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Lung function in oil spill response workers 1–3 years after the *Deepwater Horizon* disaster

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Abstract

Background—Little is known about the effects of inhalation exposures on lung function among workers involved in the mitigation of oil spills. Our objective was to determine the relationship between oil spill response work and lung function 1–3 years after the *Deepwater Horizon* (*DWH*) disaster.

Methods—We evaluated spirometry for 7,775 adults living in the Gulf states who either participated in *DWH* response efforts (workers) or received safety training but were not hired (non-workers). At an enrollment interview, we collected detailed work histories including information on potential exposure to dispersants and burning oil/gas. We assessed forced expiratory volume in 1 second (FEV₁; mL), forced vital capacity (FVC; mL), and the ratio (FEV₁/FVC%) for differences by broad job classes and exposure to dispersants or burning oil/gas using multivariable linear and modified Poisson regression.

Results—We found no differences between workers and non-workers. Among workers, we observed a small decrement in FEV₁ (Beta: –71 mL, 95% CI: –127, –14) in decontamination

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The data and computer code are not available for replication because the data are not publicly available. Data may be requested through the GuLF STUDY website via the link for researchers: <https://gulfstudy.nih.gov/en/index.html>.

workers compared to support workers. Workers with high potential exposure to burning oil/gas had reduced lung function compared to unexposed workers: FEV₁ (Beta: -183 mL, 95% confidence interval[CI]: -316, -49) and FEV₁/FVC (Beta: -1.93%, 95% CI: -3.50, -0.36), and an elevated risk of having a FEV₁/FVC in the lowest tertile (prevalence ratio: 1.38, 95% CI: 0.99, 1.92).

Conclusions—While no differences in lung function were found between workers and non-workers, lung function was reduced among decontamination workers and workers with high exposure to burning oil/gas compared to unexposed workers.

Keywords

Pulmonary function test; spirometry; oil spill; disaster

Introduction

On April 20, 2010, an explosion aboard the *Deepwater Horizon (DWH)* oil rig led to the accidental release of approximately 4.9 million barrels of crude oil into the Gulf of Mexico. This resulted in the largest marine oil spill in U.S. history and elicited a large response effort that included and tens of thousands of workers.^{1,2} Response activities included stopping the oil release, burning of crude oil/natural gas, drilling of relief wells, application of chemical dispersants (Corexit EC9527A and EC9500A), clean-up activities on land and water, decontamination of vessels, and administrative and operational support.^{2,3}

Oil spill response workers were exposed to hazardous airborne chemicals from crude oil and from chemicals associated with oil mitigation techniques.^{3,4} Volatile petroleum hydrocarbons, one component of crude oil, have been shown to induce oxidative stress and airway inflammation in cell and animal models and have been associated with reduced lung function in humans.⁵⁻⁸

Little is known about the impact of oil spill response exposures on lung function. Prior studies have reported higher rates of respiratory symptoms among oil spill clean-up workers compared to non-workers, immediately after and up to 5 years following a major oil spill.⁹⁻¹² Fewer studies have investigated respiratory health among these workers using clinical measurements, and findings among such studies are mixed.^{10,13-15} One study identified reductions in lung function among workers compared to non-workers directly following oil spill response work, though these decrements were not observed at 1-year follow-up.¹³ Other studies did not detect differences in lung function, but did observe elevated levels of the airway inflammation marker 8-isoprostane in exhaled breath condensate of workers compared to non-workers.^{10,15} Previous studies of lung function related to oil spill work were small (n=50 – 700) and no study has yet focused on the *DWH* disaster. The objective of our study was to determine the relationship between oil spill response work and lung function 1–3 years following the 2010 *DWH* disaster.

Methods

Study Design and Participants

We used data from the Gulf Long-term Follow-up Study (GuLF STUDY), a large prospective cohort of adults who worked on the oil spill response effort or who received safety training but were not hired.³ Briefly, persons who participated in oil spill response and clean-up work (workers) and others who were not hired (non-workers) enrolled in the study (n=32,608) by completing a telephone interview about oil spill jobs and tasks, demographics, lifestyle, and health. A total of 11,193 enrolled participants out of 25,304 eligible participants who spoke English or Spanish and lived in Alabama, Florida, Louisiana, Mississippi, and eastern Texas completed a home visit between May 2011 and May 2013 that included pulmonary function testing. We restricted our analysis to participants with complete spirometry testing data that met specific quality definitions (see *Pulmonary function*) and complete covariate information (n=7,775).

Ethical Approval

This research was approved by the Institutional Review Board of the National Institute of Environmental Health Sciences. Participants who completed a home visit provided written informed consent.

Oil spill response and clean-up work

We used a structured interview at enrollment to collect information on jobs and tasks performed as a *DWH* response worker including information on potential exposure to dispersants used to break up the oil and to particulate matter from burning crude oil and natural gas. Those who worked at least one day on any job or task related to the oil spill were classified as workers and all others were classified as non-workers. Industrial hygienists grouped jobs and tasks, based on an approximate intensity of exposure reported as level of total hydrocarbons, into one of six broad hierarchical job classes:^{3,16} response (highest exposure), operations, clean-up on water, decontamination, clean-up on land, and support work (lowest exposure). An overview of job/tasks in each job class can be found in the supplement (eAppendix 1). Questionnaire responses were used to identify workers with possible exposure to dispersants.¹⁶ Workers' potential exposure to burning oil/gas was assessed from questions on the task of *in situ* burning on the water's surface or for those working near the oil/gas flaring, the name of the vessel on which they worked and classified into high, medium, low, and no potential exposure to burning oil/gas, based on proximity to the oil/gas flaring and the controlled burns.¹⁶

Pulmonary function

Spirometry was conducted by trained examiners at the home visit using a portable ultrasound transit-time based spirometer (Easy On-PC; NDD Medical Technologies, Chelmsford MA, USA) following 2005 American Thoracic Society guidelines.¹⁷ Participants performed spirometry in a seated position wearing a nose-clip, until achieving either three acceptable maneuvers or a maximum of eight maneuvers. Real-time feedback was provided to examiners and all tests were reviewed for quality control.

Among home visit participants (n=11,193), some did not complete a spirometry test due to refusal (n=110), early home visit termination (n=75), or a technical problem (n=74) and others were not eligible for spirometry due to an American Thoracic Society or study-specific medical exclusion (n=716). For a small number of participants, the reason for missing spirometry test data was not recorded (n=178). Of the 10,040 home visit participants who completed a spirometry test, 10,019 had complete over-read data, and 8,428 had at least two acceptable forced expiratory volume (FEV₁) and two acceptable forced vital capacity (FVC) maneuvers and were eligible for inclusion in our analysis. Acceptability was defined as meeting the 2005 American Thoracic Society within-maneuver criteria.¹⁷ Participants who had an FEV₁ or FVC value that was implausible (n=2) were assigned the next highest value from the remaining curves available. We also excluded one participant with a FEV₁/FVC value that exceeded 1.0.

We analyzed the forced expiratory volume in 1 second (FEV₁; milliliters), forced vital capacity (FVC; milliliters), and the ratio of FEV₁: FVC (FEV₁/FVC%). Using a standard approach, we used the best FEV₁ and best FVC measure regardless of the maneuver from which they originated to derive the FEV₁/FVC ratio.¹⁷ We did not require reproducibility criteria for our primary analytic sample, but assessed the impact of this in a sensitivity analysis with a subsample of workers with full covariate data who met the 2005 American Thoracic Society acceptability and reproducibility criteria (n=5,596).¹⁷

Confounder Assessment

We selected covariates for adjusted models using a minimally sufficient adjustment set determined by a Directed Acyclic Graph¹⁸ (eFigure 1). Demographic, socioeconomic, lifestyle, and health data collected at enrollment included age (years), gender (male, female), race (White, Black, Asian, Other), Hispanic ethnicity (Hispanic, Non-Hispanic), smoking status (heavy current (≥ 20 cigarettes per day), light current (<20 cigarettes per day), former, never), educational attainment (less than high school, high school equivalent, some college, 4 years college or greater), employment at enrollment (employed, looking for work, other), pre-spill physician diagnosis of lung disease (yes, no), and pre-spill physician diagnosis of diabetes (yes, no). Information on previous oil industry experience (yes, no) or if a participant was involved in previous oil spill clean-up (yes, no), was also collected. We defined residential proximity to the spill at enrollment as living in a county directly adjacent to the Gulf of Mexico (direct), living in a county adjacent to a coastal county (indirect), or living in a Gulf state county further from the spill (other Gulf state). Information on secondhand smoke (currently living with at least one person who smokes or not) was collected at the home visit. Examiners measured standing height (inches) and weight (pounds); the mean of three measurements was used in statistical analyses. Analyses combined participants who reported their race as Asian or Other due to the small number of Asians in our sample (n=51 total; n=33 workers). Age at the time of the pulmonary function test was derived from the age at enrollment. Height squared was used based on a previously reported quadratic relationship with lung function.^{19,20} Additionally, more variation in lung function was explained by models including height squared than those with height alone. Body mass index was required to meet the minimal sufficient adjustment set, so weight was included in the model since height squared was already present.

Statistical Analysis

We used multivariable linear and modified Poisson regression to estimate the adjusted relationship between oil spill response and clean-up exposures and lung function. We treated FEV₁, FVC, and FEV₁/FVC as continuous measures and conducted multivariable linear regression to determine mean differences and 95% confidence intervals for lung function measurements by exposure status. We also categorized each of FEV₁, FVC, and FEV₁/FVC into lowest versus highest tertiles and used a modified Poisson regression approach to estimate prevalence ratios (PR) and 95% confidence intervals for having each measurement of lung function in the lowest tertile compared to highest tertiles by exposure status. We conducted all analyses on the entire subsample and then restricted to workers (n=6,294) to address a possible healthy hire bias. We combined low and medium potential exposure to burning oil/gas exposure categories because there were too few in the low category to analyze separately (n=14). Statistical Analysis Software (SAS) version 9.3 was used to conduct analyses (Cary, NC).

Sensitivity Analyses

We repeated analyses restricted to workers who lived in coastal Gulf counties as a subpopulation of interest. To assess the impact of using stricter pulmonary function test quality inclusion criteria, we repeated analyses in a subgroup of 5,596 workers who had a pulmonary function test that met the 2005 American Thoracic Society acceptability and reproducibility criteria. Included participants had three acceptable tests with 150mL difference between the largest and second largest FVC curve and 150mL difference between the largest and second largest FEV₁ curve (n=5,196) or were deemed to have met comparable criteria by a spirometry expert (n=400).¹⁷ We also analyzed a subgroup of workers who worked during the time that oil was being released, before the well was capped on July 15th, 2010. Additionally, we stratified analyses by self-reported respiratory protection equipment use defined as having worn a respirator or mask on at least one job during the oil spill response. We also restricted to non-smokers, and separately, to those without pre-spill lung disease. To account for potential differences in previous occupational exposures we stratified analyses by previous work in the oil industry. To address a potential healthy worker survivor bias, we restricted analyses to workers who worked more than 2 weeks, excluding those who may not have been able to work under the stressful work conditions. We stratified analyses by time since the participant last worked on the oil spill (< 1 year, >1 to 2 years, >2 years) to evaluate whether associations were stronger closer to the time of work. Finally, we fit weighted models using inverse probability for participation weights to generalize results to the full sample of 10,040 (n=8,081 workers) participants who completed a pulmonary function test.^{21,22} Weights were estimated using a logistic model for participation as a function of age, gender, BMI, income, worries a lot about future health, previous oil industry experience, and pre-spill lung disease, race, and exposure to burning oil/gas.

Results

Workers were slightly younger than non-workers (43.5 vs. 46.7 years of age) (Table 1). A higher proportion of workers than non-workers were men (80% vs 69%) and were employed

at the time of enrollment (57% vs. 51%). Fewer workers than non-workers had 4 or more years of higher education (15% vs. 19%) and a slightly higher percentage of workers than non-workers had done previous oil spill clean-up work (8.3% vs. 5.8%). Also, fewer workers than non-workers had a pre-spill lung disease diagnosis (14% vs. 16%) or a pre-spill diabetes diagnosis (5.3% vs. 8.2%). Support workers differed in several aspects from non-workers and from other non-support workers (eAppendix 2). Adjusted lung function measurements by oil spill response and clean-up work exposures are shown in Table 2. FEV₁, FVC, and FEV₁/FVC were slightly better in workers compared to non-workers. After adjustment for potential confounders, no differences in FEV₁, FVC, or FEV₁/FVC were found between workers and non-workers (Table 3). After restricting to workers, adjusted analyses showed slightly lower FEV₁ among decontamination workers compared to support workers (Beta: -71 mL, 95% CI: -127, -14) (Table 4). No other differences were found in lung function measurements by job class compared with support work. Results from the categorical analysis were similar with a suggestive increase in poor lung function (lowest tertile) among decontamination workers (PR: 1.11, 95% CI: 1.00, 1.24) but no other differences in job class were found.

Those who likely had exposure to dispersants had modestly lower FEV₁, FVC, and FEV₁/FVC measurements compared to other workers. We separately assessed the sub-cohort who reported personally using dispersants (in contrast to being on a vessel that used dispersant) and found a suggestive inverse association with FEV₁/FVC (Beta: -0.76%, 95% CI: -1.33, -0.18). No difference in lung function measurements was found across tertiles of dispersant exposures.

Among workers, those who had the highest potential exposure to burning oil/gas had lower lung function measurements compared to workers who did not have exposure to burning oil/gas: FEV₁ (Beta: -183 mL, 95% CI: -316, -49), and FEV₁/FVC (Beta: -1.93%, 95% CI: -3.50, -0.36). Analogous tertile analyses showed increased prevalence ratios for FEV₁ (PR: 1.16, 95% CI: 0.81, 1.64) and FEV₁/FVC (PR: 1.38, 95% CI: 0.99, 1.92) for having a lung function measurement in the lowest tertile with high versus no potential exposure to burning oil/gas.

In an analysis restricted to workers from coastal counties, associations between lung function measurements and job class and dispersants were largely unchanged (except the association with decontamination work which was not seen) (eAppendix 3). However, lung function decrements associated with high potential exposure to burning oil/gas were enhanced (FEV₁ (Beta: -256 mL, 95% CI: -448, -65)). Additionally, the risk of having a FEV₁ measurement (PR: 1.44, 95% CI: 1.01, 2.04), a FVC measurement (PR: 1.52, 95% CI: 1.11, 2.08), and a FEV₁/FVC measurement (PR: 1.57, 95% CI: 1.00, 2.47) in the lowest tertile was elevated among those with high potential exposure to burning oil/gas.

Results were also similar when restricted to those workers whose pulmonary function test quality met the stricter 2005 American Thoracic Society criteria (n=5,596). Point estimates and 95% confidence intervals were similar though slightly attenuated compared to those in our primary analysis (eAppendix 4). Restriction to only those who worked during the period when the oil was being released (n=6,026) had little impact on results (eAppendix 5).

89% of workers provided information on respiratory protective equipment and 15% of workers reported wearing a respirator or mask on at least one job. The association between high exposure potential to burning oil/gas and FEV₁ appeared to be attenuated among workers who reported use of respiratory protective equipment (Beta: -49 mL, 95% CI: -242, 143) compared to those who did not report use of respiratory protective equipment (Beta: -289 mL, 95% CI: -485, -92) (eAppendix 6). This pattern was similar for FVC. In contrast, for workers with high potential exposure to burning oil/gas, those who reported respiratory protective equipment use had a lower FEV₁/FVC average (Beta: -2.47%, 95% CI: -4.75, -0.19) than did those who did not report respiratory protective equipment usage (Beta: -1.33%, 95% CI: -3.64, 0.99).

Inverse associations between high burning oil/gas and FEV₁ (Beta: -223 mL, 95% CI: -409, -37) and FEV₁/FVC (PR: 1.63, 95% CI: 1.16, 2.27) remained after restricting our analysis to never-smoking workers (eAppendix 7). We also observed decrements of all lung function measurements in decontamination workers in this subgroup. Findings from analyses among those without pre-spill lung disease also were substantively unchanged from findings in the primary analytic sample (eAppendix 8).

Associations among those who did not have previous oil industry experience were stronger than associations among those with previous oil industry experience (eAppendix 9). Analyses restricted to those who worked more than 2 weeks (n=5,991) showed similar associations as observed in the primary analysis.

Analyses stratified by time since last day of oil spill response work showed that decrements in lung function measurements associated with high versus no potential exposure to burning oil/gas were generally largest within 1 year of last exposure for FEV₁ (Beta: -420 mL; 95% CI: -830, -10), and FEV₁/FVC (Beta: -5.62%; 95% CI: -10.72%, -0.52) (eAppendix 10). This pattern was not seen in analyses of job class, where decrements for decontamination workers were greater 1–2 years since last day of work. Analyses incorporating inverse probability for participation weights conducted among the larger sample of all participants who completed a home visit spirometry test (n=10,040 overall; n=8,081 workers) were similar in magnitude and direction as those observed among the analytic subsample (n=7,775 overall; n=6,294 workers) (eAppendix 11).

Discussion

We estimated the relationships between oil spill work and lung function measured after the *DWH* disaster in a large cohort of persons who worked on the oil spill and others who were not hired. Overall, we found no differences in lung function between workers and non-workers. Among workers, we observed that decontamination workers had a small decrement in FEV₁ compared to support workers. Most notably, FEV₁, and FEV₁/FVC were reduced in workers with the highest potential exposure to burning oil/gas compared to workers without likely burning oil exposure. Moreover, lung function decrements associated with high potential exposure to burning oil/gas tended to be largest within 1 year since the oil spill and the magnitude of these decrements decreased over time, which may suggest lung recovery following initial exposures. Decontamination work and high exposure to burning oil/gas

remained associated with poorer lung function measurements in analyses among never-smoking workers and workers without pre-spill lung diseases.

Previous studies on oil spill workers and lung function are limited and have inconsistent findings.^{10,13–15} Men performing oil spill work had reduced lung function compared to unexposed controls directly following the *Tasman Spirit* oil spill, but no differences were observed at 1-year follow-up.¹³ No differences in lung function were found among fishermen who engaged in oil spill clean-up activities following the *Prestige* oil spill compared to those who did not though elevated levels of lung injury markers were observed.¹⁰ We analyzed raw lung function measurements to allow for comparison to existing studies of oil spill clean-up workers and to studies of other environmental exposures such as smoking and air pollution^{23–26}. Findings in our study may differ from previous work due to our ability to distinguish between subgroups of workers with differing levels of exposure potential to total hydrocarbons, burning oil/gas, and dispersants or the substantially larger number of study subjects in our study.

The decrement of FEV₁ (–71 mL) among decontamination workers exceeds that observed among smokers.^{23,27} This large decrement may be attributable to the nature of decontamination work following the *DWH* disaster, which required the spraying of cleaning chemicals to contaminated equipment, sometimes in areas with inadequate ventilation; such spraying can generate aerosolized exposures. The relative lung function comparing decontamination workers versus other workers was lowest among those examined between 1–2 years following the spill. This pattern might be explained by differences in timing of exposure; decontamination workers experienced highest/more frequent exposures, towards the end of the clean-up period.

Workers with high exposure to burning oil/gas had the greatest difference in lung function observed in our study compared to unexposed workers (–183 mL in FEV₁; –125 mL in FVC), a difference that greatly exceeds expected annual decrement differences among smokers compared to non-smokers.^{23,27} This association between high potential exposure to burning oil/gas and reduced lung function is consistent with existing literature on lower lung function linked with exposure to particulate matter found in cigarette smoke and air pollution.^{28–30} It is also consistent with literature on lung inflammation observed in mice following exposure to particulate matter sampled from the *DWH* crude oil burn-off.²⁹

The associations between high burning oil/gas exposure and lung function measurements were attenuated for FEV₁ and FVC (but not for FEV₁/FVC) among those who reported wearing respiratory protective equipment. Respiratory protective equipment use was not required as a standard procedure for any task identified in the personal protective equipment matrices during the *DWH* response. Respiratory protective equipment was used as a general indicator of respiratory protection, and use must be interpreted with caution due to lack of details on the usage. Further, it cannot be determined whether workers were wearing respirators because of pre-existing symptoms or because of true or perceived high exposures. Respiratory protective equipment use might be underestimated in our study because an early version of our questionnaire had a more restrictive definition that asked specifically about employee required respiratory protective equipment use.

Our study has several limitations. We assessed a single measurement of lung function 1–3 years following the *DWH* oil spill, which may not have captured acute transient effects. Further, without a measure of lung function prior to the spill, it is difficult to be certain that observed deficits in lung function were due to oil spill exposures. If individuals with poor lung function prior to the spill were less likely to have higher exposures, effects could be biased toward the null. Participants within an exposure group may have experienced variation in oil spill chemical exposures since these analyses did not account for exposure duration or account for multiple jobs/tasks. Unmeasured confounders, such as residential or occupational exposures, could also bias effect estimates, although there is little reason to suspect that such exposures other than previous oil industry or oil spill work would be correlated with exposures during the *DWH* oil spill response and clean-up. Future use of time-weighted exposure metrics currently being developed may help us to better describe risk of adverse lung function associated with unit-changes in exposure to individual oil spill chemicals.

Recall could also be a potential source of bias in our study as jobs were ascertained at an enrollment interview between 1–3 years after the oil spill. However, we did not see any evidence of a correlation between job class and time of enrollment and expect any recall bias to be minimally influential because classifications were based on self-reported job/tasks performed by participants that were combined and ranked using objective air monitoring data that was collected at the time of the spill.

We excluded non-workers *a priori* to address a potential healthy hire bias based on a higher prevalence of pre-spill lung disease and diabetes among non-workers compared to workers. Support workers were considered a potentially more appropriate referent group since they had similar prevalence of prior disease compared to other workers, and because they also may have experienced other response-related exposures that could have impacted lung function.

There could still be a healthy hire bias even among workers. Workers in the most physically demanding jobs were likely to have had some of the highest exposures, yet may have been healthier at the outset than support workers, which would lead to potential underestimation of effects. However, prevalence of prior chronic diseases did not differ by job class. Also, support workers had higher education and lower rates of smoking than non-support workers and these factors were accounted for in all analyses.

Workers performing response and operations jobs that required technical skills included those with prior training and professional experience. These workers had possible previous exposure to oil. In analyses stratified on previous oil industry experience, associations were stronger among those without previous oil industry experience, consistent with potential healthy worker survivor bias. However, results were similar in an analysis restricted to those who worked more than 2 weeks.

This is the largest study to examine lung function in oil spill workers, allowing for more power to detect smaller differences compared to previous studies. Exposure metrics in our study may aid in a more direct interpretation of risks and are better characterized than

previous studies. Our exposure classification roughly accounts for total hydrocarbons measured during the time of the oil response and considers unique exposures such as burning oil, which improves upon previous studies. Detailed questionnaire data allowed for adjustments and restrictions to control for potential confounding not found in other studies.

To our knowledge, this study is the first to examine lung function among *DWH* oil spill response workers and the largest to examine lung health in individuals working in remediation efforts following a major oil spill. Results indicate possible elevated risks of lower lung function associated with decontamination work and high potential for exposure to burning crude oil/gas. Longitudinal assessment of lung function is needed to further characterize the risk of adverse pulmonary health outcomes attributed to the oil spill response work.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. National Commission on the BP Deepwater Horizon Oil Spill and Offshore Drilling. Deep Water: The Gulf Oil Disaster and the Future of Offshore Drilling, Report to the President. Washington, DC: 2011.
2. United States Coast Guard. On Scene Coordinator Report Deepwater Horizon Oil Spill: Submitted to the National Response Team. 2011
3. Kwok RKEL, Miller AK, Blair A, Curry MD, Jackson WB, Stewart PA, Stenzel MR, Birnbaum LS, Sandler DP. The GuLF STUDY: A Prospective Study of Persons Involved in the *Deepwater Horizon* Oil Spill Response and Clean-up. *Environmental Health Perspectives*. 2016; 125(4):570–578.
4. Goldstein BD, Osofsky HJ, Lichtveld MY. The Gulf Oil Spill. *New England Journal of Medicine*. 2011; 364(14):1334–48. [PubMed: 21470011]
5. ATSDR. Toxicological Profile for Total Petroleum Hydrocarbons (PB/99/163370). US Department of Health and Human Services. 1999
6. Wang F, Li C, Liu W, Jin Y. Effect of exposure to volatile organic compounds (VOCs) on airway inflammatory response in mice. *Journal of Toxicological Sciences*. 2012; 37(4):739–48. [PubMed: 22863854]
7. Mogel I, Baumann S, Bohme A, Kohajda T, von Bergen M, Simon JC, Lehmann I. The aromatic volatile organic compounds toluene, benzene and styrene induce COX-2 and prostaglandins in human lung epithelial cells via oxidative stress and p38 MAPK activation. *Toxicology*. 2011; 289(1):28–37. [PubMed: 21801798]
8. Cakmak S, Dales RE, Liu L, Kauri LM, Lemieux CL, Hebborn C, Zhu J. Residential exposure to volatile organic compounds and lung function: Results from a population-based cross-sectional survey. *Environmental Pollution*. 2014; 194:145–151. [PubMed: 25108490]

9. Zock JP, Rodriguez-Trigo G, Pozo-Rodriguez F, Barbera JA, Bouso L, Torralba Y, Anto JM, Gomez FP, Fuster C, Vereá H. Prolonged respiratory symptoms in clean-up workers of the prestige oil spill. *American Journal of Respiratory and Critical Care Medicine*. 2007; 176(6):610–6. [PubMed: 17556713]
10. Rodriguez-Trigo G, Zock JP, Pozo-Rodriguez F, Gomez FP, Monyarch G, Bouso L, Coll MD, Vereá H, Anto JM, Fuster C, Barbera JA. Health changes in fishermen 2 years after clean-up of the Prestige oil spill. *Annals of Internal Medicine*. 2010; 153(8):489–98. [PubMed: 20733177]
11. Zock JP, Rodriguez-Trigo G, Rodriguez-Rodriguez E, Espinosa A, Pozo-Rodriguez F, Gomez F, Fuster C, Castano-Vinyals G, Anto JM, Barbera JA. Persistent respiratory symptoms in clean-up workers 5 years after the Prestige oil spill. *Occupational and Environmental Medicine*. 2012; 69(7):508–13. [PubMed: 22539655]
12. Meo S, Al-Drees A, Rasheed S, Meo I, Al-Saadi M, Ghani H, Alkandari J. Health complaints among subjects involved in oil cleanup operations during oil spillage from a Greek tanker "Tasman Spirit". *International Journal of Occupational Medicine and Environmental Health*. 2009; 22(2): 143–148. [PubMed: 19546094]
13. Meo SA, Al-Drees AM, Meo IM, Al-Saadi MM, Azeem MA. Lung function in subjects exposed to crude oil spill into sea water. *Marine Pollution Bulletin*. 2008; 56(1):88–94. [PubMed: 18031764]
14. Meo SA, Al-Drees AM, Rasheed S, Meo IM, Khan MM, Al-Saadi MM, Alkandari JR. Effect of duration of exposure to polluted air environment on lung function in subjects exposed to crude oil spill into sea water. *International Journal of Occupational Medicine and Environmental Health*. 2009; 22(1):35–41. [PubMed: 19351614]
15. Zock JP, Rodriguez-Trigo G, Rodriguez-Rodriguez E, Souto-Alonso A, Espinosa A, Pozo-Rodriguez F, Gomez FP, Fuster C, Castano-Vinyals G, Anto JM, Barbera JA. Evaluation of the persistence of functional and biological respiratory health effects in clean-up workers 6 years after the prestige oil spill. *Environment International*. 2014; 62:72–7. [PubMed: 24184661]
16. Stewart P, Stenzel Mark R, Ramachandran Gurumurthy, Banerhee Sudipto, Huynh Tran, Groth Caroline, Kwok Richard K, Blair Aaron, Engel Lawrence S, Sandler Dale P. Development of an Ordinal Job-Exposure Matrix for Workers Responding to the Deepwater Horizon Disaster: The GuLF STUDY.
17. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CPM, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas D, Pedersen OF, Pellegrino R, Viegi G, Wanger J. Standardisation of spirometry. *European Respiratory Journal*. 2005; 26(2):319–338. [PubMed: 16055882]
18. Greenland S, Pearl J, Robins JM. Causal Diagrams for Epidemiologic Research. *Epidemiology*. 1999; 10(1):37–48. [PubMed: 9888278]
19. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. *American Journal of Respiratory and Critical Care Medicine*. 1999; 159(1):179–87. [PubMed: 9872837]
20. Quanjer PH, Stanojevic S, Cole TJ, Baur X, Hall GL, Culver BH, Enright PL, Hankinson JL, Ip MS, Zheng J, Stocks J. Multi-ethnic reference values for spirometry for the 3–95-yr age range: the global lung function 2012 equations. *Eur Respir J*. 2012; 40(6):1324–43. [PubMed: 22743675]
21. Hernan, MA., Robins, JM. Causal inference. CRC; Boca Raton, FL; 2010.
22. Cole SR, Hernan MA. Constructing inverse probability weights for marginal structural models. *Am J Epidemiol*. 2008; 168(6):656–64. [PubMed: 18682488]
23. Xu X, Dockery DW, Ware JH, Speizer FE, Ferris BG Jr. Effects of cigarette smoking on rate of loss of pulmonary function in adults: a longitudinal assessment. *Am Rev Respir Dis*. 1992; 146(5 Pt 1): 1345–8. [PubMed: 1443894]
24. Ackermann-Lieblich U, Leuenberger P, Schwartz J, Schindler C, Monn C, Bolognini G, Bongard JP, Brändli O, Domenighetti G, Elsasser S, Grize L, Karrer W, Keller R, Keller-Wossidlo H, Künzli N, Martin BW, Medici TC, Perruchoud AP, Schöni MH, Tschopp JM, Villiger B, Wüthrich B, Zellweger JP, Zemp E. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. *American Journal of Respiratory and Critical Care Medicine*. 1997; 155(1):122–129. [PubMed: 9001300]

25. Laffon B, Pasaro E, Valdiglesias V. Effects of exposure to oil spills on human health: Updated review. *J Toxicol Environ Health B Crit Rev*. 2016; 19(3–4):105–28. [PubMed: 27221976]
26. Aguilera F, Méndez J, Pásaro E, Laffon B. Review on the effects of exposure to spilled oils on human health. *Journal of Applied Toxicology*. 2010; 30(4):291–301. [PubMed: 20499335]
27. Kerstjens HA, Rijcken B, Schouten JP, Postma DS. Decline of FEV1 by age and smoking status: facts, figures, and fallacies. *Thorax*. 1997; 52(9):820–827. [PubMed: 9371217]
28. Paulin L, Hansel N. Particulate air pollution and impaired lung function. *F1000Research*. 2016; 5:F1000. Faculty Rev-201.
29. Jaligama S, Chen Z, Saravia J, Yadav N, Lomnicki SM, Dugas TR, Cormier SA. Exposure to Deepwater Horizon Crude Oil Burnoff Particulate Matter Induces Pulmonary Inflammation and Alters Adaptive Immune Response. *Environ Sci Technol*. 2015; 49(14):8769–76. [PubMed: 26115348]
30. Dockery DW, Pope CA. Acute respiratory effects of particulate air pollution. *Annual review of public health*. 1994; 15(1):107–132.

Table 1

Characteristics of GuLF STUDY home visit participants with acceptable pulmonary function test quality by work status (n=7,775)

Characteristics	Workers (n=6,294)	Non-workers (n=1,481)
	Mean (SD)	Mean (SD)
Age, years	43.5 (12.7)	46.7 (13.1)
Height, inches	68.4 (3.6)	67.8 (3.8)
Weight, lbs	199.9 (47.2)	193.0 (47.7)
	n (%)	n (%)
Gender		
Female	1281 (20)	460 (31)
Male	5013 (80)	1021 (69)
Race		
White	3566 (57)	828 (56)
Black	2094 (33)	468 (32)
Other	634 (10)	185 (12)
Ethnicity		
Hispanic	395 (6.3)	102 (6.9)
Non-Hispanic	5899 (94)	1379 (93)
Smoking status		
Heavy current smoker	773 (12)	170 (11)
Light current smoker	1540 (24)	322 (22)
Former smoker	1292 (21)	361 (24)
Never smoker	2689 (43)	628 (42)
Secondhand smoke		
Yes	1838 (29)	431 (29)
No	4456 (71)	1050 (71)
Education		
Less than high school/equivalent	1214 (19)	297 (20)
High school diploma/GED	2141 (34)	461 (31)
Some college/2-year degree	1988 (32)	436 (29)
4-year college graduate or more	951 (15)	287 (19)
Employment		
Working now	3576 (57)	749 (51)
Looking for work or unemployed	1657 (26)	362 (24)
Other	1061 (17)	370 (25)
Residential county proximity to Gulf of Mexico ^a		
Direct	4554 (72)	1044 (70)
Indirect	443 (7.0)	132 (8.9)
Other Gulf state residence	1297 (21)	305 (21)
Previous oil spill clean-up work		

Characteristics	Workers (n=6,294)	Non-workers (n=1,481)
	Mean (SD)	Mean (SD)
Yes	522 (8.3)	86 (5.8)
No	5772 (92)	1395 (94)
Previous oil industry experience		
Yes	1075 (17)	237 (16)
No	5219 (83)	1244 (84)
Reported pre-spill lung disease diagnosis		
Yes	855 (14)	240 (16)
No	5439 (86)	1241 (84)
Reported pre-spill diabetes diagnosis		
Yes	331 (5.3)	121 (8.2)
No	5963 (95)	1360 (92)

Abbreviations: GuLF STUDY, Gulf Long-Term Follow-up Study; SD, standard deviation; GED, General Equivalency Diploma

^aDirect proximity is defined as living in a county directly adjacent to the Gulf of Mexico; indirect is defined as living in a county adjacent to coastal counties; other Gulf state residence is defined as living in a Gulf state further from the spill.

Table 2

Lung function measurements in GuLF STUDY home visit participants by oil spill response and clean-up exposures (n=7,775)

		FEV ₁ , mL	FVC, mL	FEV ₁ /FVC%
	n	Mean (SE) ^a	Mean (SE) ^a	Mean (SE) ^a
Non-worker	1481	3007 (26)	3803 (30)	79.17 (0.31)
Worker	6294	3020 (23)	3814 (27)	79.26 (0.28)
Job class among workers ^b				
Support	606	3110 (36)	3872 (41)	80.27 (0.42)
Land Clean-up	1029	3039 (31)	3840 (36)	79.29 (0.37)
Decontamination	1248	2991 (31)	3788 (36)	79.04 (0.37)
Water Clean-up	920	3073 (33)	3864 (38)	79.65 (0.39)
Operations	1298	3032 (31)	3846 (36)	79.01 (0.37)
Response	1193	3063 (31)	3858 (36)	79.41 (0.37)
Potential exposure to dispersant among workers ^c				
No	5242	3010 (23)	3801 (27)	79.25 (0.28)
Yes	790	2992 (31)	3786 (36)	79.06 (0.37)
Potential exposure to burning oil/gas among workers ^d				
None	5609	3049 (29)	3848 (34)	79.38 (0.34)
Low/Medium	507	3058 (38)	3858 (44)	79.30 (0.45)
High	68	2881 (75)	3728 (87)	77.74 (0.89)

Abbreviations: GuLF STUDY, Gulf Long-Term Follow-up Study; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; mL, milliliters; SE, standard error

^aAdjusted for age, height, height², race, gender, and ethnicity

^bJob class was hierarchical, starting with the highest class first (response)

^cn=262 missing due to unknown dispersant status

^dn=110 missing due to unknown burning oil/gas status

Table 3

Oil spill response and clean-up work status and lung function among GuLF STUDY home visit participants (n=7,775)

	Linear Regression		Poisson Regression	
	n	Beta (95% CI) ^a	n ^c	PR (95% CI) ^{a,b}
Non-worker	1481	Ref	621	Ref
FEV ₁ , mL	6294	8 (-24, 40)	1964	0.96 (0.90, 1.02)
Non-worker	1481	Ref	620	Ref
FVC, mL	6294	11 (-26, 48)	1969	0.96 (0.90, 1.02)
Non-worker	1481	Ref	523	Ref
FEV ₁ /FVC%	6294	-0.03 (-0.42, 0.35)	2069	1.00 (0.92, 1.07)

Abbreviations: GuLF STUDY, Gulf Long-Term Follow-up Study; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; mL, milliliters; CI, confidence interval; PR, prevalence ratio

^a Adjusted for: age, height, height², weight, gender, ethnicity, race, pre-spill diabetes, pre-spill lung disease, education, employment, previous oil industry experience, previous oil spill clean-up work, residential proximity to the Gulf of Mexico, smoking, secondhand smoke

^b Prevalence ratio of having a lung function measurement in the lowest vs. highest tertiles

^c Indicates number of those in the specified exposure group who have lung function in the lowest tertile

Table 4
Oil spill response and clean-up work and lung function among workers (n=6,294)

Job Class	Linear Regression			Poisson Regression		
	n	Beta (95% CI) ^a	n ^c	n	PR (95% CI) ^{a,b}	
FEV ₁ , mL	Support	606	Ref	230	Ref	
	Land	1029	-22 (-79, 36)	417	1.02 (0.91, 1.13)	
	Decon	1248	-71 (-127, -14)	522	1.11 (1.00, 1.24)	
	Water	920	11 (-47, 70)	268	0.98 (0.86, 1.11)	
	Operations	1298	-21 (-78, 35)	354	1.05 (0.93, 1.19)	
	Response	1193	0.1 (-56, 57)	307	0.98 (0.87, 1.11)	
FVC, mL	Support	606	Ref	237	Ref	
	Land	1029	-4 (-71, 63)	428	0.99 (0.89, 1.11)	
	Decon	1248	-60 (-126, 6)	538	1.08 (0.97, 1.20)	
	Water	920	22 (-47, 90)	255	0.95 (0.84, 1.08)	
	Operations	1298	8 (-58, 73)	330	0.98 (0.87, 1.11)	
	Response	1193	16 (-49, 82)	309	0.97 (0.86, 1.10)	
FEV ₁ /FVC%	Support	606	Ref	190	Ref	
	Land	1029	-0.26 (-0.94, 0.42)	340	1.04 (0.90, 1.20)	
	Decon	1248	-0.43 (-1.09, 0.24)	394	1.04 (0.90, 1.20)	
	Water	920	0.08 (-0.62, 0.77)	337	1.01 (0.88, 1.16)	
	Operations	1298	-0.45 (-1.11, 0.22)	458	1.08 (0.94, 1.24)	
	Response	1193	-0.21 (-0.88, 0.45)	379	1.02 (0.88, 1.17)	
Potential exposure to dispersant ^d						
FEV ₁ , mL	No	5242	Ref	1777	Ref	
	Yes	790	-6 (-48, 37)	238	0.99 (0.90, 1.10)	
FVC, mL	No	5242	Ref	1762	Ref	
	Yes	790	-4 (-53, 46)	260	1.07 (0.97, 1.17)	
FEV ₁ /FVC%	No	5242	Ref	1766	Ref	

	Linear Regression			Poisson Regression		
	n	Beta (95% CI) ^a	n ^c	PR (95% CI) ^{a,b}	n ^c	PR (95% CI) ^{a,b}
Yes	790	-0.09 (-0.60, 0.41)	229	0.95 (0.85, 1.06)		
Potential exposure to burning oil/gas ^e						
FEV ₁ , mL	5609	Ref	1909	Ref		
None						
Low/medium	507	8 (-43, 59)	139	0.98 (0.86, 1.11)		
High	68	-183 (-316, -49)	20	1.16 (0.81, 1.64)		
FVC, mL	5609	Ref	1908	Ref		
None						
Low/medium	507	14 (-45, 73)	141	0.99 (0.87, 1.12)		
High	68	-125 (-280, 31)	19	1.04 (0.74, 1.48)		
FEV ₁ /FVC%	5609	Ref	1883	Ref		
None						
Low/medium	507	-0.18 (-0.78, 0.42)	159	1.02 (0.90, 1.16)		
High	68	-1.93 (-3.50, -0.36)	25	1.38 (0.99, 1.92)		

Abbreviations: FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; mL, milliliters; CI, confidence interval; PR, prevalence ratio; Decon, decontamination; Land, land clean-up; Water, water clean-up

^a Adjusted for: age, height, height², weight, gender, ethnicity, race, pre-spill diabetes, pre-spill lung disease, education, employment, previous oil industry experience, previous oil spill clean-up work, residential proximity to the Gulf of Mexico, smoking, secondhand smoke

^b Prevalence ratio of having a lung function measurement in the lowest vs. highest tertiles

^c Indicates number of those in the specified exposure group who have lung function in the lowest tertile

^d n=262 missing due to unknown dispersant status

^e n=110 missing due to unknown burning oil/gas status