, although exposed subjects "had significant differences in various hematological and hepatic indices, our results indicate that exposure to the oil spill did not uniformly affect subjects who participated in oil spill [response] activities." This observation is consistent with the hallmark axiom of toxicology: *Dose plus host (susceptibility) makes the poison*.

95. No attempt was made to distinguish between these exposures, but other studies found "it unsurprising that those exposed to dispersants were more likely to have also been exposed to levels of airborne THC > 3.0 ppm (53% vs. 7% unexposed), and those exposed via dermal contact were also "substantially more likely to have also come into contact with oil or tar (97% vs. 31% unexposed).[83] Therefore, "exposed" is likely to be CE-oil exposed. This is consistent with my observations during the year following the BP DHOS disaster: If you were there, you were likely exposed to oil and CE-oil.

96. Notably, the initial study makes this observation: "The health complaints reported by those involved in the oil spill clean-up operations are consistent with previously reported studies of other major spills [1996 *Sea Empress,* 1997 *Nakhodka*, 2003 *Tasman Spirit,* 2007 *Hebei Spirit*]. *However, the prevalence of symptoms appears to be higher in the present study compared with the earlier findings of other investigators."* [84] This is also consistent with my observations. I was shocked by the rapid onset, severity, and frequency of symptoms that I had anticipated, based on my experience with and later understanding of the *Exxon Valdez* oil spill, 21 years earlier. During my year spent in Gulf coast communities after the BP DHOS, I concluded that the observed rapid onset, severity, and frequency of symptoms was dispersant-mediated and related to the solvent properties of these Corexit products, as discussed in my earlier testimony.

97. The follow-up medical evaluations were conducted 7 years post disaster. The cohort consisted of 44 of the original (CE)-oil spill exposed group (who elected to return and/or could be located) and data from 44 unexposed subjects in the comparative group. The long-term follow-up study found that response workers experienced persistent alterations or worsening of their hematological, hepatic, pulmonary, and cardiac functions, and prolonged or worsening illness symptoms, 7

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[82] Id., D'Andrea and Reddy 2013, Health consequences, FN 79.

[83] McGowan CJ, Kwok RK, Engel LS, et al. Respiratory, dermal, and eye irritation symptoms associated with Corexit™ EC9527A/EC9500A following the BP DHOS: Findings from the GuLF STUDY. *Environ Health Perspect.* **2017** Sep, 125(9): 097015. Epub 15 Sept 2017. doi: [10.1289/EHP1677]

[84] D'Andrea and Reddy 2013, Health consequences, FN 79.

years after their spill exposure. Key findings of the 7-year follow-up evaluations are as follows.[85]

- Hematological alterations included increased mean WBC [white blood cell] counts, hemoglobin, hematocrit, and reduced platelet counts as well as BUN [blood urea nitrogen] levels.
- Hepatic alterations included increased ALP [alkaline phosphatase], AST [aspartate amino transferase], and ALT [alanine amino transferase] levels in the serum.
- Most workers were found to have developed chronic rhinosinusitis (91%) and reactive airway dysfunction syndrome (45%) as new symptoms up to 7 years post disaster. These symptoms had not been routinely reported during the initial clinic visit.
- The incidence of increased deteriorated pulmonary function was found in oil spill exposed workers increased over 2-fold from their initial clinic visit. The moderate-to-severe pulmonary function abnormalities were more common 7 years after their oil spill exposure, "indicating a prolonged and persistent adverse health effect due to oil spill exposure."
- Incidence and persistence of cardiac abnormalities were found and were unexpected, given the age of the exposed subjects.
- Prolonged or worsening of illness symptoms were still present 7 years after their initial exposure. Shortness of breath was the most frequently reported symptom among oil exposed subjects at both their initial (75%) and their 7-year (84%) follow-up visits. Headaches was the second most frequently reported symptom, followed by skin rash, chronic cough, weakness, dizzy spells, painful joints, and chest pains.

98. These findings of long-term harm from oil spill exposure to blood and liver function, oncogenesis, respiratory and pulmonary function, and cardiac function are supported by other longitudinal studies and are discussed separately.

Genotoxicity and Oncogenesis

99. Several longitudinal studies were conducted on genotoxicity 2 years,[86] 6 years,[87] and 7 years[88] after the 2002 *Prestige* oil spill off the northern coast of Spain. These

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[85] D'Andrea MA, Reddy GK. The development of long-term adverse health effects in oil spill cleanup workers of the BP DHOS offshore drilling rig disaster. *Front Public Health.* **2018** Apr 26; 6:117. doi: 10.3389/fpubh.2018.00117.

[86] Rodriquez-Trigo G, Zock JP, Pozo-Rodriquez F, et al. Health changes in fishermen 2 years after clean-up of the *Prestige* oil spill. *Ann Intern Med.* **2010**, 153:489–499. doi: 10.7326/0003-4819-53-8-201010190-00279 PMID: 20733177

Monyarch G, de Castro RF, Zock JP, et al. Chromosomal bands affected by acute oil exposure and DNA repair errors. **2013**, 8(11):e81276. doi: 10.1371/journal.pone.0081276 PMID: 24303039

studies used chromosomal damage as biomarkers to detect genotoxic effects and to predict an increase in cancer risk. In all these studies, an increase in chromosome damage was detected. The persistence of genotoxic damage suggested stem cells of the bone marrow had been affected. Another 7-year post disaster study on the *Prestige* cohort identified four chromosome bands that were especially prone to breakage upon replication stress in oil exposed individuals only.[89] This type of chromosome instability and breakage is a driving force of oncogenesis – the early stages of cancer development or tumor formation. The study noted that "a significant number of chromosome alterations in hematological diseases, such as patients with T-cell lymphoma, acute lymphoblastic leukemia and acute myeloid leukemia, are associated with the four chromosome bands identified as being damage by oil spill exposure. Further, the study observed that of the full study cohort (n = 622), 6 of the 7 persons who had developed various cancers were oil exposed individuals (unpublished data); studies are planned to assess the effect of acute oil exposure on cancer.

100. Several longitudinal studies were conducted on genotoxicity after the 2007 *Hebei Spirit* oil spill. In one study conducting during the 8-month spill response effort, urine samples were collected before and after spill response work on 150 university student volunteers.[90] The samples were analyzed for 3 urinary metabolites: t,t-muconic acid, a biomarker of benzene; mandelic acid, a biomarker of ethylbenzene and xylene; and 1-hydroxyprene, a metabolite biomarker of PAHs. The levels of all 3 metabolites was significantly higher in samples after oil exposure than those measured before participation.

101. In another study, urinary oxidative stress biomarkers were measured on 671 residents who participated in *Hebei Spirit* response work for over 100 days.[91] Levels of PAH metabolites (1-hydroxypyrene and 2-naphthol) and levels oxidative stress

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Biern g, Gialdo J, Zock JP et al. Human genotoxic study carried out two years after oil exposure during the clean-up activities using two different biomarkers. *J Mar Sci.* **2015**, 3:1334–1348. doi: 10.3390/jmse3041334

[87] Hildur K, Templado C, Zock JP, et al. Follow-up genotoxic study: Chromosome damage two and six years after exposure to the *Prestige* oil spill. *PLoS One*. **2015**, 10:e0132413. doi: 10.1371/journal.pone.0132413 PMID: 26221948

[88] Laffon B, Aguilera F, Rios-Vazquez J, et al. Follow-up study of genotoxic effects in individuals exposed to oil from the tanker *Prestige,* 7 years after the accident. *Mutat Res.* **2014**, 760:10–16.

[89] Frances A, Hildur K, Barbera JA, et al. Persistence of Breakage in Specific Chromosome Bands 6 Years after Acute Exposure to Oil. *PLoS ONE* **2016**, 11(8): e0159404. doi:10.1371/journal. pone.0159404

[90] Ha M, Kwon H, Cheong HK, et al. Urinary metabolites before and after cleanup and subjective symptoms in volunteer participants in cleanup of the Hebei Spirit oil spill. *Sci Total Environ.* **2012**, 429:167–173. doi: 10.1016/j.scitotenv.2012.04.036. Epub 15 May 2012

[91] Noh SR, Cheong HK, Ha M, et al. Oxidative stress biomarkers in long-term participants in clean-up work after the Hebei Spirit oil spill. *Sci Total Environ. **2015** May 15, 515-516:207-14.* doi: 10.1016/j.scitotenv.2015.02.039. Epub 26 Feb 2015

biomarkers (malondialdehyde, MDA, an indicator of lipid peroxidation; and 8-hydroxy-2'-deoxyguanosine, 8-OHdG, an indicator of oxidative DNA damage) were significantly increased with longer involvement in oil spill response work. The biomarker levels decreased as more time elapsed since the last involvement in spill response work. Significant associations among biomarkers was maintained for up to 12 months after the last oil spill response work.

102. In a longitudinal study, urinary oxidative stress biomarkers were collected 1.5 years and 6 years post disaster on residents who lived near (n = 476) and far (n = 152) from the coast impacted by the *Hebei Spirit* oil spill.[92] Levels of the biomarkers (MDA and 8-OHdG) were significantly higher in the "near" group and had a significant association with duration of spill response activities even 6 years post disaster, indicating consequences of oil pollution may last for years.

103. After finding consistent evidence of adverse health effects among Taean County residents impacted by the 2007 *Hebei Spirit* oil spill, including findings of elevated levels of oxidative DNA stress markers indicative of cancer risk in high-exposed groups even 6 years post disaster, researchers in South Korea decided to investigate the cancer incidence trend in Taean County.[93] Five major cancers and leukemia were selected for analysis. Cancers of the stomach, lung, and colon were selected in both men and women. In addition, in men, liver and prostate cancers were selected and, in women, thyroid and breast cancers were selected. The exposure area, Taean County, was classified into a high-exposure area, comprised of 4 towns within 10 km of the oil-impacted coast, and a low-exposure area, comprised of 4 towns 10–45 km from the coast. The comparison area included 3 coastal areas with a demographics and coastal area similar to that of Taean County. The observed cancer incidence rates and trends in the exposed and comparison areas were compared with those observed nationwide from 1999 through 2014.

104. The study found that the incidence rate of prostate cancer has increased in Taean since 2007 and is higher in Taean than that observed in other coastal areas in 2009–2014 and, further, that the incident rates were higher in the high-exposure areas than in the low-exposure areas.[94] Further, the annual percent change of the incidence rates in Taean was higher than that observed nationwide and in coastal areas: Taean, 39.3% [2007–2010]; nationwide 13.5% [1999–2009]; and coastal areas, 15.6% [1999–2009]. The study also found that the incidence rate of

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[92] Kim JA, Noh SR, Cheong HK, et al. Urinary oxidative stress biomarkers among local residents measured 6 years after the *Hebei Spirit* oil spill. *Sci Total Environ.* **2017** Feb 15, 580:946-952. doi: 10.1016/j.scitotenv.2016.12.044. Epub 16 Dec 2016

[93] Choi KH, Park MS, Ha M, et al. Cancer incidence trend in the *Hebei Spirit* oil spill area, from 1999 to 2014: An Ecological Study. *Int. J. Environ. Res. Public Health* **2018**, 15:1006. doi:10.3390/ijerph15051006

[94] Id., Choi et al. 2018, Cancer incidence trend, FN 93.

leukemia in women Taean residents had increased and was higher than in the other coastal areas and nationwide in 2009–2014 and that the incident rates were higher in the high-exposed areas than the low-exposed areas. However, the incident rate ratio was not significantly high, because leukemia is not a common disease and Taean has a small population. The study concluded that the incidence of prostate cancer among Taean County residents had increased since the *Hebei Spirit* oil spill.

Respiratory and Pulmonary Functions

105. In longitudinal studies conducted to assess respiratory symptoms 2 years and 5 years after the 2002 *Prestige* oil spill,[95] investigators reported that oil spill exposed workers had persistent symptoms including shortness of breath, wheeze, cough, and phlegm initially and 5 years post disaster.

106. Building on these *Prestige* oil spill studies, two studies evaluated cytotoxic and genotoxic *in vitro* effects on cultures of human lung epithelial cells, following various exposures to water-accommodated fractions of dispersed oil alone and with the Corexit dispersants 9500A or 9527A.[96] Besides cell survival, the study measured induction of DNA damage and the effect on DNA repair capability of exposures. The study found that the WAF-dispersed oil at low level exposures (100 to 300 ppm) induced DNA damage in the form of single and double strand breaks. The latter can lead to cell death, genomic instability, and malignant transformation. Addition of dispersants further increased the genotoxic effects of the oil at low levels (at high levels, cytotoxic effects were observed). Double strand breakage DNA repair pathways appeared unaffected by oil-dispersant mixtures tested. The study concluded there was a potential that many of the exposed and damaged surviving cells would accumulate mutations, positioning them as potential sources for future disease states such as cancer.

107. Further building on the *Prestige* studies, Liu et al. performed the first RNA-sequencing study using cultured human airway epithelial cells at concentrations of WAF-dispersed oil of 300 ppm.[97] The study found that effects of WAF-dispersed oil

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[95] Zock JP, Rodriguez-Trigo G, Pozo-Rodriguez F, et al. 2007, Prolonged respiratory symptoms in clean-up workers of the *Prestige* oil spill. *Am J Respir Crit Care Med.* **2007**, 176(6):610–6. doi:10.1164/rccm.2007o1-016OC

Zock JP, Rodriquez-Trigo G, Rodriguez -Rodriguez E, et al. Persistent respiratory symptoms in clean-up workers 5 years after the *Prestige* oil spill. **2012**, 69(7):508–13. doi:10.1136/oemed-2001-100614

[96] Major D, Derbes RS, Wang H, Roy-Engel AM. Effects of corexit oil dispersants and the WAF of dispersed oil on DNA damage and repair in cultured human bronchial airway cells, BEAS-2B. *Gene Rep*. **2016**, 3:22-30. Published online 2016 Feb 11. doi: [10.1016/j.genrep.2015.12.002]

[97] Liu YZ, Roy-Engel AM, Baddoo MC, et al. The impact of oil spill to lung health-insights from an RNA-seq study of human airway epithelial cells. *Gene*. **2016**, 578:38–51. doi: 10.1016/j.gene.2015.12.016. Epub 9 Dec 2015.

was different for Corexit 9500A and Corexit 9527A. 9500A alone had the largest effect at the individual gene level (84 response genes) and 9527A had the weakest effect (0 response genes), while oil alone had a medium effect (26 response genes). However, when mixed with oil, 9527A had a stronger synergistic effect (46 response genes), than 9500A (4 response genes). Significantly, "the response pathways were characterized by enhanced angiogenesis (formation of new blood vessels) and immune response and weakened cell junctions and steroid synthesis. These signature pathways/gene sets correspond to some of the key pathological features for asthma, cystic fibrosis, or COPD." [98] A key feature of cystic fibrosis and COPD is excess mucus production and airway mucous obstruction.

108. Recent studies from the BP DHOS disaster show further evidence of persistent respiratory effects. The Coast Guard studies drew from a cohort of white (76.8%) males (85.7%) who were members of the Coast Guard between the start of the disaster (20 April 2010) and the end of the *transition* phase on 17 December 2010 (n = 53,519 with n = 8,696 responders and n = 44,823 non-responders).[99] Most were younger than 34 years of age (63.8%).[100]

109. Prospective and cross-sectional analyses were based on the subgroup of responders who completed the (second) exit survey (n = 4,855).[101] Crude oil exposure was reported for 55% of the responders and dispersant exposure for 40.4% (or 22% with direct contact) with considerable overlap between oil and dispersant exposure (91.1%). (Exhaust exposure was reported by 75.3% of responders.) The two most reported missions (jobs) were administrative support and incident command support/command post (n = 2,064 and 1,685, respectively), which were categorized as not likely exposed to crude oil. The most commonly reported missions with a high likelihood of exposure to crude oil were booming/skimming operations (n = 1,497), spill clean-up/decontamination operations (n = 1,128), and safety/environmental health (n = 970).

110. In the cross-sectional comparison of those,[102] statistically significant associations for crude oil were found with:
    - respiratory symptoms of coughing, shortness of breath, and wheezing, including an exposure-response relationship, (i.e., increased exposure was associated

[98] Liu et al. 2016, RNA-sequence study, FN 97.
[99] Rusiecki J, Alexander M, Schwartz EG, et al. The Deepwater Horizon oil spill Coast Guard cohort study. *Occup Environ Med.* **2018** Mar, 75(3):165-175. doi: 10.1136/oemed-2017-104343
[100] Id., Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99. Medical encounter data were available for all active-duty cohort members, dating back to October 2007 and allowing for prospective follow-up health assessments; serum samples were collected after exposure ended and are available for future studies on biomarkers of biological effects from exposure to crude oil and/or dispersants and other oil contaminants.
[101] Id., Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.
[102] Id., Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.

with increased prevalence of symptoms), with a possible threshold effect for coughing;

- neurological symptoms of headaches and light-headedness/dizziness, including a possible exposure-response relationship with possible threshold effects;
- dermal symptoms of skin rash/itching with similar elevated prevalence ratios across exposures (i.e., no exposure-response relationship);
- gastrointestinal symptoms of diarrhea and stomach pain, including exposure-response relationships, and nausea/vomiting, which was only statistically significant only at the highest exposure level; and
- genitourinary symptoms of burning or painful urination, which was only statistically significant only at the highest exposure level, with evidence of an exposure-response trend (the number of cases was small).

111. There was an indication of increased prevalence for the cardiovascular symptom, sudden heartbeat changes, in the highest exposure category, but it was not statistically significant.[103] Supporting evidence of atrial arrhythmias in large pelagic fish (tuna) was discussed in my previous testimony and is supported with new evidence the GuLF studies, discussed below under the Cardiac Function subsection. This symptom was not anticipated, given the age of the cohort.

112. Longitudinal analyses were conducted on relative risks for health outcomes to compare incidence between active-duty responders with non-responders, and oil-exposed responders with non-oil-exposed in the first 2.5 years post disaster. The longitudinal analyses revealed that responders had elevated relative risk for dermal condition, as did oil-exposed responders for chronic respiratory conditions, asthma, and dermal conditions. Elevated relative risks were also found for chronic obstructive pulmonary disease and headaches/migraines, with estimates approaching statistical significance. The researchers noted that, "Analysis of the health encounter data (medical records) indicated that health effects associated with the oil spill response may be more apparent when comparing exposed and non-exposed responders, rather than when comparing responders with non-responders," as the responders were a heterogeneous group. It may also stem from a healthy worker effect.[104]

113. Further, it should be noted that the health symptoms were categorized using ICD-9 coding, which did not include categories for chemical illness symptoms. ICD-10 coding came into effect in October 2015 in the U.S. It includes vomiting in both

[103] Id., Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.
[104] Id., Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.

the stomach category and the nervous system category,[105] the latter being more likely for symptoms related to chemical illnesses, in my opinion.

114. A cross-sectional study of acute respiratory health symptoms was analyzed for the Coast Guard cohort of spill responders.[106] The most common acute respiratory symptom was coughing (19.4%), followed by shortness of breath (5.5%) and wheezing (3.6%). Adjusted analyses found:

- An exposure-response relationship existed between increasing deployment duration and likelihood of these 3 symptoms for any oil exposure in the pre-capping period (before July 15, 2010), and a similar pattern in the post-capping period for coughing and wheezing.
- Increased prevalence ratios for all 3 symptoms for any oil exposure. Increasing frequency of inhalation of oil was associated with increased likelihood of all 3 symptoms. A similar pattern was observed for contact with oil dispersants for coughing and shortness of breath.
- The combination of CE-oil (i.e., oil plus dispersants) presented associations that were much greater in magnitude than oil alone for all 3 symptoms, corroborating findings from the *in vitro* studies with human lung epithelial cells.

115. Coast Guard cohort responders routinely reported any use of oil exposure-reducing Personal Protective Equipment, PPE (72.4%), including safety glasses, safety boots, protective headgear, gloves, Tyvek suits, waders and/or respirators. Prevalence ratios for all 3 acute respiratory symptoms were higher among responders who did not report any use of PPE compared to those who did report any use of PPEs. A similar pattern was found for responders reporting use of a respirator; i.e., those who did not report use had higher prevalence for shortness of breath and wheezing.

116. Dispersant models were similarly adjusted by use of dispersant-related PPE, period of response in relation to the well capping (i.e., before/after), and smoking status. Higher prevalence ratios were found among those did not report use of PPE; those who responded after the capping, with particularly strong associations among responders with the longest deployments (> 60 days); and those who smoked.

117. The first finding can be easily explained: it demonstrates PPE actually works when used (properly) to reduce inhalation of dangerous airborne oil contaminants, as recommended on the Material Safety Data Sheets for Corexit dispersants. Photo

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[105] Marsh D. ICD-10-CM coding for 'vomiting' is simple, except for when it's not. *tci SuperCoder* blog, 11 May **2018**. www.blog.supercoder.com/my-skill-sharpener/icd-10-cm-coding-for-vomiting-is-simple-except-when-its-not/ Visited on 2 Dec 2018.

[106] Alexander M, Engel LS, Olaiya N, et al. The BP DHOS Coast Guard cohort study: A cross-sectional study of acute respiratory health symptoms. *Environ Res.* **2018** Apr, 162:196-202. doi: 10.1016/j.envres.2017.11.044. Epub 2018 Jan 11.

documentation provides evidence to support widespread reports from Gulf coast residents that family and friends who worked BP DHOS response were told by BP safety trainers that, if they wore respirators, their jobs would be terminated (Appendix B, p. 2). Contract workers during the 2010 Enbridge Pipeline tar sands/diluent (dilbit) spill into the Kalamazoo, River, in Michigan were not provided respirators either, while government employees were provided with respirators, as photo-documented (Appendix B, p. 10). Implications of these safety discrepancies are discussed in subsection 4.3.

118. The second finding is not surprising to me or to any Gulf coast resident with family and friends who worked the BP DHOS response. The Coast Guard study (and all other BP DHOS studies) assume that aerial spraying of dispersants began and ended when officially reported: i.e., Corexit 9527A between April 22 and May 22 and Corexit 9500A from April 27 to July 19.[107] This assumption is flat incorrect. I was in Gulf coast communities in Mississippi and Alabama in July before and after the well was capped, and *I personally witnessed more aerial- and boat-spraying of Corexit dispersants in coastal state waters AFTER the well was capped, than before it was capped, and the heavy-duty coastal spraying continued into September.* I believe that this heavy-duty spraying was a misguided multi-state effort to disperse/sink the oil still on the water surface from coming ashore, during the height of the summer tourist season, as most of the aerial spraying was done at night. I was not the only one to witness this. Local residents photo-documented dispersant use of both Corexit 9500A and 9527A well outside the "official" windows of use and in areas where dispersants were not "officially" used, such as at decontamination staging areas (Appendix B, pages 5–6).

119. The third finding – that prevalence ratios were also strongest among non-smokers compared to smokers – can be explained, based on intolerance of non-smokers to high levels of particulate matter and, conversely, tolerance of smokers to those same levels.

120. Finally, the Gulf Long-Term Follow up (GuLF) study also investigated a suite of health symptoms associated with exposure to Corexit dispersants. Unlike the Coast Guard study cohort, the GuLF study cohort is representative of a unique population that is culturally, ethnically, and linguistically diverse, and includes areas with some of the highest rates of poverty and unemployment and the lowest rates of access to health care in the United States.[108] Many individuals in coastal communities suffered psychological stress, depression, and financial and

[107] Steward et al. 2017, GuLF Study job-exposure matrix, FN 14.

[108] Resnik DB, Miller AK, Kwok RK, et al. Ethical issues in environmental health research related to public health emergencies: Reflection on the GuLF Study. *Environ. Health Propect*. **2015** Sep, 123(9):A227-31. doi: 10.1289/ehp.1509889 Epub 1 Sep 2015.

community-level trauma from the BP DHOS disaster, as well as from previous natural and man-made disasters.

121. The GuLF study cohort included 32,608 study participants who completed the BP-contracted safety training program, or other safety training programs as required of oil and gas industry professionals, and who performed oil-spill related work.[109] This represented 23–30% of the total workforce of 110,000 to 140,000. Of these, 7,671 individuals took the training but were not hired. They were enrolled in the study as nonworkers to form a comparison population to reflect the unique social demographics. Of the full cohort (workers plus nonworkers), 82.3% lived in Gulf coast states (Texas, Louisiana, Alabama, Mississippi, and Florida). Enrollment occurred between March 2011 and May 2013. Home visits were completed within 2 months of enrollment on a sub-cohort of 11,193 individuals, most of whom resided in more highly affected counties or parishes along the coast.[110] During home visits, biological and environmental samples, along with other measurements and information, were collected to support investigation of a range of biomarkers to assesses health effects in relation to the oil spill response experiences of this sub-cohort.[111]

122. The GuLF study found that potential exposure to either of the Corexit dispersants used during the BP DHOS response was significantly associated with the now familiar suite of suspected health symptoms from oil spill exposure, including cough wheezing, shortness of breath, skin irritation, burning in nose/throat/lungs, tightness of chest, and burning eyes. The last 3 had the strongest associations. Also, weaker, but still significant, associations were found between dispersant exposure and all outcomes except cough and itching eyes at the time of study enrollment.[112]

123. The findings of the GuLF study are remarkable on three counts: unexplained association of dispersant and symptoms at enrollment; overreporting or misreporting of symptoms; and misclassification of exposures. Each is explained with available evidence.

124. *Unexplained association of dispersants and health symptoms at enrollment*. As described, 82% of the GuLF study cohort was from Gulf coast states. There was considerable overlap between oil and dispersant exposure (91.1%).[113] Multiple studies found significant associations among biomarkers and/or symptoms were

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[109] Kwok et al. 2017, GuLF Study prospective, FN 1.
[110] Id., Kwok et al. 2017, GuLF Study prospective, FN 1.
[111] Engel LS, Kwok RK, Miller AK, et al. The Gulf Long-Term Follow-Up Study (GuLF STUDY): Biospecimen collection at enrollment. *J Toxicol Environ Health A*. **2017**, 80(4):218-229.
[112] McGowan et al. 2017, GuLF Study: Health symptoms associated with Corexit, FN 83.
[113] Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.

maintained for 1 to 6 years post disaster or after the last oil spill response work.[114] The enrollment for the GuLF study occurred between 1- and 3-years post disaster. Therefore, it was actually <u>*to be expected*</u> that Gulf coast residents, especially from or near the coast, would be exhibiting symptoms associated with oil spill exposure at the time of enrollment. *Further, as a corollary, the GuLF study finding provides evidence of widespread, persistent harm from oil spill exposure 1 to 3 years post disaster*.

125. <u>*Over-reporting or misreporting of symptoms*</u>. GuLF study investigators "used self-reported excessive **hair loss** at the time of oil spill response [work], to identify participants who may have over-reported their symptoms because there is no known biological mechanism that would relate dispersant exposure to excessive hair loss." I personally observed and heard numerous reports of excessive hair loss in communities in the four most oil-impacted states. In my opinion, the volume of observations tipped the weight of evidence to factual. Local residents provided photo-documentation of the phenomena in children (Appendix B, p. 4, middle row, left, "Louisiana"). In my opinion, a possible mechanism for excessive hair loss from dispersant exposure could well be that dispersants disperse oil, including the small oil-producing sebaceous glands attached to hair follicles and abundantly present on human scalps. Alternatively, sudden emotional trauma or long-term stress have also been associated with hair loss, due to changes in eating and sleeping patterns that disrupt hair growth cycles and cause shedding. There is currently no known mechanism related to dispersant exposure only because no one has looked for a mechanism. In my opinion, the evidence is there and overwhelming.

126. GuLF study investigators conducted sensitivity analyses excluding exposed workers who reported being exposed **outside the known dates of dispersant use** and exposed workers whose only exposure was reported **handling of dispersants on land and** who also reported active participation in equipment **decontamination activities**.[115] Investigators were concerned that cohort members may have confused dispersants with degreasers and solvents used to decontaminate and clean equipment. As evidenced earlier in this testimony (paragraphs 37–38, 63, 65), both dispersants were, in fact, used outside of the official dates of dispersant use and both were, in fact, used in oil spill decon activities (Appendix B, pp. 5–6). Further, other products were used that were not even on the list of eligible products for use in oil spill response (Appendix B, p. 7).

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[114] Hildur et al. 2015, Follow-up genotoxic study: *Prestige*, FN 87.
Noh et al. 2015, Oxidative stress biomarkers: *Hebei Spirit,* FN 91.
[115] McGowan et al. 2017, GuLF study: Health symptoms associated with Corexit, FN 83.

127. Finally, GuLF study investigators found the "inverse association between skin/clothing contact with dispersant during spill response work and **skin irritation** reported at the time of enrollment was also unexpected and is difficult to explain. Many media reports at the time of the study linked **skin lesions** with work or recreational activities involving contact with water from the Gulf of Mexico. We were unable to account for current recreational contact with the water in this analysis." [116] In my opinion, there is abundant evidence to suggest that Gulf waters remained toxic for years after the BP DHOS disaster. Corexit dispersants were found to enrich the presence of *Vibrio* bacteria in Gulf surface water, CE-oiled beach sands, marine CE-oil snow, and CE-tar balls/mats.[117] Public advisories were posted in Gulf coast states 2- and 3-years post disaster to warn beach-goers to avoid contact with tar balls. Marine oil snow accumulated in sediments. Local residents provided photos of 'the day's catch' in New Orleans in April 2012 – shrimp with blackened gills and tumors related to ongoing oil exposure in bottom sediments (Appendix B, p. 9). NOAA tracked unusual cetacean mortalities for 4 years in the oil-spill impacted coastal region before the dolphins began to recovery (Appendix B, p. 9). Fluorescent light illuminated CE-oil sands at night that otherwise looked white and clean during daylight – beach-goers would not even have been aware of exposures, as shown in Appendix B, p. 9 (top photos). People were encouraged to use the beach – at their own risk, but were not informed of the dangers (Appendix B, p. 3, all photos). Oily debris was disposed in public landfills across the Gulf coast, especially in economically-disadvantages communities, and it seeped into adjacent property, prolonging exposure to oil contaminants (Appendix B, p. 8). Home furniture, clothes, carpet, toys, books, and more absorb and then emit toxic oil contaminants: ExxonMobil purchased at least 37 homes that were not considered safe for human inhabitation (Appendix B, p. 11).[118] Again, in my opinion, there is ample evidence to support findings of CE-oil skin irritation, as well as other oil spill exposure-related symptoms, at the time of enrollment.

128. *Misclassification of exposures*. In my opinion, several of the job-exposures were misclassified. For example, in my opinion and the known toxic properties of oil mists and oil aerosols, all jobs that involved spraying or burning – *in situ* burn teams, decon activities, dispersant spraying, or working near the surface hot zone – were jobs with a very high risk of exposure. Instead, *in situ* burn teams were rated as low risk of exposure and decon workers were classified as a median level exposure (category 4 of 6 with category 6 as land-based, low exposure work.[119] In

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[116] Id., McGowan et al. 2017, GuLF study: Health symptoms associated with Corexit, FN 83.
[117] Doyle et al. 2018, Rapid formation of aggregates, FN 36.
Suja et al. 2017, Role of EPS, FN 34.
[118] Price V. Mayflower oil spill: 5 years later. KARK.com 28 Feb **2018.** www.kark.com/news/local-news/mayflower-oil-spill-five-years-later/999021561 Visited on 2 Dec. 2018.
[119] Steward et al. 2017, GuLF Study job-exposure matrix, FN 14.

the Coast Guard study, decontamination work was classified as high exposure.[120] Misclassification of exposure can lead to erroneous or misleading findings. For example, longitudinal GuLF studies were conducted to assess the relationship between exposure as total hydrocarbons (THC) and lung function in exposed workers 1- and 3-years post disaster. The study found no differences between workers and nonworkers, but workers with high potential exposure to burning oil/gas and decontamination workers had reduced lung function when compared to unexposed workers.[121] These results suggest that the risk category assigned to the decon workers does not reflect the exposure risk.

Cardiac Function

129. Longitudinal GuLF studies were conducted to assess associations between oil spill exposures and nonfatal myocardial infarctions approximately during 2010 and 2-to 3-years post disaster.[122] Exposures of interest included participation in response work; during of response work; highest exposure response job; maximum overall THC (total hydrocarbons) exposure during spill response work; potential work exposure to burning crude oil; having to stop work due to heat; and residential proximity to the oil spill. Among the full GuLF study cohort without history of myocardial infarction prior to the BP DHOS disaster (n = 31,109), spill response work and living in proximity to the oil spill (vs. further away) were suggestively associated with a possible increased risk of nonfatal myocardial infarction. Among oil spill workers (n = 24,006), working > 180 days was associated with nonfatal myocardial infarctions, as was stopping work due to heat. There were suggested associations of maximum THC (≥ 3.00 ppm) and working on decontamination.

130. These findings are supported by studies conducted 1 year after the *Hebei Spirit* oil spill that found a significant association between exposure to the oil spill during response work, distance from initially-impacted coastline, and the risk of metabolic syndrome, a predictor of cardiovascular disease and Type II diabetes, in an exposure-response manner.[123] Remarkable findings in the GuLF study include the association between maximum THC and decon work, which I believe is valid and indicates a misclassification of decon workers job-exposure category, and the stronger association with nonfatal myocardial infarction after the active cleanup period than for the whole study period, which I believe is also valid and indicates

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[120] Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.

[121] Gam KB, Kwok RK, Engel LS, et al. Lung function in oil spill response and clean-up workers 1-3 years after the BP DHOS. *Epidemiology*. **2018** May, 29(3):315-322. doi: 10.1097/EDE.0000000000000808.

[122] Strelitz J, Engel LS, Kwok RK, et al. BP DHOS exposures and nonfatal myocardial infarction in the GuLF study. *Environmental Health* **2018**, 17:69 doi: 10.1186/s12940-018-0408-8

[123] Lee I-J, Jang B-K, Lee J-W, et al. Association between metabolic syndrome and participation in clean-up work at the *Hebei Spirit* oil spill. *Korean J Environ Health Sci.* **2015**, 41:335–348. doi: 10.5668/JEHS.2015.41.5.335

the influence of overwhelming mental health stressors after the spill response drama.

131. Longitudinal GuLF studies were also conducted to assess associations between oil spill exposures and fatal myocardial infarctions during the first 5-years post disaster.[124] Results were similar to findings for nonfatal myocardial infarctions, in that residential proximity to the spill and duration of response work were associated with a 29–43% higher hazard of heart disease events.

132. In summary, the main findings of this subsection are:

- There is a consistent suite of acute respiratory, neurological, dermal, and gastrointestinal symptoms, among others like cardiovascular and genitourinary, associated with oil spill exposures and dispersant exposure.
- Dispersant use increases the prevalence and severity of oil spill exposure-related health symptoms and may contribute to a more rapid onset of symptoms.
- Where aerial spraying of dispersants is conducted, there is considerable overlap of oil and dispersant exposures to the point of being indistinguishable in the field and, hence, in health studies based on field exposures.
- Dispersed oil and chemically-enhanced (CE-) dispersed oil can have synergistic effects; exposure to dispersants alone can initiate or elevate cell death, genomic instability, and malignant transformations.
- Biomarkers can be used to link evidence of past oil exposures to end organ damage, as statistically significance associations were found between oil exposure and proximity to oiled coasts and duration of response work.
- In longitudinal studies with and without biomarkers, often initial health symptoms were found to have persisted and worsened; new health symptoms were found in follow-up visits; and initial damage to organ function (blood, liver, pulmonary, and cardiac) was found to have persisted and worsened.
- The persistence of genotoxic damage in oil spill-exposed individuals suggests elevated risk of blood-related cancers; DNA and RNA-sequencing damage linked with illnesses such as asthma, COPD, cystic fibrosis; and fatal and nonfatal cardiac infarctions.
- Proper use of Personal Protective Equipment, appropriate for the situation, can reduce exposures to oil contaminants, including dispersants.
- Dispersant use continued, at least in state waters, well after federal officials claimed use had stopped, as evidenced by associations between human exposure and data analyzes stratified for dispersant use.

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[124] Strelitz J, Keil AP, Richardson DB, et al. Self-reported myocardial infarction and fatal coronary heart disease among oil spill workers and community members 5 years after Deepwater Horizon. Environ Res. **2018** Sep 22, 168:70–79. doi: 10.1016/j.envres.2018.09.026.

- In some studies, particularly the GuLF study, there was tendency to discount unusual information from cohort members, such as consistently and frequently reported health symptoms that had not yet been reported in the scientific literature or tasks that conflicted with official reports, rather than to find explanations for the field observations.
- Bias from exposure misclassification was evident in one study (GuLF study) when findings were compared with other large-scale studies.

133. In conclusion, even short exposures to oil spill contaminants (days to weeks) can result in persistent life-time adverse health effects. Oil spills do not uniformly affect individuals, as dose plus host (susceptibility) makes the poison.

3.0 Impacts to Public Health from Oil Spills

3.1 Evidence from the BP DHOS Disaster

134. Of the 3 large-scale longitudinal studies initiated in the aftermath of the BP DHOS disaster, one involved only Coast Guard members and two involved study cohorts who were mostly from the oil-impacted area. Of the latter, the GuLF study included mostly workers who were residents of Louisiana, Alabama, Mississippi, and western Florida (and to a lesser extent, Texas), while the Women and Their Children's Health (WaTCH) study involved women and their children who lived in southern Louisiana, including a small number of responders. Despite these differences in demographic characteristics and (presumably) exposure between workers and residents, the findings of all 3 studies were comparable and supported by findings from studies of other oil spills.

135. The WaTCH study cohort was comprised of women who lived in 7 of the most heavily oil-impacted parishes (counties) in Louisiana on the day of the BP DHOS disaster.[125] Six of the 7 parishes were used in the southeast Louisiana study of ambient air concentrations of benzene and PAHs.[126] Only women who completed a baseline telephone questionnaire were eligible (n = 2,852), and of these, most were white (58.3%), some were smokers (20%), a few had worked oil spill response (2.2%). Physical health symptoms were recorded for 13 symptoms, based on a suite of symptoms characteristic of oil spill exposures during the 8-month time period immediately following the BP DHOS and corresponding to the core emission period, as evidenced by air, water, and human blood levels of VOCs and

[125] Peters ES, Rung AL, Bronson MH, et al. The women and their children's Health (WaTCH) study: Methods and design of a prospective cohort study in Louisiana to examine the health effects from the BP oil spill. *BMJ Open.* **2017** Jul 10, 7(7):e014887. doi: 10.1136/bmjopen-2016-014887.

[126] Nance et al. 2016, Ambient air concentrations, FN 5.

PAHs. Oil spill exposures were characterized by 6 questions, including spill impact on financial and recreational resources, resource losses relative to others in the community, physical contact with oil other than work-related, and perceived strength of oil smell.

136. In the WaTCH study, statistically significant associations between health and spill exposure were found for all 13 physical health symptoms with the strongest associations for burning in the nose, throat, or lungs; sore throat; dizziness; and wheezing.[127] Women who had high-economic exposure were significantly more likely to report wheezing; headaches; watery, burning, itchy eyes; and stuffy, itchy, runny nose.

137. These findings are supported by the dangerously high levels of dangerous oil compounds reported in the southeast Louisiana ambient air concentration study;[128] the findings of significant overlap among oil and dispersant exposure;[129] and findings of statistically-significant associations between health symptoms and persistent dispersant/oil exposure 1- to 3- years post disaster.[130]

138. The findings are also supported with photo-documentation of a high public health risk of exposure to oil and dispersant contaminates from inhalation, dermal contact, and ingestion. For example, beaches remained open to the public throughout most of summer 2010 and children played around the spill responders (Appendix B, p. 3). The white sand on the beaches was coated with CE-oil, visible only under fluorescent light; coastal water had CE-dispersed oil and the dispersant component facilitated transfer of oil across the skin, visible only under fluorescent light (Appendix B, p. 4).[131] Often people developed skin rashes and lesions after contact with the Gulf water or sitting on the beach sand. Exposed persons described stinging and burning skin, and the sensations did not go away during or after showers, because, as evidenced by the "blue leg" photo, the CE-oil was already under the skin (photos, p. 4).

139. I spoke at length with Steven Aguinaga of Hazelhurst, Mississippi, who encountered marine CE-oil snow, along with his friend Merrick Vallian, while were swimming off Fort Dalton Beach, Florida, in July 2010 while on vacation with their wives. Aguinaga told *Al Jazeera*, "I swam underwater, then found I had orange slick

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[127] Peres LC, Trapido E, Rung AL, et al. The Deepwater Horizon oil spill and physical health among adult women in southern Louisiana: The Women and Their Children's Health (WaTCH) Study. *Environ Health Perspect.* **2016**, 124(8):1208–13. doi: [10.1289/ehp.1510348]

[128] Nance et al. 2016, Ambient air concentrations, FN 5.

[129] Rusiecki et al. 2018, The BP DHOS Coast Guard cohort study, FN 99.

[130] D'Andrea and Reddy, 2018, Long-term adverse health effects, FN 85. Stewart et al. 2017, GuLF Study job-exposure matrix, p. 9, FN 14.

[131] Id., Kirby 2012, Persistent PAHs, see esp. Figures 6 and 7, p. 16, FN 36.

stuff all over me. At that time, I had no knowledge of what dispersants were, but within a few hours, we [Aguinaga and Villian] were drained of energy and not feeling good. I've been extremely sick ever since." [132] Aguinaga told *Al Jazeera*, "I have terrible chest pain, at times I can't seem to get enough oxygen, and I'm constantly tired with pains all over my body. At times I'm pissing blood, vomiting dark brown stuff, and every pore of my body is dispensing water." Aguinaga's blood contained levels of hydrocarbons in the upper 95th percentile of a nationally representative NHANES study (as discussed in subsection 2.2.3). Aguinaga's friend Merrick Vallian, a physically-fit 33-year-old, fared worse from his encounter with the marine CE-oil snow. Within a month, he was dead.[133]

140. To further increase the risk of exposure for the general public, land-based support for dispersant spraying operations in coastal waters were staged in neighborhood marinas (Appendix B, p. 5). This secondary source of oil contaminants was literally in people's backyards and neighborhoods – and immediately offshore in coastal waters. This created a huge risk of exposure to dangerous levels of dangerous chemicals for local residents.

141. Further, decontamination operations were also staged in residential areas – in neighborhoods and near local public boat ramps. The cleaning solvents and degreasers included Corexit dispersants (Appendix B, p. 5–6). One resident was coated in dispersant mist and aerosols carried by a sea breeze. She received treatment for chemical burns and the skin rash resulting from exposure to Corexit 9527A. Symptoms and exposure-related illnesses have persisted for 8 years, at the time of this writing.

142. The general public was also at risk of exposure to other unauthorized solvents and degreasers that were used in spill response (Appendix B, p. 7). Local residents were also exposed 24/7 to oil contaminants, as were many of the offshore workers remained on their boats for weeks or months at a time. Chronic exposures of long duration can be deadly. Captain RB died of cancer in September 2014 (Appendix B, p. 7).

143. Oil and oily debris were disposed of in public landfills after the oil spill response was declassified from a hazardous waste cleanup (for political expedience as there were few hazardous waste landfills along the Gulf coast). Landowners adjacent to public landfills reported oily substances seeping from the landfill – another source of exposure to oil contaminates.

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[132] Jamail D. Gulf spill sickness wrecking lives. *Huffington Post*, March 9, 2011. www.aljazeera.com/indepth/features/2011/03/201138152955897442.html

[133] Id., Jamail, Gulf spill sickness, 2011, FN 39.

144. Ingestion of contaminated seafood was another potential source of exposure for local residents. Oiled sediment continued to contaminate seafood like shrimp caught and being sold on the market 2 years post disaster (Appendix B, p. 9). Regardless of FDA standards and assurances of seafood safety, eating local oil-impacted seafood involved risk-taking – just like visiting the local oil impacted beaches.

145. Finally, indirect harm to public health and economic wellbeing may also occur as dispersant-initiated red tides like the longest-running bloom now affecting the oil-impacted Gulf coast of western Florida.[134]

3.2 Collaborating Evidence of Human Health Effects from Dolphin Studies

146. In my previous testimony, I discussed acute disease conditions, including lung disease, impaired stress response, serum biochemical abnormalities, low levels of adrenal hormones, and other diseases consistent with crude oil exposure and toxicity, for 32 bottlenose dolphins that were temporarily captured and given health assessments in Barataria Bay, Louisiana, in August 2011, a year after the flow of oil had ceased from the BP DHOS disaster. NEW evidence is now provided on long-term health assessments and population recovery of these same dolphins and comparisons are drawn with human health.

147. Barataria Bay was one of the heaviest oiled coastal regions of the Gulf of Mexico following the BP DHOS disaster. Barataria Bay extends seaward from three of the heavily oil-impacted parishes that were part of the southeast Louisiana ambient air concentration study, discussed in this testimony.[135] The other three parishes in that study are adjacent to the coastal parishes.

148. The bottlenose dolphins found in Barataria Bay before, during, and after the BP DHOS were found to be homebodies, staying mostly within the bay or within 1.75 km of the outer Gulf shores.[136] No Barataria Bay dolphins were tracked or observed more than 14 km beyond their overall home range. This multi-year site fidelity suggests long-term, year-round residency, making this a unique population for long-term health studies.

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[134] Wei-Haas M. Red tide is devastating Florida sea life: Are humans to blame? *NationalGeographic.com* Aug. 8, **2018**. www.nationalgeographic.com/environment/2018/08/news-longest-red-tide-wildlife-deaths-marine-life-toxins/

[135] Nance et al. 2016, Ambient air concentrations, FN 5.

[136] Wells R, Schwacke L, Rowles TK et al. Ranging patterns of common bottlenose dolphins (Tursiops truncatus) in Barataria Bay, Louisiana, following the BP DHOS. *Endangered Species Research*. **2017,** 33.159–180. 10.3354/esr00732.

149. During the initial study, 10 of the 32 dolphins were found to be pregnant with due dates in spring and summer 2012. Only 20% of the pregnant dolphins produced viable calves, compared to a previously reported pregnancy success rate of 83% in a comparable population.[137] Of the pregnant females that did not successfully produce a calf, 57% had been previously diagnosed with moderate to severe lung disease. Also, the estimated annual survival rate of the Barataria Bay dolphins was low (86%) as compared with survival rates of 95.1% and 96.25 from previously studies populations.

150. Survival rates for Barataria Bay dolphins were low and varied between 80 and 0.85 during the first 3 years (2011–2013) after the BP DHOS, as compared to historical averages of about 95% in unoiled bays in Florida and North Carolina.[138] Abundance of Barataria Bay dolphin was unknown prior to the spill, but after the spill it declines from 3,100 to ~1,600 to ~2,400 individuals and remained low until fall 2013/spring 2014. The lowest populations coincided with an Unusual Mortality Event for bottlenose dolphins throughout the oil-impacted Gulf coast region. The highest recorded dolphin strandings on record were in Louisiana from April 2010 to December 2011, and a large number of these were in and around Barataria Bay. The number of dolphin strandings decreased in 2014, and the Unusual Mortality Event officially ended in July 2014. The Barataria Bay dolphin population increased to ~3,100 individuals in summer 2014.

151. Simply counting dead dolphins does not consider long-term health impacts to populations, such as the loss of future reproductive potential from death of reproducing females, or the chronic health effects that continue to compromise survival long after acute effects subside. A model was created to account for lost cetacean years, the difference between baseline trajectories had the injury not occurred and injured population size, summed over the time period, and time to stock recovery to within 95% of baseline.[139] This is the dolphin equivalent to the years lived with disability metric, developed by the South Korean researchers to quantify the diminished quality of life from *Hebei Spirit* oil spill exposures (discussed in my previous testimony). For Barataria Bay dolphins, estimated time to recovery was 39 years ($CI_{95\%}$ 24 to 80 years). The estimated lost cetacean years was substantial: 30,347 ($CI_{95\%}$ 11,511 to 89,746).

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[137] Lane SM et al. Reproductive outcome and survival of common bottlenose dolphins sampled in Barataria Bay, Louisiana, USA, following the BP DHOS. *Proc. R. Soc. B* **2015**, 282:20151944. doi: 10.1098/rspb.2015.1944

[138] McDonald TL, Hornsby FE, Speakman TR, et al. Survival, density, and abundance of common bottlenose dolphins in Barataria Bay following the BP DHOS. *Endangered Species Research*. **2017**, 33:193–209. 10.3354/esr00806.

[139] Schwacke L, Thomas L, Wells RS, et al. Quantifying injury to common bottlenose dolphins from the BP DHOS using an age-, sex- and class-structured population model. *Endangered Species Research*. **2017**, 33:265–279. 10.3354/esr00777.

152. Acute and longitudinal studies for Barataria Bay dolphins and humans show striking similarities among health symptoms, biochemical alterations predictive of long-term diseases, and population-level impacts from the BP DHOS disaster. Cross-boundary collaborative efforts among researchers might prove very productive – as they did during the large-scale ecosystem studies in the aftermath of the *Exxon Valdez* oil spill. Potential harm to reproductive rates, pregnancy success rates, and survival rates of offspring have not been examined yet in humans in the oil-impacted Gulf coast region. This might be a productive avenue of future research.

4.0 Opinion on Health Risk from Oil Spills in Urbanized Areas & Minimizing That Risk

4.1 The Risks of a Spill

153. It seems fairly obvious that increased tanker traffic will increase the risk and likelihood of an oil spill, since calculations of spill risk are based on volume of traffic, among other things. I discussed the risks of a spill in my previous testimonty.

154. NEW evidence is now submitted on potential consequences.

4.2 The Consequences of an Oil Spill

155. On June 15, 2016, the government of Canada authorized use of two Corexit dispersants – Corexit 9500A and Corexit 9580A – by listing them as surface treating agents for oil spill response.[140] Canada listed these dispersants for use before the consequences on the environment and human health were more fully understood, based on the human health and ecosystem studies now emerging on the BP DHOS, and discussed in this testimony.

156. Canadian Merv Fingas, an internationally recognized expert on dispersant use in cold marine waters and formerly with Environment Canada, consistently opposed

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[140] Canadian regulations establishing a list of spill-treating agents (Canada Oil and Gas Operations Act). SOR/2016-108 May 25. *Canadian Gazette.* **2016**. 15(12): June 15, 2016. www.gazette.gc.ca/rp-pr/p2/2016/2016-06-15/html/sor-dors108-eng.html

Genwest Systems, Inc., 2015, Oil spill trajectory model report in Burrard Inlet for the Trans Mountain Expansion Project, prepared for City of Vancouver and others, May. http://vancouver.ca/green-vancouver/neb-evidence-library.aspx

Levelton Consultants, Ltd., 2015, Air Quality Impacts from Simulated Oil Spills in Burrard Inlet & English Bay, An Air Quality Dispersion Modeling Report, prepared for Metro Vancouver. http://twnsacredtrust.ca/wp-content/uploads/2015/05/TWN-Assessment-Appendix-5.pdf

dispersant use in Canadian waters throughout his entire career, based on his research that dispersants simply don't work effectively in cold marine environments and that dispersants have a great potential to do more environmental harm than good. His latest literature reviews synthesize the dispersant literature into 2018.[141, 142]

157. While there are minor obstacles still must be overcome by a company that wishes to actually use dispersants, like approval by the NEB, I imagine that the next step for oil companies that ship oil in Canadian waters would be to seek pre-authorization of these toxic products, as they have successfully done in the United States.

158. Given the increased risk of a large spill in Burrard Inlet, commensurate with projected increased tanker traffic for the Trans Mountain Pipeline Expansion, I offer my opinion of the consequences of a large oil spill in Burrard Inlet, without and with Corexit dispersants as STA. Basically the only NEW comments to my previous assessment are:

- The current understanding of human and ecosystem health impacts is that even short exposures to oil spill contaminants (days to weeks) can result in persistent life-time adverse health effects.
- Therefore, everything would be worse than I previously projected – for oil spill responders, local residents, and wildlife, based on the new understanding of human health impacts from the BP DHOS, the longitudinal studies on human health effects from, primarily, the 2002 *Prestige* oil spill in Spain and the 2007 *Hebei Spirit* oil spill in South Korea, and the longitudinal health assessments on Barataria Bay dolphins.
- The current understanding of Corexit dispersant use on human and ecosystem health impacts is that dispersed oil and chemically-enhanced (CE-) dispersed oil can have synergistic effects and that dispersant use increases the prevalence and severity of oil spill exposure-related health symptoms and may contribute to a more rapid onset of symptoms associated with oil spill exposure.
- Therefore, Corexit dispersants should be banned from use as STA during oil spill response in heavily-urbanized waterways like Burrard Bay.
- Alternatively, if dispersants are used in Burrard Inlet with its surrounding heavily urbanized areas, the consequences would be comparable to what happened – *and what is still happening* – in the Gulf of Mexico coastal region impacted

[141] Fingas M. A review of literature related to human health and oil spill dispersants, 2014–2018, on behalf of Prince William Sound Regional Citizens' Advisory Council, Anchorage, Alaska. PWSRCAC Contract Number 955.18.01. www.pwsrcac.org/programs/environmental-monitoring/dispersants/dispersant-literature-reviews/

[142] Fingas M. A review of literatures related to oil spill dispersants, on behalf of Prince William Sound Regional Citizens' Advisory Council, Anchorage, Alaska. PWSRCAC Contract Number 955.17.03. www.pwsrcac.org/programs/environmental-monitoring/dispersants/dispersant-literature-reviews/

from the BP DHOS disaster. Besides the direct risk from CE-oil exposures, there would risk of exposures from the residential dispersant staging operations and decontamination operations and waste disposal allowances. It really can become unbelievable bad, unbelievable fast, for an unbelievably long time.

4.3 Oil Spill Response Planning and Preparation to Mitigate Harm

159. Nance et al. (2016) offer recommendations based on their Southeast Louisiana ambient air concentration study that are relevant for other areas. They recommend:

- Government agencies involve local officials and the public in discussions about health-based and regulatory air quality levels that should apply during an environmental disaster.
- Discussions could also cover the types of emergency monitoring equipment that would be acceptable so that as much data as possible would be recognized as valid in the context of a disaster.
- Plans should include ways for people to shelter in place safely and identify sensitive populations that may need to be temporarily evacuated.
- Adapting health-based disaster thresholds would facilitate decision-making, enhance public awareness, and reduce potential public health impact during a man-made disaster.[143]

160. To this I will add:

- Independent (of industry and government) regional citizens' advisory councils, modeled after the Prince William Sound Regional Citizens' Advisory Council in Alaska, could be created to minimize risk to public health and wellbeing from oil industry activities.
- Citizen-science projects could be established with local schools and universities participating in and conducting regional air monitoring projects to establish a real-time baseline and provide real-time preventative health announcements and precautions during an oil spill disaster.
- Industrial worker safety training programs for oil spill response contract workers should be required to cover costs of blood BTEX levels tests, provided through the contract worker's health care provider and conducted before the worker is deployed for response duty and upon termination of duties.
- Standards for public health should be used to assess potential occupational health impacts for contract workers, since many are exposed 24/7 to oil contaminants.

[143] Nance et al. 2016, Ambient air concentrations, FN 5.

Appendix A — Curriculum vitae of Riki Ott, PhD

RIKI OTT, PHD

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PROFESSIONAL SUMMARY

Accomplished in civic activism and grassroots engagement, empowering youth and adults with accessible, science- and civics-based trainings to inform decisions and actions towards a healthy democracy and a healthy energy future.

EDUCATION & OTHER PROFESSIONAL QUALIFICATIONS

2015	Make It Safe Coalition	Grace Lee Boggs Pillar Award for work in oil spill-impacted communities to increase science literacy on health impacts
1985	University of Washington	PhD, Marine Toxicology
1980	University of South Carolina	MS, Marine Toxicology
1978	Sea Education Association	Thomas Watson Fellowship, marine oil pollution fates & effects
1976	Colby College	BS, Biology-Geology major

EXPERIENCE (SELECTED)

2017-2018	Organized and presented panels on Toxic Trespass at the Public Interest Environmental Law Conference with Gulf Coast environmental justice leaders
2010-2017	Conducted annual or bi-annual visits to Gulf Coast communities impacted by 2010 BP Deepwater Horizon oil disaster to increase scientific literacy on health impacts among those most affected (local residents)
2015 August	Invited speaker on behalf of Haida Nation Raven Clan at historic potlatch to strip two chiefs of title for supporting Enbridge's Northern Gateway Pipeline project and to restore clan and tribal unity
2015 August	Drafted comments on behalf of Living Oceans Society and other Canadian non-governmental organizations in response to the Canadian rulemaking on proposed regulations establishing a list of spill-treating agents
June 2015	*U.S. EPA Region 6 Environment Justice Training Workshop, Little Rock, AR:* Gave panel on US oil spill response regime & opportunities for citizen engagement
2015 Jan-April	*What's the Plan? When Oil Disasters Hit Your Backyard* (tour & webinars) Science & legal training that empowered nearly 600 ordinary citizens to submit individual comments during EPA rulemaking on National Contingency Plan Subpart J, dispersant use; ALERT submitted 300 pages of technical comments
2014 - present	*Founder & Director of ALERT, a project of Earth Island Institute* Develop environmental justice leadership to build towards a healthy energy future
2012 February	Testified as an expert witness on behalf of the Council of the Haida Nation at National Energy Board Joint Review Panel hearing in Old Massett, Haida Gwaii
2010 May	Invited speaker at First Nation summit in Kitimaat Village (Haisla Nation); birthed into new *Solidarity Clan* & granted Matriarch of High Standing status in Clan