

# Effectiveness of a portable air cleaner in removing aerosol particles in homes close to highways

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## Abstract

Outdoor traffic-related airborne particles can infiltrate a building and adversely affect the indoor air quality. Limited information is available on the effectiveness of high efficiency particulate air (HEPA) filtration of traffic-related particles. Here, we investigated the effectiveness of portable HEPA air cleaners in reducing indoor concentrations of traffic-related and other aerosols, including black carbon (BC), PM<sub>2.5</sub>, ultraviolet absorbing particulate matter (UVM) (a marker of tobacco smoke), and fungal spores. This intervention study consisted of a placebo-controlled cross-over design, in which a HEPA cleaner and a placebo "dummy" were placed in homes for 4-weeks each, with 48-hour air sampling conducted prior to and during the end of each treatment period. The concentrations measured for BC, PM<sub>2.5</sub>, UVM, and fungal spores were significantly reduced following HEPA filtration, but not following the dummy period. The indoor fraction of BC/PM<sub>2.5</sub> was significantly reduced due to the HEPA cleaner, indicating that black carbon was particularly impacted by HEPA filtration. This study demonstrates that HEPA air purification can result in a significant reduction of traffic-related and other aerosols in diverse residential settings.

## KEY WORDS

black carbon, fungi, HEPA air cleaner, PM<sub>2.5</sub>, tobacco smoke, traffic-related air pollution (TRAP)

## 1 | INTRODUCTION

Traffic is a major source of outdoor air pollution. Approximately 11.3 million people (or 3.7% of the US population) live within 150 m of a major highway placing them at increased likelihood of exposure to traffic-related air pollution (TRAP).<sup>1</sup> The primary particles from vehicle exhaust emissions typically fall within the size range of

particulate matter less than 2.5  $\mu\text{m}$  (PM<sub>2.5</sub>) or ultrafine particles less than 0.1  $\mu\text{m}$  (UFP), as they consist mainly of carbonaceous agglomerates with diameters in the size range from 0.05 to 1  $\mu\text{m}$ .<sup>2</sup> Traffic-related aerosol particles are an important component of PM<sub>2.5</sub>.<sup>3</sup> In larger metropolitan areas that are affected by year-round particle pollution, motor vehicle traffic was identified as a major source of PM<sub>2.5</sub>.<sup>4,5</sup> Black carbon (BC) is a commonly used as a marker for

TRAP, as it is typically associated with incomplete combustion of fossil fuels, and has rare indoor sources with the exception of possibly candles, kerosene lamps or charring of food.<sup>6,7</sup> While there are other indoor or outdoor sources of PM<sub>2.5</sub> or UFP,<sup>8,9</sup> traffic can be a major component, particularly in locations nearby major roads.<sup>10,11</sup> The fraction of BC/PM<sub>2.5</sub> can be an indicator of incomplete combustion sources and combustion efficiency.<sup>12</sup>

Poor indoor air quality has become important health concern, especially since people in the United States spend 87% of their time indoors.<sup>13</sup> Traffic-related airborne particles can infiltrate a building and adversely affect the indoor air quality.<sup>14,15</sup> Exposure to these particles has been associated with enhanced aeroallergen sensitization, exacerbation of existing asthma, and the incidence of asthma among young and adolescent children.<sup>16-21</sup> Studies have shown an increased risk of cardiovascular disease in individuals living near major roadways, implicating traffic air pollutants such as PM<sub>2.5</sub>, UFP, and BC as potential sources.<sup>22-27</sup> Indoor exposure to microorganisms, microbial cell debris, allergens, and other particles in house dust has also become an area of interest in asthma and allergy research because of the potential adverse health effects.<sup>28,29</sup>

Portable air cleaners with high efficiency particulate air (HEPA) filtration and other high efficiency media have been evaluated for use in homes for the removal of smoke, dust, and fungal spores. Cheng et al. showed that the air cleaner is useful in removing pollen grains and fungal spores; however, these were the only aerosols evaluated.<sup>30</sup> Batterman et al. evaluated particulate matter removal utilizing HEPA air cleaners in asthmatic children's bedrooms finding that filters reduced total PM levels.<sup>31</sup> Padró-Martínez et al. investigated HEPA filtration reduction of UFP in public housing near a highway and found a median particle percent reduction to be 47%.<sup>23</sup> PM<sub>2.5</sub> concentrations have been shown to be reduced by 36%<sup>32</sup> and 60%<sup>33</sup> when using portable HEPA air cleaners in TRAP-impacted homes. Although aforementioned intervention studies<sup>23,32,33</sup> were conducted in homes close to traffic sources, the targeted particulate pollutants (PM<sub>2.5</sub> and UFP) were not specific to traffic pollution, and no other traffic pollution indicators were evaluated.

Recent studies have also revealed that air filtration is beneficial for the health of occupants with the largest potential benefits being reductions in morbidity and mortality. Several groups have examined the effect of air filtration interventions on asthma and allergy symptoms.<sup>34-44</sup> We have previously demonstrated that decreased exposure to TRAP, calculated by a land-use regression model, has a clinically significant impact on asthma control in adults.<sup>45</sup> Living near a major roadway makes this exposure essentially unavoidable. Thus, there is a strong need for HEPA air cleaners to be fully validated through an intervention study for the reduction of traffic-related aerosols for the health benefit of the occupants.

Recently, our group evaluated several portable HEPA air cleaners in a controlled laboratory setting and identified one model for further investigation through this intervention study. The purpose of the present study was to investigate the effectiveness of the selected HEPA air cleaner in reducing traffic-related air pollutants, in which black carbon (BC) was used as a surrogate for TRAP,

## Practical Implications

- Traffic-related air pollution (TRAP) represents a growing public health concern worldwide as a large portion of the population is moving to major metropolitan areas and reside near major roadways.
- Chronic and acute diseases have been associated with traffic aerosols, and therefore, a viable solution to improve the indoor air quality for residences with unavoidable exposures to traffic sources is important.
- This study demonstrated that HEPA air cleaners address the need in reducing exposure to TRAP, measured as black carbon in the current study, while also reducing exposure to other aerosols including PM<sub>2.5</sub>, tobacco smoke, and fungal spores.

especially diesel particles. Other studied particulate pollutants included PM<sub>2.5</sub> and UFP that can have strong traffic-related sources, as well as tobacco smoke and fungal spores that are not emitted from traffic. We hypothesized that the HEPA air cleaner significantly reduced traffic-related airborne particles, such as BC, PM<sub>2.5</sub>, and UFP, along with other aerosols, such as fungal spores and tobacco smoke. The overall goal was to determine the effectiveness of removing black carbon (a surrogate for traffic-related particles), and other aerosols of concern, in the indoor environment with the utilization of a HEPA air cleaner under real-world conditions.

## 2 | MATERIALS AND METHODS

### 2.1 | Home selection and study design

This intervention study consisted of a randomized placebo-controlled cross-over design. Study subjects were recruited from participants of the Cincinnati Childhood Allergy and Air Pollution Study and Cincinnati Children's Hospital Medical Center Asthma Clinic. Eligibility criteria for study enrollment included children (age 10-15) with asthma having a primary residence <500 m from a major roadway or an elevated concentration of elemental carbon as estimated using a previously validated land-use regression model (ECAT score of at least 0.33).<sup>46</sup> Major roads were defined as state highways or federal interstates with an average daily truck count of more than 1000. Participants were randomly selected to have either "HEPA" treatment or a placebo "dummy" period first which lasted 4 weeks. The dummy period was a placebo-control, in which the carbon prefilter remained in the air cleaner, but the HEPA filter was removed; the device was turned on during the 4-week timeframe. Subjects were not aware whether a HEPA treatment was implemented or it was a dummy period. After the first 4-weeks, there was a 4-week washout period in which no device was in the home. Previous studies have demonstrated that a

1-week washout period was utilized to restore particulate and ambient exposures to baseline levels.<sup>47</sup> Subsequently, the alternative, either the HEPA air cleaner or the dummy air cleaner, was in the home for 4-weeks. Using a checklist, the research team recorded observations regarding the characteristics of the study home, including age and type of building, number of occupants, type of ventilation, indications of moisture damage, size of home, frying food, candle burning, wood burning fire, and the presence of dogs, cats, and smokers. Conditions during sampling such as opening windows, odors, and cleaning activities were also recorded. This study required and received approval from the Institutional Review Board of the University of Cincinnati.

## 2.2 | Air cleaner selection

In a previous laboratory study, 21 air cleaners were considered, and 6 air cleaners were selected for the assessment of their efficiency in removing airborne diesel particles, cost (including unit cost, replacement filters, and energy usage), and noise level. A community advisory board ranked the units based on this information, and the Whirlpool Whispure (Model AP51030K, Austin, TX) was selected for the intervention study. The Whirlpool Whispure HEPA filter has been designed to capture 99.97% of 0.3  $\mu\text{m}$  particles. For diesel particles, the selected air cleaner had a clean air delivery rate (CADR) ranging from 217 to 343  $\text{ft}^3/\text{min}$  (0.10–0.16  $\text{m}^3/\text{s}$ ) (minimum to maximum speed).<sup>36</sup> The air cleaner was placed in the asthmatic child's bedroom, and the setting was chosen to provide an appropriate CADR based on the size of each bedroom. During the dummy treatment, the lowest setting was always selected yielding an exhaust of 2.9 m/s. For HEPA treatment, the exhaust velocity ranged from 11.8 to 14.1 m/s, based on the size of the room to provide an appropriate CADR. HEPA and dummy air cleaners were placed 38 cm from walls and corners, not within 15 cm of a vent or intake, with no obstructions in front of or above it, and not directly adjacent to air sampling devices. Electric monitors (P3 International, New York, NY) were attached to monitor the use of the air cleaner during the HEPA treatment and dummy period.

## 2.3 | Sampling and analysis methods

Air sampling was performed indoors in the child's bedroom as well as outdoors of each residence for 48 hours prior to ("baseline") and during the end of treatment of each 4-week installation of both the HEPA air cleaner and the dummy unit. A baseline was collected prior to both the HEPA and dummy treatments, and each baseline was analyzed with the respective treatment. The HEPA and dummy units were operating during the respective treatment 48-hour sampling period. Outdoor sampling occurred on the same premises typically on a deck, patio, or porch. Sampling stations provided a consistent 1 meter sampling height. Personal Modular PM<sub>2.5</sub> impactors (SKC Inc., Eighty-Four, PA) with 37-mm polytetrafluoroethylene (PTFE) filters with a polymethylpentene ring and a pore size of 2  $\mu\text{m}$  (Pall, Port Washington, NY) were deployed operating at a flow rate of 3 L/

min. The filters were analyzed gravimetrically before and after sampling for PM<sub>2.5</sub> mass on an Measurement Technology Laboratories automated weighing system after equilibration for 24 hours at constant temperature and humidity and by optical absorption technique for BC and ultraviolet absorbing particulate matter (UVPM).<sup>48</sup> Ultraviolet absorbing PM is an indicator of organics such as cigarette smoking, cooking, incense, or wood smoke.

Airborne inhalable fungal spores were collected onto 25-mm diameter, 1- $\mu\text{m}$  pore size PTFE filters (Merck Millipore, Billerica, MA) using a Button™ sampler (SKC, Inc., Eighty-Four, PA). Air samples operated at a flow rate of 4 L/min. Within the sampling station, Button™ and PM<sub>2.5</sub> sampling inlets were at least 10 cm apart from each other. After the sampling, the Button™ samplers were returned to the laboratory, and each filter was placed into a 2-mL extraction tube containing 0.3 g of glass beads and the DNA extracted, as previously described.<sup>49</sup> Subsequently, each of the 36 fungal species that make up Environmental Relative Moldiness Index (ERMI) panel of indicator fungi was quantified using mold specific quantitative PCR (MSQPCR) assays. The MSQPCR assays for the 36 species have been described previously.<sup>49–54</sup> Results were reported as spore equivalents (SE) per filter for each of the 36 species, summed per filter and then divided by the volume of air per sample yielding a concentration of summed MSQPCR-fungi in spore equivalents per cubic meter (SE/ $\text{m}^3$ ). A metric called the ERMI-like value for each sample was also calculated, similar to the calculation of ERMI itself, as described previously.<sup>52,53</sup>

In 21 homes, co-located Button™ samples were also analyzed for total DNA utilizing Qubit® 3.0 Fluorometer and for total fungal DNA utilizing universal fungal primers in quantitative PCR (qPCR).<sup>49,55</sup> The methodology for the total fungal DNA is described in detail in the Supplemental Information.

Ultrafine particles were monitored indoors and outdoors using a P-Trak® ultrafine particle counter (TSI, Shoreview, MN), and results were expressed as particles per  $\text{cm}^3$  (pt/ $\text{cm}^3$ ). Monitoring occurred eight times for each home for approximately 15 minutes while indoor and outdoor air sampling stations were set-up and taken down. Temperature and humidity were recorded (HOBO Humidity Data Logger, Onset, Bourne, MA) for the entire 1-month duration of the HEPA treatment and dummy period, including the baseline and treatment sampling periods.

## 2.4 | Quality control

Media and field blanks were collected in parallel of 10% of all (indoor and outdoor) PM<sub>2.5</sub> SKC® and Button™ samples. The traditional surrogate analyte for diesel particulate matter is elemental carbon (EC) measured by thermal optical techniques, as described in NIOSH method 5040. In this study, we collected quality control samples of EC to validate BC as a comparable diesel surrogate. Elemental carbon samples were collected on prefired 37-mm quartz filters at 3 L/min with a personal Modular PM<sub>2.5</sub> impactor (SKC Inc., Eighty-Four, PA) in parallel at a rate of 10% of the indoor BC samples. All filters were stored at -20°C before analysis.

## 2.5 | Statistical analysis

The program R (version 3.1.1) was utilized for statistical analysis. Wilcoxon signed-rank test was used to compare the concentrations measured at baseline-HEPA and HEPA treatment, as well as between baseline-dummy and dummy treatment for BC, PM<sub>2.5</sub>, UFP, UVPM, BC/PM<sub>2.5</sub> ratio, UVPM/PM<sub>2.5</sub> ratio, summed MSQPCR-fungi, total fungal DNA, total DNA, temperature, and humidity. The comparison was performed for indoor and outdoor values separately. Additionally, indoor/outdoor (I/O) ratios of all particle pollutants at the baseline and at the end of the treatments were compared.

A best subset regression was performed utilizing the lowest Akaike information criterion (AIC) to determine the best models during HEPA and dummy treatments separately for the four exposure variables (BC, PM<sub>2.5</sub>, UVPM, and summed MSQPCR-fungi). The list of variables used in each regression included all inside and outside concentrations, all concentrations at the baseline and all concentrations at the end of the treatment (HEPA and dummy separately), number of smokers in the home, housing type, number of occupants, number of people with asthma, construction year, size of home, size of room, type of flooring in child's room, presence of central air, presence of gas heat, pets, reported infestations (cockroaches, mice, rats, bedbugs), reported mold (visible or odor), ECAT score, distance to highway, distance to interstate, number of trucks within 400 m, and conditions during sampling. The latter included the following: season, operating air conditioning, open windows, operating kitchen fan, operating clothes dryer, operating gas heat, operating humidifier, operating dehumidifier, frying food, operating fireplace, burning candles, and cleaning activities. Given that the study was conducted over a 3-month time-period, factors such as outdoor concentration, heating, air conditioning, opening windows, smoking, and candle or fireplace use could not be controlled baseline to treatment sampling events. However, each condition was recorded for the duration of the 48-hour sampling events, and all parameters were entered into the models.

Spearman's correlation coefficient was calculated to determine whether there was a correlation between EC and BC values, and between total DNA, total fungal DNA, and summed MSQPCR-fungi. Bonferroni adjustment for multiple comparisons was performed on the P-values to determine significance. As four primary parameters were evaluated (BC, PM<sub>2.5</sub>, UVPM and summed MSQPCR-fungi), P-values less than 0.0125 (0.05/4) were considered significant for all tests.

## 3 | RESULTS

### 3.1 | Study participants

A total of 43 of the homes completed the entire 3-month study, and an additional 3 homes completed a portion ( $\leq$ 1-month) of the study ( $n = 46$ ) (Figure S1). The homes were built between 1865 and 2016. The number of occupants per home ranged from 2 to 10 people. The size of the homes ranged from 65 to 334 m<sup>2</sup> and the size of the sampling rooms ranged from 5 to 32 m<sup>2</sup>. While the number of

smokers per home was determined, personnel did not ask if the occupants typically smoked indoors, outdoors (potentially near our sampling station), or if they allowed visitors to smoke in their home. It was noted at least once that while in a home without a smoker, smoking paraphernalia was evident. The percent of homes with different building conditions is listed in Table S1 and the percent of homes with reported conditions during specific sampling periods is indicated in Table S2.

### 3.2 | Reduction in concentrations of BC, PM<sub>2.5</sub>, UFP, UVPM, and summed MSQPCR-fungi

The pollutant concentrations did not follow a normal distribution, and therefore, median concentrations, rather than the means, were determined to be representative of the dataset. The median concentrations of the indoor BC obtained at baseline and at the end of HEPA treatment were 0.6 and 0.1  $\mu\text{g}/\text{m}^3$ , respectively. The corresponding median concentrations of the indoor PM<sub>2.5</sub> were 7.6 and 3.4  $\mu\text{g}/\text{m}^3$ . For the indoor UFP, the median concentrations were 4996 and 3507  $\text{pt}/\text{cm}^3$ , obtained at baseline and at the end of HEPA treatment, respectively. The median concentrations measured at baseline and at the end of HEPA treatment were 2.1 and 0.4  $\mu\text{g}/\text{m}^3$ , respectively, for indoor UVPM. The respective values were 166 and 112  $\text{SE}/\text{m}^3$  for the summed MSQPCR-fungi, and 3.9 and 1.6 for the ERMI-like values. The median values for outdoor and indoor/outdoor (I/O) ratios of BC, PM<sub>2.5</sub>, UFP, UVPM and summed MSQPCR-fungi at the different time points are presented in Table 1. The distribution of the indoor BC, PM<sub>2.5</sub>, UVPM and summed MSQPCR-fungi data can be seen in Figure 1, and the distribution of the outdoor and I/O ratio data is presented in Figures S2-S5. The distribution of the indoor, outdoor, and I/O ratio of UFP data is shown in Figure S6, and the distribution of the indoor ERMI-like data is in Figures S7. A summary of the average concentrations and standard deviations for BC, PM<sub>2.5</sub>, UVPM and summed MSQPCR-fungi concentrations for each building variable and sampling condition can be seen in Table S3.

A significant reduction was found in the indoor concentrations of BC, PM<sub>2.5</sub>, and UVPM between the baseline-HEPA and HEPA treatment ( $P < 0.001$ ) (Table 1, Figure 1). In contrast, there were no significant differences in indoor BC, PM<sub>2.5</sub>, and UVPM concentrations between the baseline-dummy and dummy treatment, or in outdoor concentrations between the baselines and either respective treatments (Table 1). There was a significant reduction between the baseline and HEPA treatment of I/O ratios for BC, PM<sub>2.5</sub>, and UVPM ( $P < 0.001$ ), whereas there was no significant difference between the baseline and dummy treatment (Table 1, Figures S2, S3, S4). No significant differences were found in indoor or outdoor concentrations of UFP between the baselines and either treatment (Table 1, Figure S6). There was a reduction, albeit not significant, in the median I/O ratios of UFP from the baselines to either treatment (Figure S6).

During the baseline-HEPA sampling, the median BC/PM<sub>2.5</sub> ratio was 0.05, and the median UVPM/PM<sub>2.5</sub> ratio was 0.3. During the HEPA treatment, the median BC/PM<sub>2.5</sub> ratio had significantly decreased to 0.02 ( $P < 0.001$ ). The median UVPM/PM<sub>2.5</sub> ratio also

**TABLE 1** Median values of parameters at HEPA-baseline, HEPA, dummy-baseline, and dummy sampling time points

| Parameter                                                                                         | Location (n = HEPA/Dummy)        | Baseline-H | HEPA  | Baseline-D | Dummy |
|---------------------------------------------------------------------------------------------------|----------------------------------|------------|-------|------------|-------|
| Black carbon (BC) ( $\mu\text{g}/\text{m}^3$ )                                                    | Indoor(n = 41/38)                | 0.6*       | 0.1*  | 0.7        | 0.6   |
|                                                                                                   | Outdoor(n = 41/36)               | 1.1        | 0.9   | 1.1        | 1.0   |
|                                                                                                   | Indoor/Outdoor ratio (n = 40/36) | 0.6*       | 0.1*  | 0.6        | 0.5   |
| Particulate matter less than 2.5 $\mu\text{m}$ ( $\text{PM}_{2.5}$ ) ( $\mu\text{g}/\text{m}^3$ ) | Indoor(n = 41/39)                | 7.6*       | 3.4*  | 9.6        | 8.2   |
|                                                                                                   | Outdoor(n = 41/36)               | 10.8       | 9.1   | 10.4       | 11.0  |
|                                                                                                   | Indoor/Outdoor ratio (n = 40/35) | 0.9*       | 0.3*  | 0.7        | 0.7   |
| Ultrafine particulate matter (UFP) (pt/cm <sup>3</sup> )                                          | Indoor(n = 40/39)                | 4996       | 3507  | 8336       | 6399  |
|                                                                                                   | Outdoor(n = 36/31)               | 8147       | 8014  | 8347       | 7825  |
|                                                                                                   | Indoor/Outdoor ratio (n = 34/30) | 0.8        | 0.4   | 1.2        | 0.6   |
| Ultraviolet absorbing particulate matter (UVPM) ( $\mu\text{g}/\text{m}^3$ )                      | Indoor(n = 41/38)                | 2.1*       | 0.4*  | 2.7        | 2.4   |
|                                                                                                   | Outdoor(n = 41/36)               | 2.4        | 2.2   | 2.5        | 2.5   |
|                                                                                                   | Indoor/Outdoor ratio (n = 40/35) | 0.7*       | 0.2*  | 1.2        | 0.8   |
| Summed MSQPCR-fungi (SE/m <sup>3</sup> )                                                          | Indoor(n = 43/44)                | 166*       | 112*  | 292        | 139   |
|                                                                                                   | Outdoor(n = 39/40)               | 1818       | 2128  | 2653       | 1872  |
|                                                                                                   | Indoor/Outdoor ratio (n = 38/40) | 0.1*       | 0.04* | 0.1        | 0.1   |
| ERMI-like                                                                                         | Indoor(n = 43/44)                | 3.9        | 1.6   | 3.4        | 2.7   |

Due to Bonferroni correction, *P*-value <0.0125 was considered significant.

\**P* < 0.001.

Baseline-H or Baseline-D indicates 48 hours prior to HEPA or Dummy installation and HEPA or Dummy indicates the last 48 hours during the treatment.

decreased to 0.1 (*P* = 0.05), but the *p*-value did not meet the criteria determined to be significant (*P* < 0.0125). The ratios during baseline-dummy were 0.06 for BC/PM<sub>2.5</sub> and 0.3 for UVPM/PM<sub>2.5</sub>. The ratios of BC/PM<sub>2.5</sub> and UVPM/PM<sub>2.5</sub> were not significantly different from the baseline-dummy to the dummy treatment (0.06 and 0.3, respectively). Outdoor ratios remained consistent being 0.1 for BC/PM<sub>2.5</sub> and 0.2 for UVPM/PM<sub>2.5</sub> throughout the sampling (at baselines and at the end of both treatments).

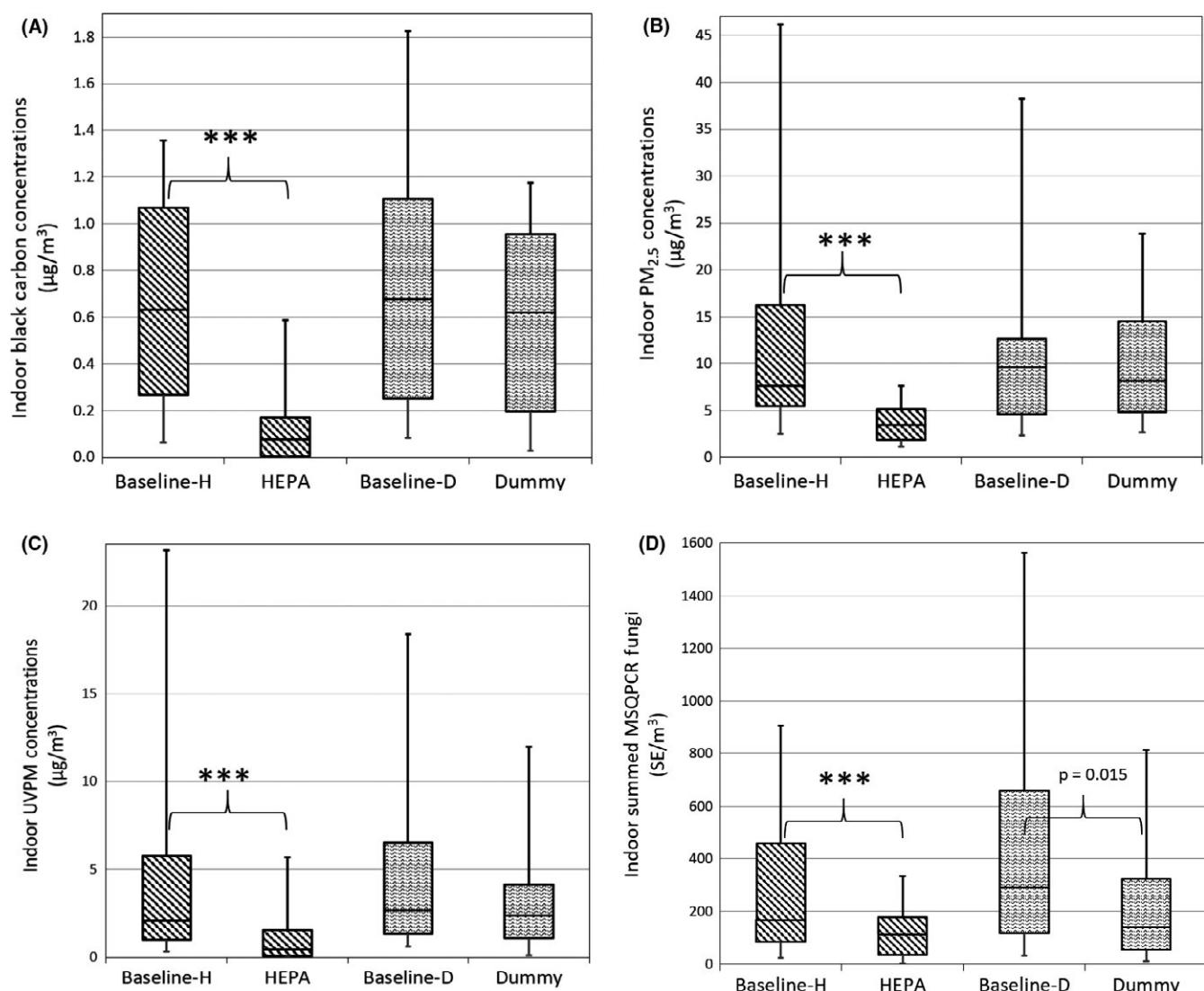
There was a significant reduction in indoor summed MSQPCR-fungi between baseline-HEPA and HEPA treatment (*P* < 0.010) and a borderline significant reduction at baseline-dummy and dummy treatment (*P* = 0.015) (Table 1). The outdoor summed MSQPCR-fungi had no statistically significant differences between the baseline and treatment concentrations for either HEPA or dummy periods (Figure S5). The I/O ratios between baseline-HEPA and HEPA treatment for summed MSQPCR-fungi were statistically significant (*P* < 0.001). There was a reduction in the median I/O ratios between the baseline-dummy and dummy treatment, but the reduction was not statistically significant. The indoor ERMI-like values at baseline-HEPA and at the end of the HEPA treatment had borderline significance (*P* = 0.04), whereas the respective dummy values were not significantly different (Figure S7).

Samples taken in a subset of 21 homes with co-located Button™ samplers were analyzed for total fungal DNA with qPCR and total

DNA with Qubit. The concentrations in these samples were also compared with the concentration of summed MSQPCR-fungi (Figure S8). Although the results from all three methods demonstrated a reduction in the median concentrations from baseline-HEPA to HEPA treatment, none were significantly different. The concentrations of the summed MSQPCR-fungi in this subset of homes had a borderline significant reduction from baseline-HEPA to HEPA treatment (*P* = 0.019) (Figure S8).

### 3.3 | Regression models

Eight best subset linear regression models for HEPA and dummy treatments using the lowest AIC were developed for the four exposure parameters (BC, PM<sub>2.5</sub>, UVPM, summed MSQPCR-fungi) (Table S4). The eight final models (after the best subset variable selection) included the variable for all baseline concentrations (HEPA and dummy treatments separately) in the regression estimate. However, only the models utilizing the HEPA data for BC, PM<sub>2.5</sub>, UVPM, and summed MSQPCR-fungi, found the baseline concentrations to be significantly higher compared to the HEPA concentration values (Table S4). In all four models utilizing the dummy data, the baseline concentrations were not found to be statistically significant compared to the data at the end of the dummy treatment. We note that the significance of the baseline sampling period only in the HEPA



**FIGURE 1** Indoor concentrations of A, Black carbon (BC) ( $\mu\text{g}/\text{m}^3$ ) (HEPA n = 41 homes; Dummy n = 38 homes), B, Particulate matter  $<2.5\text{ }\mu\text{m}$  ( $\text{PM}_{2.5}$ ) ( $\mu\text{g}/\text{m}^3$ ) (HEPA n = 41 homes; Dummy n = 39 homes), C, Ultraviolet absorbing particulate matter (UVPM) ( $\mu\text{g}/\text{m}^3$ ) (HEPA n = 41 homes; Dummy n = 38 homes), D, Fungal spores determined by the summed mold specific quantitative PCR (MSQPCR) fungi ( $\text{SE}/\text{m}^3$ ) (HEPA n = 43 homes; Dummy n = 44 homes). Baseline-H and Baseline-D indicate 48 hours prior to HEPA or Dummy installation and HEPA or Dummy indicates the last 48 hours during the treatment. Horizontal lines in the box plot represent the 10%, 25%, 50%, 75%, and 90% percentiles. Due to Bonferroni correction, P-value  $<0.0125$  was considered significant. \*\*\*P < 0.001

models and not in the models with dummy data, is consistent with our findings described in the previous section. Similarly, the four models utilizing the HEPA data demonstrated that outdoor concentrations were significantly higher than indoor concentrations. Utilizing the dummy data, the BC and summed MSQPCR-fungi models found all outdoor concentrations significantly higher than the indoor concentrations; however, the  $\text{PM}_{2.5}$  and UVPM outdoor concentrations were not found to be statistically significant from the indoor concentrations.

Additionally, the variables selected as significant in either the HEPA or dummy models for BC regression analyses included baseline concentrations, outdoor concentrations, infestations in the last 12 months, type of HVAC, floor area of home, ECAT score, distance to highway, distance to interstate, summer

season, open windows, operating humidifier, and burning candles. The variables selected as significant in either the HEPA or dummy models for the  $\text{PM}_{2.5}$  included baseline concentrations, outdoor concentrations, distance to highway, operating clothes dryer, operating humidifier, and frying food. For the summed MSQPCR fungal regression analyses, the variables selected as significant in either the HEPA or dummy models included baseline sampling concentrations, outdoor concentrations, hardwood flooring, floor area of home, distance to highway, total number of people with asthma, and operating clothes dryer. The variables that were significant in either the HEPA or dummy regression models for the UVPM included baseline sampling concentrations, outdoor concentrations, ECAT score, and total number of people with asthma.

### 3.4 | Temperature and relative humidity data

The temperature and humidity values tended to follow a normal distribution; therefore, reporting average values seemed representative of the dataset. The averages, minimums, and maximums of temperature and relative humidity for each treatment period are shown in Table S5. The means of indoor and outdoor temperature and humidity during the HEPA and dummy months were similar. There were no significant differences between the two treatment periods for either indoor or outdoor values.

### 3.5 | Quality control

The data from electric monitors showed that the air cleaners in the homes were turned on an average of 88% during the HEPA treatment. Three electric monitors showed that the air cleaners operated less than 70% of the time; these three units operated for an average of 24% of the time. All sampling efforts were included in the study due to the high percentage of filter usage, and all air cleaners were operational during sampling. The results from blank samples of  $\text{PM}_{2.5}$ , BC, UVPM and summed MSQPCR-fungi were averaged ( $1.25 \mu\text{g}/\text{m}^3$ ,  $0.03 \mu\text{g}/\text{m}^3$ ,  $0.10 \mu\text{g}/\text{m}^3$ ,  $0.35 \text{ SE}/\text{m}^3$ , respectively) and subtracted from the respective sample values. Eighteen elemental carbon samples were collected and compared to co-located black carbon samples. The median EC concentration was  $0.23 \mu\text{g}/\text{m}^3$  (ranging from  $<0.04$  to  $3.03 \mu\text{g}/\text{m}^3$ ) and the median BC concentration from the same co-located samples was  $0.41 \mu\text{g}/\text{m}^3$  (ranging from  $<0.01$  to  $6.08 \mu\text{g}/\text{m}^3$ ). The EC and BC results had a significant and strong correlation ( $r = 0.94$ ,  $P < 0.001$ ) (Figure S9).

## 4 | DISCUSSION

The overall concentrations of traffic-related aerosol particles, as expressed via BC and  $\text{PM}_{2.5}$ , and other investigated aerosol particles, that is, UVPM and summed MSQPCR-fungi, were significantly reduced after a HEPA cleaner was operated in the home. Our observed  $\text{PM}_{2.5}$  reduction following HEPA treatment is consistent with findings by Allen et al. who reported a reduction of  $\text{PM}_{2.5}$  after a 7-day installation of HEPA air cleaners.<sup>56</sup> In a 21-day HEPA treatment, Padró-Martínez et al. demonstrated a reduction of UFP; however, they did not include a flush period between the HEPA and the dummy periods and did not perform outdoor sampling for comparison.<sup>23</sup> While our study did not show a significant reduction in UFP, it did encompass a flush period to ensure there was not crossover from each of the treatments, and also included outdoor sampling. In addition, the participants in the aforementioned study by Padró-Martínez et al. were located in 2 apartment complexes within 400 meters of each other and 200 meters of an interstate and a highway providing a clear and consistent source of traffic pollution. Our study did not strictly stay within 200 m of an interstate or highway providing for a direct source of air pollution but instead utilized the ECAT score to estimate the level of traffic pollution. Utilizing the ECAT score allowed us to sample homes further away from known TRAP sources and to include a broad variety of buildings

throughout the Cincinnati metropolitan area, illustrating the applicability of HEPA air cleaner in diverse settings.

Black carbon sources include combustion processes such as burning of fossil fuels or biomass, which is mostly attributed to outdoor sources. In urban settings, black carbon can also be a major chemical constituent of  $\text{PM}_{2.5}$ .<sup>57,58</sup> The BC/ $\text{PM}_{2.5}$  ratio of 10% obtained outdoors during the present study is consistent with the one reported by Rattigan et al. for an outdoor urban setting (7%-10% for Rochester, NY).<sup>59</sup> In our study, the indoor fraction of BC/ $\text{PM}_{2.5}$  was significantly reduced with the HEPA cleaner, indicating that black carbon was particularly impacted by the HEPA filtration. The BC and UVPM portion measured before the HEPA treatment accounted for nearly one-third of the collected  $\text{PM}_{2.5}$  (by mass). After applying the HEPA treatment, only about one-seventh of the  $\text{PM}_{2.5}$  was found attributable to BC and UVPM. The lack of change between the concentrations at baseline-dummy and dummy treatment emphasize the large impact a HEPA filter can have on  $\text{PM}_{2.5}$  reductions, especially UVPM and BC fractions. We believe that the present effort is the first intervention study that has more comprehensively demonstrated that HEPA air cleaners significantly reduce traffic-related aerosols, while previous investigations evaluated the effectiveness of HEPA air purification for various types of particles [e.g.,  $\text{PM}_{2.5}$  and cigarette smoke<sup>27,43,44,60</sup>], and did not address such a variety of building characteristics and sampling conditions.

Cheng et al.<sup>30</sup> showed that a HEPA air cleaner reduced the concentration of fungal spores indoors. However, the quoted study was limited to microscopic counts of five fungal genera that were only measured for two hours and did not consider or evaluate the influence of fungal spores in the outdoor air. In our study, the indoor concentrations of the 36-fungal species were shown to be significantly reduced with the HEPA treatment, based on MSQPCR analysis, over a 48-hour period, even after accounting for the outdoor fungal-spore populations. The summed MSQPCR-fungi made up about 90% of the total fungal DNA. Therefore, these 36-fungi represented a major portion of the total fungal burden. In addition, the ERMI-like values themselves showed reductions (borderline significant) as a result of the HEPA treatment but not the dummy treatment (Figure S7). There was also a reduction in summed MSQPCR-fungi (borderline significant) after the dummy period. The prefilter used in these HEPA units is capable of removing larger particles. Therefore, one possible reason for the reduction due to dummy treatment could be the removal of spores by the prefilter, especially if the spores are large or attached to larger particles or the prefilter became heavily loaded, as was often observed. The conidia of *E. nigrum* measure 15-25  $\mu\text{m}$  in physical diameter and 11.8  $\mu\text{m}$  in aerodynamic diameter, and the conidia of *C. cladosporioides* measure 3.6-4  $\mu\text{m}$  in physical diameter and 2.8-5.5  $\mu\text{m}$  in aerodynamic diameter.<sup>61-65</sup> We have previously reported that these large conidia were common in a subset of the study homes.<sup>66</sup> Another potential contributing factor to the reduction was the lower level of outdoor fungi during the dummy sampling time.

The best subset, linear regression models demonstrated that the concentrations at the baseline-HEPA were significantly higher compared to the concentrations at the end of the HEPA treatment for

the following parameters: BC, PM<sub>2.5</sub>, UVPM, and summed MSQPCR-fungi. Conversely, the concentrations at the baseline-dummy were included in the regression models but were not significantly different to the concentrations at the end of the dummy treatment. This supports our data that the HEPA treatment significantly reduced BC, PM<sub>2.5</sub>, UVPM, and the summed MSQPCR-fungi with the baseline-HEPA concentrations significantly impacting these results. The dummy treatment did not significantly reduce the BC, PM<sub>2.5</sub>, UVPM, and summed MSQPCR-fungi, and the concentrations at the baseline-dummy did not significantly impact these results. Outdoor concentrations significantly impacted all four HEPA regression models and two of the dummy regression models (BC and summed MSQPCR-fungi) compared to indoor concentrations, demonstrating the impact of the infiltration of outdoor air particles into these homes.

In the PM<sub>2.5</sub> models, frying food had a statistically significant coefficient for the dummy regression and had a similar but not significant coefficient for the HEPA regression analyses, indicating this parameter is a consistent contributor to PM<sub>2.5</sub> levels in the home. Frying food can be a major contributor to indoor airborne particulate matter, and various studies have attributed 25%-50% of indoor PM<sub>2.5</sub> to cooking sources.<sup>67-69</sup> In the BC model, burning candles was also a significant coefficient for the dummy period. This indicates there was a significant difference between burning candles and not burning candles during the dummy treatment. Burning candles has been shown to be a contributor to indoor black carbon levels in residential environments.<sup>70,71</sup> The models that examined summed MSQPCR-fungi, the variable "year of construction" had similar negative coefficients in both HEPA and dummy models. This indicates that the newer the home, the fewer summed MSQPCR-fungi would occur regardless of HEPA or dummy treatment. This is likely due to the fact that newer homes have less air-leakage and less interchange with the outside air.<sup>72</sup> All other variables in these models did not appear significant in either HEPA or dummy regression analyses, included very small estimates and/or were potentially analytical artifacts due to the wide number of parameters considered.

Indoor/outdoor ratios provided the context of the level of outdoor air pollution that penetrated inside each home. Indoor/outdoor ratios of BC, PM<sub>2.5</sub>, UVPM and summed MSQPCR-fungi were significantly reduced during the HEPA treatment but not during the dummy period. The median I/O ratios of BC, UVPM and summed MSQPCR-fungi were mostly below 1, indicating the absence of a substantial indoor sources. However, the median I/O ratio value prior to the installation of the dummy air cleaner for UVPM was above 1, which indicates that there was a contributing indoor source, for example, smoking and/or cooking within the home. In our study, self-reported prevalence of smokers in the home was 22%.

Ultrafine particles were not sampled similarly to PM<sub>2.5</sub>, UVPM, BC or summed MSQPCR-fungi, as they were sampled briefly (approximately 15-minute intervals versus 48 hours) when the sampling team was adjacently setting up and tearing down equipment. The lack of long-term sampling and the close proximity of the sampling team may not accurately reflect the true levels of UFP. The potential elevated bias generated by the presence of the sampling team, however, is expected

to be consistent through all sampling days. The overall trend showed a decrease in both treatments. The outdoor medians being relatively consistent suggest that, despite UFP fluctuations, an overall typical outdoor level of ultrafine particles was stable at these locations. The limited capture of data of ultrafine particles, however, did not provide sufficient information to draw significant conclusions. Another limitation of the study was associated with the sample size for the total DNA and total fungal DNA analysis. The results demonstrated a decreasing trend from baseline-HEPA to the end of the HEPA treatment, and with additional data, the reduction could have become statistically significant.

The traditional surrogate analyte for diesel particulate matter is elemental carbon (EC) utilizing NIOSH method 5040. In recent years, studies have demonstrated that EC and BC define a similar fraction of the carbonaceous aerosol and are relatively comparable.<sup>48,73</sup> In the present investigation, we sampled EC to validate BC as a comparable diesel surrogate. The strong correlation between these two analyses supports utilizing BC as the surrogate for diesel particulate matter in this study.

Traffic-related aerosol particles is a growing public health concern as a large percentage of the world population is moving closer to the cities and major roadways.<sup>1</sup> Chronic illnesses and diseases have been associated with TRAP,<sup>16,17,19,21</sup> and therefore, a solution to provide healthy indoor air is critical regardless of location. Using BC as a surrogate for TRAP and especially diesel particulate matter, this study demonstrated that HEPA air cleaners provide a solution in reducing these aspects of TRAP. Significant reductions were also seen in the tobacco smoke, PM<sub>2.5</sub>, and fungal spores. This project successfully demonstrated that the outdoor air pollution impacts our indoor air quality, and the utilization of a HEPA air cleaner effectively reduces exposure to traffic and other aerosols of concern in real-world situations under varying conditions.

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## COMPETING INTERESTS AND DISCLAIMER

The U.S. Environmental Protection Agency (EPA) through its Office of Research and Development collaborated in the research described here. It has been subjected to the Agency's peer-review and has been approved as an EPA publication. Mention of trade names or commercial products does not constitute endorsement or recommendation by the EPA for use. The findings and the conclusions in this report are those of the authors and do not necessarily represent the views of the US EPA. MSQPCR is a U.S. EPA patented technology, and its commercial application can provide royalties to the US EPA.

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## REFERENCES

1. Centers for Disease Control And Prevention. Conclusion and future directions: CDC health disparities and inequalities report—United States. *Morb Mortal Wkly Rep*. 2013;2013(62):189.
2. Jamriska M, Morawska L, Mergersen K. The effect of temperature and humidity on size segregated traffic exhaust particle emissions. *Atmos Environ*. 2008;42:2369-2382.
3. Hu S, McDonald R, Martuzevicius D, et al. UNMIX modeling of ambient PM2. 5 near an interstate highway in Cincinnati, OH, USA. *Atmos Environ*. 2006;40:378-395.
4. Thurston GD, Ito K, Lall R. A source apportionment of US fine particulate matter air pollution. *Atmos Environ*. 2011;45:3924-3936.
5. American Lung Association. *State of the Air* 2016. Chicago, IL: American Lung Association; 2016.
6. Briggs N, Long CM. Critical review of black carbon and elemental carbon source apportionment in Europe and the United States. *Atmos Environ*. 2016;144:409-427.
7. Andreae MGelencsér A. Black carbon or brown carbon? The nature of light-absorbing carbonaceous aerosols. *Atmos Chem Phys*. 2006;6:3131-3148.
8. Hussein T, Glytsos T, Ondráček J, et al. Particle size characterization and emission rates during indoor activities in a house. *Atmos Environ*. 2006;40:4285-4307.
9. Wan M-P, Wu C-L, To G-NS, et al. Ultrafine particles, and PM2. 5 generated from cooking in homes. *Atmos Environ*. 2011;45:6141-6148.
10. Qin Y, Kim E, Hopke P. The concentrations and sources of PM2. 5 in metropolitan New York City. *Atmos Environ*. 2006;40:312-332.
11. Cyrys J, Heinrich J, Hoek G, et al. Comparison between different traffic-related particle indicators: elemental carbon (EC), PM 2.5 mass, and absorbance. *Journal of Exposure Science and Environmental Epidemiology*. 2003;13:134.
12. Gatari MJ, Kinney PL, Yan B, et al. High airborne black carbon concentrations measured near roadways in Nairobi, Kenya. *Transportation Research Part D: Transport and Environment*. 2017; <https://doi.org/10.1016/j.trd.2017.10.002>.
13. Klepeis NE, Nelson WC, Ott WR, et al. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *J Expo Anal Environ Epidemiol*. 2001;11:231-252.
14. Mejia JF, Choy SL, Mengersen K, et al. Methodology for assessing exposure and impacts of air pollutants in school children: data collection, analysis and health effects—a literature review. *Atmos Environ*. 2011;45:813-823.
15. Tong Z, Chen Y, Malkawi A, et al. Quantifying the impact of traffic-related air pollution on the indoor air quality of a naturally ventilated building. *Environ Int*. 2016;89:138-146.
16. Codispoti CD, LeMasters GK, Levin L, et al. Traffic pollution is associated with early childhood aeroallergen sensitization. *Ann Allergy Asthma Immunol*. 2015;114:126-133.
17. Brunekreef B, Janssen NA, De Hartog J, et al. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology*. 1997;8:298-303.
18. Gauderman WJ, Avol E, Gilliland F, et al. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med*. 2004;351:1057-1067.
19. McConnell R, Islam T, Shankardass K, et al. Childhood incident asthma and traffic-related air pollution at home and school. *Environ Health Persp*. 2010;118:1021-1026.
20. McConnell R, Berhane K, Yao L, et al. Traffic, susceptibility, and childhood asthma. *Environ Health Persp*. 2006;114:766-772.
21. Beelen R, Hoek G, Van Den Brandt PA, et al. Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). *Environ Health Persp*. 2008;116:196.
22. Brugge D, Durant JL, Rioux C. Near-highway pollutants in motor vehicle exhaust: a review of epidemiologic evidence of cardiac and pulmonary health risks. *Environ Health*. 2007;6:23.
23. Padró-Martínez LT, Owusu E, Reisner E, et al. A randomized cross-over air filtration intervention trial for reducing cardiovascular health risks in residents of public housing near a highway. *Int J Environ Res Public Health*. 2015;12:7814-7838.
24. Polichetti G, Cocco S, Spinali A, et al. Effects of particulate matter (PM10, PM2. 5 and PM1) on the cardiovascular system. *Toxicology*. 2009;261:1-8.
25. Urch B, Silverman F, Corey P, et al. Acute blood pressure responses in healthy adults during controlled air pollution exposures. *Environ Health Perspect*. 2005;113:1052.
26. Gan WQ, Koehoorn M, Davies HW, et al. Long-term exposure to traffic-related air pollution and the risk of coronary heart disease hospitalization and mortality. *Environ Health Perspect*. 2011; 119:501.
27. Day D, Xiang J, Mo J, et al. Combined use of an electrostatic precipitator and a high-efficiency particulate air filter in building ventilation systems: effects on cardiorespiratory health indicators in healthy adults. *Indoor Air*. 2018;28:360-372.
28. Karvonen A, Hyvärinen A, Rintala H, et al. Quantity and diversity of environmental microbial exposure and development of asthma: a birth cohort study. *Allergy*. 2014;69:1092-1101.
29. Ryan PH, Bernstein DL, Lockey J, et al. Exposure to traffic-related particles and endotoxin during infancy is associated with wheezing at age 3 years. *Am J Resp Crit Care*. 2009;180:1068-1075.
30. Cheng Y, Lu JC, Chen TR. Efficiency of a portable indoor air cleaner in removing pollens and fungal spores. *Aerosol Sci Tech*. 1998;29:92-101.
31. Batterman S, Du L, Mentz G, et al. Particulate matter concentrations in residences: an intervention study evaluating stand-alone filters and air conditioners. *Indoor Air*. 2012;22:235-252.
32. Kajbafzadeh M, Brauer M, Karlen B, et al. The impacts of traffic-related and woodsmoke particulate matter on measures of cardiovascular health: a HEPA filter intervention study. *Occup Environ Med*. 2015;72:394-400.
33. Brauner EV, Forchhammer L, Møller P, et al. Indoor particles affect vascular function in the aged: an air filtration-based intervention study. *Am J Resp Crit Care*. 2008;177:419-425.
34. Sublett JL. Effectiveness of air filters and air cleaners in allergic respiratory diseases: a review of the recent literature. *Curr Allergy Asthma Rep*. 2011;11:395-402.
35. Sublett JL, Seltzer J, Burkhead R, et al. Air filters and air cleaners: rostrum by the American Academy of Allergy, Asthma & Immunology Indoor Allergen Committee. *J Allergy Clin Immunol*. 2010;125:32-38.
36. McDonald E, Cook D, Newman T, et al. Effect of air filtration systems on asthma: a systematic review of randomized trials. *CHEST J*. 2002;122:1535-1542.

37. Institute Of Medicine. Cleaning the air: asthma and indoor air exposures. National Academy Press: National Academy of Sciences; 2000.

38. Reisman RE. Do air cleaners make a difference in treating allergic disease in homes? *Ann Allergy Asthma Immunol*. 2001;87:41-43.

39. Reisman RE, Mauriello PM, Davis GB, et al. A double-blind study of the effectiveness of a high-efficiency particulate air (HEPA) filter in the treatment of patients with perennial allergic rhinitis and asthma. *J Allergy Clin Immunol*. 1990;85:1050-1057.

40. Wood RA. Air filtration devices in the control of indoor allergens. *Curr Allergy Asthma Rep*. 2002;2:397-400.

41. Francis H, Fletcher G, Anthony C, et al. Clinical effects of air filters in homes of asthmatic adults sensitized and exposed to pet allergens. *Clin Exp Allergy*. 2003;33:101-105.

42. Sulser C, Schulz G, Wagner P, et al. Can the use of HEPA cleaners in homes of asthmatic children and adolescents sensitized to cat and dog allergens decrease bronchial hyperresponsiveness and allergen contents in solid dust? *Int Arch Allergy Immunol*. 2009;148:23-30.

43. Butz AM, Matsui EC, Breysse P, et al. A randomized trial of air cleaners and a health coach to improve indoor air quality for inner-city children with asthma and secondhand smoke exposure. *Arch Pediat Adol Med*. 2011;165:741-748.

44. Lanphear BP, Hornung RW, Khoury J, et al. Effects of HEPA air cleaners on unscheduled asthma visits and asthma symptoms for children exposed to secondhand tobacco smoke. *Pediatrics*. 2011;127:93-101.

45. Epstein TG, Ryan PH, LeMasters GK, et al. Poor asthma control and exposure to traffic pollutants and obesity in older adults. *Ann Allergy Asthma Immunol*. 2012;108:423-428.

46. Ryan PH, LeMasters GK, Levin L, et al. A land-use regression model for estimating microenvironmental diesel exposure given multiple addresses from birth through childhood. *Sci Total Environ*. 2008;404:139-147.

47. Stillerman A, Nachtsheim C, Li W, et al. Efficacy of a novel air filtration pillow for avoidance of perennial allergens in symptomatic adults. *Ann Allergy Asthma Immunol*. 2010;104:440-449.

48. Yan B, Kennedy D, Miller RL, et al. Validating a nondestructive optical method for apportioning colored particulate matter into black carbon and additional components. *Atmos Environ*. 2011;45:7478-7486.

49. Haugland R, Vesper S. *Method of identifying and quantifying specific fungi and bacteria*. Washington, DC: US Environmental Protection Agency; 2002. US Patent 6,387,652 B1

50. Haugland RA, Varma M, Wymer LJ, et al. Quantitative PCR analysis of selected *Aspergillus*, *Penicillium* and *Paecilomyces* species. *Syst Appl Microbiol*. 2004;27:198-210.

51. Vesper S, McKinstry C, Haugland R, et al. Higher environmental relative moldiness index (ERMI) values measured in Detroit homes of severely asthmatic children. *Sci Total Environ*. 2008;394:192-196.

52. Vesper S, McKinstry C, Haugland R, et al. Development of an environmental relative moldiness index for US homes. *J Occup Environ Med*. 2007;49:829-833.

53. Vesper S, McKinstry C, Cox D, et al. Correlation between ERMI values and other moisture and mold assessments of homes in the American Healthy Homes Survey. *J Urban Health*. 2009;86:850-860.

54. Haugland RA, Brinkman N, Vesper S. Evaluation of rapid DNA extraction methods for the quantitative detection of fungi using real-time PCR analysis. *J Microbiol Methods*. 2002;50:319-323.

55. Dannemiller KC, Lang-Yona N, Yamamoto N, et al. Combining real-time PCR and next-generation DNA sequencing to provide quantitative comparisons of fungal aerosol populations. *Atmos Environ*. 2014;84:113-121.

56. Allen RW, Carlsten C, Karlen B, et al. An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community. *Am J Resp Crit Care*. 2011;183:1222-1230.

57. Miguel AH, Kirchstetter TW, Harley RA, et al. On-road emissions of particulate polycyclic aromatic hydrocarbons and black carbon from gasoline and diesel vehicles. *Environ Sci Technol*. 1998;32:450-455.

58. Snyder DC, Rutter AP, Worley C, et al. Spatial variability of carbonaceous aerosols and associated source tracers in two cities in the Midwestern United States. *Atmos Environ*. 2010;44:1597-1608.

59. Rattigan OV, Civerolo K, Doraiswamy P, et al. Long term black carbon measurements at two urban locations in New York. *Aerosol Air Qual Res*. 2013;13:1181-1196.

60. Batterman S, Godwin C, Jia C. Long duration tests of room air filters in cigarette smokers' homes. *Environ Sci Technol*. 2005;39:7260-7268.

61. Yamamoto N, Nazaroff W, Peccia J. Assessing the aerodynamic diameters of taxon-specific fungal bioaerosols by quantitative PCR and next-generation DNA sequencing. *J Aerosol Sci*. 2014;78:1-10.

62. Reponen T, Grinshpun S, Conwell K, et al. Aerodynamic versus physical size of spores: measurement and implication for respiratory deposition. *Grana*. 2001;40:119-125.

63. Cole GT, Samson RA. Mould allergy. In: Al-Doory Y, Domson JF, eds. *The conidia*. Philadelphia, PA: Lea & Febiger; 1984:66-104.

64. Mims CW, Richardson EA. Ultrastructure of sporodochium and conidium development in the anamorphic fungus *Epicoccum nigrum*. *Can J Botany*. 2005;83:1354-1363.

65. Yamamoto N, Schmechel D, Chen BT, et al. Comparison of quantitative airborne fungi measurements by active and passive sampling methods. *J Aerosol Sci*. 2011;42:499-507.

66. Cox J, Indugula R, Vesper S, et al. Comparison of indoor air sampling and dust collection methods for fungal exposure assessment using quantitative PCR. *Environ Sci Process Impacts*. 2017;19:1312-1319.

67. Evans G, Peers A, Sabaliauskas K. Particle dose estimation from frying in residential settings. *Indoor Air*. 2008;18:499-510.

68. Zhao W, Hopke PK, Norris G, et al. Source apportionment and analysis on ambient and personal exposure samples with a combined receptor model and an adaptive blank estimation strategy. *Atmos Environ*. 2006;40:3788-3801.

69. Ozkaynak H, Xue J, Spengler J, et al. Personal exposure to airborne particles and metals: results from the Particle TEAM study in Riverside, California. *J Expo Anal Environ Epidemiol*. 1996;6:57-78.

70. Habre R, Coull B, Moshier E, et al. Sources of indoor air pollution in New York City residences of asthmatic children. *J Exposure Sci Environ Epidemiol*. 2014;24:269-278.

71. Sørensen M, Loft S, Andersen HV, et al. Personal exposure to PM<sub>2.5</sub>, black smoke and NO<sub>2</sub> in Copenhagen: relationship to bedroom and outdoor concentrations covering seasonal variation. *J Expo Anal Environ Epidemiol*. 2005;15:413-422.

72. Chan WR, Price PN, Sohn MD, et al. Analysis of US residential air leakage database. Lawrence Berkeley National Laboratory. 2003.

73. Lavanchy V, Gäggeler H, Nyeki S, et al. Elemental carbon (EC) and black carbon (BC) measurements with a thermal method and an aethalometer at the high-alpine research station Jungfraujoch. *Atmos Environ*. 1999;33:2759-2769.

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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