Respiratory Effects of Indoor Heat and the Interaction with Air Pollution in Chronic Obstructive Pulmonary Disease

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Abstract

Rationale: There is limited evidence of the effect of exposure to heat on chronic obstructive pulmonary disease (COPD) morbidity, and the interactive effect between indoor heat and air pollution has not been established.

Objectives: To determine the effect of indoor and outdoor heat exposure on COPD morbidity and to determine whether air pollution concentrations modify the effect of temperature.

Methods: Sixty-nine participants with COPD were enrolled in a longitudinal cohort study, and data from the 601 participant days that occurred during the warm weather season were included in the analysis. Participants completed home environmental monitoring with measurement of temperature, relative humidity, and indoor air pollutants and simultaneous daily assessment of respiratory health with questionnaires and portable spirometry.

Measurements and Main Results: Participants had moderate to severe COPD and spent the majority of their time indoors. Increases in maximal indoor temperature were associated with worsening of daily Breathlessness, Cough, and Sputum Scale scores and increases in rescue inhaler use. The effect was detected on the same day and lags of 1 and 2 days. The detrimental effect of temperature on these outcomes increased with higher concentrations of indoor fine particulate matter and nitrogen dioxide ($P < 0.05$ for interaction terms). On days during which participants went outdoors, increases in maximal daily outdoor temperature were associated with increases in Breathlessness, Cough, and Sputum Scale scores after adjusting for outdoor pollution concentrations.

Conclusions: For patients with COPD who spend the majority of their time indoors, indoor heat exposure during the warmer months represents a modifiable environmental exposure that may contribute to respiratory morbidity. In the context of climate change, adaptive strategies that include optimization of indoor environmental conditions are needed to protect this high-risk group from the adverse health effects of heat.

Keywords: chronic obstructive pulmonary disease; particulate matter; nitrogen dioxide; climate change; heat

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Understanding the health effects of climate change has been identified as a research priority by the American Thoracic Society and other leading health organizations (1–7). The anticipated increases in temperature represent one aspect of climate change that has been associated with adverse health consequences. Global average temperatures are projected to increase by 1.4–5.8% by the end of the century, and heat waves are projected to be more frequent, intense, and longer lasting (8). It is critical to understand the health implications of heat exposure to protect those at greatest risk (9).

Previous population-level studies have demonstrated that heat waves are associated with increases in mortality (10, 11) and that certain populations, including those with underlying respiratory and cardiac disease, are likely to be at increased risk (12, 13). Studies investigating the impact of heat on morbidity using hospitalization and emergency visit records have also identified high-risk subgroups (14–17). To date, disease-specific indicators of morbidity have not been assessed, and studies have rarely used individual-level exposure assessment (18). Few studies have investigated the effect of indoor temperature on respiratory health, and the interactive effect between indoor temperature and indoor air pollution is unknown. It is important to understand the effects of the indoor environment because individuals spend the majority of their time indoors and this is projected to increase in the context of climate change (8, 19). Furthermore, actions such as using air conditioning, cooling centers, and energy-efficient building designs can reduce indoor heat exposure, and these can be deployed immediately at the individual and local levels.

To develop strategies to protect individuals from the adverse consequences of heat exposure, it is necessary to improve our understanding of the specific health consequences among high-risk groups. In this study, we sought to understand the health effects of heat among individuals with chronic obstructive pulmonary disease (COPD), using disease-specific respiratory health outcomes. We hypothesized that (*1*) increases in indoor and outdoor temperature during the warmer months would be associated with increases in daily respiratory symptoms and rescue medication use and decreases in lung function among participants with COPD, and (*2*) increases in air pollution exposure would modify the effects of temperature, enhancing the detrimental effects of increases in temperature on these daily indicators of COPD morbidity.

Methods

Participant Recruitment and Study Design

Participants provided written informed consent, and the Johns Hopkins Medical Institutional Review Board approved the protocol. Participants and methods have been described previously (20). Briefly, participants were former smokers with COPD who were recruited from the Baltimore area and studied at baseline and at 3 and 6 months as part of the COPD and Domestic Endotoxin Study. To determine the health effects of heat, we restricted analysis to the warm weather season, defined as the time between the first and last day that the maximal outdoor temperature exceeded 90°F in Baltimore for each calendar year. Sixty-nine of 84 participants were monitored during this warm weather season. Fifty-four were monitored for 1 week, and 15 were monitored for 2 weeks. Participants completed health and demographic questionnaires, and spirometry was performed according to American Thoracic Society criteria (21, 22).

Environmental Monitoring of Heat and Air Quality

A home environmental assessment was completed; this included a home inspection and continuous environmental monitoring over a 1-week period to capture daily indoor temperature and humidity and weekly particulate matter (PM) and nitrogen dioxide (NO_2). Participants completed a daily activity diary during the environmental monitoring period. Air sampling occurred in the main living area, identified as a room other than the bedroom where the participant reported spending the most time. Additional methods are provided in the online supplement and in a prior publication (20). Outdoor temperature, humidity, and pollution concentrations (PM, NO_2, and ozone) were obtained from publicly available data sets provided by the National Oceanic and Atmospheric Administration and the Environmental Protection Agency (*see* Table E1 in the online supplement) (23, 24).

COPD Daily Respiratory Health Outcomes

Participants completed daily questionnaires and performed spirometry during home environmental monitoring. The validated Breathlessness, Cough, and Sputum Scale (BCSS) contains three questions, each of which assesses a symptom using a Likert-type scale ranging from 0 to 4. A change in total score of 0.3–0.4 is considered mild, whereas a change of 1.0 is considered substantial (25). Handheld spirometry was also performed daily (PiKo-1; nSpire Health, Inc., Longmont, CO). Frequency of rescue inhaler medication use was captured in a daily diary as zero, one, two, three, or four or more times daily.

Statistical Analysis

Descriptive statistics were analyzed using Spearman correlations, chi-square tests, and *t* tests. At each time point, daily maximal temperature was used as the primary exposure variable in generalized estimating equation models to account for repeated measures. Models for indoor and outdoor temperature were run separately and were adjusted for age, sex, education, visit (baseline or 3 or 6 mo), and baseline percent predicted FEV_1. Pack-years of smoking were used to account for disease severity for models in which the primary outcome was lung function.

Models were constructed to account for pollutant concentrations; models of indoor temperature included indoor daily average humidity and weekly average indoor $PM_{2.5}$ and NO_2. Models of outdoor temperature included daily average outdoor humidity, $PM_{2.5}$, NO_2, and ozone. Lag terms were created to assess same-day and subsequent-day health effects. To assess effect modification, interaction terms were created between pollution and temperature variables. To illustrate the temperature effects at given pollution concentrations, models were used to calculate the outcomes of interest for pollutant concentrations at the 25th, 50th, and 75th percentiles, using average or mode values for other variables.

Interaction terms and stratified models were created to investigate whether time spent outdoors modified the effect of outdoor temperature on COPD. Sensitivity analyses were performed using the 95th percentile values of temperature, rather than

the maximal values, and excluding extreme outliers. Analyses were performed with Stata SE statistical software, version 11.0 (Stata Corp, College Station, TX). Statistical significance was defined as a P value <0.05.

Results

Study participants were older individuals with moderate or severe COPD and impaired pulmonary function (Table 1). Between 2009 and 2011, there were 601 participant study days in the warm weather season. Participants spent a substantial amount of time indoors and only went outdoors on 46% of these days. On the days during which participants went outdoors, the mean (SD) time outdoors was 2.0 (2.1) hours. The mean (SD) daily maximal indoor and outdoor temperatures were 80 (7)°F and 85(9)°F, respectively (Figure 1). There was only moderate correlation between daily indoor and outdoor maximal temperatures (Spearman's $\rho = 0.44$, $P < 0.01$).

Eighty-five percent of participants had either central air conditioning or window units. Central air conditioning was reported to have been used on 50% of the study days, and window air conditioning unit use was reported to have been used on 18% of the study days. Participants reported that they did not use air conditioning at all on 37% of the study days that occurred during the warm season. The maximal indoor temperature was lower on days on which participants reported using central air conditioning compared with days on which they did not (mean [SD], 81.4 [6.4] vs. 79.0 [7.0]°F, respectively, $P < 0.01$)

Effect of Indoor Temperature on COPD Morbidity

Increases in maximal daily indoor temperature were associated with increases in symptoms as measured using the BCSS, and with increases in the frequency of rescue inhaler use. These associations persisted even after accounting for indoor pollutant concentrations, including indoor NO_2 and indoor $PM_{2.5}$. For example, a 10°F increase in indoor temperature was associated with a 0.38 increase in BCSS score (95% CI, 0.01–0.67; $P = 0.01$), even after adjustment for indoor relative humidity, $PM_{2.5}$, and NO_2. Inclusion of lag terms in these models suggested that increases in indoor temperatures had an immediate (same-day) effect, as well as an effect that was detectable 1–2 days after exposure.

We found no detectable effect of daily changes in indoor temperature on daily measurements of lung function, using either morning or evening FEV_1 (evening values are shown in Table 2). Sensitivity analyses conducted excluding extreme temperature outliers (>110°F) and using the 95th percentile value of daily indoor temperature yielded similar results (Table E2). An evaluation of interaction terms and stratified models suggested an enhanced effect of indoor temperature on the use of rescue inhalers among participants who had more advanced COPD (coefficient 0.27, $P = 0.06$ for $FEV_1 \leqslant 50\%$ predicted; coefficient 0.01, $P = 0.816$ among those with $FEV_1 \geqslant 50\%$ predicted; P for interaction = 0.048) but did not suggest that disease severity influenced the effect of indoor heat on symptoms. Stratified models also suggested an enhanced effect of indoor temperature on symptoms and rescue inhaler use on days during which participants did not go outdoors (Table E3).

Interactive Effect of Indoor Pollution and Indoor Temperature

In models investigating the effect of indoor temperature on BCSS and on rescue inhaler use, significant positive interactions were detected between $PM_{2.5}$ and temperature (interaction term $P < 0.001$ in both models). Similarly, significant positive interactions were detected between indoor NO_2 and temperature (interaction term $P < 0.05$ in the BCSS model and <0.001 in the rescue inhaler model).

To illustrate the positive interactive effect, Table 3 provides estimates of the effect of a 10°F increase in temperature at given percentiles of indoor pollutant concentrations and demonstrates that the effect of temperature is greater with increasing indoor pollutant concentrations. For example, a participant residing in a home that had an indoor $PM_{2.5}$ concentration at the 25th percentile of the study homes (5 $\mu g/m^3$) would experience an increase of 0.4 in BCSS score (indicative of a mild increase in symptoms) for every 10°F increase in indoor temperature,

Table 1. Participant characteristics (n = 69)

Age, yr	69 (8)
Sex, % male	57
White race, %	90
Smoking, pack-years	56 (30)
Baseline post-bronchodilator FEV_1 % predicted	54 (16)
GOLD stage, %	
II	48
III	40
IV	12
Daily health assessment average values	
BCSS score	2.7 (2.2)
Inhaler use, puffs/d	0.88 (1.3)
Morning FEV_1, L	1.3 (0.6)
Evening FEV_1, L	1.3 (0.6)
Environmental characteristics (n = 601 study days)	
Daily maximal indoor temperature, °F	80.1 (6.7)
Daily average indoor relative humidity, %	40.8 (8.5)
Weekly average indoor $PM_{2.5}$, $\mu g/m^3$	13.1 (15.4)
Weekly average indoor NO_2, ppb	11.3 (11.7)
Days of reported central air conditioning use, %	50
Days of reported window-unit air conditioning use, %	18
Days reported with no air conditioning use, %	37
Days that participants went outdoors, %	47
Time outdoors on days that participants went outdoors, h	1.95 (2.07)
Daily maximal outdoor temperature, °F	84.9 (9.9)
Daily average outdoor $PM_{2.5}$, $\mu g/m^3$	12.8 (6.3)
Daily average outdoor NO_2, ppb	27.5 (11.1)
Daily average outdoor ozone, ppb	35.2 (8.7)
Daily average outdoor relative humidity, %	58.3 (13.1)

Definition of abbreviations: BCSS = Breathlessness, Cough, and Sputum Scale; GOLD = Global Initiative for Chronic Obstructive Lung Disease; PM = particulate matter.
Data are presented as mean (SD) unless otherwise indicated.

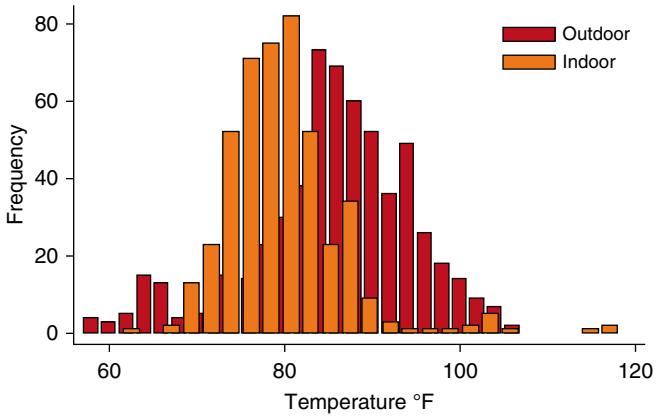

Figure 1. Distribution of daily indoor and outdoor maximal temperatures during the warm weather season. Participants spent most of their time indoors; they went outside on only 47% of study days and spent about 2 hours outdoors on those days. Maximal daily indoor temperatures averaged 80°F, whereas maximal daily outdoor temperature averaged 85°F.

whereas an individual in a home at the 75th percentile (16 μg/m^3) would experience an increase of 1 in BCSS score (indicative of a severe increase in symptoms) (25). Models investigating lung function as the outcome did not demonstrate an interactive effect between indoor temperature and pollutants.

Effect of Outdoor Temperature on COPD Morbidity

Daily maximal outdoor temperature was not significantly associated with respiratory symptoms, rescue medication use, or lung function in the overall cohort (Table 4). Because participants reported going outdoors on fewer than one-half of the study days in the warm weather season, we performed analyses stratified by days on which participants reported going outdoors and assessed for interaction by time outdoors. Stratified models suggested a significant association between increasing outdoor temperature and respiratory symptoms on days during which participants went outdoors. There were no statistically significant interactions between outdoor temperature and outdoor air pollutants, including $PM_{2.5}$, NO_2, and ozone (data not shown).

Table 2. Association between indoor temperature and COPD symptoms, rescue medication use, and lung function

Outcome	Coefficient*	95% Confidence Interval	*P* Value
Breathlessness, Cough, and Sputum Scale			
Daily temperature (limited model)†	0.30	0.00–0.59	0.048
Daily temperature (with humidity, NO_2, $PM_{2.5}$)	0.38	0.01–0.67	0.013
Lag 0	0.30	0.00–0.59	0.048
Lag 1	0.36	0.01–0.70	0.042
Lag 2	0.48	0.12–0.85	0.010
Lag 3	0.10	−0.27–0.47	0.602
Rescue inhaler use			
Daily temperature (limited model)†	0.26	0.09–0.42	0.002
Daily temperature (with humidity, NO_2, $PM_{2.5}$)	0.23	0.06–0.41	0.008
Lag 0	0.26	0.09–0.42	0.002
Lag 1	0.17	-0.02–0.36	0.077
Lag 2	0.21	-0.01–0.42	0.058
Lag 3	−0.02	−0.24–0.20	0.845
Lung function (evening FEV_1)			
Daily temperature (limited model)†	−0.02	−0.05–0.02	0.419
Daily temperature (with humidity, NO_2, $PM_{2.5}$)	−0.01	−0.05–0.02	0.439
Lag 0	−0.02	−0.05–0.02	0.419
Lag 1	−0.01	−0.04–0.03	0.610
Lag 2	−0.01	−0.05–0.02	0.418
Lag 3	−0.02	−0.06–0.02	0.262

Definition of abbreviations: COPD = chronic obstructive pulmonary disease; PM = particulate matter.
*Models include visit, age, sex, education, baseline FEV_1. (Number of pack-years was used instead of baseline FEV_1 in the lung function models).
†Changes are expressed per 10°F increase in indoor temperature.

Discussion

This study is among the first to describe the effect of heat exposure on disease-specific morbidity outcomes in those with COPD, a group that has been identified as at a high risk of the detrimental health effects of heat, and, to the best of our knowledge, it is the first to report an interactive effect between indoor temperature and indoor pollution.

In a cohort of participants with moderate to severe COPD, we found that increases in home indoor temperature during warmer weather were associated with increases in daily indicators of COPD morbidity, including respiratory symptoms and rescue inhaler medication use. There was a positive interaction between temperature and indoor air pollution, including $PM_{2.5}$ and NO_2, such that the effect of indoor heat was greater in the presence of higher indoor air pollutant concentrations.

In this study population, participants spent a great deal of time indoors and ventured outdoors on only one-half of the study days. Outdoor temperature was associated with increased respiratory symptoms on these days. In the context of the anticipated increase in temperatures related to climate change, these findings suggest that adaptive strategies targeting the indoor environment provide an opportunity to minimize the health risks for those with COPD.

Our results are consistent with and extend previous findings that have demonstrated adverse health consequences of heat exposure. Previous studies have largely used ambient data to assign exposure and have linked this to population health

Table 3. Illustration of the interactive effects between indoor temperature and pollutants

Percentile of Indoor Air Pollutant Concentrations	Estimated Effect of 10°F Increase in Temperature at Increasing Indoor $PM_{2.5}$ Concentrations		Estimated Effect of 10°F Increase in Temperature at Increasing Indoor NO_2 Concentrations	
	BCSS Score	Rescue Inhaler	BCSS Score	Rescue Inhaler
25 $PM_{2.5}$ 4.99 μg/m^3 NO_2 4.37 ppb	0.36	0.25	0.12	0.05
50 $PM_{2.5}$ 8.24 μg/m^3 NO_2 6.84 ppb	0.54	0.42	0.20	0.13
75 $PM_{2.5}$ 16.20 μg/m^3 NO_2 13.00 ppb	0.98	0.85	0.41	0.34
95 $PM_{2.5}$ 38.36 μg/m^3 NO_2 34.58 ppb	2.21	2.02	1.12	1.08

Definition of abbreviations: BCSS = Breathlessness, Cough, and Sputum Scale; PM = particulate matter.
The estimated effect of a 10°F increase in temperature becomes larger with increasing indoor pollutant concentrations (NO_2 and $PM_{2.5}$), demonstrated for given percentiles. Models used to predict estimated effect included age, sex, education, baseline FEV_1, indoor humidity, and either indoor $PM_{2.5}$ or indoor NO_2.

effects with compelling results. Such studies have identified elderly individuals and those with underlying cardiac and respiratory diseases, including COPD, as at an increased risk of adverse health effects of heat exposure (12, 15, 26–29).

For example, a time series study across 12 cities in the United States demonstrated increases in deaths attributable to COPD during hot weather, with differences between hot and cold cities. In cold cities, hot temperatures were associated with an increase in the risk of death attributable to COPD by as much as 25% (12) with immediate, same-day effects. In hot cities, the effect of hot temperatures was attenuated and delayed, with a 6% increase in COPD deaths at lags of 3 and 4 days.

A study in New York City examined COPD morbidity using hospitalization data and found that the same-day risk of COPD hospitalization increased by 7.6% for every one-degree Celsius increase above a threshold temperature of 29°C (17), and that there was a detectable but smaller association between temperature and respiratory hospitalization when applying a 1-day lag.

Other studies have used Medicare data to provide estimates of effect that are representative of a broader portion of the population of the United States (15, 30). In a study that included 12.5 million elderly individuals in 213 urban counties in the United States, there was a 4.7% increased risk of hospitalization for COPD for every 10°F increase in ambient temperature, and the findings were not attributable to air pollution health effects (15). Heat and respiratory hospitalizations were associated most strongly on the day of exposure in this study; the effect was still present and significant at a lag of 1 day and no longer detectable at 2 days. The ecologic design of such studies and the potential for bias caused by measurement error in exposure assignment have been limitations; these limitations are addressed in this study, which provides individual-level data.

Researchers who previously investigated the interactive effect of temperature and air pollution have reported mixed results, with little evidence for differences in COPD outcomes. For example, in a study conducted in Brisbane, Australia, PM_{10} modified the effect of temperature on respiratory hospital admissions, but there was no interactive effect for respiratory emergency visits (31). Basu and colleagues examined temperature and mortality in California and did not find that pollution modified the effect of temperature (32). In a study of nine European cities included in the EuroHEAT project, the effect of heat on overall mortality and cardiovascular mortality was increased on high-PM_{10} and high-ozone days, but the interactive effect of these pollutants and heat on respiratory mortality was less evident (33).

Ren and colleagues (31) analyzed data from 98 urban communities in the U.S. National Morbidity, Mortality, and Air Pollution Study and found that ozone modified the risk of cardiovascular mortality. However, their results did not include respiratory mortality as an outcome measure. Zanobetti and Schwartz studied nine cities in the United States during the warm season and did not find evidence that outdoor $PM_{2.5}$ or ozone modified the relationship between increasing outdoor temperature and risk of death (34).

In the current study, we did not find an interaction between the effects of outdoor temperature and ozone or PM. However, there was a consistent signal demonstrating that increases in indoor pollution enhanced the adverse effect of increasing indoor temperature on COPD symptoms and rescue medication use. To our knowledge, this is the first study to report interactive effects of indoor temperature and air pollution on COPD morbidity.

Previous studies linking outdoor heat with hospitalization or death have demonstrated same-day health effects of heat exposure or lag times of 1 day (15). Our findings suggest the health effect of indoor

Table 4. Daily maximal outdoor temperature and COPD health outcomes stratified by whether the participant went outdoors on a given day

Outcomes	Overall (n = 485)		Days Participants Stayed Indoors (n = 250)		Days Participants Went Outdoors (n = 229)		*P* Value for Interaction
	Coefficient (95% Confidence Interval)	*P* Value	Coefficient (95% Confidence Interval)	*P* Value	Coefficient (95% Confidence Interval)	*P* Value	
BCSS score	0.13 (−0.06–0.32)	0.18	0.00 (−0.29–0.29)	0.985	0.38 (0.122–0.628)	0.004	0.045
Rescue inhaler	0.01 (−0.09–0.10)	0.912	0.01 (−0.14–0.16)	0.899	0.03 (−0.10–0.17)	0.619	0.554
Evening FEV_1*	0.00 (−0.02–0.03)	0.830	0.01 (−0.03–0.04)	0.749	−0.01 (−0.06–0.03)	0.534	0.477

Definition of abbreviations: BCSS = Breathlessness, Cough, and Sputum Scale; COPD = chronic obstructive pulmonary disease; PM = particulate matter. Multivariate analysis adjusted for age, sex, education, baseline lung function or smoking history, visit, outdoor $PM_{2.5}$, outdoor NO_2, outdoor ozone, and outdoor relative humidity.
*n = 479 for overall, 187 for days indoors, and 211 for days participants went outdoors for evening FEV_1.

heat exposure is immediate and may persist for 1 to 2 days. Although the mechanisms of health effects in COPD remain incompletely understood, proposed mechanisms include both thermoregulatory responses (35) and bronchoconstrictive effects of heat.

Patients with COPD may have an impaired ability to respond to heat stress. It has been proposed that heatstroke leads to increases in intravascular coagulation caused by the release of IL-1 or IL-6 into the systemic circulation with activation of microvascular thrombosis, which may trigger a respiratory distress syndrome (36, 37). Studies in asthma suggest that breathing hot humid air may result in bronchoconstriction and increased airways resistance that is mediated via cholinergic pathways (38, 39). We did not detect an association between indoor heat and FEV_1. This suggests either that bronchoconstriction did not affect the airways resistance captured by the FEV_1 outcome measurement or that bronchoconstriction was not the mechanism by which participants were affected by the heat.

Limitations

Because this study was limited to the Baltimore region, the results may not be generalizable to other areas of the country. We used education as an indicator of socioeconomic status because household income was not reflective of socioeconomic status in our largely retired population, and this approach may have resulted in some misclassification of socioeconomic status. Indoor air monitoring did not include ozone because previous studies in Baltimore city have documented that this exposure is typically low in homes (40).

Although each participant provided comprehensive and daily characterization of the indoor environment and COPD health outcomes, outdoor measurements of temperature and pollutant concentrations were obtained from central site monitors, which may have resulted in measurement error in assigning outdoor exposure to each individual. This may have contributed to the weaker associations between outdoor temperature and COPD morbidity, although the daily activity diary data would suggest that the short amount of time spent outdoors was also a key contributing factor.

Conclusions

The findings of this study suggest that additional indoor cooling may improve COPD respiratory health during the warmer months and that consideration should be given when traveling outdoors in warmer weather. Furthermore, our finding that increases in indoor air pollution exaggerated the adverse health effects of indoor heat exposure highlights the opportunity to improve COPD health through optimization of the indoor environment. The participants with COPD in this study spent the overwhelming majority of their time indoors during warm weather days.

Although 86% of the participants had some form of air conditioning available, air conditioning was not used on 37% of the study days. We did not find that education level was associated with the use of air conditioning; however, we were limited in our ability to understand the extent to which the use of air conditioning was influenced by financial hardship.

Addressing barriers to air conditioning use may have potential health benefits for those with COPD, and future studies may be needed to fully elucidate the impact of air conditioning use on respiratory health in COPD. However, although air conditioning may provide a short-term solution for this high-risk group (41), it is not without consequences. The use of air conditioning ultimately contributes to the cycle that perpetuates climate change and the steady rise in outdoor temperatures (42). Furthermore, the associated costs are problematic for disadvantaged populations in the United States and around the world.

Ultimately, in addition to adaptive strategies at the individual and population level, mitigation strategies are needed at a policy level to intercept the alarming rise in outdoor temperatures. To effectively address this and to provide the foundation for such policy will require the interdisciplinary work of urban planners, public health officials, engineers, economists, and climate scientists, in addition to medical practitioners. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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