Continuous in-home PM$_{2.5}$ concentrations of smokers with and without a history of respiratory exacerbations in Iowa, during and after an air purifier intervention

Emma M Stapleton, PhD$^1$, Jacob E Simmering, PhD$^1$, Robert B Manges, BA$^1$, Octav Chipara, PhD$^2$, Elizabeth A Stone, PhD$^3$, Joseph Zabner, MD$^1$, Thomas M Peters, PhD$^4$, Ted Herman, PhD$^2$, Phil M Polgreen, MD$^1$, Alejandro P Comellas, MD$^1$

$^1$Department of Internal Medicine, Roy J. and Lucille A. Carver College of Medicine, University of Iowa, Iowa City, Iowa, USA

$^2$Department of Computer Science, College of Liberal Arts and Sciences, University of Iowa, Iowa City, Iowa, USA

$^3$Department of Chemistry, College of Liberal Arts and Sciences, University of Iowa, Iowa City, Iowa, USA

$^4$Department of Occupational and Environmental Health, College of Liberal Arts and Sciences, University of Iowa, Iowa City, Iowa, USA

Abstract

Background—Americans spend most of their time indoors. Indoor particulate matter 2.5 microns and smaller (PM$_{2.5}$) concentrations often exceed ambient concentrations. Therefore, we tested whether the use of an air purifying device (electrostatic precipitator, ESP) could reduce PM$_{2.5}$ in homes of smokers with and without respiratory exacerbations, compared to baseline.

Methods—we assessed PM$_{2.5}$ concentrations in homes of subjects with and without a recent (≤3 years) history of respiratory exacerbation. We compared PM$_{2.5}$ concentrations during one month of ESP use to those during one month without ESP use.

Results—Our study included 19 subjects (53–80 years old), nine with a history of respiratory exacerbation. Geometric mean (GM) PM$_{2.5}$ and median GM daily peak PM$_{2.5}$ were significantly lower during ESP deployment compared to the equivalent time-period without the ESP (GSD=0.50 and 0.37 μg/m$^3$, respectively, p<0.001). PM$_{2.5}$ in homes of respiratory exacerbators tended (p<0.14) to be higher than PM$_{2.5}$ in homes of those without a history of respiratory exacerbation.
Conclusion—Subjects with a history of respiratory exacerbation tended to have higher: mean, median and mean peak PM\textsubscript{2.5} concentrations compared to homes of subjects without a history of exacerbations. The ESP intervention reduced in-home PM\textsubscript{2.5} concentrations, demonstrating its utility in reducing indoor exposures.

INTRODUCTION

Increases in ambient particulate matter (PM) concentration averaged over 24 hours are associated with adverse respiratory outcomes, including increased asthma exacerbations, emergency room visits, and acute lower respiratory tract infections, with susceptible populations especially at risk.\textsuperscript{(1–5)} Vulnerable populations, such as those with chronic obstructive pulmonary disease (COPD), are at a heightened risk of experiencing acute respiratory exacerbations upon exposure to PM, especially fine PM (aerodynamic diameter <2.5 μm), or PM\textsubscript{2.5}.\textsuperscript{(6–9)}

Considerably fewer studies assess continuous PM\textsubscript{2.5} concentrations in homes compared to ambient PM\textsubscript{2.5}, and these may be more strongly correlated with adverse health outcomes than ambient PM because Americans spend most of their time indoors.\textsuperscript{(10–13)} This is exceedingly important since indoor PM\textsubscript{2.5} has been shown to exceed ambient concentrations with indoor peaks surpassing 24-hr average ambient concentrations.\textsuperscript{(14, 15)} Moreover, peak exposures, despite their short duration, are suggested as potentially more effective indicators of health effects than 24-hour samples.\textsuperscript{(16, 17)} However, most studies present average daily, or weekly, PM\textsubscript{2.5} concentrations,\textsuperscript{(18–20)} as opposed to peak exposures.\textsuperscript{(21)} Additionally, while air purification studies exist, we have found relatively few studies assessing its effect on the vulnerable population of current and ex-smokers who are especially susceptible to respiratory exacerbations.\textsuperscript{(9, 22)}

To this end, we characterized continuous indoor PM\textsubscript{2.5} concentrations in homes of 21 current and ex-smokers, with and without respiratory exacerbations by measuring average and peak PM\textsubscript{2.5} during and after an air purifier intervention (one month). This allowed us to test whether the intervention reduced PM\textsubscript{2.5}, and whether homes of respiratory exacerbators had higher indoor PM\textsubscript{2.5} than non-exacerbators.

SUBJECTS AND METHODS

Cohort summary

We selected subjects from an NIH funded cohort (COPDGene, http://www.copdgene.org/) based on respiratory exacerbation history and their house location (≤30 miles of the University of Iowa Hospital). The study was approved by the University of Iowa Institutional Review Board. The selected subjects had similar GOLD (Global Initiative for Obstructive Lung Disease Criteria for COPD) stage and male/female ratio. This is a pilot study with limited eligible participants and resources, so the number of subjects enrolled provide a baseline estimate for future studies. We obtained informed consent from and enrolled twenty-one subjects including 13 females and 8 males. Two of the subjects were active smokers and 19 were former smokers We had access to the history of their respiratory exacerbations over the last three years. The composition of the final study population.
included: 11 females and 8 males, 9 enrollees with a history of exacerbation and 10 without, and subjects ranged in age from 53–80 years old. Due to subjects’ smoking history, from hereafter they will be referred to as smokers. The exacerbator group was comprised of subjects who experienced ≥1 exacerbation per year within the previous three years (2015–2017), while non-exacerbators had not experienced any exacerbations within the prior three years. The subject enrollment was conducted October 23rd-December 6th, 2016, and continuous particle monitoring occurred until May 2017. Subjects completed a survey describing potential PM generating sources and personal habits that may affect exposures, such as cleaning methods, cooking materials and combustion sources.(23)

**Aerosol intervention**

We removed PM ≥20 nm using an electrostatic precipitator device (ESP, OION B-1000, ©OION Technologies) in each home for the first month (average 33 ±3 days) after each participants’ enrollment. (24) The collection electrode’s efficiency varies based on particle type and size; however, use of ESPs for industrial air purification has occurred for decades due to their low electrical demand and high efficiency at capturing combustion particles. (25–27)

The ESP generated 0.036 mg/min of ozone, translating to NAAQS exceedance after approximately 7 hours in an unventilated room; however, at a ventilation rate of 230 L/min the ESP would not exceed NAAQS exposure limits, and all homes participating had active ventilation systems in place. Additionally, the ESP generated 45.8 dB of noise. (25)

**Real-time aerosol monitoring**

During and following ESP deployment, we asked subjects to place a particle monitor (Foobot®—Airboxlab S.A.S.—© 2019, San Francisco, USA and Luxembourg) in the room of their house the subject spends the most time (in the same room as the ESP). After the ESP was removed, the Foobot device remained in situ for the remaining five months. We have previously validated the Foobot device for accuracy and precision.(28) The Foobot measures PM size 0.3 μm to 2.5 μm (called PM$_{2.5}$ by the manufacturer), with a range of 0 to 1300 μg/m$^3$ and precision of ±4 μg or ±20% (https://foobot.io/foobotspecs.pdf). Additionally, the Foobot device records PM$_{2.5}$ continuously and reports an average every 5 minutes. The device transmits this information in real-time using a pre-attached cellular modem connecting the Foobot device to Foobot’s servers. We later retrieved the data using the Foobot application programming interface.

For each subject, we computed the mean and maximum (peak) PM$_{2.5}$ recorded each day. We define the subject’s typical exposure as the average of the daily means, and the subject’s typical daily peak as the average of each subject’s daily peaks over the study period. To provide robustness against measurement error, or potentially aberrant PM$_{2.5}$ concentrations, we also computed a typical baseline exposure as the mean of the daily medians. We also computed a 24-hour moving average of PM$_{2.5}$ when at least 80% of the points that should have been collected during the previous 24-hours were available. Lastly, we compared PM$_{2.5}$ concentrations between groups based on exacerbation status.
The advantage of the Foobot is that it monitors PM$_{2.5}$ continuously; however, participants were asked to set them up, which may have at times led to their placement in high-PM locations. Therefore, we inspected data for outliers. Mean PM$_{2.5}$ in house #15 was 74.6 μg/m$^3$, which was over 15 standard deviations (SD) greater than the remaining sample means, therefore data from this house was removed. Additionally, beginning on the 120th sampling day (March 15th, 2018), multi-hour sustained measurements of PM$_{2.5} > 1000$ μg/m$^3$ were observed in house #13, leading us to remove subsequent data due to apparent measurement error. Both houses were homes of female subjects without a history of exacerbation.

**Effect of electrostatic precipitation on PM$_{2.5}$ concentrations**

We evaluated the mean and peak PM$_{2.5}$ concentrations during and following ESP deployment to assess the efficacy of the ESP as an air purifying device. Specifically, for each subject, the average PM$_{2.5}$ concentration during the first month (ESP deployment) was compared to the PM$_{2.5}$ concentration during the following month (post ESP deployment). For logistical reasons, we were unable to deploy the ESP after the first month of monitoring, and therefore the intervention occurred prior to the “baseline” PM$_{2.5}$ monitoring.

We tested PM$_{2.5}$ data for normality, and differences in geometric means and between the geometric mean of the peaks were normally distributed, while the medians were not. Therefore, we used a paired two-sided t-test for differences in geometric means and geometric means of peaks. A Wilcoxon signed rank test was used to assess differences in the medians.

**Comparison of PM$_{2.5}$ concentrations in homes for patients with and without history of respiratory exacerbations**

We also assessed the potential for differences in PM$_{2.5}$ in the homes of those with a history of exacerbations versus those without, using daily measurements from the entire six-month study period (including when ESP was present). Our hypothesis was that people with a history of exacerbation have greater exposure to PM$_{2.5}$ than people with no history. We divided the sample into the cohort of exacerbators (n=9) and the cohort of non-exacerbators (n=10). As PM$_{2.5}$ was not expected to be normally distributed, we used a generalized linear model with a gamma distribution and identity link function to estimate differences between the groups. This model accounted for both the skew and strictly positive attributes of PM$_{2.5}$ concentrations.

**Code availability**

To access the available code used in our analysis, please contact the corresponding author.

**RESULTS**

**Cohort summary**

Summary demographics of study participants are described in Supplemental Table 1. Fifty-three percent of subjects were female, 60% of females had a history of exacerbation, while five out of nine males had exacerbation (Fisher’s exact test p-value = 1.00). The average age
of female subjects was 70.2 years, whereas the average age of male subjects was 66.2 years (female SD = 8.8, male SD = 6.7 years, p = 0.29).

**Effect of electrostatic precipitation on PM$_{2.5}$ concentrations**

We compared paired overall PM$_{2.5}$ concentrations during the first month of the study, while the ESP was deployed, to the following month (ESP absent). Table 1 describes all study subjects’ mean daily PM$_{2.5}$ concentrations as well as mean and median daily peak PM$_{2.5}$ concentrations during and after ESP deployment. While the ESP was deployed, geometric mean (GM) PM$_{2.5}$ concentration was 1.37 μg/m$^3$ compared with 1.87 μg/m$^3$ (+0.50 μg/m$^3$, 95% CI: 0.29, 0.71 μg/m$^3$) after the ESP was removed. The GM daily peak PM$_{2.5}$ concentrations during deployment were 3.56 μg/m$^3$ and increased to 3.70 μg/m$^3$ following ESP removal (difference = 0.13 μg/m$^3$, 95% CI: –0.04 μg/m$^3$, +0.30 μg/m$^3$). Median daily peak PM$_{2.5}$ concentrations during deployment were 2.79 μg/m$^3$ compared to 3.16 without ESP (p<0.001).

Paired PM$_{2.5}$ concentrations during and after ESP deployment are shown in Figure 1. ESP deployment significantly reduced log-transformed mean daily PM$_{2.5}$ concentrations, and median daily peak PM$_{2.5}$ concentrations (Table 1).

**Comparison of PM$_{2.5}$ in homes for subjects with and without history of respiratory exacerbations**

Daily log PM$_{2.5}$ concentrations in homes of subjects with a history of exacerbation and subjects without a history of exacerbation can be seen in Figure 2.

All analyses showed increased PM$_{2.5}$ in the exacerbator group, whereas only average non-transformed daily peak concentrations were significantly different between exacerbator groups (data not shown). These results demonstrated a trend of increased PM$_{2.5}$ concentrations in the homes of respiratory exacerbators (p<0.14), Supplemental Table 2. No GSDs are provided for differences, as they are not paired in this instance.

**DISCUSSION**

The ESP intervention significantly reduced PM$_{2.5}$ concentrations. GM PM$_{2.5}$ concentrations during intervention were 1.37 μg/m$^3$, while the removal of the ESP increased mean PM$_{2.5}$ concentrations to 1.87 μg/m$^3$ (p=0.0001). GM daily peaks during deployment were 3.56 μg/m$^3$ and 3.70 μg/m$^3$ after deployment; however, this difference was not significant (p=0.11), Table 1. Our results demonstrate variance in daily PM$_{2.5}$ peaks, and relatively low daily averages (GM 1.75–2.10 μg/m$^3$, 95% CI –0.04–0.73 μg/m$^3$, no history of exacerbation and history of exacerbation respectively), Supplemental Table 2.

In-home PM$_{2.5}$ concentrations of subjects with a history of acute respiratory exacerbations tended (p<0.14) to have higher mean and median levels of PM$_{2.5}$ compared to those with no respiratory exacerbation history. GM peak concentrations were greater in homes of subjects with a history of respiratory exacerbations compared to homes of subjects with no history of exacerbations by 0.6 μg/m$^3$, or 17%. Indeed, of the metrics assessed, GM daily peak PM$_{2.5}$ was the most different between homes of subjects with a history of respiratory exacerbations.
and those without (p<0.07), Supplemental Table 2. We are not the first to show significant within-day variability in indoor PM$_{2.5}$ concentrations, and that short-term (within 15 minutes) peaks can well-exceed 24-hour PM$_{2.5}$ concentrations.(14)

We speculate that in-home peak PM$_{2.5}$ may disproportionately contribute to increased risk of respiratory exacerbations in smokers, while mean and median exposures may not independently affect respiratory exacerbation outcomes. For example, it has been proposed that small alterations to lung physiology induced by PM inhalation enhances morbidity in sensitive populations, such as those with COPD,(29) and risk of admission for acute exacerbation of COPD increases with increasing PM$_{2.5}$ exposure (2.3% increased risk per unit increase PM$_{2.5}$ within 9 days).(7) PM$_{2.5}$ exposure is thought to increase pulmonary inflammation by mechanisms including increased levels of pro-inflammatory cytokines, as well as altered phagocytosis.(6, 30, 31) Acute PM$_{2.5}$ exposure can activate polymorphonuclear leukocytes, leading to the secretion of pro-inflammatory cytokine IL-8.(32, 33) We hypothesize that acute peak PM$_{2.5}$ exposures may enhance sensitivity to future exposures in a susceptible population, and that long-term acute (peak) exposures augment the pro-inflammatory effects of PM$_{2.5}$ in this population. Therefore, peak PM$_{2.5}$ exposures may be more clinically relevant.

Focusing on peak exposures poses a statistical analysis challenge. By definition, maximums (peaks) are extreme values with substantially more noise and variability than means. Because peak exposures are so much higher than typical exposures, an air purifying device intervention is unlikely to dramatically change the average peak value, as seen in our study (Table 1). Historically, PM$_{2.5}$ health effects have been assessed using the mean exposure of a pre-determined exposure time-interval, e.g. 8 or 24 hours,(34, 35) and it has been difficult to capture the effect of seemingly arbitrary peak-exposures. However, with the rise of inexpensive sensors capable of logging near real-time PM$_{2.5}$ concentrations (such as the Foobot used in this study), new opportunities exist to better understand the health effects of peak, as well as long-term, exposures to PM$_{2.5}$.

ESP's have historically been utilized in industrial air purification because ESP's are efficient capturers of submicron particles, even in large airstreams.(26, 36) While indoor filter-based air purifiers can be popular due to the lack of ozone generated, the ESP's we selected generate minimal ozone (0.036 mg/min), are relatively quiet (45.8 dB), have low electrical energy demands, do not require filter changes, and are capable of collecting large quantities of PM.(25) Additionally, filter-based air purifiers are typically loud (due to the necessary air pump), have a higher pressure drop, and become less efficient over time,(37) therefore, we considered them to be potentially more intrusive and result in less study compliance throughout our month-long sampling period.

The ESP's we selected were tested for collection efficiency using Arizona Road Dust (ARD), nebulized NaCl, and diesel fumes and preferentially recovered the ARD (70%), which we consider the most similar aerosol in Iowa homes given the high prevalence of crustal metals in our samples.(24, 25) Additionally, the sub-micron (40–400 nm) size range ESP collection efficiency of all PM was ~45%, and in a literature search of submicron collection efficiency, no non-charged filter based technologies (nor techniques not rooted in ESP technology)
reporting submicron collection efficiency was found.\textsuperscript{(25, 37–39)} Smaller particles are thought to likely disproportionately affect health,\textsuperscript{(40, 41)} so for these reasons we determined the O-ION ESP to be the most suitable air purifier to be used in our study.

Limitations of our study include its study design. Our observed association between exacerbation risk and PM\textsubscript{2.5} is ecologic: the assessed PM\textsubscript{2.5} concentrations occurred after collecting exacerbation history (which occurred over the previous three years). While it is unlikely that the indoor environment is radically altered from the time of the exacerbations, concurrent measurement of indoor air quality and exacerbation frequency would provide a more robust measure of association and more importantly, could help us link peaks with exacerbation. Although study participants were requested to place the Foobot in the room where they spent the most time, we were unable to assess compliance in this regard. An additional limitation is that the Foobot devices used in our study were not co-located prior to field use. Our evaluation of the effect of the ESP utilized a crossover design whereby the period after ESP deployment acted as a control for the deployment period. However, this order was not randomized – everyone had the ESP for the first month and the following month served as the control days. It is also possible that external factors, such as seasonal patterns in behavior (e.g., opening windows, or the use of forced air heating), or ambient PM\textsubscript{2.5} concentrations confounded our measured association. The ESP is known to generate ozone, a potential environmental confounder; however, we assessed self-reported symptom severity from the previous day (better, worse, about the same) among study participants, and found no difference. Additionally, comorbidities, such as obesity, have been implicated in enhancing airway and systemic inflammation due to PM\textsubscript{2.5} on respiratory exacerbations in former smokers,\textsuperscript{(42)} which we were unable to account for in this analysis.

Despite these study limitations, we believe the strength of our study include its granular characterization of in-home environments of individuals susceptible of developing respiratory exacerbations (smokers). To our knowledge, no study detailing long-term PM\textsubscript{2.5} concentrations (mean, median and peak) in homes of smokers relative to respiratory health risk, while evaluating the efficacy of an air purifying intervention, exists.

Our data show that subjects with a history of respiratory exacerbation tend to have more PM; however, further testing and better symptom data may further elucidate causality between specific symptom-data and PM\textsubscript{2.5} concentrations. Additionally, our results indicate the use of an air purifying device, such as an ESP, may improve chronic in-home mean and median (peak) PM\textsubscript{2.5} concentrations; however, the ESP did not significantly improve acute exposures (mean peak concentrations), which as discussed above, may be a more important metric for assessing the development of respiratory exacerbations than mean PM\textsubscript{2.5} concentrations.

The use of an ESP reduced average in-home PM\textsubscript{2.5} concentrations, signaling its potential utility in reducing indoor exposures. Homes of subjects with a history of respiratory exacerbation tended to have higher mean, median and mean peak PM\textsubscript{2.5} concentrations compared to the homes of subjects with no history of respiratory exacerbations. Real-time PM monitors continue to improve in price and accuracy and may help identify exposure nuances to the environment of susceptible populations.
Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

We would like to thank all study participants for their willingness to maintain both the Foobot and ESP device throughout the study period.

Funding

This work was partly supported by the Origins of Cystic Fibrosis Airway Disease PPG grant (HL091842–11) funded by the National Institutes of Health. Additional funding for this study was provided by The University of Iowa Institute for Clinical and Translational Science, NIH U54 TR001356 and the Environmental Health Sciences Research Center, NIH P30 ES005605.

REFERENCES

1. Horne BD, Joy EA, Hofmann MG, Gesteland PH, Cannon JB, Lefler JS, et al. Short-term elevation of fine particulate matter air pollution and acute lower respiratory infection. American Journal of Respiratory and Critical Care Medicine. 2018;198(6).
2. Dockery DW, Pope CA. Acute respiratory effects of particulate air pollution. Annual Review of Public Health. 1994;15(1):107–32.
3. Peel JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Todd K, et al. Ambient air pollution and respiratory emergency department visits. Epidemiology. 2005;164–74. [PubMed: 15703530]
4. Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B. Traffic-related air pollution near busy roads: the East Bay Children’s Respiratory Health Study. American Journal of Respiratory and Critical Care Medicine. 2004;170(5):520–6. [PubMed: 15184208]
5. Clark NA, Demers PA, Karr CJ, Koehoorn M, Lencar C, Tamburic L, et al. Effect of early life exposure to air pollution on development of childhood asthma. Environmental Health Perspectives. 2010;118(2):284–90. [PubMed: 20123607]
6. Ni L, Chuang C-C, Zuo L. Fine particulate matter in acute exacerbation of COPD. Frontiers in Physiology. 2015;6:294. [PubMed: 26557095]
7. Kumar N, Liang D, Comellas A, Chu AD, Abrams T. Satellite-based PM concentrations and their application to COPD in Cleveland, OH. Journal of Exposure Science and Environmental Epidemiology. 2013;23(6):637–46. [PubMed: 24045428]
8. Sint T, Donohue JF, Ghio AJ. Ambient air pollution particles and the acute exacerbation of chronic obstructive pulmonary disease. Inhalation Toxicology. 2008;20(1):25–9. [PubMed: 18236218]
9. Hansel NN, McCormack MC, Belli AJ, Matsui EC, Peng RD, Aloe C, et al. In-home air pollution is linked to respiratory morbidity in former smokers with chronic obstructive pulmonary disease. American Journal of Respiratory and Critical Care Medicine. 2013;187(10):1085–90. [PubMed: 23525930]
10. Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, et al. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. Journal of Exposure Science and Environmental Epidemiology. 2001;11(3):231–52.
11. Wheeler AJ, Wallace LA, Kearney J, Van Ryswyk K, You H, Kulka R, et al. Personal, indoor, and outdoor concentrations of fine and ultrafine particles using continuous monitors in multiple residences. Aerosol Science and Technology. 2011;45(9):1078–89.
12. Gao M, Cao J, Seto E. A distributed network of low-cost continuous reading sensors to measure spatiotemporal variations of PM2.5 in Xi’an, China. Environmental Pollution. 2015;199:56–65. [PubMed: 25618367]
13. Jiao W, Hagler G, Williams R, Sharpe R, Brown R, Garver D, et al. Community Air Sensor Network (CAIRSENSE) project: evaluation of low-cost sensor performance in a suburban environment in the southeastern United States. Atmospheric Measurement Techniques. 2016;9(11):5281–92. [PubMed: 32802212]
14. Ramachandran G, Adgate JL, Hill N, Sexton K, Pratt GC, Bock D. Comparison of short-term variations (15-minute averages) in outdoor and indoor PM2.5 concentrations. Journal of the Air & Waste Management Association. 2000;50(7):1157–66. [PubMed: 10939209]

15. Fromme H, Diemer J, Dietrich S, Cyrys J, Heinrich J, Lang W, et al. Chemical and morphological properties of particulate matter (PM10, PM2.5) in school classrooms and outdoor air. Atmospheric Environment. 2008;42(27):6597–605.

16. Michaels RA, Kleinman MT. Incidence and apparent health significance of brief airborne particle excursions. Aerosol Science & Technology. 2000;32(2):93–105.

17. Adams H, Nieuwenhuijsen M, Colville R, McMullen M, Khandelwal P. Fine particle (PM2.5) personal exposure levels in transport microenvironments, London, UK. Science of the Total Environment. 2001;279(1–3):29–44. [PubMed: 11712603]

18. Kingham S, Meaton J, Sheard A, Lawrenson O. Assessment of exposure to traffic-related fumes during the journey to work. Transportation Research Part D: Transport and Environment. 1998;3(4):271–4.

19. Ruuskanen J, Tuch T, Ten Brink H, Peters A, Khlystov A, Mirme A, et al. Concentrations of ultrafine, fine and PM2.5 particles in three European cities. Atmospheric Environment. 2001;35(21):3729–38.

20. He K, Yang F, Ma Y, Zhang Q, Yao X, Chan CK, et al. The characteristics of PM2.5 in Beijing, China. Atmospheric Environment. 2001;35(29):4959–70.

21. Onabat, Stakeeva B. Personal exposure of commuters in public transport to PM2.5 and fine particle counts. Atmospheric Pollution Research. 2013;4(3):329–35.

22. Karottki DG, Spilak M, Frederiksen M, Gunnarsen L, Brauner EV, Kolarik B, et al. An indoor air filtration study in homes of elderly: cardiovascular and respiratory effects of exposure to particulate matter. Environmental Health. 2013;12(1):116. [PubMed: 24373585]

23. National Academies of Sciences, Engineering, Medicine, editors. Sources of Indoor Particulate Matter. Health Risks of Indoor Exposure to Particulate Matter: Workshop Summary; 2016; Washington (DC).

24. Parker GJ, Ong CH, Manges RB, Stapleton EM, Comellas AP, Peters TM, et al. A Novel Method to Collect and Chemically Characterize Milligram Quantities of Airborne Indoor Particulate Matter. Aerosol and Air Quality Research. 2019.

25. Ong CH. Electrostatic precipitator to collect large quantities of particulate matter [MS]. Iowa City, IA: University of Iowa; 2017.

26. Parker KR. Why an electrostatic precipitator? Applied Electrostatic Precipitation: Springer; 1997 p. 1–10.

27. McCain JD, Gooch JP, Smith WB. Results of field measurements of industrial particulate sources and electrostatic precipitator performance. Journal of the Air Pollution Control Association. 1975;25(2):117–21.

28. Sousan S, Koehler K, Hallett L, Peters TM. Evaluation of consumer monitors to measure particulate matter. Journal of aerosol science. 2017;107:123–33. [PubMed: 28871212]

29. Gordon T, Nadziejko C, Schlesinger R, Chen LC. Pulmonary and cardiovascular effects of acute exposure to concentrated ambient particulate matter in rats. Toxicology Letters. 1998;96:285–8. [PubMed: 9820679]

30. Miyata R, van Eeden SF. The innate and adaptive immune response induced by alveolar macrophages exposed to ambient particulate matter. Toxicology and applied pharmacology. 2011;257(2):209–26. [PubMed: 21951342]

31. Xing Y-F, Xu Y-H, Shi M-H, Lian Y-X. The impact of PM2.5 on the human respiratory system. Journal of Thoracic Disease. 2016;8(1):E69. [PubMed: 26904255]

32. McCaill RW, Holden EP, Blackwell TR, Christman JW. Leukotriene B4 stimulates human polymorphonuclear leukocytes to synthesize and release interleukin-8 in vitro. American journal of respiratory cell and molecular biology. 1994;10(6):651–7. [PubMed: 8003341]

33. Marchini T, Magnani ND, Paz ML, Vanasco V, Tasat D, Maglio DG, et al. Time course of systemic oxidative stress and inflammatory response induced by an acute exposure to Residual Oil Fly Ash. Toxicology and applied pharmacology. 2014;274(2):274–82. [PubMed: 24321338]
34. Aarnio P, Yli-Tuomi T, Kousa A, Mäkelä T, Hirsioko A, Hämeri K, et al. The concentrations and composition of and exposure to fine particles (PM2.5) in the Helsinki subway system. Atmospheric Environment. 2005;39(28):5059–66.
35. Pillarisetti A, Carter E, Rajkumar S, Young BN, Benka-Coker ML, Peel JL, et al. Measuring personal exposure to fine particulate matter (PM2.5) among rural Honduran women: A field evaluation of the Ultrasonic Personal Aerosol Sampler (UPAS). Environment international. 2019;123:50–3. [PubMed: 30496981]
36. Mizuno A Electrostatic precipitation. IEEE Transactions on Dielectrics and Electrical Insulation. 2000;7(5):615–24.
37. Lehtimäki M, Heinonen K. Reliability of electret filters. Building and Environment. 1994;29(3):353–5.
38. Jaworek A, Krupa A, Sobczyk AT, Marchewicz A, Szudyga M, Antes T, et al. Submicron particles removal by charged sprays. Fundamentals. Journal of Electrostatics. 2013;71(3):345–50.
39. Saiyasitpanich P, Keener TC, Khang S-J, Lu M. Removal of diesel particulate matter (DPM) in a tubular wet electrostatic precipitator. Journal of Electrostatics. 2007;65(10–11):618–24.
40. Knol AB, de Hartog JJ, Boogaard H, Slottje P, van der Sluijs JP, Lebret E, et al. Expert elicitation on ultrafine particles: likelihood of health effects and causal pathways. Particle and Fibre Toxicology. 2009;6(1):19. [PubMed: 19630955]
41. Kumar P, Morawska L, Birmili W, Paasonen P, Hu M, Kulmala M, et al. Ultrafine particles in cities. Environment international. 2014;66:1–10. [PubMed: 24503484]
42. McCormack MC, Belli AJ, Kaji DA, Matsui EC, Brigham EP, Peng RD, et al. Obesity as a susceptibility factor to indoor particulate matter health effects in COPD. European Respiratory Journal. 2015;45(5):1248–57. [PubMed: 25573407]
Novelty of study

Our work characterizes particulate matter (PM) air pollution concentrations in homes of study subjects with and without respiratory exacerbations. We demonstrate that PM concentrations tend to be higher in homes of participants with respiratory exacerbations, and that the use of an inexpensive air purifier resulted in significantly lower daily average PM concentrations than when the purifier was not present. Our results provide a helpful intervention strategy for purifying indoor air and may be useful for susceptible populations.
Figure 1.
Log-transformed PM$_{2.5}$ concentrations during and after ESP deployment. Each line connects a single subject’s PM$_{2.5}$ without and with an ESP. Differences in PM$_{2.5}$ before- and after-ESP deployment were significant, except for the mean daily peak PM$_{2.5}$. 
Figure 2. Average daily, average peak daily, and median peak daily PM$_{2.5}$ concentrations, grouped by respiratory exacerbation status; to be considered within the exacerbator group, subjects had experienced $\geq$1 exacerbation per year within the previous three years (2015–2017), while non-exacerbators had not experienced any exacerbations within the prior three years. The box plots represent the interquartile range (Q1-Q3, or 25–75 percentile) and lines inside indicate means. Whiskers represent 95% confidence intervals.
Table 1.
Mean, median and maximum PM$_{2.5}$ concentrations during and proceeding ESP deployment. Mean, median, and maximum measurements were calculated using average daily measurements.

| Daily Measure          | No ESP (μg/m$^3$) [GSD] | +ESP (μg/m$^3$) [GSD] | Difference (μg/m$^3$) [GSD] | 95% CI         | p     |
|------------------------|-------------------------|-----------------------|-----------------------------|----------------|-------|
| Mean PM$_{2.5}$        | 1.87 [0.40]             | 1.37 [0.59]           | 0.50 [0.44]                 | 0.29–0.71      | 0.0001|
| Mean daily peak PM$_{2.5}$ | 3.70 [0.87]             | 3.56 [0.91]           | 0.13 [0.35]                 | −0.04–0.30     | 0.1144|
| Median daily peak PM$_{2.5}$ | 3.16 [0.85]             | 2.79 [1.04]           | 0.37 [0.53]                 | NA             | 0.0008|