Manufacturer's Statements, Facts, and Findings, and Tables 1–5

Appendix A in: ALERT Project, Earth Island Institute, Government Accountability Project, 2024. Petition requesting EPA to remove the dispersants Corexit 9527A and Corexit 9500A from the product schedule of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) pursuant to 40 CFR § 300.970.

NOTE: Manufacturer's statements were selected from the 2019 Safety Data Sheets (SDSs) for Corexit 9500A1 and Corexit 9527A. 2 Statements from Sections 4 and 11 of the SDSs are identical for each of the Corexit dispersants except where noted. Two TRUE statements (††) are included for discussion. Statements contrary to new or relevant information published after the SDSs (Aug. 30, 2019) are considered outdated.

EXCERPTS OF SAFETY DATA SHEETS FOR COREXIT DISPERSANTS 9500A & 9527A

Section: 4. First Aid Measures

Notes to physician	:	Treat symptomatically.
Most important symptoms and effects, acute and delayed	:	See Section 11 for more detailed information on health effects and symptoms.

Section: 11. Toxicological Information

Potential Health Effects

†9527A – Skin	:	Harmful in contact with skin. (††)
†9527A – Inhalation	:	Health injuries are not known or expected under normal use.
†9500A – Inhalation	:	Harmful if inhaled. (††)
†9500A – Skin	:	Health injuries are not known or expected under normal use.
Chronic Exposure	:	Health injuries are not known or expected under normal use.

Experience with Human Exposure

Skin contact, 9500A	:	No symptoms known or expected.
Skin contact, 9527A	:	No information available.
Inhalation, 9500A	:	No information available.
Inhalation, 9527A	:	No symptoms known or expected.

Toxicity – Product

Respiratory or skin sensitization	:	no data available
Carcinogenicity	:	no data available
Reproductive effects	:	no data available
Teratogenicity	:	no data available

Specific Target Organ Toxicity (STOT) – Repeated or Prolonged Exposure

STOT - repeated exposure	:	no data available

(1) FACTS: Regarding statements on First Aid Measures.

It was known since 2011 (study) that Corexit 9500A was a potent skin sensitizer, and that DOSS, the active ingredient in 9500A, was a moderate skin sensitizer. Since Corexit 9527A also contains DOSS as an active ingredient, this makes 9527A a suspected skin sensitizer as well.

The OSHA HAZCOM standard recommended in 2012 that exposure to respiratory or skin sensitizers should be treated comprehensively, not symptomatically, with a clinical history that includes both medical and occupational history to determine the relationship between the current exposure and the development of hypersensitivity.

FINDING: These First Aid Measures statements are misleading and inaccurate.

(2) FACTS: Regarding statements on Potential Health Effects (Table 1).

It was known since 1991 (study) that, in humans during whole body exposure to *airborne* 2-butoxyethanol, an ingredient in Corexit 9527A, the primary route of uptake into the blood—accounting for 75% of the total uptake—was *absorption across the skin*, not inhalation.

It was known since 2011–2015 (studies) that show these dispersants possess an inherent capacity, as potent surfactants, to render cell membranes fully permeable, across species including humans, thereby rapidly and efficiently facilitating the transfer of dispersant and oil across skin and/or lungs into the bloodstream and across the blood-brain barrier into the brain.

FINDING: The Potential Health Effects statements are incorrect and outdated.

(3) FACTS: Regarding statements on Experience with Human Exposure (Tables 2 and 3).

It was known since 2012–2019 (studies) that Corexit dispersants remained associated with oil and were persistent in harmful concentrations—as fine coatings of sand grains, residual tar balls and weathered materials, and submerged sediments or coarsely aggregated material in coastal waters where people walked, waded, and swam throughout the spill area—during the response and for up to at least 1.5 years after the disaster.

It was known since 2012–2017 (studies) that these weathered oil-dispersant mixtures were readily absorbed across human skin, especially moist or wet skin and that PPE, even when used, was inadequate to protect workers from harm.

It was known since 2013 (testimonials) and 2016 (studies) that extraordinarily high levels of oil contaminants in crude oil and dispersants, associated with end organ damage, were found in the blood of workers and coastal residents during the months of peak oil spill emissions in 2010 and that these levels had returned to background 1–3 years later (studies, 2019), while retaining a signature of formerly high concentrations.

It was known since 2013 (testimonials) and 2017–2022 (studies) that exposure to Corexit dispersants only or as oil-dispersant mixtures via skin contact or inhalation was strongly

associated with acute symptoms of skin and respiratory irritation and neurological harm and chronic conditions of respiratory damage—and that these associations were much stronger than for exposure to oil alone. Also, harm occurred at low levels of exposure, and worsened over time with reoccurring skin rashes, headaches, and persistent coughs, consistent with chemical intolerance from respiratory and/or skin sensitization.

FINDING: The statements on Experience with Human Exposure are incorrect and outdated.

(4) FACTS: Regarding statements on Product Toxicity (Tables 4A to C).

It was known since 2011 (study) that Corexit 9500A is a potent skin sensitizer and that its active ingredient, DOSS, is a moderate skin sensitizer. Since DOSS is an active ingredient in Corexit 9527A, this makes 9527A a suspect skin sensitizer as well. Testimonials (and medical records) from individuals who experienced skin contact with 9527A or oil-dispersants are consistent with exposure to respiratory and skin sensitizers.

It was known since 2013—2020 that both Corexit products are potent carcinogens: Tests on various mammal cells from mice to whales, and humans, found that tests with Corexit 9527A and 9500A with or without oil consistently promoted genotoxicity by damaging DNA and triggering multiple cancer pathways; tests with 9527A generally elicited more pronounced responses than 9500A.

It was known since 2016 that Corexit 9527A is a potent teratogen as it interferes with retinol signaling and neuronal differentiation that are critical to embryo survival, and that the surfactant ingredient DOSS was largely responsible for this harm. Since Corexit 9500A also contains DOSS as an active ingredient, this makes 9500A a suspected teratogen as well.

There is cause for concern that these Corexit products may be reproductive toxins as aerosols were implicated in epidemiology studies on dolphins, responder, and residents from the oil-impacted region that found poor live birth and health outcomes from oil spill (oil, dispersant, and oil-dispersant) exposures in general.

FINDING: The cited Product Toxicity statements are incorrect and outdated.

(5) FACTS: Regarding statements on Specific Target Organ Toxicity (Tables 5A and B).

It was known since 2011–2020 that Corexit dispersants are potent neurotoxins that cause *central nervous system damage*, experienced by responders and residents as brain damage and loss of function, bad headaches, hypersensitivities to odors, light, and sound, fatigue, irregular heartbeat, and *peripheral nervous system damage*, experienced as numbness or tingling in the appendages, blurred or double vision, and nausea.

It was known since 2013–2018 that Corexit dispersants are blood system and respiratory system toxins as oil-dispersant exposure significantly altered blood profiles in ways indicative of cellular level damage associated with (spill-related) benzene damage and liver dysfunction and damage; and further, that progressive deterioration of the respiratory

system occurred over time with development of (among other conditions) chronic reactive airways disease indicative of chemical intolerances.

It was known since 2019–2022 that Corexit dispersants are potent cardiovascular system toxins as oil-dispersant exposure increased risk of severe heart disease (heart attacks) and coronary heart disease after only five and a half years—even in a young, fit population; and, further, symptoms and conditions were generally stronger among workers who reported oil-dispersant exposure (vs. oil alone) and among residents who lived in oil-impacted areas.

FINDING: The statements on Specific Target Organ Toxicity are incorrect and outdated.

Table 1. Potential human health effects

Dispersant-only exposure

- Whole body exposure of male human volunteers to 2-butoxyethanol resulted in uptake rates and concentrations in blood that were 3–4 times higher from dermal absorption than from inhalation; OSHA Sweden affiliated authors cautioned that respirators alone were unlikely to adequately protect workers from 2-butoxyethanol vapors.[1]
- In a 2015 lab study across species, Corexit 9500A altered membrane permeability of respiratory epithelial cells from human and mice lungs and gills of zebrafish and blue crab through inflammation of cell tissue and cleavage of key proteins, leading to cell death (apoptosis).[2]
- In a 2011 lab study with male rats, whole-body inhalation exposure to Corexit 9500A altered the permeability of the blood-brain barrier, allowing toxic chemicals to flood into the brain and disrupting neurotransmitter signaling in the brain in ways that would change the behavior and performance of the affected individual.[3]
- In a 2014 lab study, Corexit 9500A altered intracellular oxidative states and led to mitochondrial dysfunction and apoptosis (cell death) in five different types of mammalian cells including human embryo and adult kidney cells, human glial cells, rat nerve cells from the hippocampus (an area of the brain involved in memory, learning, and emotion), and mice skin cells.[4]

[1] See note 50, Johanson, Boman (OSHA Sweden), 1991, Percutaneous absorption of 2-butoxyethanol, at 792.

[2] Li FJ, Duggal RN, Oliva OM, et al., 2015. Heme oxygenase-1 protects Corexit 9500A-induced respiratory epithelial injury across species. PLOS ONE 10(4):e0122275. https://doi.org/10.1371/journal.pone.0122275

[3] Sriram K, Lin GX, Jefferson AM, Goldsmith WT, Jackson M, Mckinney W, et al. 2011. Neurotoxicity following acute inhalation exposure to the oil dispersant COREXIT EC9500A. *J Toxicol Environ Health A* 74: 1405–1418. https://www.tandfonline.com/doi/full/10.1080/15287394.2011.606796

[4] Zheng M, Ahuja M, Bhattacharya D, Clement TP, Hayworth JS, Dhanasekaran M, 2014. Evaluation of differential cytotoxic effects of the oil spill dispersant Corexit 9500A. *Life Sci* 95: 108–117. https://www.sciencedirect.com/science/article/abs/pii/S0024320513007571

Table 2A. Experience with human exposure: Skin contact
Dispersant-only exposure
• Direct skin contact with Corexit 9527A caused skin corrosion (lesions, rashes, and dermatitis) with scarring, reoccurring itchy rashes, and hair loss.[5]
• Direct skin contact with Corexit 9527A also led to feeling sick and fatigued all the time, memory loss, bad headaches, dizziness, vertigo, bouts of seizures, and blackouts.[6]
• An NIH GuLF study found that exposure from skin/clothing with Corexit 9527A/9500A was significantly associated with skin irritation despite "relevant PPE use" reported by 97% of the participants in the dermal analysis group.[7] There was also a positive but nonsignificant association with excessive hair loss.

[5] See note 2, Lori Bosarge Affidavit, 2012; In: GAP, 2015, Deadly Dispersants Addendum.

[6] Ibid. See notes 129–153 and discussion.*****

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

Table 2B. Experience with human exposure: Skin contact
Oil-dispersant exposure
• Corexit dispersants remain associated with oil in the environment and were found to persist on oiled Gulf of Mexico beaches for about four years[8] or longer for larger tar mats and balls.[9]
• Weathered oil-dispersant material that washed ashore between Waveland, Mississippi, and Cape San Blas, Florida (some 330 miles), was still highly toxic 11 to 19 months (~1 to 1.5 years) after the BP Deepwater Horizon oil disaster.[10] Also from this study:
◦ Dispersants act as a "built-in absorption accelerant," making uptake of oil by skin absorption, especially for wet skin, rapid and highly efficient.
• Crude oil volatile organic compounds and n-hexane were found in the blood of workers, coastal residents, and children during peak emissions of the BP oil disaster at very high levels associated with end organ damage.[11]
• Residual levels of oil contaminants were still evident in blood 1–3 years later, and they still carried the signature of once-high levels of oil components even as the overall levels returned to background.[12]
• Numerous and consistent acute and chronic reports from exposed Gulf coast residents of an intensely itchy rash of small red bumps, later dubbed the "Suicide Itch."[13]

[8] See note 112, White et al., 2014, Long-term persistence of dispersants.

[9] See note 111, Bociu et al., 2019, Decomposition of sediment-oil agglomerates.

[10] See note 109, Kirby, 2012, Persistent PAHs.

[11] Summarco PW, et al., 2016. Concentrations in human blood of petroleum hydrocarbons associated with the BP Deepwater Horizon oil spill, Gulf of Mexico. *Arch Toxicol* 90(4):829-37. doi: 10.1007/s00204-015-1526-5

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

Werder EJ, et al., 2019. Blood BTEX levels and neurologic symptoms in Gulf states residents. *Environ Res* 175:100-107. https://pubmed.ncbi.nlm.nih.gov/31108353/

[13] See note 2, Affidavits of Kindra Arnesen, A.C. Cooper, Jorey Danos, John Gooding, Jamie Griffin, Steve Kolian, Betsey Miller, Michael Robichaux MD, Wilma Subra; In: GAP, 2015, Deadly Dispersant Addendum.

See note 2, GAP, 2020, Ten Years After the Deepwater Horizon, at 11, suicide itch.

Table 3A. Experience with human exposure: Inhalation

Dispersant-only exposure

- An NIH GuLF study found airborne dispersant exposure was significantly associated with adverse respiratory and eye irritation despite PPE use (absent respirators) reported by 48% of the participants in the respiratory analysis group.[14] Other findings indicated presence of a respirator sensitizer:
  - While direct work with dispersants was more strongly associated with symptoms of respiratory and eye irritation than indirect exposure, i.e., working in an area where dispersants were used, indirect exposure was still significantly associated with most of the same symptoms.
  - The associations between dispersant exposure and symptoms of either respiratory or eye irritation remained significant at all work locations from land to offshore, regardless of airborne concentrations of oil exposure.
  - At the time of study enrollment 1–3 years later, dispersant exposure remained significantly associated with the prevalence of most symptoms for respiratory and eye irritation among those who had reported initial symptoms—and among those who had not reported initial symptoms.

[14] See note 191, McGowan et al., 2017, Symptoms associated with Corexit dispersants.

Table 3B. Experience with human exposure: Inhalation

Oil-dispersant exposure

- A USCG study on dispersant exposure via inhalation (Alexander et al.) found that relationships between oil-dispersant exposures and symptoms of coughing, shortness of breath, and wheezing among disaster responders were much greater in magnitude than for oil alone.[15] Other findings indicated presence of a respirator sensitizer:
  - Associations with coughing, shortness of breath, and wheezing were present before *and after* the well was capped (on July 15) when *offshore* dispersant spraying by plane and boats largely stopped, and associations were particularly strong among responders with the longest deployments (>60 days).
  - Oil-dispersant exposures had at least twice the prevalence (number of cases) of coughing and five times the prevalence ratios of shortness of breath and wheezing than exposure to crude oil alone, and the associations generally followed an exposure-response relationship relating to duration of exposure and increased frequency of inhalation or dermal contact.
  - Although skin irritant outcomes were not analyzed in this study, significant trends were found between respiratory symptoms of coughing and shortness of breath and increased frequency of dermal dispersant contact (as duration), suggesting that dermal exposure also contributed to respiratory symptoms and followed an exposure-response relationship.
- A USCG follow up study on dispersant exposure via inhalation (Rusiecki et al.) found that associations between inhalation of crude oil-dispersant vapors and chronic respiratory *conditions* (as diagnosed illnesses) after 5 years[16] were "appreciably greater" than for crude oil-only exposures.

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

[16] Rusiecki J, Denic-Roberts H, Thomas DL, Collen J, Barrett J, Christenbury K, Engel LS. 2022. Incidence of chronic respiratory conditions among oil spill responders: Five years of follow-up in the Deepwater Horizon oil spill Coast Guard cohort study. *Environ Res.* Jan; 203:111824. doi: 10.1016/j.envres.2021.111824.

Table 4A. Product Toxicity: RESPIRATORY OR SKIN SENSITIZATION
Dispersant-only exposure
• In lab studies, dermal exposure of mice to Corexit 9500A and one of its active ingredients, dioctyl sodium sulfosuccinate (DOSS), at working concentrations used during the BP Deepwater Horizon disaster response, induced a Th1-cell-mediated immunological response that led to classification of Corexit 9500A *as a potent sensitizer* and DOSS *as a moderate sensitizer*.[17]
• Direct contact from Corexit 9527A led to chronic reoccurring rashes, impaired memory function and loss, seizures, headaches, blurry vision, chemical sensitivities to smells, and sensitivities to light and sound.[18]
• Direct contact from Corexit dispersants led to chemical induced asthma, chronic reactive airways disease, chronic reoccurring rashes and other chronic skin conditions, and severe reoccurring headaches.[19]

[17] See note 169, Anderson et al., 2011, Immunological effects from Corexit 9500A.

[18] See note 2, Lori Bosarge Affidavit, 2020; In: GAP, 2020, Ten Years After Deepwater Horizon.

[19] See note 2, John Maas Affidavit; In: GAP, 2024, DEEP IMPACT.

Table 4B. Product Toxicity: CARCINOGENICITY
Dispersant-only exposure
• In lab studies with human bronchial epithelial cells, Corexit dispersants 9500A and 9527A triggered enhanced production of reactive oxygen species at the highest test level and significantly higher cell death with more pronounced response in the 9527A tests.[20]
• In lab studies with sperm whale skin cells, Corexit 9500A and 9527A were cytotoxic and genotoxic; 9527A was less cytotoxic but more genotoxic than 9500A.[21]
Oil-dispersant exposure
• In lab studies with human bronchial epithelial cells, oil-dispersant mixtures (whole and water-accommodated fractions) promoted more double- and single-stranded DNA breaks and activation of DNA damage response mechanisms than oil alone; oil-9527A mixtures produced more double-stranded DNA breaks than oil-9500A mixtures.[22]
• In lab studies with human bronchial epithelial cells, an oil-9527A mixture induced a pattern of change towards cancer development by promoting a greater number of RNA transcription errors that blocked various receptors for protein processing and signaling than found in cells after tests with oil-9500A.[23]
• In lab studies with human bronchial epithelial cells, an oil-9527A mixture elicited the most pronounced effects on DNA damage and proliferation by initiating 27 cancer pathways compared to 8 for the oil-9500A mixture; also, oil-9527A functionally shifted the small lung cancer pathway to a smaller set of genes that have even more cancer pathways.[24]
• In lab studies with mice models, exposure to oil-dispersant mixtures promoted more genotoxicity and DNA damage, cell death, inflammation, and tumor formation in the pulmonary system than exposures to oil or dispersant alone; also, tests with Corexit 9527A triggered more cancer pathways than tests with Corexit 9500A (19 vs. 7, respectively).[25]

[20] See note 251, Shi et al., 2013, Corexit dispersants on cytotoxicity in human airway cells.

[21] See note 253, Wise et al., 2014, Corexit dispersants on genotoxicity to sperm whales.

[22] Major D, et al., 2016. Effects of Corexit oil dispersants and the WAF [water-accommodated fraction] of dispersed oil on DNA damage and repair in cultured human bronchial airway cells, BEAS-2B. *Gene Rep* 3:22-30. doi: 10.1016/j.genrep.2015.12.002

[23] Liu YZ, Roy-Engel AM, Baddoo MC, et al., 2016. The impact of oil spill to lung health – Insights from an RNA -seq study of human airway epithelial cells. *Gene* 578(1):38-51. doi: 10.1016/j.gene.2015.12.016

[24] See note 183, Liu et al., 2017, Carcinogenic effects of oil dispersants in humans.

[25] Liu YZ, Miller CA, Zhuang Y, et al., 2020. The Impact of the Deepwater Horizon Oil Spill upon Lung Health-Mouse Model-Based RNA-Seq Analyses. *Int J Environ Res Public Health*. Jul 29;17(15):5466. doi: 10.3390/ijerph17155466

Table 4C. Product Toxicity: TERATOGENICITY AND REPRODUCTIVE EFFECTS
Dispersant-only exposure

• A lab study with mouse P19 embryonal pluripotent cells found Corexit 9527A interferes with retinol signaling and neuronal differentiation that are critical to survival.[26] Three specific mechanisms were identified including:
  • 9527A blocked biosynthesis of retinol acid from retinol by disrupting an enzyme involved in formation of retinol acid needed for differentiation of embryonic stem cells into neurons.
  • 9527A interfered with enzymatic binding (by blocking receptors) of some proteins during the conversion process of retinol, which inhibited production of retinoic acid.
  • 9527A jammed the neuro signaling required for guiding differentiation (by altering membrane permeability), which inhibited neuronal differentiation.
• Further, this study found that the surfactant ingredient DOSS was a major, if not the only, ingredient responsible for the observed adverse effects Corexit 9527A in the mouse P19 cells.
• This study also found that Corexit 9500A was more cytotoxic than Corexit 9527A to mouse P19 embryonal pluripotent cells.

[26] See note 270, Chen, Reese, 2016, Corexit 9527A disrupts mice embryonal pluripotent cells.

Table 5A. Specific Target Organ Toxicity (STOT) – Repeated or Prolonged Exposure

CENTRAL AND PERIPHERAL NERVOUS SYSTEMS

Dispersant-only exposure

- As reported in Table 1, in a 2011 lab study, whole-body inhalation exposure of male rats to Corexit 9500A altered the permeability of the blood-brain barrier, allowing toxic chemicals to flood into the brain and disrupting neurotransmitter signaling in the brain in ways that would change the behavior and performance of the affected individual.[27]
- As reported in Table 4A, direct skin contact with Corexit 9527A led to feeling sick and fatigued all the time, memory loss, bad headaches, blurry vision, dizziness, vertigo, bouts of seizures, blackouts,[28] and hypersensitivities to odors, light, and sound.[29]

Oil-dispersant exposure

- A USCG study that assessed acute neurological symptoms during oil spill response found positive associations and significant trends between increased frequency of crude oil exposure via inhalation or skin contact and increased likelihood of headaches, lightheadedness, difficulty concentrating, numbness/ tingling sensation, blurred vision, and memory loss/ confusion; the highest prevalence ratios occurred for numbness/tingling sensations and blurred/double vision, in particular. Significantly,
  - "Exposure to both oil and oil dispersants yielded associations that were appreciably greater in magnitude than for oil alone for all neurological symptoms."[30]
- An NIH GuLF study that assessed chronic neurological function 4–6 years after the oil spill found modest decreases in neurobehavioral function, especially in sustained attention, memory, executive function, and coding (response speed) associated with both airborne exposures to oil spill vapors and job class.[31] Further,
  - The magnitude of the deficit in one measure (delay in response) was the equivalent of aging 4 to 9 years, and it varied across the job classes with the greatest magnitude for land cleanup workers (9 years).

[27] See note 183, Sriram et al., 2011, Neurotoxicity with acute inhalation exposure, Corexit 9500A.

[28] See note 2, Lori Bosarge Affidavit, 2012; In: GAP, 2013, Deadly Dispersants.

[29] See note 2, Lori Bosarge Affidavit, 2020; In: GAP, 2020, Ten Years After Deepwater Horizon.

[30] Krishnamurthy JK, Engel LS, Wang L, et al., 2019. Neurological symptoms associated with oil spill response exposures: Results from the Deepwater Horizon oil spill Coast Guard cohort study. *Environ Intl.* 163:104963. doi: 10.1016/j.envint.2019.104963.

[31] Quist AJL, Rohlman DS, Kwok RK, et al. 2019. Deepwater Horizon oil spill exposures and neurobehavioral function in GuLF STUDY participants. *Environ Res.* Dec;179(Pt B):108834. doi: 10.1016/j.envres.2019.108834.

Table 5B. STOT – Repeated or Prolonged Exposure

HEMATOLOGICAL, RESPIRATORY AND CARDIOVASCULAR SYSTEMS

Oil-dispersant exposure

- A clinical study[32] and follow up study[33] 7 years after the oil spill assessed prevalence of symptoms, including hematologic and hepatic biomarkers, and pulmonary and cardiac function, in oil spill workers who participated in response activities along the Louisiana coast. These studies found:
  - The most reported symptoms by workers during their initial visits were frequent headaches (77%), shortness of breath (71%), skin rash (59%), chronic cough, dizzy spells, and fatigue (51–49); the incidence of their occurrence was comparable 7 years later.
  - Workers exposed to oil-dispersants had significantly altered blood profiles, significant amounts of phenol in their urine (indicating benzene exposure), and higher levels of three liver enzymes that are biomarkers of hepatic dysfunction and damage, compared to the unexposed group; no improvement was found after 7 years.
  - Most workers had progressive deterioration of their respiratory system over 7 years—91% had developed chronic rhinosinusitis and 45% had chronic reactive airways dysfunction syndrome.
  - During their initial visit, ECGs (electrocardiograms) revealed over half (52%) of the workers experienced some type of cardiac function abnormalities indicative of increased risk of heart failure from cardiovascular diseases, an unexpected finding given the average age (35.8 years); 7 years later, cardiac function abnormalities were slightly decreased (41%).
- A USCG study found increased cardiovascular symptoms (chest pain, arrhythmia or irregular heartbeats) were associated with increased exposures to crude oil and oil-dispersant from direct skin contact and inhalation; symptoms and conditions were generally stronger among workers who reported oil-dispersant exposure (vs. oil or dispersant alone).[34]
- NIH GuLF studies 5-years after the oil spill found increased risk of heart attacks and fatal coronary heart disease were associated with longer duration of response work, residential proximity of the spill,[35] and higher estimated exposure to total hydrocarbons.[36]

---

[32] D'Andrea MA, Reddy GK, 2013. Health consequences among subjects involved in Gulf oil spill clean-up activities. *Amer J Med* 126(11):966–74. doi: 10.1016/j.amjmed.2013.05.014.

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

[34] Denic-Roberts H, Rowley N, Haigney MC, et al., 2022. Acute and longer-term cardiovascular conditions in the Deepwater Horizon oil spill Coast Guard cohort. *Environ Intl.* 158: doi.org/10.1016/j.envint.2021.106937

[35] Strelitz J, Keil AP, Richardson DB, et al. 2019. Self-reported myocardial infarction and fatal coronary heart disease among oil spill workers and community members 5 years after Deepwater Horizon. *Environ Res.* Sep 22, 168:70–79. doi: 10.1016/j.envres.2018.09.026.

[36] Strelitz J, Sandler DP, Keil AP, et al., 2019. Exposure to total hydrocarbons during cleanup of the Deepwater Horizon oil spill and risk of heart attack across 5 years of follow-up. May. *Amer J Epidemiology* 188(5:917–927. https://doi.org/10.1093/aje/kwz017