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Personal and community-level exposure to air pollution and daily changes in respiratory symptoms and oxygen saturation among adults with COPD

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Abstract

Background: Air pollution exposure is associated with hospital admissions for Chronic Obstructive Pulmonary Disease (COPD). Few studies have investigated whether daily personal exposure to air pollutants affects respiratory symptoms and oxygenation among COPD patients.

Methodology: We followed 30 former smokers with COPD for up to 4 non-consecutive 30-day periods in different seasons. Participants recorded worsening of respiratory symptoms (sub-categorized as breathing or bronchitis symptoms) by daily questionnaire, and oxygen saturation by pulse oximeter. Personal and community-level exposure to fine particulate matter (PM_{2.5}), nitrogen dioxide (NO₂), and ozone (O₃) were measured by portable air quality monitors and stationary monitors in the Boston area. We used generalized and multi-level linear mixed-effects models to estimate associations of the 24-hour average of each pollutant in the previous day with changes in respiratory symptoms and oxygen saturation.

Results: Higher community-level exposure to air pollutants was associated with worsening respiratory symptoms. An interquartile range (IQR) higher community-level O₃ was associated with a 1.35 (95% CI: 1.07–1.70) higher odds of worsening respiratory symptoms. The corresponding ORs for community-level PM_{2.5} and NO₂ were 1.18 (95% CI: 1.02–1.37) and 1.06 (95% CI: 0.90–1.25), respectively. Community-level NO₂ was associated with worsening

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Author contributions

A.G. performed data analysis and interpretation and wrote the initial draft of the manuscript. A.J.S., C.K., L.N., C.S. assisted in data analysis and manuscript writing. B.A.C. provided expertise in statistical methodology and development of the analytical plan. P.K. guided personal and community-level exposure assessment, calibration of the monitors, and interpretation of the exposure data. M.B.R. was responsible for designing the study and supervised all aspects of the project including data collection, analysis, interpretation, and writing of the manuscript. All authors contributed to the critical revision of the manuscript and approved of the final submitted version.

Supplementary materials

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bronchitis symptoms (OR=1.25, 95% CI: 1.00–1.56), but not breathing symptoms. Personal PM_{2.5} exposure was associated with lower odds of worsening respiratory symptoms (OR=0.91; 95% CI: 0.81–1.01). Personal exposure to NO₂ was associated with 0.11% lower oxygen saturation (95% CI: –0.22, 0.00) per IQR.

Conclusions: In this COPD population, there was a pattern of worsening respiratory symptoms associated with community-level exposure to O₃ and PM_{2.5}, and worsening oxygenation associated with personal exposure to NO₂.

Keywords

Air pollution; Particulate matter; Nitrogen dioxide; Ozone; Chronic obstructive pulmonary disease; Symptoms

1. Introduction

Exposure to air pollution has been identified as one of the greatest public health concerns worldwide (Kelly and Fussell, 2015). A broad range of adverse respiratory health effects have been associated with exposure to ambient air pollution, including lower pulmonary function, higher risk of respiratory infections, and risk of exacerbations of chronic lung diseases, such as asthma and chronic obstructive pulmonary disease (COPD) (Thurston et al., 2017). COPD is characterized by an abnormal inflammatory response to noxious particles and gasses (Gómez and Rodriguez-Roisin, 2002), and adults with COPD appear to be at increased risk of experiencing adverse respiratory effects of ambient air pollution exposure, even at relatively low levels. (Duan et al., 2020)

While several studies have found that outdoor levels of particulate matter less than 2.5 µm in aerodynamic diameter (PM_{2.5}), nitrogen dioxide (NO₂), and ozone (O₃) are associated with higher risk of COPD hospitalization (Gan et al., 2013; Moore et al., 2016; Song et al., 2014), few studies have evaluated if day-to-day differences in exposure to these pollutants affect daily symptoms or oxygenation among non-hospitalized COPD patients living at home. Most epidemiological studies investigating the effects of air pollution on COPD patients have relied only on outdoor pollution measurements from fixed central monitoring locations, which may differ from the personal exposure of the individual to pollution from both outdoor and indoor pollution sources (Butland et al., 2019a). Capturing personal exposure may be especially important in COPD patients, who spend more time at home compared to the general population (Leech and Smith-Doiron, 2006). Nonetheless, outdoor monitors provide relevant exposure information even for those who remain home, because pollution from outdoor sources such as traffic and industry enter the home and pollute the indoor environment. (Huang et al., 2018)

To address these knowledge gaps, we conducted the Study of Pollution and COPD Exacerbation (SPACE) to evaluate whether personal and community-level pollutant exposures are associated with daily worsening of respiratory symptoms and oxygen saturation among patients with COPD living in the Boston area, where pollution levels are generally in compliance with national ambient air quality standards. We hypothesized that

both personal and community-level exposure to PM_{2.5}, NO₂, and O₃ would be associated with worsened daily respiratory symptoms and oxygen saturation among COPD patients.

2. Material and methods

2.1. Study population

The study population consists of 30 former smokers with COPD who were recruited as part of the Study of Pollution and COPD Exacerbation (SPACE) at Beth Israel Deaconess Medical Center in Boston, Massachusetts (MA), United States of America. To be eligible, study participants were required to be former smokers with a ≥ 10 pack-year smoking history and a clinical diagnosis of COPD with at least moderate GOLD Stage II airflow obstruction, defined as forced expiratory volume in 1 second (FEV₁)/forced vital capacity (FVC) ratio of <70% and FEV₁ <80% predicted, using NHANES III prediction formula (HANKINSON et al., 1999). In order for the community-level pollution data to be applicable, all participants were required to have a home address within 50 km of the Harvard air pollution supersite in Boston. Participants with a history of lung cancer, interstitial lung disease, or bronchiectasis were ineligible to take part in the study.

2.2. Data collection

Participants entered the study between 02/24/2017 and 01/17/2019. At study entry, demographic information, height, weight, past medical history, medication history, baseline lung function, and oxygen saturation were obtained. In addition, participants were instructed on how to use a personal air quality monitor (PAM), portable oximeter and were shown how to complete a daily electronic questionnaire (full questionnaire available in online supplement, Appendix A).

Study participants were then observed for up to four non-consecutive months in different seasons in a twelve-month period. During these observation periods, participants recorded daily changes in respiratory symptoms and oxygen saturation using a daily questionnaire, accessed electronically through Research Electronic Data Capture (REDCap) (Harris et al., 2009). This questionnaire was developed to identify self-reported changes in respiratory symptoms (Aaron et al., 2012; Seemungal et al., 1998; Wilkinson et al., 2004). The questionnaire asks about the occurrence of any of the following within the previous 24 h compared to usual state of health: increased breathlessness; increased sputum color; increased sputum amount; cold-like symptoms (such as a runny or blocked nose); increased wheeze or chest tightness; sore throat; increased cough; fever. Furthermore, as in previous work (Scheerens et al., 2022), we categorized respiratory symptoms into breathing symptoms (breathlessness, chest tightness, and wheeze) (“Global Initiative for Chronic Obstructive Lung Disease,” 2019) and bronchitis symptoms (cough, sputum color, and sputum amount) (Miravitlles et al., 2012). In addition, participants measured their daily oxygen saturation (SpO₂) using Nonin GO2 LED Achieve 9571 Finger Pulse Oximeter (Plymouth, Minnesota). The Nonin GO2 device uses the same PureSAT[®] SoO₂ technology as the medical grade oximeters and has a declared oxygen saturation accuracy range of 70%–100% SpO₂ + 2 digits. As an estimate of physical activity, we measured personal 24-hour step count using the portable Fitbit[®] Charge 2 worn by participants each day for

all hours, except during bathing (during which the participants were instructed to charge the device). We also recorded inhaler use during the participants' entry visit into the study, which included inhaled corticosteroids, short-acting beta agonists, long-acting beta agonists, short-acting muscarinic antagonist, long-acting muscarinic antagonist.

2.3. Exposure assessment

We measured personal pollutant exposures using PAMs, and community-level pollutant exposures using an average of Boston-area stationary monitors. For community-level PM_{2.5} and NO₂, we averaged daily concentrations from state-owned monitors in Boston. For O₃, we used the state monitor in the Roxbury neighborhood of Boston, which measures O₃, as previously described (Nurhussien et al., 2021). The PAMs, developed by Atmospheric Sensors Ltd (model 520) in collaboration with investigators in the U.K., were equipped with sensors for temperature and relative humidity, Alphasense[®] electrochemical cells for NO₂ and O₃, and an Alphasense[®] optical particle counter for fine PM_{2.5} (Liu et al., 2019). All personal 24-hour exposure measures for PM_{2.5}, NO₂, O₃, temperature, and relative humidity collected during our study were calibrated against reference monitors prior to data analysis, as previously described (Nurhussien et al., 2021).

2.4. Statistical analyses

We calculated mean 24-hour exposure to pollutants from the PAMs (personal exposure) and outdoor state-owned stationary monitors in the Boston area (community-level exposure). We assessed associations between 24-hour exposure to personal and community-level pollutants and odds of worsening of any respiratory symptoms, and also examined associations with odds of worsening breathing symptoms (defined as breathlessness, chest tightness, wheeze), and bronchitis symptoms (defined as cough, sputum color, and sputum amount) separately. We constructed generalized logistic mixed-effects models using the PROC GLIMMIX function in SAS with a participant-level random effect to account for intra-individual correlations between repeated measurements among the same person, and a participant-observation month-specific random effect to allow daily observations from a single observation month within the same person to be more highly correlated than observations from a different observation month on the same person. To examine associations between 24-hour exposures and oxygen saturation (a continuous outcome), we constructed similar multi-level linear mixed-effects (PROC MIXED) models with a participant-specific random effect, a participant-observation month specific random effect, and normally distributed errors assumed to follow an autoregressive [AR(1)] process to model the serial correlation among the daily time series within the same person's observation month, as previously described (Nurhussien et al., 2021). All models were adjusted for previous-day temperature, humidity, season, age, sex, race, height, weight, total pack-year smoking history, and education. Season was categorized as winter, spring, summer, and fall based on the calendar start dates of each observation month. Moreover, we conducted a secondary analysis to examine if physical activity and inhaler use could affect associations of pollutant exposures with symptoms and oxygenation by additionally adjusting for 24-hour step count and inhaler use in our models. All statistical analyses were performed using SAS 9.4 (Cary, North Carolina, United States of America).

3. Results

3.1. Characteristics of study participants

Baseline measures of study participants were collected at the time of their respective study entry visit and are summarized in Table 1. We collected 3619 daily observations with exposure and outcome data among 30 participants with a mean age of 71.1 ± 8.4 years and were 80% white and 20% black. Household income and education status were generally low. A large majority of participants had a household income of less than \$50,000, and fewer than one third of participants had attained a bachelor's degree of education or higher. Participants were former heavy smokers with more than 50 pack-years of smoking on average. As we have previously noted, the overall frequency of worse-than-baseline respiratory symptoms was low (Sun et al., 2020). Out of the 3619 daily observations, participants reported any worsening of baseline respiratory symptoms on 490 days (13.5%), including 227 days (6.3%) with worsening breathing symptoms and 330 days (9.1%) with worsening bronchitis symptoms.

3.2. Concentration levels of air pollutants

Table 2 displays the distribution of personal and community-level exposure to air pollutants. The median previous-day 24-hour personal exposure to $\text{PM}_{2.5}$ by personal ($8.8 \mu\text{g}/\text{m}^3$) and community-level monitor ($6.0 \mu\text{g}/\text{m}^3$) were within the EPA 24-hour $\text{PM}_{2.5}$ standard of $35 \mu\text{g}/\text{m}^3$ and the annual standard of $12 \mu\text{g}/\text{m}^3$. Median previous-day 24-hour personal and community-level NO_2 concentrations were similar (approximately 7 ppb), while median personal exposure to O_3 was very low (9.6 ppb) compared to the community-level O_3 concentration (26.2 ppb), which was well within the 8-hour ambient air quality standard for O_3 (70 ppb) (Cox et al., 2015; US EPA, 2013). As previously published by our group (Nurhussien et al., 2021), community-level exposures of these pollutants were positively associated with personal exposures measured by the PAMs, however, the magnitude of these associations was modest. For example, for each $10 \mu\text{g}/\text{m}^3$ increase in community-level $\text{PM}_{2.5}$, there was 2 (0.9–3.2) $\mu\text{g}/\text{m}^3$ increase in personal exposure to $\text{PM}_{2.5}$.

3.3. Pollution exposure and respiratory symptoms

The associations between previous-day exposure to air pollutants and worsening respiratory symptoms are shown in Fig. 1. In fully adjusted models, we observed a pattern of positive (adverse) associations between higher community-level exposure to air pollutants and worsening of respiratory symptoms. Every IQR higher community-level exposure to O_3 was associated with a 1.35 (95% CI: 1.07–1.70) higher odds of worsening of any respiratory symptom. The corresponding odds ratios (ORs) for community-level $\text{PM}_{2.5}$ and NO_2 were 1.18 (95% CI: 1.02–1.37) and 1.06 (95% CI: 0.90–1.25), respectively. The association for community-level O_3 was greater for breathing symptoms (OR=1.52, 95% CI: 1.14–2.02), than bronchitis symptoms (OR=1.11, 95% CI: 0.81–1.52), while community-level NO_2 was associated with worsening bronchitis symptoms (OR=1.25, 95% CI: 1.00–1.56) but not breathing symptoms [OR=1.04, 95% CI: 0.86–1.26]. Community-level $\text{PM}_{2.5}$ had similar associations with both breathing and bronchitis symptoms with ORs of 1.17 (95% CI: 0.98–1.40) and 1.23 (95% CI: 1.00–1.51), respectively. Every IQR higher personal exposure to $\text{PM}_{2.5}$ was associated with lower odds of worsening of any respiratory symptoms (OR=0.91;

95% CI: 0.81–1.01) including breathing symptoms (OR=0.91; 95%: 0.81–1.03). Personal exposure to NO₂ and O₃ were not associated with worsening respiratory symptoms (Fig. 1). After adjusting for physical activity and inhaler use in our secondary analyses, we found no significant changes in our primary associations. For example, the association between personal PM_{2.5} and worsening of any respiratory symptoms was unchanged [OR 0.91 (CI 0.81, 1.01)], as was the association between community-level O₃ and worsening of any respiratory symptoms [OR 1.35 (CI 1.07, 1.70) vs. 1.33 (CI 1.04, 1.69)] in models adjusting for prior-day step count and inhaler use. Full results available in supplementary material (Appendix B).

3.4. Pollution exposure and oxygenation

Fig. 2 shows the association between previous-day exposure to air pollutants and daily change in oxygen saturation. Per IQR of previous-day personal NO₂ exposure, oxygen saturation was on average 0.10 (95% CI: -0.21, 0.01) percentage points lower. Personal and community-level exposure to PM_{2.5} and O₃ were not associated with daily oxygen saturation. Full results available in supplementary material (Appendix C).

4. Discussion

In our study population of 30 former smokers with moderate-to-severe COPD living in the Greater Boston area, we found positive associations between community-level air pollutants, including PM_{2.5}, O₃ and NO₂ concentrations and daily worsening of respiratory symptoms. Community-level O₃ and PM_{2.5} were associated with worsening of any respiratory symptoms, while community-level NO₂ was only associated with worsening bronchitis symptoms. On the other hand, personal exposure to PM_{2.5} was paradoxically associated with a lower odds of worsening symptoms. Personal exposure to NO₂, but not PM_{2.5} or O₃, was associated with lower daily oxygen saturation.

Our findings are consistent with a growing number of studies reporting adverse effects of community-level exposure to air pollution on respiratory symptoms among COPD patients. For example, Peacock et al. investigated the effects of outdoor air pollution, measured by local monitors in London, among 94 COPD subjects who recorded their symptoms by filling daily diaries (Peacock et al., 2011). The authors reported that an IQR increase in PM₁₀ (19 µg/m³) was significantly associated with dyspnea (OR=1.13, 95% CI: 1.04–1.23, *p*=0.008) and symptomatic fall in peak expiratory flow (OR=1.12, 95% CI: 1.02–1.25, *p*=0.029). No significant association was observed between outdoor O₃ and symptoms in the Peacock et al. (2011) study, in contrast to our study. On the other hand, a multi-center panel study of patients with COPD and asthma in 4 European cities found that a 10 µg/m³ increase in outdoor O₃ for the same day, previous-day and previous 2 days were positively associated with cough (OR=1.06, 95% CI: 1.01–1.11 for same day and similar for previous 1–2 days), while associations of PM_{2.5} and NO₂ with respiratory symptoms were inconsistent, suggesting that O₃ gas is a more powerful respiratory irritant at ambient concentrations (Karakatsani et al., 2012). In another three-month prospective study among 40 COPD patients living in New Zealand, an IQR increase in outdoor PM₁₀ (35.04 µg/m³) was associated with nighttime chest symptoms (RR=1.38, 95% CI: 1.07–1.78) and an

IQR increase in outdoor NO₂ (9.74 µg/m³) was associated with increased daily rescue medication use (RR=1.42, 95% CI: 1.13–1.79) (Harre et al., 1997). Taken together, these findings support the notion that outdoor gaseous and particulate pollutants can exacerbate daily respiratory symptoms in COPD patients. Differences in the findings of studies on the respiratory effects of ambient PM_{2.5}, NO₂ and O₃ may be partially explained by different pollution levels between regions and over time, and different pollutant mixtures that comprise the ambient PM_{2.5} and gaseous pollution.

Despite the accumulating evidence that exposure to outdoor air pollutants affects COPD symptoms, few studies have focused on indoor exposures. In a Baltimore-based study, Hansel et al. (2013) found that each 10 µg/m³ increase in indoor PM_{2.5} in the main living area was associated with worse dyspnea ($\beta=0.11$, 95% CI 0.01–0.24, $p = 0.06$), more wheezing ($\beta=0.27$, 95% CI 0.11–0.43, $p = 0.001$), and higher risk of exacerbations (OR=1.50, 95% CI 1.04–2.18, $p = 0.03$) among former smokers with COPD (Hansel et al., 2013). Their results showed that exposure to indoor air pollutants, despite overall relatively low concentrations, was associated with worsening respiratory symptoms and increased risk of COPD exacerbations. Nevertheless, in our study population, despite previously finding negative associations between personal exposure to pollutants (especially NO₂) and lower lung function (Nurhussien et al., 2021), we did not observe any adverse effects of personal exposure to air pollution measured by PAMs and respiratory symptoms.

Studies on effects of exposure to air pollutants on oxygen saturation are less common and have shown variable results (Jansen et al., 2005; Pope et al., 1999). A repeated-measures study on elderly subjects living in Steubenville, OH, found that oxygen saturation decreased by 0.18% (95% CI –0.31, –0.06) per 13.4 µg/m³ increase in PM_{2.5} in the previous day (Luttmann-Gibson et al., 2014). A chamber study involving controlled exposure of participants to different concentrations of air pollutants found that exposure to concentrated ambient particles, mainly PM_{2.5} at a concentration of 200 µg/m³ over 2 h, was associated with a statistically significant decrease in oxygen saturation estimated as 0.7% for healthy subjects ($n = 6$) and 0.3% for COPD patients ($n = 18$). In that study, co-exposure to NO₂ did not significantly change the responses to particles, which led the authors to conclude that the observed decreases in oxygen saturation were driven primarily by PM_{2.5} (Gong et al., 2005). In contrast, our study found that personal exposure to NO₂, not PM_{2.5}, was associated with lower oxygen saturation.

Most studies of air pollution health effects have relied on fixed monitoring site data, which may not accurately measure personal exposure to air pollution (Butland et al., 2019b). Stationary, state-owned monitors are the gold standard used for monitoring of ambient air quality and for regulation of air quality standards. Studies in Boston and elsewhere have shown that outdoor air quality is a major determinant of indoor air quality (Brown et al., 2009; Freijer and Bloemen, 2011; Sarnat et al., 2002; Tang et al., 2017; Zou et al., 2009). Estimates from the state-owned monitors provide an unbiased estimate of community-level daily exposure that is not influenced by person-level factors. However, personal exposure to air pollution depends on several factors, including individual activity patterns, housing quality, indoor pollution (e.g. from cooking), and nearby outdoor pollution sources. Recent advances in measurement technologies, such as the portable PAMs used in our study, present

an opportunity to assess the associations between personal exposure to air pollution and health outcomes (Mead et al., 2013). These personal monitors have the advantage of also measuring indoor air quality, where people actually spend most of their time, and may be able to offer a more individualized exposure assessment by capturing differences in exposure due to behaviors and housing characteristics. However, while the personal monitors have been calibrated to stationary monitors, the measurements are more variable and sensitive to environmental and technical factors (Chatzidiakou et al., 2019; Liu et al., 2019; Nurhussien et al., 2021).

A recent study conducted in London with a similar design to the present Boston study, including deployment of PAMs, found that personal exposure to NO₂, but not PM_{2.5}, was associated with a higher odds of COPD exacerbations (Evangelopoulos et al., 2021). Our study found that personal exposure to NO₂ was associated with lower daily oxygen saturation, however, no association was found with symptoms, possibly due to smaller sample size and the low frequency of worsening respiratory symptoms in our study. Results of both studies suggest that personal exposure to NO₂ might be more consistently harmful than PM_{2.5} to the daily respiratory health of COPD patients.

The negative association between personal exposure to PM_{2.5} and COPD symptoms observed in our study was also reported in the London study (Evangelopoulos et al., 2021). A potential reason could be that the total personal exposure to PM_{2.5} measured by the PAMs does not distinguish between indoor- and outdoor-generated particles. These particles come from different sources and therefore have different composition, characteristics, and effects on respiratory health (Evangelopoulos et al., 2020). Additionally, this unexpected finding could be explained by the “cloud-dust effect” phenomenon. Indoor activities on less symptomatic days may be associated with resuspension of the house dust, resulting in increased PM_{2.5} captured by PAMs (Ferro et al., 2004). More studies are needed in the future to identify impacts of particles from different sources on the respiratory health of COPD patients.

Our study has several limitations. Since our study population consists of former smokers with COPD living in an urban environment, our findings may not be generalizable to current smokers or those living in other settings. While we collected a large amount of longitudinal data (up to 120 observation-days per participant), our study only included 30 individuals with moderate-to-severe COPD who did not have a high frequency of symptom exacerbation, which also limits generalizability. We did not ascertain whether a participant ever left home without the PAM, which likely resulted in some misclassification of the personal exposure estimates. We used stationary monitors to estimate community-level exposure to the pollutants. Community-level, state-owned monitors are the gold standard used for monitoring and regulation of air quality standards. While this captures day to day variability in outdoor exposure, it may not capture contributions from more local sources including traffic. This is especially relevant for NO₂ exposure among participants living nearer to major roadways. Lastly, our results could have been affected by unmeasured pollutants, including constituents of PM_{2.5} such as black carbon and ultrafine particles.

Our study also has several strengths. Our longitudinal study design with daily personal measures of exposure, respiratory symptoms, and oxygen saturation allowed us to evaluate how day-to-day variability in individual-level exposures relate to daily respiratory health in a vulnerable population while adjusting for a robust list of potential individual-level and seasonal confounders. We used lightweight, portable exposure monitors, calibrated to gold standard stationary monitors, to measure exposure at the individual level for a prolonged period (4 months) spanning all four seasons. Most studies have relied on community-level monitors or brief sampling periods to estimate exposure to pollution, which may not capture personal exposure to pollutants, especially among COPD patients who spend most of their time at home.

5. Conclusions

In this study of former smokers with COPD living in an urban environment, there was a pattern of worsening respiratory symptoms associated with previous-day community-level exposure to O₃ and PM_{2.5}, and worsening oxygenation associated with personal exposure to NO₂. However, personal exposure to PM_{2.5} was associated with lower odds of worsening symptoms, possibly due to differences in the composition of indoor PM_{2.5}. Future work is needed to determine the effects of personal exposure to air pollution on the respiratory health of COPD patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Declaration of Competing Interest

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Abbreviations:

COPD	chronic obstructive pulmonary disease
PM_{2.5}	fine particulate matter less than 2.5 µm in aerodynamic diameter
NO₂	nitrogen dioxide
O₃	ozone

FEV1	forced expiratory volume in 1 second
FVC	forced vital capacity
PAM	personal air quality monitor
SpO2	oxygen saturation
SD	standard deviation
OR	odds ratio
CI	confidence interval
IQR	interquartile range

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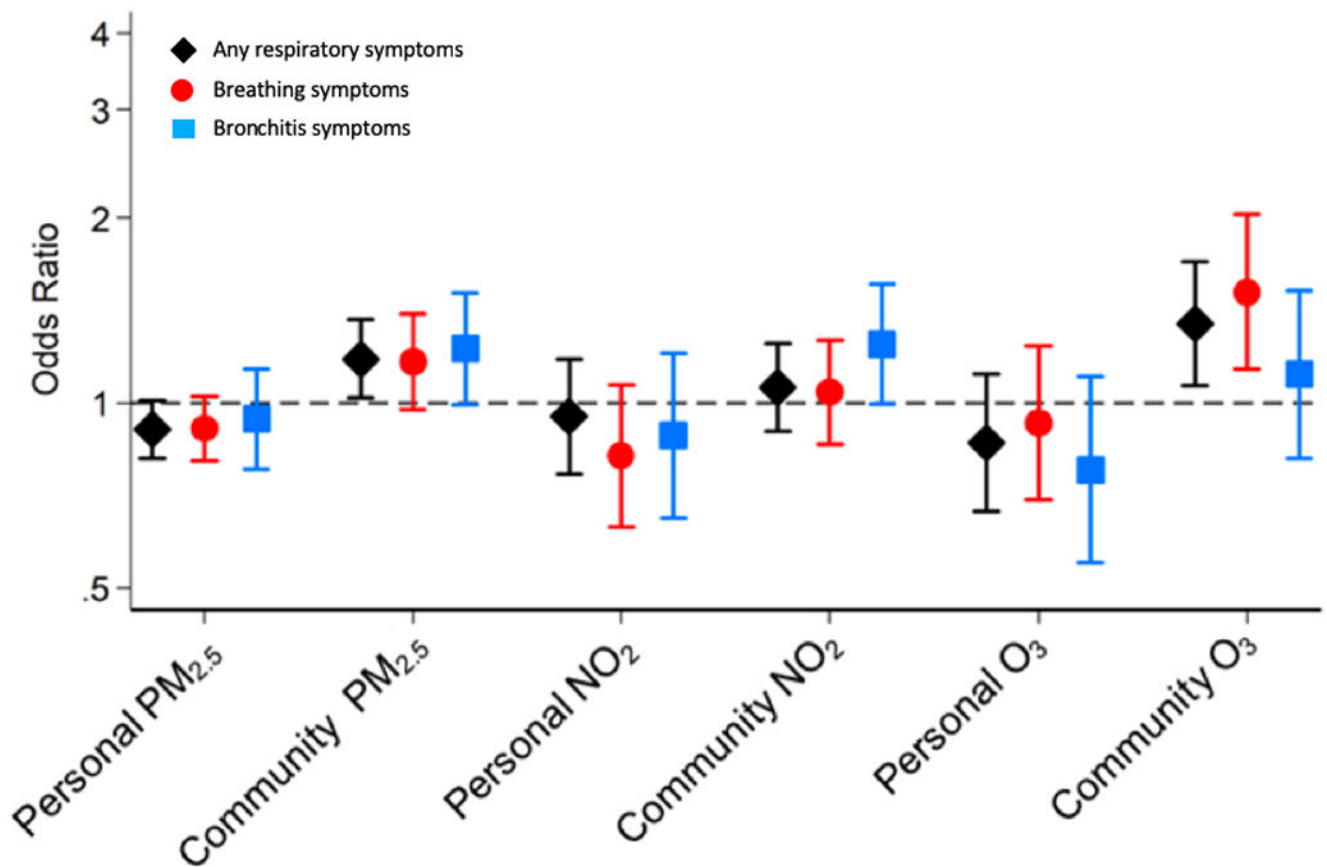


Fig. 1.

Associations of personal and community-level exposure to air pollutants with odds of worsening respiratory symptoms

Legend: Mixed-model analyses showing odds of worsening respiratory symptoms per interquartile range (IQR) difference in previous-day pollutant exposure. All models are adjusted for previous-day temperature, humidity, season, age, sex, race, height, weight, total pack-year smoking history, and education. Black: results for any respiratory symptom, red: results for breathing symptoms (breathlessness, wheeze, tightness of the chest), blue: results for bronchitis symptoms (cough, sputum color, sputum amount). Abbreviations: OR, odds ratio, PM_{2.5}, fine particulate matter less than 2.5 μm in diameter; NO₂, nitrogen dioxide; O₃, ozone.

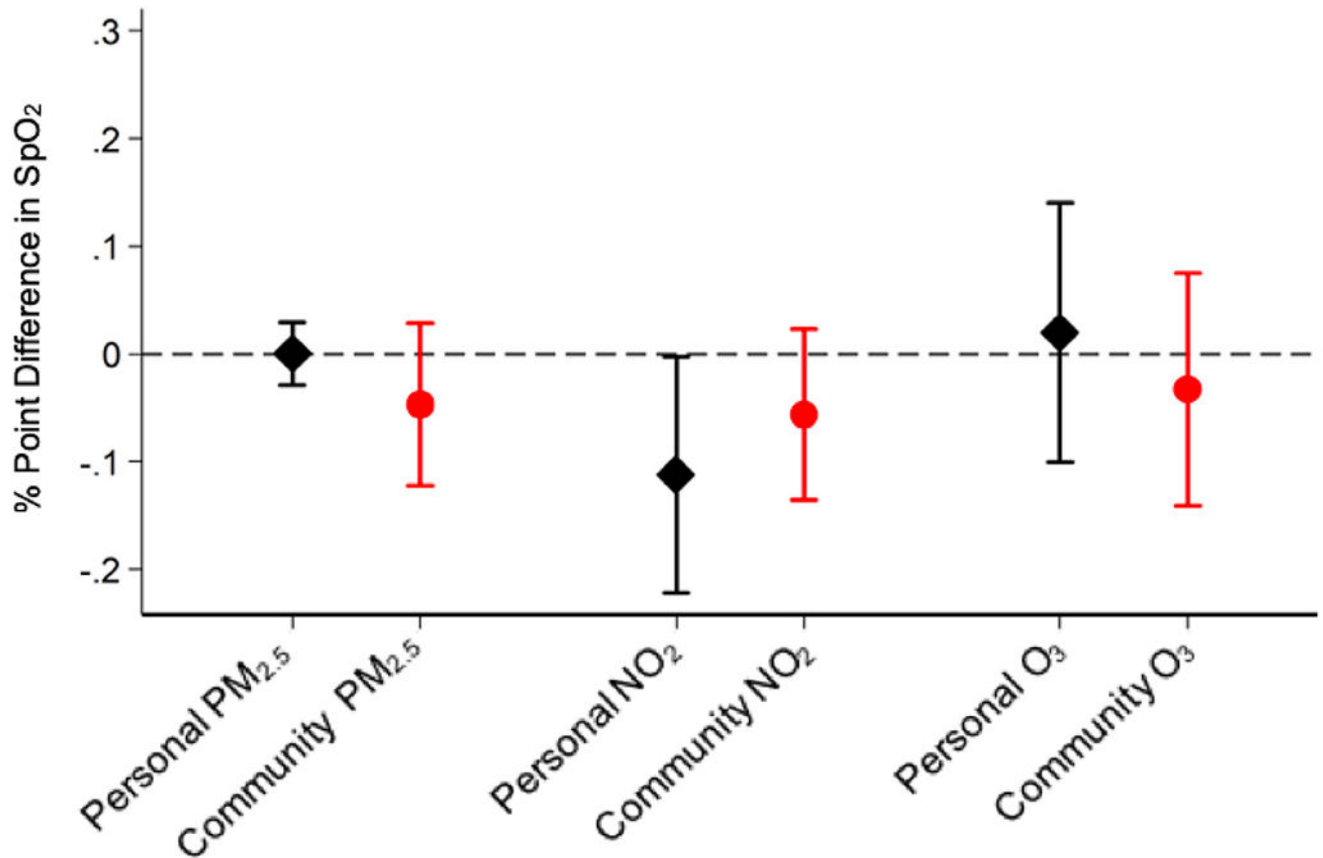


Fig. 2.

Associations of personal and community-level exposure to air pollutants with daily oxygen saturation

Legend: Linear mixed-model analysis showing percentage point difference in oxygen saturation per interquartile range (IQR) difference in previous-day pollutant exposure.

All models are adjusted for previous-day temperature, humidity, season, age, sex, race, height, weight, total pack-year smoking history, and education. Abbreviations: PM_{2.5}, fine particulate matter less than 2.5 μm in diameter; NO₂, nitrogen dioxide; O₃, ozone; SpO₂, blood oxygen saturation.

Table 1Baseline characteristics of participants ($N = 30$).

	Mean (SD) or N (%)
Demographics	
Age, years	71.1 (8.4)
Height, cm	165.1 (10.2)
Weight, kg	85.3 (17.7)
Sex	
Male	14 (46.7)
Female	16 (53.3)
Race	
White	24 (80.0)
Black or African American	6 (20.0)
Income	
<\$25,000	10 (33.3)
\$25,000-\$49,999	10 (33.3)
> \$50,000	9 (30.0)
Other/Missing	1 (3.3)
Education	
Up to grades 12 or GED	8 (26.7)
Some college – Associate’s degree	13 (43.3)
Bachelor’s degree and above	9 (30.0)
Total Pack-Year Smoking History	54.4 (30.7)
Baseline Clinical Measures	
FEV1% predicted	54.3 (14.5)
FVC% predicted	79.9 (15.7)
FEV1/FVC	51.1 (11.5)
Number of observations	
Total number of daily observations	3619 (100)
Days with worsening of any respiratory symptom	490 (13.5)
Days with worsening of breathing symptoms	227 (6.3)
Days with worsening of bronchitis symptoms	330 (9.1)

Data are presented as mean (SD, standard deviation) or as n (% , column percentage), as indicated. FEV1: forced expiratory volume in 1 second; FVC: forced vital capacity.

Table 2

Previous-day community-level and personal exposure to air pollutants.

Exposure	N	Median	IQR
PM _{2.5} , µg/m ³			
Community-Level	3619	6.0	3.6
Personal	2943	8.8	3.8
NO ₂ , ppb			
Community-Level	3619	7.2	4.7
Personal	2943	6.7	5.3
O ₃ , ppb			
Community-Level	3601	26.2	11.8
Personal	2943	9.6	5.2

Abbreviations: PM_{2.5}, fine particulate matter less than 2.5 µm in diameter; NO₂, nitrogen dioxide; O₃, ozone; IQR, interquartile range.