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Fine Particulate Matter and Lung Function among Burning-Exposed *Deepwater Horizon* Oil Spill Workers

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Background: During the 2010 *Deepwater Horizon* (*DWH*) disaster, controlled burning was conducted to remove oil from the water. Workers near combustion sites were potentially exposed to increased fine particulate matter [with aerodynamic diameter ≤2.5 μm ($PM_{2.5}$)] levels. Exposure to $PM_{2.5}$ has been linked to decreased lung function, but to our knowledge, no study has examined exposure encountered in an oil spill cleanup.

Objective: We investigated the association between estimated $PM_{2.5}$ only from burning/flaring of oil/gas and lung function measured 1–3 y after the *DWH* disaster.

Methods: We included workers who participated in response and cleanup activities on the water during the *DWH* disaster and had lung function measured at a subsequent home visit ($n = 2{,}316$). $PM_{2.5}$ concentrations were estimated using a Gaussian plume dispersion model and linked to work histories via a job-exposure matrix. We evaluated forced expiratory volume in 1 s (FEV1; milliliters), forced vital capacity (FVC; milliliters), and their ratio (FEV1/FVC; %) in relation to average and cumulative daily maximum exposures using multivariable linear regressions.

Results: We observed significant exposure–response trends associating higher cumulative daily maximum $PM_{2.5}$ exposure with lower FEV1 (p-trend = 0.04) and FEV1/FVC (p-trend = 0.01). In comparison with the referent group (workers not involved in or near the burning), those with higher cumulative exposures had lower FEV1 [−166.8 mL, 95% confidence interval (CI): −337.3, 3.7] and FEV1/FVC (−1.7, 95% CI: −3.6, 0.2). We also saw nonsignificant reductions in FVC (high vs. referent: −120.9, 95% CI: −319.4, 77.6; p-trend = 0.36). Similar associations were seen for average daily maximum $PM_{2.5}$ exposure. Inverse associations were also observed in analyses stratified by smoking and time from exposure to spirometry and when we restricted to workers without prespill lung disease.

Conclusions: Among oil spill workers, exposure to $PM_{2.5}$ specifically from controlled burning of oil/gas was associated with significantly lower FEV1 and FEV1/FVC when compared with workers not involved in burning. https://doi.org/10.1289/EHP8930

Introduction

The 2010 *Deepwater Horizon* (*DWH*) disaster was the largest marine oil spill in U.S. history (National Commission on the BP Deepwater Horizon Oil Spill and Offshore Drilling 2011). An estimated 4.9 million barrels of crude oil were discharged into the Gulf of Mexico until the wellhead was mechanically capped on 15 July 2010 (National Commission on the BP Deepwater Horizon Oil Spill and Offshore Drilling 2011). The spill also saw one of the largest oil spill response and cleanup (OSRC) operations in maritime history (Kwok et al. 2017).

To remove oil from the ocean surface, controlled burning was used as a spill remediation method in addition to other mechanical means of removing the oil (U.S. Coast Guard 2011). Two controlled burning activities took place: *a*) flaring of oil/natural gas, and *b*) *in situ* burning of oil on the water surface (U.S. Coast Guard 2011). Between 17 May 2010 and 16 July 2010, two drilling rigs (the *Discoverer Enterprise* and the *Helix Q4000*) and a production/offloading vessel (the *Helix Producer I*) flared oil/gas at the wellhead (U.S. Coast Guard 2011). The *Discoverer Enterprise*, capable of separating natural gas from the captured oil, processed ∼18,000 barrels of oil per day and flared the separated gas (U.S. Coast Guard 2011) from 17 May to 25 May 2010 and from 5 June to 11 July 2010. The other two vessels joined the effort later. The *Helix Q4000* flared ∼10,000 barrels of combined oil and gas per day between 17 June and 16 July 2010, and the *Helix Producer I* flared ∼25,000 barrels of the oil/gas mixture per day from 13 July to 16 July 2010 (U.S. Coast Guard 2011). The spill also saw the largest *in situ* burn (ISB) operation in US history (Allen et al. 2011). From 28 April 2010 to 19 July 2010, workers in the ISB Group attempted 411 burns offshore and removed nearly 300,000 barrels of oil, amounting to ∼6% of the total discharged oil (Allen et al. 2011). Unlike flaring, which occurred almost continuously throughout the period, ISBs were episodic. The number of burns conducted on a single burn day ranged from 1 to 26, with each combustion event lasting anywhere from 4 min to 23 h (Allen et al. 2011).

Despite being an efficient way to eliminate oil, controlled burning can produce particulate and gaseous emissions that could endanger the health of nearby workers (Barnea 2011; U.S. EPA

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1999). Of particular concern is fine particulate matter (PM), particles with aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$). $PM_{2.5}$ is a universal air pollutant produced by incomplete combustion of fuel. Common anthropogenic sources of emissions include vehicles and engines, power plants, other industrial processes, and indoor use of fireplaces and woodstoves (U.S. EPA 2020). The particles can penetrate deeply into human lungs and even enter the bloodstream, causing cardiorespiratory diseases (Brook et al. 2010; Xing et al. 2016). During the *DWH* disaster, PM and its components (soot particles, black carbon, dioxins) were detected in smoke plumes produced by *in situ* burning, although no attempt was made to quantify the level of $PM_{2.5}$ specifically (Gullett et al. 2016; Middlebrook et al. 2012; Perring et al. 2011; Schaum et al. 2010). Nance et al. reported elevated concentrations of $PM_{2.5}$ in coastal/urban areas of Louisiana during the time frame of the oil spill (Nance et al. 2016); however, most of the equipment used to monitor $PM_{2.5}$ did not meet federal regulatory criteria, which added uncertainty to the measurements and limited their use (Nance et al. 2016). Recently, exposure to $PM_{2.5}$ from burning was estimated for oil spill response and cleanup (OSRC) workers using an air model recommended by the U.S. Environmental Protection Agency (U.S. EPA) (Pratt et al. 2020), providing a new opportunity to study potential health effects associated with the burning.

A link between short- and long-term ambient particulate air pollution and respiratory effects is well documented (Liu et al. 2017; Xing et al. 2016). Ambient exposure has been associated with emergency department visits, hospital admissions, and exacerbated symptoms for chronic respiratory diseases, including asthma and chronic obstructive pulmonary disease (COPD) (U.S. EPA 2020). In a review by the U.S. EPA, objective spirometric measures of lung function were found in many, but not all, studies to be inversely associated with short-term ambient $PM_{2.5}$ exposure (U.S. EPA 2020). Most of these studies, however, examined participants' lung function immediately after the exposure. Persistent decreases in lung function have been observed in some studies of firefighters, up to several months (and in one cohort, years) after exposure to combustion emissions, as reviewed by Groot et al. (2019). To our knowledge, no study has examined $PM_{2.5}$ from controlled burning of oil/gas, a novel emission source that can expose oil spill workers to short-term concentrations that may exceed the daily National Ambient Air Quality Standard (Pratt et al. 2020). In a previous study of the *DWH* disaster in which workers were assigned to one of four burning exposure rankings based on self-reported work activities (Stewart et al. 2018), we found that higher ranked workers had worse lung function than workers with no exposure (Gam et al. 2018b). To our knowledge, the impact on lung function of quantitatively assessed $PM_{2.5}$ exposure during an oil spill has not yet been investigated. The objective of this study was to assess the relationship between estimates of quantitative $PM_{2.5}$ exposure resulting specifically from burning activities and lung function among *DWH* disaster OSRC workers to provide information for future responders who are considering controlled burning as an option for mitigating the effects of an oil spill. Other sources of $PM_{2.5}$ exposure were not considered.

Methods

Study Population

The GuLF Study (Gulf Long-Term Follow-up Study) is a prospective cohort study of the potential health effects of the *DWH* disaster on OSRC workers ($N = 32{,}608$) (Kwok et al. 2017). Eligible individuals included adults ≥21 y of age at enrollment who either had participated in OSRC for at least 1 d (workers) or had completed safety training but were not hired (nonworkers) (Kwok et al. 2017). Participant enrollment started in March 2011, approximately 8 months after the spill was mitigated, and continued through May 2013. At enrollment, all participants completed a computer-assisted telephone interview in which they provided detailed information on sociodemographics, lifestyle, and health, and a work history describing the OSRC activities they performed. In addition, participants who spoke English or Spanish and lived in one of the Gulf States (Louisiana, Mississippi, Alabama, Florida, and parts of Texas) were invited to complete a home visit, which included a more detailed interview, biological sample collection, anthropometric measurements, and spirometry.

Between May 2011 and May 2013 (1–3 y after the oil spill), 11,193 cohort members, including 8,968 workers, completed the home visit. A total of 887 workers did not complete spirometry, mostly due to medical exclusions ($n = 539$) but also because of refusal ($n = 81$), early home visit termination ($n = 59$), or technical problems ($n = 62$). For a small group of participants ($n = 146$), reasons for not completing the test were not recorded. Among the 8,081 workers who completed spirometry, we selected for analysis the 6,048 workers who had at least three acceptable maneuvers of forced vital capacity (FVC; mL) and forced expiratory volume in 1 s (FEV1; mL). A maneuver was deemed acceptable if it met the criteria established by the American Thoracic Society/European Respiratory Society (ATS/ERS) in 2005 (Miller et al. 2005) or was approved by an expert over-reader. We examined workers who conducted any response or cleanup activities on water (i.e., water workers, $n = 2{,}954$) and excluded 3,094 land workers from the main analysis because workers on land were additionally exposed to $PM_{2.5}$ emissions from land equipment engines, but we lacked information to characterize the magnitude or pattern of this background exposure. We further restricted our main analysis to 2,513 water workers who worked at least 1 d between 15 May and 15 July 2010, the primary period in which burning occurred. Finally, we removed 197 workers with any missing covariate data, arriving at a final analytical sample of 2,316 participants. Participants provided written informed consent during the home visit, and the study was approved by the institutional review board of the National Institute of Environmental Health Sciences (NIEHS).

$PM_{2.5}$ Exposure Assessment

The method for developing $PM_{2.5}$ exposure estimates for workers in the GuLF Study has been described elsewhere (Pratt et al. 2020). While working on water, workers were potentially exposed to three sources of $PM_{2.5}$ emissions: flaring at the wellhead, *in situ* burning offshore, and operation of thousands of mostly diesel-powered vessel engines. However, because of uncertainties in the locations of workers and vessels, it was not possible to consider background emissions from the vessel exhaust or other sources in the development of individual exposure estimates. Here, we summarize the approach by which $PM_{2.5}$ exposure from controlled burning of oil and gas was assessed.

Potential exposure to $PM_{2.5}$ from burning activities was estimated from 15 May to 15 July 2010. Emissions for each ISB or flaring episode were calculated based on emission factors reported in previous studies (Fingas et al. 1995; U.S. EPA 2017) and the estimated volume of oil/gas burned. The resulting primary emissions data were used along with meteorological data and source characterizations as inputs in the Gaussian air dispersion model, AERMOD (Cimorelli et al. 2005), to estimate air concentrations of $PM_{2.5}$ across the Gulf. Meteorological data were obtained from meteorological stations in the Gulf area, and emission sources were optimized by comparing potential AERMOD simulation options with photographs/videos of plumes recorded during the

DWH cleanup to see which options best represented the photographic evidence. Using AERMOD, hourly $PM_{2.5}$ concentrations were modeled for 3,960 geospatial model receptors in the Gulf area for each day that burning occurred. From the modeled hourly concentrations, two daily air concentration estimates at each receptor were retained in the exposure assessment database: the maximum 1-h concentration (to represent peak concentrations) and the maximum of two 12-h (0:00–11:59 and 12:00–23:59) average concentrations (to represent work shift concentrations).

To link workers with these concentration estimates, industrial hygienists created exposure groups based on work locations in the Gulf: hot zone [$\leq$1 nautical mile (nmi) from the wellhead], source ($>$1 and $\leq$5 nmi from the wellhead), offshore ($>$5 nmi from the wellhead to $>$3 nmi from shore), near shore ($\leq$3 nmi from shore), and land. These areas were delineated by 10×10 nmi grid squares, along with a finer grid of 1×1 nmi squares in the 10×10 nmi square containing the wellsite for higher resolution in the hot zone and source areas. Workers in the offshore exposure group were further divided by their reported activity into ISB workers and non-ISB offshore workers to underscore the higher exposure experienced by the ISB Group from *in situ* burning. A job-exposure matrix was created by assigning each exposure group an exposure estimate that represented a spatiotemporal average of the daily maximum concentrations across all days of burning over the period of 15 May to 15 July 2010 (i.e., average daily maximum exposure). For ISB workers, industrial hygienists first averaged daily concentrations (either maximum 1-h or maximum 12-h average) across receptors within grid squares that contained ISBs on each burn day and then took the (arithmetic) mean of these area-average daily estimates across all ISB days ($n = 30$). For the other exposure groups (i.e., non-ISB workers), exposure was calculated by first averaging daily concentrations across all receptors in the grid squares that delineated the work location on each burn/flaring day and then averaging these daily values across all 57 d during which ISB/flaring occurred.

To match individual workers to the exposure groups and the corresponding average daily maximum exposure estimates, industrial hygienists relied on work histories obtained from the enrollment interviews and external administrative data maintained by BP, p.l.c., and its contractors. Participants who worked in multiple locations and/or performed multiple activities (i.e., ISB and others) were matched to the exposure group with the highest exposure estimate. Besides estimates of average daily maximum exposure, industrial hygienists also created "cumulative daily maximum exposure" estimates, a proxy for the total exposure burden received in the exposure period, by multiplying average daily maximum exposure by the number of days exposed to $PM_{2.5}$. To estimate days of exposure, the number of days worked in the exposure period was multiplied by the proportion of (either flare or ISB) burn days in the exposure period. By applying the two exposure metrics (i.e., average and cumulative) to each of the two daily concentration estimates (i.e., maximum 1-h, maximum 12-h average), four measures of $PM_{2.5}$ exposure were available for analysis. Exposure estimates using the maximum 1-h daily concentration and the maximum 12-h average daily concentration had nearly identical distributions (Pearson $r > 0.99$), so we chose to examine only the average maximum 12-h exposure (micrograms per cubic meter) and the cumulative maximum 12-h exposure [micrograms per cubic meter-day ($\mu g/m^3$-d); henceforth, average daily maximum and cumulative daily maximum exposures] in all analyses.

Pulmonary Function

Trained certified medical assistants conducted prebronchodilator spirometry with home visit participants using a portable, ultrasonic transient time–based spirometer (Easy on-PC; NDD Medical Technologies) (Gam et al. 2018b). If applicable, participants with preexisting lung disease were asked to refrain from using an inhaler on the day of the home visit. Spirometry was conducted according to the 2005 ATS/ERS guidelines (Miller et al. 2005). Participants performed the test seated while wearing a disposable nose clip and repeated the test until a minimum of three acceptable maneuvers or a total of eight maneuvers was achieved. Acceptability of a maneuver was defined by the ATS/ERS within-maneuver acceptability criteria as follows: free from artifacts, had a good start, and showed satisfactory exhalation for both FVC and FEV1 (Miller et al. 2005). To ensure the quality of tests, a spirometry expert reviewed all tests and flagged overrides of device-generated quality scores. We chose the best FEV1 and FVC measures, regardless of whether they originated from the same maneuver, and calculated the ratio between the two (FEV1/FVC; %) to be collectively examined in the main analysis.

Covariates

We identified covariates for adjustment based on a directed acyclic graph (Greenland et al. 1999) and included the sufficient adjustment set in the models (Figure S1). Covariates that were ascertained in the enrollment interview included sex (male; female), race (elaborated below), Hispanic ethnicity (Hispanic; non-Hispanic), cigarette smoking status (heavy current ($\geq$20 cigarettes/d); light current ($<$20 cigarettes/d); former; never), highest education attainment (less than high school; high school diploma or general equivalency diploma; some college or 2-y degree; 4-y college graduate or more), employment at the time of enrollment (employed; looking for work or unemployed; other), previous oil spill cleanup experience (yes; no), previous oil industry experience (yes; no), prespill lung disease diagnosis (asthma, chronic bronchitis, emphysema) (yes; no), prespill diabetes diagnosis (yes; no), and residential proximity to the spill (living in a coastal county directly affected by the spill; living in a county adjacent to the coastal counties; living in a Gulf or non-Gulf state further from the spill). Race and ethnicity were included in the models as proxies for unmeasured differences in socioeconomic and environmental factors that were predictive of lung function and occurred as a result of institutional racism (Celedón et al. 2017). To obtain information on race, participants were provided the following choices and permitted to select multiple responses: "American Indian or Alaskan Native," "Asian," "Black or African American," "Native Hawaiian or Pacific Islander," "White," and "other" (with the option to specify race as free text). We aggregated participants who self-identified as "American Indian or Alaskan Native," "Asian," "Native Hawaiian or Pacific Islander," or "other (races)" and who selected multiple races into the "other/multiracial" group because of the small numbers of workers in these groups. We used age at the home visit. At the home visit, exposure to secondhand smoking (yes; no) was elicited by a question that asked how many regular smokers the participant currently lived with. Height and weight were measured three times, and the average values were used in the analysis. We modeled age and height in quadratic form after considering model fit (R^2), visual relationship (LOWESS plots), and the modeling approach described in a study that established spirometric reference values for the general U.S. population (Hankinson et al. 1999). Last, to account for body mass index (BMI) as a potential confounder, we included weight as a covariate in addition to the height parameters that were already in the model.

Statistical Modeling

We examined mean values of lung function by categorized exposures and used multivariable linear regressions to estimate adjusted

mean differences in lung function parameters (FEV1, FVC, and FEV1/FVC ratio) and 95% confidence intervals (95% CIs) associated with increasing levels of estimated $PM_{2.5}$ among water workers. We checked assumptions of linear regression via diagnostic plots of the residuals, and we assumed observations were independent of one another. In the main analysis, we adjusted for all covariates in the sufficient adjustment set (i.e., fully adjusted model) and examined average and cumulative daily maximum exposures in separate models. We also examined adjusted mean differences in lung function in minimally adjusted models that accounted for age at home visit (quadratic form), sex, race, ethnicity, height (quadratic form), and weight to explore the magnitude of confounding. Because burning-related $PM_{2.5}$ exposure of the nearshore and non-ISB offshore workers were substantially lower than those of the other water workers, the first two groups were combined as the "referent group" in the analyses for comparison with the other workers with higher burning-related exposures (henceforth, "burning-exposed workers") (Table S1). For average daily maximum exposure, we collapsed exposure levels for the ISB workers ($10.4\ \mu g/m^3$) and workers at the source ($28.7\ \mu g/m^3$) due to the small number of ISB workers ($n = 10$). Average daily maximum exposure then became a three-level categorical variable: referent ($0.8\ \mu g/m^3$), low ($10.4–28.7\ \mu g/m^3$), and high, with high corresponding to the exposure level for hot zone workers ($96.9\ \mu g/m^3$). The cumulative daily maximum exposure metric, which was determined by both the exposure level and the exposure duration (median = 26 d, range = 0–49 d), had greater individual variability. To model it in the analysis, we employed the same referent group and categorized the remainder of workers (i.e., burning-exposed group) into tertiles by the exposure distribution to create a four-level categorical variable: the referent group ($<10\ \mu g/m^3$-d), low (10–689 $\mu g/m^3$-d), medium (718–1,406 $\mu g/m^3$-d), and high (1,551–3,199 $\mu g/m^3$-d). Because of numerous tied values at the tertile cutoffs (Figure S2), the number of burning-exposed workers in each tertile is not evenly distributed. In addition to the aforementioned models, we also investigated exposure–response trends by assessing continuous exposures in relation to lung function in covariate-adjusted models. Exposure–response trends were analyzed separately for average (per $10\ \mu g/m^3$ increase) and cumulative daily maximum exposures (per $10\ \mu g/m^3$-d increase).

We then investigated potential effect measure modification (EMM) by cigarette smoking status (ever vs. never) to explore whether exposure to $PM_{2.5}$ from burning of oil/gas had similar effects among participants with or without respiratory burden from smoking. We were underpowered to stratify analysis by preexisting lung disease and examine associations in the diseased group. Instead, we restricted this analysis to those without prespill lung disease. Because studies have demonstrated improved lung function in association with reductions in $PM_{2.5}$ exposure (Downs et al. 2007; Paulin and Hansel 2016), we stratified the analysis by time from cessation of exposure (15 July 2010, i.e., the end of the exposure period) to spirometry test (median: ≤ 1.99 and >1.99 y) to see whether the association was stronger among participants with shorter time between exposure and exam. For all stratified analyses, we reported *p*-values for the product terms of categorized exposures and effect measure modifiers to formally assess heterogeneity of the associations. In a sensitivity analysis, we loosened the spirometry quality criteria by examining participants with at least *two* acceptable maneuvers that met the criteria defined by the ATS/ERS guidelines to address possible selection bias, as studies have linked poor lung function with spirometry of poor quality (Eisen et al. 1984). In addition, we compared characteristics between workers selected into our study with the remainder of water workers who were eligible for the home visit. Because volatile components of the crude oil also had the potential to induce lung inflammation (ATSDR 1999), we additionally adjusted for cumulative exposure to total hydrocarbons, measured as cumulative total petroleum hydrocarbons, to see whether results differed. Exposure to total hydrocarbons was estimated via a job-exposure matrix based on personal air sample measurements and OSRC work histories collected at enrollment (Groth et al. 2017, 2021; Huynh et al. 2020a, 2020b, 2021; Ramachandran et al. 2021). To capture residual confounding from smoking, we also characterized cigarette smoking as continuous pack-years, which was ascertained at enrollment via questions on smoking history. Because workers were also exposed to the unquantified $PM_{2.5}$ from engine exhaust, we assessed the potential impact of bias by performing sensitivity analyses that excluded non-ISB offshore workers, the group with the largest potential vessel exhaust exposure variability, and separately, that included land workers as an additional exposure category to quantify the potential bias from land equipment emissions. All analyses were performed using SAS, (version 9.4; SAS Institute Inc.). An alpha level of 0.05 was considered statistically significant for all analyses.

Results

In comparison with the referent group ($n = 1{,}798$), burning-exposed workers ($n = 518$) were younger (40.6 vs. 44.6 y old) at the time of the home visit and more likely to be Black (44.4% vs. 16.7%) (Table 1). Although the proportion of ever smokers was similar between the two groups, a larger percentage of burning-exposed workers was light current smokers in comparison with the referent group (30.7% vs. 21.3%). Compared with the referent group, burning-exposed workers were more likely to have a high school diploma as the highest educational attainment (38.0% vs. 30.9%) and less likely to have attained a college or graduate degree (10.4% vs. 14.6%); more of them were unemployed at the time of enrollment (30.3% vs. 19.2%). In comparison with the referent group, burning-exposed workers were less likely to live in a county directly impacted by the spill (63.3% vs. 75.6%) (i.e., they lived outside of the coastal counties). With regard to baseline health, a slightly smaller proportion of burning-exposed workers reported a prespill lung disease diagnosis (12.7% vs. 15.1%) or diabetes diagnosis (4.1% vs. 5.6%) in comparison with the referent group. The other selected characteristics were similar between the two groups. We also examined characteristics of our study population by average daily maximum exposure, dividing burning-exposed workers into low- and high-exposure groups for presentation purposes (Table S2). In comparison with the low-exposure and referent groups, the high-exposure group had a higher proportion of males (high: 97.1%; low: 89.8%; referent: 87.8%) and non-White workers (high: 61.8%; low: 54.7%; referent: 27.7%). Only 29.4% of highly exposed workers received post-secondary education, in comparison with 43.6% and 46.5% in the low exposure and referent groups, respectively. Workers in the high-exposure group were also more likely to live farther away from the spill (high: 38.2%; low: 26.2%; referent: 17.6%) and have previous experience working in the oil industry (high: 29.4%; low: 16.9%; referent: 22.1%) or in another oil spill cleanup (high: 13.2%; low: 10.0%; referent: 9.4%) in comparison with those with lower exposure.

In the minimally adjusted models, workers with higher average and cumulative daily maximum $PM_{2.5}$ exposures had lower FEV1, FVC, and FEV1/FVC, although no significant trends were observed (Table 2). After accounting for additional covariates, the inverse associations appeared stronger, and most point estimates showed a change greater than 10% in the fully adjusted models (Table 2). In these models, we observed statistically significant exposure–response trends relating average daily maximum $PM_{2.5}$

Table 1. Characteristics of DWH disaster oil spill water workers who had acceptable lung function test quality by $PM_{2.5}$ exposure group, at home visit, 2011–2013 ($n = 2,316$).

Characteristic	Burning-exposed workers[a] ($n = 518$)	Referent group[a] ($n = 1,798$)
	Mean (SD)	Mean (SD)
Age (y)	40.6 (12.0)	44.6 (13.0)
Height (in)	69.1 (3.3)	68.7 (3.5)
Weight (lb)	202.0 (48.2)	201.4 (45.2)
BMI (kg/m^2)	29.7 (6.7)	30.0 (6.4)
	n (%)	*n* (%)
Sex		
Male	470 (90.7)	1,578 (87.8)
Female	48 (9.3)	220 (12.2)
Race		
White	230 (44.4)	1,300 (72.3)
Black	230 (44.4)	301 (16.7)
Asian	6 (1.2)	13 (0.7)
Other	37 (7.1)	133 (7.4)
Multiracial	15 (2.9)	51 (2.8)
Ethnicity		
Hispanic	35 (6.8)	101 (5.6)
Non-Hispanic	483 (93.2)	1,697 (94.4)
Smoking Status		
Heavy current smoker	45 (8.7)	265 (14.7)
Light current smoker	159 (30.7)	383 (21.3)
Former smoker	96 (18.5)	436 (24.3)
Never smoker	218 (42.1)	714 (39.7)
Secondhand smoker		
Yes	144 (27.8)	544 (30.3)
No	374 (72.2)	1,254 (69.7)
Education		
Less than high school	105 (20.3)	405 (22.5)
High school diploma/GED	197 (38.0)	556 (30.9)
Some college/2-y degree	162 (31.3)	574 (31.9)
4-y college graduate or more	54 (10.4)	263 (14.6)
Employment		
Working now	282 (54.4)	1,122 (62.4)
Looking for work or unemployed	157 (30.3)	345 (19.2)
Other	79 (15.3)	331 (18.4)
Residential county proximity to Gulf of Mexico[b]		
Direct	328 (63.3)	1,359 (75.6)
Indirect	46 (8.9)	123 (6.8)
Other residence	144 (27.8)	316 (17.6)
Previous oil spill cleanup work		
Yes	54 (10.4)	169 (9.4)
No	464 (89.6)	1,629 (90.6)
Previous oil industry experience		
Yes	96 (18.5)	397 (22.1)
No	422 (81.5)	1,401 (77.9)
Reported prespill lung disease diagnosis		
Yes	66 (12.7)	271 (15.1)
No	452 (87.3)	1,527 (84.9)
Reported prespill diabetes diagnosis		
Yes	21 (4.1)	100 (5.6)
No	497 (96.0)	1,698 (94.4)

Note: Acceptable pulmonary function test quality is defined as having at least 3 FEV1 and FVC curves that met the 2005 American Thoracic Society/European Respiratory Society acceptability criteria or were approved by a spirometry expert. No missing in covariates as results describe the analytical sample. BMI, body mass index; *DWH*, *Deepwater Horizon*; GED, general equivalency diploma; in, inches; lb, pounds; SD, standard deviation.

[a]The referent group consisted of nearshore workers and offshore workers who did not work on in situ burns. Burning-exposed workers consisted of the remaining water workers (i.e. in situ burn workers, workers at the source, and workers at the hot zone).

[b]Direct 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 residence is defined as living in a Gulf or non-Gulf state further from the spill.

exposure to lower FEV1 (p-trend = 0.04) and FEV1/FVC (p-trend = 0.03). When examining cumulative daily maximum exposure, we again found significant inverse trends for FEV1 (p-trend = 0.04) and FEV1/FVC (p-trend = 0.01). Workers in the high cumulative daily maximum exposure category had marginally significant decreases in mean FEV1 (−166.8 mL, 95% CI: −337.3, 3.7) and FEV1/FVC (−1.7%, 95% CI: −3.6, 0.2) in comparison with the referent group. We also saw an inverse association between cumulative daily maximum exposure and FVC, but the trend test was not significant (p-trend = 0.36).

When we restricted analyses to workers without lung disease diagnosis before the spill, we saw statistically significant trends that associated FEV1 with average (p-trend = 0.04) and cumulative daily maximum exposures (p-trend = 0.02). Effect measures among workers with higher exposure were similar to those observed in the main analysis (Table 3). We also observed inverse but nonsignificant associations with FVC and FEV1/FVC.

When analyses were stratified by smoking status, trends were generally more pronounced among never smokers (Table 4). In this subgroup, we saw a significant trend between average daily maximum exposure and FEV1 (p-trend = 0.02) and significantly lower FEV1 (−228.3 mL, 95% CI: −431.3, −25.3) among workers in the high-exposure group. We also observed consistently lower FVC among never smokers with higher average and cumulative daily maximum exposures. In contrast, the associations with FEV1 and FVC were weaker in the ever-smokers subgroup. Among ever smokers, we saw a statistically significant trend for the association between cumulative daily maximum exposure and FEV1/FVC (p-trend = 0.01) accompanied by nonsignificantly lower FEV1/FVC in the high-exposure group (−2.7%, 95% CI: −5.6, 0.1).

When we stratified analyses by time from cessation of exposure to spirometry, inverse associations appeared to be stronger among workers whose spirometry tests were closer to their exposure (≤1.99 y), especially in the high exposure category, although tests of interaction were not significant (Table 5). Among workers who completed tests earlier (≤1.99 y), those in the highest average and cumulative daily maximum exposure categories had substantially lower FEV1, FVC, and FEV1/FVC in comparison with the referent group. Among workers who completed tests later (>1.99 y), we also observed decreases in all three lung function measures, including a significant test of trend between cumulative daily maximum exposure and FEV1/FVC, although the relationship was not monotonic.

When we relaxed the spirometry quality criteria and included participants with at least two acceptable maneuvers, results were not substantively different (Table S3). When we compared water workers examined in the analysis vs. the remainder of water workers who were eligible for the home visit but not selected, population characteristics were similar except for a small difference in employment at enrollment (Table S4). Adjusting for cumulative exposure to total hydrocarbons, which was moderately correlated with average (Pearson $r = 0.30$) and cumulative daily maximum $PM_{2.5}$ exposures (Pearson $r = 0.36$), produced minimal differences in the observed associations (Table S5). Replacing cigarette smoking status with a more quantitative pack-year measure did not produce noticeable differences in results (Table S6). When we excluded non-ISB offshore workers from the analytical sample, associations were somewhat attenuated, but interpretations remained similar (Table S7). Last, when we expanded the study population to include land workers as a separate exposure group, we observed significantly lower values for all three lung function metrics among land workers in comparison with the referent group (Table S8). A comparison between land workers and the referent group showed that land workers were more likely to be Black and female but otherwise had BMIs and proportions of lung disease and diabetes diagnoses that were similar to those of the referent group (Table S9).

Table 2. $PM_{2.5}$ exposure and lung function among *DWH* disaster oil spill water workers at home visit ($n = 2,316$).

				Minimally adjusted[a]		Fully adjusted[b]	
Lung function measures	$PM_{2.5}$ exposure	n (%)	Mean (SD)	β (95% CI)	p-Value	β (95% CI)	p-Value
	Average exposure						
FEV1 (mL)	Ref[c]	1,798 (77.6)	3,334.9 (786.0)	—	—	—	—
	Low	450 (19.4)	3,300.5 (798.4)	−42.9 (−105.6, 19.7)	0.18	−50.9 (−112.2, 10.4)	0.10
	High	68 (2.9)	3,325.6 (825.5)	−86.2 (−229.1, 56.6)	0.24	−114.2 (−254.3, 26.0)	0.11
	Per 10 $\mu g/m^3$ increase	—	—	−10.4 (−23.4, 2.7)	0.12	−13.3 (−26.1, −0.5)	0.04
FVC (mL)	Ref[c]	1,798 (77.6)	4,260.6 (934.4)	—	—	—	—
	Low	450 (19.4)	4,193.3 (953.0)	−40.7 (−112.6, 31.3)	0.27	−42.3 (−113.6, 29.1)	0.25
	High	68 (2.9)	4,280.3 (1075.8)	−47.5 (−211.6, 116.5)	0.57	−55.5 (−218.7, 107.7)	0.50
	Per 10 $\mu g/m^3$ increase	—	—	−7.1 (−22.1, 7.8)	0.35	−7.9 (−22.8, 7.0)	0.30
FEV1/FVC (%)	Ref[c]	1,798 (77.6)	78.3 (6.7)	—	—	—	—
	Low	450 (19.4)	78.7 (7.1)	−0.3 (−1.0, 0.3)	0.32	−0.5 (−1.2, 0.2)	0.14
	High	68 (2.9)	78.0 (7.0)	−0.9 (−2.5, 0.7)	0.26	−1.4 (−3.0, 0.1)	0.07
	Per 10 $\mu g/m^3$ increase	—	—	−0.1 (−0.2, 0.04)	0.16	−0.2 (−0.3, −0.02)	0.03
	Cumulative exposure						
FEV1 (mL)	Ref[c]	1,798 (77.6)	3,334.9 (786.0)	—	—	—	—
	Low	216 (9.3)	3,291.6 (824.6)	−62.0 (−146.3, 22.4)	0.15	−80.8 (−163.2, 1.6)	0.05
	Medium	257 (11.1)	3,337.1 (807.1)	−24.9 (−103.3, 53.5)	0.53	−21.8 (−98.4, 54.7)	0.58
	High	45 (1.9)	3,207.6 (683.6)	−116.7 (−290.3, 56.9)	0.19	−166.8 (−337.3, 3.7)	0.06
	Per 10 $\mu g/m^3$-d increase	—	—	−0.4 (−0.9, 0.1)	0.10	−0.5 (−0.9, −0.01)	0.04
FVC (mL)	Ref[c]	1,798 (77.6)	4,260.6 (934.4)	—	—	—	—
	Low	216 (9.3)	4,142.7 (999.8)	−86.3 (−183.1, 10.6)	0.08	−96.7 (−192.6, −0.7)	0.05
	Medium	257 (11.1)	4,273.9 (967.9)	6.1 (−83.8, 96.1)	0.89	13.4 (−75.7, 102.5)	0.77
	High	45 (1.9)	4,139.7 (848.9)	−97.9 (−297.2, 101.3)	0.34	−120.9 (−319.4, 77.6)	0.23
	Per 10 $\mu g/m^3$-d increase	—	—	−0.2 (−0.8, 0.3)	0.37	−0.3 (−0.8, 0.3)	0.36
FEV1/FVC (%)	Ref[c]	1,798 (77.6)	78.3 (6.7)	—	—	—	—
	Low	216 (9.3)	79.5 (7.0)	0.2 (−0.7, 1.1)	0.67	−0.1 (−1.0, 0.9)	0.91
	Medium	257 (11.1)	78.0 (7.3)	−0.9 (−1.7, 0.004)	0.05	−0.9 (−1.8, −0.1)	0.03
	High	45 (1.9)	77.6 (6.6)	−0.9 (−2.8, 1.0)	0.37	−1.7 (−3.6, 0.2)	0.08
	Per 10 $\mu g/m^3$-d increase	—	—	−0.01 (−0.01, 0.0001)	0.06	−0.01 (−0.01, −0.002)	0.01

Note: —, no data; β, adjusted mean difference; CI, confidence interval; *DWH*, *Deepwater Horizon*; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; mL, milliliters; PM, particulate matter; $PM_{2.5}$, PM with aerodynamic diameter less than or equal to 2.5 μm; Ref, Referent.

[a]Adjusted for age, age^2, sex, race, ethnicity, height, height^2, and weight.

[b]Adjusted for age, age^2, sex, race, ethnicity, height, height^2, smoking (former, light, heavy), secondhand smoke, weight, prespill diabetes, prespill lung disease, education, employment, previous oil industry experience, previous oil spill cleanup work, and residential proximity to the Gulf of Mexico.

[c]The referent group consisted of nearshore workers and offshore workers who did not work on *in situ* burns.

Discussion

In this study, we examined the relationship between potential exposure to $PM_{2.5}$ specifically from controlled burning of oil and gas and lung function among OSRC workers up to 3 y after the *DWH* disaster. We observed lower FEV1 and FEV1/FVC among workers who were exposed to higher levels of $PM_{2.5}$. These inverse associations persisted in analyses stratified by smoking status and time from exposure to spirometry and when we restricted to workers without prespill lung disease. The results were robust to sensitivity analyses that, separately, applied less strict spirometry quality criteria, adjusted for estimated total hydrocarbons exposure from crude oil, and restricted to nearshore workers as the referent group. We also observed a few suggestive but nonmonotonic and nonsignificant associations between $PM_{2.5}$ exposure and FVC in the main and subgroup analyses. A review of studies examining age-related lung function changes among adults without respiratory issues or tobacco use found that the annual decline in FEV1 ranged between 17.7 and 46.4 mL across studies (Thomas et al. 2019), and the rate of age-related decline in FEV1/FVC was reported to be 0.29%/year in a longitudinal study (Liao et al. 2015). The declines in lung function observed in our study are equivalent to 1 to possibly several years of lung function loss from aging, suggesting that our results are clinically meaningful.

In our study population, the average daily maximum exposure that workers experienced varied significantly across exposure groups (Pratt et al. 2020). Over a 12-h shift, ISB workers and workers in the source area were exposed to 10 and 29 $\mu g/m^3$ of shift-average $PM_{2.5}$ levels, respectively, which are similar to levels observed in high-traffic areas in developed countries (Edginton et al. 2019). On the other hand, workers in the hot zone were subject to exposures at almost 100 $\mu g/m^3$, on par with pollution levels in parts of developing countries such as India and China (Edginton et al. 2019; Kesavachandran et al. 2013). No direct measurement data for $PM_{2.5}$ on the water were available for comparison with our modeled estimates. Measurement data from the nearest 11 monitoring stations in the Gulf states showed that the 24-h average $PM_{2.5}$ concentrations in the period of OSRC ranged from 9.5 to 10.2 $\mu g/m^3$ (Pratt et al. 2020). However, even the closest station was approximately 400 km from the *DWH* wellhead, and measurements might reflect significant contributions from inland emission sources (Pratt et al. 2020).

Understanding of the mechanisms by which short-term particulate exposure impairs human lung function is still evolving. Evidence from animal toxicological studies, as reviewed by the U.S. EPA, suggests that inhaled $PM_{2.5}$ has the potential to induce injury, oxidative stress, and inflammation in the respiratory tract, which can lead to downstream effects that contribute to lung function decrements (U.S. EPA 2020). Some of the downstream effects, including allergic responses and airway remodeling, may lead to spasms and narrowing of airway walls that contribute to airway obstruction (He et al. 2017), which can be detected in spirometry as a low FEV1/FVC ratio (Pellegrino et al. 2005). Studies have shown that $PM_{2.5}$ has the potential to damage alveoli epithelium specifically, either through inflammatory response (Yang et al. 2019) or by compromising host defense and increasing the risk of respiratory infections (Yang et al. 2020; Zelikoff et al. 2003). Heavily damaged lung tissue can result in decreased lung volume, which presents in spirometry as low FVC (Pellegrino et al. 2005). In line with these proposed

Table 3. $PM_{2.5}$ exposure and lung function among DWH disaster oil spill water workers at home visit without prespill lung disease diagnosis ($n = 1,979$).

Lung function measures	$PM_{2.5}$ exposure	n (%)	β (95% CI)[a]	p-Value
	Average exposure			
FEV1 (mL)	Ref[b]	1,527 (77.2)	—	—
	Low	391 (19.8)	−55.8 (−120.7, 9.0)	0.09
	High	61 (3.1)	−119.3 (−265.4, 26.9)	0.11
	Per 10 $\mu g/m^3$ increase	—	−14.1 (−27.5, −0.8)	0.04
FVC (mL)	Ref[b]	1,527 (77.2)	—	—
	Low	391 (19.8)	−56.1 (−131.8, 19.6)	0.15
	High	61 (3.1)	−89.4 (−260.0, 81.1)	0.30
	Per 10 $\mu g/m^3$ increase	—	−11.8 (−27.4, 3.8)	0.14
FEV1/FVC (%)	Ref[b]	1,527 (77.2)	—	—
	Low	391 (19.8)	−0.4 (−1.1, 0.4)	0.33
	High	61 (3.1)	−0.9 (−2.5, 0.7)	0.28
	Per 10 $\mu g/m^3$ increase	—	−0.1 (−0.2, 0.04)	0.17
	Cumulative exposure			
FEV1 (mL)	Ref[b]	1,527 (77.2)	—	—
	Low	192 (9.7)	−76.2 (−162.8, 10.5)	0.08
	Medium	217 (11.0)	−32.3 (−114.1, 49.4)	0.44
	High	43 (2.2)	−173.8 (−346.5, −1.0)	0.05
	Per 10 $\mu g/m^3$-d increase	—	−0.6 (−1.0, −0.1)	0.02
FVC (mL)	Ref[b]	1,527 (77.2)	—	—
	Low	192 (9.7)	−89.9 (−191.1, 11.2)	0.08
	Medium	217 (11.0)	−16.2 (−111.5, 79.2)	0.74
	High	43 (2.2)	−156.6 (−358.2, 45.1)	0.13
	Per 10 $\mu g/m^3$-day increase	—	−0.5 (−1.0, 0.1)	0.11
FEV1/FVC (%)	Ref[b]	1,527 (77.2)	—	—
	Low	192 (9.7)	−0.01 (−1.0, 0.9)	0.98
	Medium	217 (11.0)	−0.6 (−1.5, 0.3)	0.17
	High	43 (2.2)	−1.2 (−3.1, 0.7)	0.23
	Per 10 $\mu g/m^3$-d increase	—	−0.005 (−0.01, 0.001)	0.08

Note: —, no data; β, adjusted mean difference; CI, confidence interval *DWH*, *Deepwater Horizon*; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; mL, milliliters; PM, particulate matter; $PM_{2.5}$, PM with aerodynamic diameter less than or equal to 2.5 μm; Ref, Referent.
[a]Adjusted for age, age^2, sex, race, ethnicity, height, height^2, smoking, secondhand smoke, weight, prespill diabetes, education, employment, previous oil industry experience, previous oil spill cleanup work, and residential proximity to the Gulf of Mexico.
[b]The referent group consisted of nearshore workers and offshore workers who did not work on *in situ* burns.

mechanisms, researchers have observed pulmonary inflammation and altered immune responses in mice exposed to PM from samples of ISB plumes collected during the *DWH* oil spill (Jaligama et al. 2015). In our study, we observed significantly lower FEV1 and FEV1/FVC among workers with higher exposures. Suggestive associations were also seen for FVC. These findings suggest that short-term exposure to burning-related $PM_{2.5}$ has the potential to induce airway obstruction and damage lungs, possibly via the inflammatory responses and immunological changes described above.

Although short-term $PM_{2.5}$ exposures have been implicated in many studies reviewed by the U.S. EPA as a major contributor to respiratory hospitalizations and mortality among the general adult population (U.S. EPA 2020), fewer studies have examined lung function as the outcome. Panel studies and cross-sectional analyses of mostly healthy populations residing near stationary monitors have generally found an inverse association between FEV1 (Rice et al. 2013; Trenga et al. 2006), FVC (Rice et al. 2013; Trenga et al. 2006; Zhou et al. 2016), or peak expiratory flow (Zhang et al. 2015) and recent $PM_{2.5}$ exposure (i.e., days) before the spirometry. However, these associations could reflect either the effect from an acute exposure or a snapshot of longer-term exposures. Other studies employed quasi-experimental designs by instructing participants to rest or exercise in sites with varying exposures and examining post-exposure changes in lung function. In studies where participants were exposed to traffic-related air pollution for short durations (<5 h) and/or at low concentrations, the authors did not find a strong association between exposure and changes in lung function (Kubesch et al. 2015; Matt et al. 2016; Strak et al. 2012; Weichenthal et al. 2011). In contrast, two randomized crossover studies that exposed participants to $PM_{2.5}$ at much higher concentrations (Huang et al. 2016) or for longer duration (5 consecutive days) (Dales et al. 2013) showed significant decrements in lung function. In the *DWH* disaster, workers were potentially exposed to $PM_{2.5}$ for 1 to up to 49 d, sometimes at levels that exceeded the daily National Ambient Air Quality Standard. Our results agree with these latter two studies in finding that workers who received high daily exposures or persistent exposure over many days had substantially lower measures of lung function. It is possible, therefore, that a threshold exists beyond which particulate exposure induces measurable changes in lung function.

Unlike the ambient air pollution studies that focused on acute changes in lung function, the longer time between exposure and spirometry in our study allowed us to evaluate decrements in lung function up to 3 y after exposure ended. Some occupational studies also have examined longer-term changes in lung function after exposure to combustion emissions, including studies of wildland firefighters. In a review of studies that compared firefighters' lung function before and after the firefighting season (Groot et al. 2019), all but one study (Gaughan et al. 2008) showed statistically significant decreases in cross-seasonal FEV1 (Betchley et al. 1997; Jacquin et al. 2011; Miranda et al. 2012; Rothman et al. 1991) or FVC (Jacquin et al. 2011; Rothman et al. 1991), with follow-up durations varying from 2 wk to 3 months after exposure. None of these studies, however, monitored levels of $PM_{2.5}$ during firefighting. One study measured levoglucosan, a proxy for $PM_{2.5}$ emissions from biomass combustion, in a 4-d firefighting session and found greater reduction in cross-shift FEV1 among firefighters with higher exposure (Gaughan et al. 2014). The persistent effect of air pollutant exposure on lung function was also evaluated in responders to the 2001 World Trade Center (WTC) disaster, who were exposed to tremendous amounts of dust (Feldman et al. 2004). In comparison with

Table 4. $PM_{2.5}$ exposure and lung function among *DWH* disaster oil spill water workers at home visit ($n = 2{,}316$), stratified by smoking status at enrollment.

Lung function measures	$PM_{2.5}$ exposure	Ever smokers ($n = 1{,}384$)			Never smokers ($n = 932$)			Interaction
		n (%)	β (95% CI)[a]	*p*-Value	*n* (%)	β (95% CI)[a]	*p*-Value	*p*-value
	Average exposure							
FEV1 (mL)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.02
	Low	262 (18.9)	−45.9 (−128.0, 36.1)	0.27	188 (20.2)	−59.7 (−150.6, 31.3)	0.20	—
	High	38 (2.8)	−34.5 (−226.0, 156.9)	0.72	30 (3.2)	−228.3 (−431.3, −25.3)	0.03	—
	Per 10 $\mu g/m^3$ increase	—	−6.7 (−24.0, 10.7)	0.45	—	−23.1 (−41.8, −4.4)	0.02	—
FVC (mL)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.05
	Low	262 (18.9)	−30.8 (−126.0, 64.4)	0.53	188 (20.2)	−60.6 (−168.8, 47.6)	0.27	—
	High	38 (2.8)	41.6 (−180.6, 263.7)	0.71	30 (3.2)	−187.5 (−428.9, 53.8)	0.13	—
	Per 10 $\mu g/m^3$ increase	—	0.8 (−19.3, 20.9)	0.94	—	−20.1 (−42.3, 2.2)	0.08	—
FEV1/FVC (%)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.23
	Low	262 (18.9)	−0.7 (−1.6, 0.3)	0.16	188 (20.2)	−0.3 (−1.3, 0.6)	0.51	—
	High	38 (2.8)	−1.3 (−3.5, 0.9)	0.24	30 (3.2)	−1.8 (−3.8, 0.3)	0.10	—
	Per 10 $\mu g/m^3$ increase	—	−0.2 (−0.4, 0.03)	0.10	—	−0.2 (−0.4, 0.03)	0.09	—
	Cumulative exposure							
FEV1 (mL)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.06
	Low	116 (8.4)	−66.2 (−180.5, 48.2)	0.26	100 (10.7)	−96.3 (−214.0, 21.4)	0.11	—
	Medium	161 (11.6)	−12.8 (−112.0, 86.4)	0.80	96 (10.3)	−42.3 (−161.7, 77.2)	0.49	—
	High	23 (1.7)	−157.6 (−401.2, 85.9)	0.20	22 (2.4)	−189.9 (−424.5, 44.7)	0.11	—
	Per 10 $\mu g/m^3$-d increase	—	−0.4 (−1.0, 0.3)	0.25	—	−0.7 (−1.3, 0.03)	0.06	—
FVC (mL)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.06
	Low	116 (8.4)	−97.6 (−230.2, 35.0)	0.15	100 (10.7)	−93.3 (−233.2, 46.5)	0.19	—
	Medium	161 (11.6)	36.1 (−79.0, 151.2)	0.54	96 (10.3)	−32.2 (−174.2, 109.7)	0.66	—
	High	23 (1.7)	−50.0 (−332.5, 232.5)	0.73	22 (2.4)	−203.6 (−482.4, 75.1)	0.15	—
	Per 10 $\mu g/m^3$-d increase	—	0.02 (−0.7, 0.8)	0.95	—	−0.7 (−1.5, 0.2)	0.11	—
FEV1/FVC (%)	Ref[b]	1,084 (78.3)	—	—	714 (76.6)	—	—	0.69
	Low	116 (8.4)	0.3 (−1.0, 1.6)	0.68	100 (10.7)	−0.5 (−1.7, 0.7)	0.44	—
	Medium	161 (11.6)	−1.2 (−2.3, −0.1)	0.04	96 (10.3)	−0.5 (−1.7, 0.8)	0.44	—
	High	23 (1.7)	−2.7 (−5.6, 0.1)	0.05	22 (2.4)	−0.7 (−3.1, 1.7)	0.56	—
	Per 10 $\mu g/m^3$-d, increase	—	−0.01 (−0.02, −0.003)	0.01	—	−0.003 (−0.01, 0.004)	0.38	—

Note: —, no data; β, adjusted mean difference; CI, confidence interval; *DWH, Deepwater Horizon*; FEV1, forced expiratory volume in 1 second; FVC, forced vital capacity; mL, milliliters; PM, particulate matter; $PM_{2.5}$, PM with aerodynamic diameter less than or equal to 2.5 μm; Ref, Referent.
[a]Adjusted for age, age^2, sex, race, ethnicity, height, height^2, smoking (former, light, heavy), secondhand smoke, weight, prespill diabetes, prespill lung disease, education, employment, previous oil industry experience, previous oil spill cleanup work, and residential proximity to the Gulf of Mexico.
[b]The referent group consisted of nearshore workers and offshore workers who did not work on *in situ* burns.

baseline measures taken prior to the event, a significant reduction in FEV1 and FVC was observed among firefighters and other rescuers 1 y after the disaster, with stronger associations found for rescuers who arrived at the site early (Banauch et al. 2006). In our study, we found exposure-related decreases in lung function up to 3 y after the spill, although decreases in the high exposure categories were larger among workers who had spirometry closer to the exposure. The somewhat attenuated effect observed among highly exposed workers who were followed longer is consistent with ambient air pollution studies that demonstrated improvement in lung function in response to reduction in $PM_{2.5}$ exposure (Downs et al. 2007; Paulin and Hansel 2016).

Many air pollution studies have associated short-term $PM_{2.5}$ exposure with lower lung function among patients with COPD (Cortez-Lugo et al. 2015; Ebelt et al. 2005; Trenga et al. 2006) and asthma (McCreanor et al. 2007; Mirabelli et al. 2015), but a few studies did not (Hsu et al. 2011; Urch et al. 2010). Although we had information on self-reported lung disease diagnoses, we were underpowered to examine associations among workers with diagnosed prespill lung disease. In analyses restricted to workers without diagnosed prespill lung disease, we observed inverse associations similar to those in the main analysis. This suggests that burning-related $PM_{2.5}$ could induce lung function decrements among adults without existing lung disease.

We also stratified the analysis by cigarette smoking, because smoking could induce hyper-responsiveness and inflammation of the respiratory airways (Willemse et al. 2004), aggravating the effect of other air pollutants. Few studies have examined short-term $PM_{2.5}$ exposure and lung function separately for smokers and never smokers. In the study of WTC responders, the authors observed differential post-exposure change in lung function by smoking status up to 13 y after the event (Aldrich et al. 2016). The reduction in FEV1 was greatest among current smokers, second greatest among former smokers, and least among never smokers. In contrast, a study of wildland firefighters (Jacquin et al. 2011) and another of adult residents of Boston (Rice et al. 2013) did not find EMM by smoking status. In our study, we saw inverse associations between exposure and lung function in both ever and never smokers, although reductions in FEV1 and FVC were more pronounced among never smokers. One possible explanation for the weaker associations among ever smokers is that the strong impact of smoking obscured the effect of the short-term burning exposure.

No previous study has examined the relationship between $PM_{2.5}$ exposure and lung function among OSRC workers. Because controlled burning had not been adopted as a major mitigation technique in previous spills, exposure in our study was unique in its emission sources and exposure patterns. Still, workers in previous spills may have been exposed to $PM_{2.5}$ from other sources (e.g., engine emissions) as well as other inhalation hazards (e.g., volatile components of the crude oil, chemical dispersants). Studies that assessed workers' respiratory health in relation to cleanup work have yielded inconsistent findings. A study of the *Tasman Spirit* oil spill found that oil spill workers had worse lung function (FEV1 and FVC) than nonworker controls 1–5 months after the spill (Meo et al. 2008), with stronger

Table 5. $PM_{2.5}$ exposure and lung function among DWH disaster oil spill water workers at home visit ($n = 2{,}316$), stratified by median time from the end of the exposure period to spirometry (1.99 y).

Lung function measures	$PM_{2.5}$ exposure	Shorter follow-up ($n = 1{,}160$) (≤1.99 y)			Longer follow-up ($n = 1{,}156$) (>1.99 y)			Interaction *p*-value
		n (%)	β (95% CI)[a]	*p*-Value	*n* (%)	β (95% CI)[a]	*p*-Value	
	Average exposure							
FEV1 (mL)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.85
	Low	207 (17.8)	−30.7 (−124.1, 62.6)	0.52	243 (21.0)	−77.2 (−158.8, 4.4)	0.06	—
	High	20 (1.7)	−315.4 (−580.4, −50.4)	0.02	48 (4.2)	−37.7 (−201.5, 126.2)	0.65	—
	Per 10 $\mu g/m^3$ increase	—	−24.2 (−46.5, −1.9)	0.03	—	−8.4 (−24.0, 7.1)	0.29	—
FVC (mL)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.82
	Low	207 (17.8)	−11.2 (−121.1, 98.7)	0.84	243 (21.0)	−74.2 (−167.8, 19.5)	0.12	—
	High	20 (1.7)	−286.5 (−598.5, 25.5)	0.07	48 (4.2)	33.7 (−154.4, 221.8)	0.72	—
	Per 10 $\mu g/m^3$ increase	—	−19.7 (−45.9, 6.6)	0.14	—	−2.2 (−20.1, 15.6)	0.81	—
FEV1/FVC (%)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.87
	Low	207 (17.8)	−0.5 (−1.5, 0.5)	0.35	243 (21.0)	−0.6 (−1.6, 0.3)	0.17	—
	High	20 (1.7)	−1.7 (−4.7, 1.2)	0.24	48 (4.2)	−1.4 (−3.3, 0.4)	0.14	—
	Per 10 $\mu g/m^3$ increase	—	−0.2 (−0.4, 0.1)	0.15	—	−0.2 (−0.3, 0.01)	0.07	—
	Cumulative exposure							
FEV1 (mL)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.81
	Low	92 (7.9)	−94.5 (−224.7, 35.6)	0.15	124 (10.7)	−79.2 (−186.1, 27.7)	0.15	—
	Medium	124 (10.7)	18.6 (−95.5, 132.6)	0.75	133 (11.5)	−67.8 (−171.0, 35.5)	0.20	—
	High	11 (1.0)	−581.7 (−934.5, −229.0)	<0.01	34 (2.9)	−53.4 (−246.1, 139.2)	0.59	—
	Per 10 $\mu g/m^3$-d increase	—	−0.5 (−1.3, 0.2)	0.18	—	−0.5 (−1.1, 0.1)	0.08	—
FVC (mL)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.87
	Low	92 (7.9)	−82.1 (−235.6, 71.5)	0.29	124 (10.7)	−113.0 (−235.7, 9.7)	0.07	—
	Medium	124 (10.7)	34.7 (−99.8, 169.2)	0.61	133 (11.5)	−11.0 (−129.5, 107.5)	0.86	—
	High	11 (1.0)	−439.7 (−855.8, −23.6)	0.04	34 (2.9)	−36.2 (−257.3, 184.9)	0.75	—
	Per 10 $\mu g/m^3$-d increase	—	−0.3 (−1.2, 0.6)	0.55	—	−0.3 (−1.0, 0.4)	0.39	—
FEV1/FVC (%)	Ref[b]	933 (80.4)	—	—	865 (74.8)	—	—	0.68
	Low	92 (7.9)	−0.6 (−2.0, 0.9)	0.45	124 (10.7)	0.21 (−1.0, 1.4)	0.73	—
	Medium	124 (10.7)	−0.3 (−1.5, 1.0)	0.65	133 (11.5)	−1.6 (−2.8, −0.5)	0.01	—
	High	11 (1.0)	−4.7 (−8.6, −0.8)	0.02	34 (2.9)	−0.9 (−3.1, 1.2)	0.40	—
	Per 10 $\mu g/m^3$-d increase	—	−0.01 (−0.01, 0.002)	0.14	—	−0.01 (−0.01, −0.002)	0.01	—

Note: —, no data; β, adjusted mean difference; CI, confidence interval; *DWH*, *Deepwater Horizon*; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; mL, milliliters; PM, particulate matter; $PM_{2.5}$, PM with aerodynamic diameter less than or equal to 2.5 μm; Ref, Referent.

[a]Adjusted for age, age^2, sex, race, ethnicity, height, height^2, smoking, secondhand smoke, weight, prespill diabetes, prespill lung disease, education, employment, previous oil industry experience, previous oil spill cleanup work, and residential proximity to the Gulf of Mexico.

[b]The referent group consisted of nearshore workers and offshore workers who did not work on *in situ* burns.

associations observed among those with longer duration of cleanup (Meo et al. 2009). In the *Prestige* oil spill, workers were significantly more likely than nonworkers to report respiratory symptoms up to 5 y after the spill, and higher risks were observed among workers who worked longer and participated in more cleanup activities (Zock et al. 2007, 2012). However, spirometry conducted in the second and sixth year did not show a clear difference in lung function between workers and nonworkers (Rodríguez-Trigo et al. 2010; Zock et al. 2014). In a previous study of the *DWH* oil spill workers, Gam et al. also did not find a difference in lung function between workers and nonworkers 1–3 y after the spill. However, participating in cleanup activities that involved high potential exposure to crude oil and burning oil/gas specifically was associated with lower FEV1 and FEV1/FVC among workers (Gam et al. 2018a, 2018b). The different findings in the oil spill literature may be explained by the varying lengths of follow-up and different control groups used. Additionally, it is possible that the adverse respiratory effects observed in some of the studies were triggered by specific exposures, although none of these studies examined quantitative estimates of chemical exposures. Our study agrees with the previous *DWH* analysis that identified burning emissions as a contributor to workers' lower lung function (Gam et al. 2018b) and further points to burning-related $PM_{2.5}$ as a potential agent responsible for the observed adverse respiratory effect.

A major strength of our study is the careful reconstruction of potential $PM_{2.5}$ exposure using the AERMOD dispersion model and detailed work histories collected from the study participants. In comparison with previous oil spill studies that relied on crude surrogates of exposure (e.g., duration of cleanup), estimates of $PM_{2.5}$ and total hydrocarbons allowed us to investigate putative exposure(s) responsible for the respiratory effects observed among our population. In comparison with other ambient pollution studies, the longer time between exposure and outcome assessment allowed us to assess the effect of $PM_{2.5}$ exposure up to 3 y after exposure ended. In addition, although spirometry was not conducted in a clinical setting, rigorous implementation of the test by trained medical staff and selection of maneuvers according to the ATS/ERS criteria ensured good quality of the lung function data. Last, available data on self-reported diagnosis of lung disease and smoking status allowed us to perform subgroup analyses and identify groups that might be particularly vulnerable to the effect of $PM_{2.5}$ exposure.

Our study also has limitations. First, the exposure estimates assigned to workers contained some degree of uncertainty. Because we lacked data on the exact location of most workers on a daily basis, we created a job-exposure matrix that assigned workers of the same exposure group an exposure estimate that reflected the average daily maximum concentration over their general work area across the burning period. However, given the substantial variation in $PM_{2.5}$ concentrations over the Gulf waters and over time, this approach reduced the variability of exposures among individuals of the same exposure group and resulted in measurement error in the exposure estimates. Exposure variability was possibly largest for workers in the vast nearshore and offshore areas, where air concentrations were higher near and downwind from the burning sites but close to nil outside of the smoke plumes and during nonburn hours. The vast majority of the model receptors in these areas had close-to-zero estimated concentrations ($<0.1\ \mu g/m^3$) on any given day and hour. Thus, average daily maximum exposure values assigned to the nearshore and non-ISB offshore groups (the referent group) were likely an overestimation of many individuals' actual exposure, because most workers in these exposure groups would not have experienced these estimated levels during most of their work period. We do not expect that overestimated exposures for the referent group biased our estimates in the analyses of categorical exposures. The uncertainty in the exposure estimates and their clustered distribution also limited our interpretation of the exposure–response trend analysis. Because a job-exposure matrix primarily based on work location was used to assign exposure estimates, distribution of the average and cumulative daily maximum exposure estimates was clustered. Given that our cumulative and average exposure estimates were not truly continuous, we could not interpret the trend analysis the way it is usually interpreted in air pollution studies with continuously distributed $PM_{2.5}$ exposures (e.g., changes in outcome corresponding to every $10\ \mu g/m^3$ increase in exposure). We focused on the significance of the associations in results to provide a sense of the exposure–response relationship. Effect estimates for the trend analysis were provided in tables to provide some indication of the strength of the association, but caution is strongly advised when trying to interpret these results or comparing them with results from other literature.

Second, because our goal was to assess health risks of exposures specifically from controlled burning to inform future oil spill responders who are considering it as an oil mitigation method, the $PM_{2.5}$ estimates reflected only exposure from ISBs and flaring and did not consider background exposures (Pratt et al. 2020). Because controlled burning activities did not occur every day in the exposure period, we only considered days with burning/flaring in the calculation of average and cumulative exposure estimates. This approach is similar to that used in many other studies that investigated occupational exposure agents that are also present in the ambient environment, whereby average and cumulative exposures were calculated based on measurements on workdays and did not include days without exposure (e.g., weekends, days off) (Andersson et al. 2019; Baker et al. 2016). Nonetheless, it is a limitation that we could not obtain total $PM_{2.5}$ exposure estimates from all sources. To partially address background sources of $PM_{2.5}$ exposures from engine exhaust that could not be accounted for, we excluded land workers in our main analysis and focused on water workers who shared the same source of background exposure (i.e., vessels). In a sensitivity analysis that included land workers, we observed significantly lower lung function measures among land workers in comparison with the referent group. This suggests that land workers might have experienced different exposures from those encountered by water workers, possibly as a result of background $PM_{2.5}$ emissions from land equipment, vehicles, or other unique sources. We lacked the data to examine these exposures. Alternatively, land workers might have differed from water workers in prespill lung function, although a comparison of key indicators of baseline health status (prespill lung disease, prespill diabetes, and BMI) showed distributions similar to those of the referent group. In another sensitivity analysis where we removed the water group with the highest potential variability in the background exposure (i.e., the non-ISB offshore group), we observed only slightly attenuated associations.

Third, there could be bias from unmeasured confounders or imperfect measurement of existing covariates in the models. We did not measure, and were thus unable to account for, coexposure to other occupational pollutants that have been related to lung impairment, including nitric oxides, ozone, and heavy metals (Kurt et al. 2016), although Pratt et al. (2020) indicated possible concentrations of other contaminants likely to have been present in the smoke from the burning based on published emission factors and correlations with $PM_{2.5}$. We adjusted analyses for total hydrocarbons, a proxy for a broad family of chemical compounds from the crude oil, in a secondary analysis and found no noticeable

difference in results. Use of protective equipment (respirators and face masks) might have mitigated inhalation exposures to $PM_{2.5}$ and other occupational pollutants that can reduce lung function. We have no evidence that respiratory protection was used routinely in any situation other than in tank entry and thus did not account for it in our analyses. There could also be a bias if workers were assigned to different jobs based on their lung function or other health factors at the time of spill that were predictive of lung function measurements at the home visit. For instance, workers with poor lung function might have been more likely to be selected into jobs with low exposures. We did not have measures of lung function before the spill, but we included indicators of baseline health (prespill lung disease, prespill diabetes, and smoking) in the model to try to reduce this bias. Also, jobs that exposed workers to high levels of $PM_{2.5}$ typically required specialized skills (Stewart et al. 2018), so prior work experience was arguably a more important predictor of exposure than was workers' baseline health. As shown in Table S2, individuals in the high-exposure group, which consisted mostly of rig workers in the hot zone, were more likely to have had previous experience working in the oil industry or another oil spill cleanup. We used self-reported race and ethnicity as proxies for the downstream effects of socioeconomic disparities that might have changed lung function, but we were not able to disaggregate race to the fullest extent because of the small number of workers in some race groups. We collected information on educational attainment and included it in the model to further account for socioeconomic factors. Last, we used covariates ascertained at enrollment or the home visit as proxies for factors at the time of exposure, although some of them (cigarette smoking, passive smoking, education, and BMI) might have changed over time. However, we expected little change in these covariates over the short span between exposure and time of their ascertainment. We ascertained the timing of some covariates (prespill lung disease and prespill diabetes) and thus knew their values at the time of exposure.

In sum, we found lower FEV1 and FEV1/FVC among workers with higher estimated $PM_{2.5}$ exposures specifically derived from ISBs and flaring of oil and gas. To our knowledge, our study is the first to evaluate the association between potential $PM_{2.5}$ exposure and lung function among oil spill workers. Additional research is needed to evaluate the persistence of effects of high-level, short-term particulate exposure on lung function among workers and among the general public, as well as any benefit conferred by the use of protective equipment and/or work practices.

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