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Effectiveness of portable HEPA air cleaners on reducing indoor endotoxin, PM₁₀, and coarse particulate matter in an agricultural cohort of children with asthma: A randomized intervention trial

Anne M. Riederer¹, Jennifer E. Krenz¹, Maria I. Tchong-French¹, Elizabeth Torres², Adriana Perez³, Lisa R. Younglove¹, Karen L. Jansen¹, David C. Hardie¹, Stephanie A. Farquhar¹, Paul D. Sampson⁴, Nervana Metwali⁵, Peter S. Thorne⁵, Catherine J. Karr^{1,6}

¹Department of Environmental and Occupational Health Sciences, University of Washington, Seattle, WA, USA

²Northwest Communities Education Center, Radio KDNA, Granger, WA, USA

³Yakima Valley Farm Workers Clinic, Toppenish, WA, USA

⁴Department of Biostatistics, University of Washington, Seattle, WA, USA

⁵Department of Occupational and Environmental Health, College of Public Health, University of Iowa, Iowa City, IA, USA

⁶Department of Pediatrics, University of Washington, Seattle, WA, USA

Abstract

We conducted a randomized trial of portable HEPA air cleaners in the homes of children age 6–12 years with asthma in the Yakima Valley, Washington. All families received asthma education while intervention families also received two HEPA cleaners (child's bedroom, living room). We collected 14-day integrated samples of endotoxin in settled dust and PM₁₀ and PM_{10–2.5} in the air of the children's bedrooms at baseline and one-year follow-up, and used linear regression to compare follow-up levels, adjusting for baseline. Seventy-one families (36 HEPA, 35 control) completed the study. Baseline geometric mean (GSD) endotoxin loadings were 1565 (6.3) EU/m² and 2110 (4.9) EU/m², respectively, in HEPA vs. control homes while PM₁₀ and PM_{10–2.5} were 22.5 (1.9) µg/m³ and 9.5 (2.9) µg/m³, respectively, in HEPA homes, and 19.8 (1.8) µg/m³ and 7.7 (2.0) µg/m³, respectively, in control homes. At follow-up, HEPA families had 46% lower (95% CI, 31%–57%) PM₁₀ on average than control families, consistent with prior studies. In the best-fit heterogeneous slopes model, HEPA families had 49% (95% CI, 6%–110%) and 89% lower

Correspondence: Anne M. Riederer, Department of Environmental & Occupational Health Sciences, University of Washington, 4225 Roosevelt Way NE, Seattle, WA 98105, USA. anneried@uw.edu.

AUTHOR CONTRIBUTIONS

AMR led formal analysis and writing; JEK conducted data curation and formal analysis; MTF, ET, AP, KLJ, DCH, and NM conducted investigation; LRY led project administration; SAF contributed to conceptualization; PDS contributed to formal analysis and writing; PST contributed to conceptualization, investigation, and writing; CJK led conceptualization and funding acquisition and contributed to writing.

CONFLICT OF INTEREST

Each author confirms that s/he has no conflict of interest.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

(95% CI, 28%–177%) $PM_{10-2.5}$ at follow-up, respectively, at 50th and 75th percentile baseline concentrations. Endotoxin loadings did not differ significantly at follow-up (4% lower, HEPA homes; 95% CI, –87% to 50%).

Keywords

asthma; children; coarse PM; endotoxin; HEPA cleaners

1 | INTRODUCTION

Endotoxin and particulate matter (PM) are common air pollutants in both agricultural and indoor residential settings and may be a particular concern in rural households of children with asthma.^{1–14} A 2015 update to the Institute of Medicine’s review on indoor environmental exposures and asthma concluded there is strong, consistent, and temporally appropriate evidence of an association between indoor endotoxin exposure and asthma exacerbations in children (and adults) stopping just short of labeling the association causal.¹⁵ Purported biologic mechanisms include inflammatory and atopic responses resulting from inhalation exposure to the bioactive lipopolysaccharide component.¹⁵ While $PM_{2.5}$ is also a known asthma trigger in children (and adults),^{16–18} a growing number of studies show associations between coarse PM (ie, $PM_{10-2.5}$) and pediatric asthma exacerbations as well.^{19–24} Coarse PM deposits in the upper airways where it induces inflammation according to animal and in vitro studies, although the relative contributions of particle size, mass, and composition are not fully understood.^{13,25–31} Endotoxin is found on both coarse and fine PM sampled from indoor residential environments^{32–37} and exposure to both may have additive and/or synergistic inflammatory effects on the airway epithelium.^{1,27,29,38–40}

In non-smoking homes with children, endotoxin is found in both airborne and settled dust and key exposure factors include the number of people living in the home and presence of pets, particularly dogs or cats.^{6,11,33,35,41–58} Despite the heterogeneity of endotoxin levels both within and between homes, the evidence for number of household occupants and presence of pets as predictors of endotoxin levels appears consistently in many studies, both large population-based cohorts and smaller panel studies, mostly from North America and Europe, showing associations regardless of whether endotoxin in indoor air and/or settled dust was measured, where in the home (eg, bedroom, living room, mattress) samples were collected, or geographic location. Additional exposure factors predicting residential endotoxin include home age and type (single or two family vs. multifamily), cleaning/cleanliness, having a vacuum with HEPA (high efficiency particulate air) filter, carpet (vs. hard flooring), presence of mold and/or pests (cockroaches, mice), smoking, heating type, crop or animal farming occupation or proximity to farms, and having a grain storage bin on the property.^{6,10,11,33,45,46,49,51,55,59–61} Relative humidity and season are associated with endotoxin in several studies, but not others (reviewed in Ref.10).

Since endotoxin is found on PM_{10} and $PM_{10-2.5}$ sampled in indoor residential air, endotoxin shares some of the same exposure factors as these PM size fractions, especially occupancy and pets (dogs, cats), but also central air conditioning (A/C), and window opening.^{32–35,37,62}

Exposure factors relevant to PM₁₀ and PM_{10-2.5}, but not necessarily endotoxin, include cooking and cleaning activities, and vigorously walking on carpet.^{37,62-64}

In our long-standing, community-engaged environmental health research partnership in the Lower Yakima Valley (Washington, USA), a region of intensive agricultural production impacted by episodes of high outdoor PM (PM_{2.5}, PM₁₀),⁶⁵⁻⁶⁷ pediatric asthma is a community concern. We previously identified adverse associations between ambient air quality (eg, PM_{2.5}, neighborhood NH₃, and a measure of animal feeding operation plume emissions) and asthma outcomes in children.⁶⁸⁻⁷⁰ Outdoor air contaminants can infiltrate homes, adding to indoor levels; one study of 40 Yakima Valley homes found that PM in household air typically exceeded paired outdoor levels.⁷¹ Protecting homes of children with asthma from infiltration of outdoor contaminants and controlling indoor sources are important features of asthma education programs in this community and others.

Evidence suggests that portable HEPA cleaners may help reduce asthma triggers in household air. To address community concerns, we conducted the Home Air in Agriculture Pediatric Intervention (HAPI) study to test the effectiveness of a commercially available portable HEPA cleaner on reducing asthma triggers in the homes of Yakima Valley children with poorly controlled asthma. The HAPI study is novel in addressing the home environment in a rural, agricultural, non-smoking setting. We previously reported the HEPA cleaners' effectiveness in reducing indoor PM_{2.5} but not NH₃ in these homes, our primary exposure outcomes.⁷² Here, we examine the effectiveness of the HEPA cleaners at reducing indoor levels of endotoxin in settled dust, and PM₁₀ and PM_{10-2.5} concentrations. Future analyses will examine whether providing the HEPA cleaners influenced asthma health outcomes in the cohort.

2 | METHODS

2.1 | Participant recruitment/enrollment and portable HEPA cleaner intervention

The HAPI study design and general procedures are detailed in Ref.⁷³ Briefly, we recruited families of children aged 6–12 years with poorly controlled asthma through the Yakima Valley Farm Worker Clinic (YVFWC) asthma education home visiting program from 2015 to 2017. Eligibility criteria included non-smoking homes within 800 m of crop and/or animal agriculture operations.

Seventy-nine families were enrolled/consented to achieve a target sample size of 66 (based on adequate power to identify ~35–50% reductions in indoor PM_{2.5}). All families received YVFWC's standard 3-module community health worker asthma education program which, among other things, instructs participants on behaviors such as low volatile organic compound cleaning, controlling dust by wet dusting/mopping and frequent vacuuming with a HEPA filter vacuum, removing pets from sleeping areas, using ventilation when cooking and showering, and other strategies that may reduce indoor contaminants such as endotoxin and PM. The study was overseen by the University of Washington (UW) Institutional Review Board and YVFWC research review committee.

At the clinic enrollment visit or during a baseline home visit, caregivers were interviewed to collect data on demographics, number of household occupants, and proximity to agricultural activities and roadways. At the end of the baseline visit, each family was randomized to either the HEPA (intervention) or control group. The intervention group received two indoor portable HEPA cleaners (Austin Air Pet Machine 410[®]; Austin Air Systems Ltd.), one for the child's sleeping area and one for the main living area. The device has three speed options (high, medium, low), is rated for 99.97% removal of particles >0.3 μm and 95% removal of particles >0.1 μm , and is rated for medium and large-sized residential rooms, with an air delivery rate of 400 ft^3/min on high speed.⁷⁴ Austin Air technicians helped install HOBO[®] Onset[®] motor on/off loggers (Onset Computer Corporation) inside each of the sleeping area units and approximately half of the living area units to monitor use. HAPI field technicians helped participants place the filters, advised them on appropriate use (ie, keep it in the same room, on at all times for the study duration, and run it at the highest speed tolerable), and gave them printed fact sheets as reminders.

2.2 | Indoor air quality (IAQ) sampling and analysis

We collected 14-day integrated samples of endotoxin in settled dust and $\text{PM}_{2.5}$ and PM_{10} in the child's main sleeping area air during the baseline visit and a follow-up visit one year later. $\text{PM}_{2.5}$ sampling and analysis methods are detailed in Ref.⁷²

2.2.1 | Endotoxin in settled dust—Electrostatic Dust Collectors (EDCs), comprised of 2–4 electret fiber electrostatic cloths inside a polypropylene folder, are simple, low-cost devices validated for passive sampling of bioaerosols in homes.^{75–77} EDCs were supplied by the Thorne Laboratory at the University of Iowa (UI) (Iowa City, IA, USA), where they were initially cleaned, depyrogenated, loaded with two cloths (one for the primary sample, the second to archive for QC analysis if needed), barcoded and packaged individually in zipper lock bags then shipped to the field laboratory. The EDCs were deployed in the child's sleeping area, alongside the IV pole, on the flat surface of a portable TV tray table or similar surface available in the room, ~1 m from the floor. Wearing clean gloves, field technicians opened the folders to expose the cloths, and secured them to the table with binder clips, careful to minimize contact with the cloths. At the end of the sampling period, EDCs were collected by closing the folder, placing it in its bag, and transporting cold to the field laboratory, where they were stored at 4°C prior to shipping cold to UI. At UI, samples were stored at –20°C then extracted and analyzed in batches of 20 for endotoxin using the kinetic chromogenic *Limulus* amebocyte lysate assay, following established methods.^{76,78,79} QA/QC measures included field blanks, and random repeat analyses. Field blanks were collected at every 7th home and treated identically as samples except they were opened for approximately one minute in the home when the primary EDCs were deployed, then placed back in the zipper lock bag, transported back to the field laboratory, and shipped to UI with other samples. The lower limit of detection (LOD) was 11.7 endotoxin units (EU)/ m^2 . Field blanks below the LOD were assigned a value of $\text{LOD}/\sqrt{2}$. Loadings were blank corrected by subtracting the median loading of the field blanks from each sample. Blank corrected loadings were adjusted for duration by dividing the measured duration by 14 days.

2.2.2 | PM₁₀ and PM_{10-2.5}—We collected gravimetric PM₁₀ and PM_{2.5} samples using RTI MicroPEMs v 3.2A (RTI International) fitted with PM₁₀ and PM_{2.5} inlets and 25 mm 3 µm pore size Teflon filters. MicroPEMs were enclosed in cages hung at the child's breathing height (approximately 1 m from the floor) from an IV pole in the sleeping area. Although MicroPEMs are designed as personal exposure samplers, one study showed good agreement between MicroPEMs deployed as gravimetric area samplers and ambient monitoring stations at PM₁₀ concentrations ranging from 20 to 140 µg/m³.⁸⁰

Pre-deployment flow rates were set to 0.5 L/min using a TSI 4140 flow meter (TSI Inc.), with flow rates checked again using the TSI at the field laboratory after the equipment was picked up from the home. If both pre-and post-deployment TSI flow rates were available, we used their mean as the primary flow rate estimate. Missing pre-deployment flow rates (~4% of samples) were imputed using the target flow rate (99% of non-missing observations were 0.5 L/min). Missing post-deployment flow rates (19% of sleeping area samples) were imputed using the median of the non-missing observations. Temperature and relative humidity (RH) in the sleeping area were also recorded every 5 min during MicroPEM deployment using a HOBO® Onset® U12-011 logger (Onset Computer Corporation). For 15.5% of samples, we did not have temperature/RH HOBOS available; in these cases, we imputed the missing data using the MicroPEM temperature and a linear regression equation fit to the HOBO vs. MicroPEM measurements from homes with both instruments deployed (Pearson $\rho = 0.87$, $p < 0.01$, $n = 118$).

We processed MicroPEM files logging timestamps, temperature, nephelometer flow rates, and other data using the rtimicropem package, with further processing in R, version 3.6.1 (R Foundation for Statistical Computing). Technicians inspected plots of pump inlet and orifice pressures to evaluate potential pump malfunctions or filter overloading; overloading was assumed if inlet pressure reached five inches of water while the orifice pressure simultaneously destabilized and the flow rate rapidly increased. In these cases, the sample duration was cut off at the date/time just before this transition occurred. For sample durations <10 days, raw data files and field notes were examined for error messages and possible explanations, including tampering or loss of electrical or battery power. A subset of files from MicroPEMs that malfunctioned or had overloaded filters (~5% of the MicroPEM data) were sent to RTI for a reliability check.

Sample air volumes were calculated by multiplying the mean flow rate by the sample duration. Filters were conditioned and weighed to the nearest 0.5 µg following standard procedures^{81,82} in a temperature and humidity controlled laboratory at UW (Seattle, WA), with filter mass changes divided by sample volumes to calculate PM_{2.5} and PM₁₀ concentrations. Quality assurance/quality control (QA/QC) procedures were described previously⁷² and included laboratory and field blank filters, reweighing of filters with negative mass changes, and flagging filters with small holes or tears. We used the method detection limit (MDL), defined as three times the median absolute deviation of the field blanks mass changes, to blank correct the primary samples. Expressed as concentrations, the PM_{2.5} and PM₁₀ MDLs were 0.2 µg/m³ and 0.1 µg/m³, respectively, at an assumed flow rate of 0.5 L/min and 14 days sample duration; no measurements were below their respective MDLs. One pair of co-located PM₁₀ samples showed good agreement between

measured concentrations ($22.60 \mu\text{g}/\text{m}^3$ and $20.0 \mu\text{g}/\text{m}^3$); precision of two pairs of co-located $\text{PM}_{2.5}$ measurements was also good and is reported in Ref.⁷² $\text{PM}_{10-2.5}$ concentrations were calculated using the subtraction method, that is, subtracting the co-located blank-corrected $\text{PM}_{2.5}$ concentration from the blank-corrected PM_{10} concentration. Following,⁸³ the $\text{PM}_{10-2.5}$ MDL was defined as the square root of the sum of the squares of the $\text{PM}_{2.5}$ and PM_{10} measurements, or $0.2 \mu\text{g}/\text{m}^3$; one observation below this value was replaced with the MDL.

2.3 | IAQ questionnaires

Caregivers were interviewed during the initial clinic visit and baseline sample period to collect information on demographic and residential proximity (eg, proximity to dairy and/or cattle operations) characteristics. Trained field technicians collected data on factors potentially affecting IAQ at the baseline and follow-up air sampling visits using an observational, 11-module Home Environmental Checklist (HEC), adapted from the EPA Asthma Home Environment Checklist⁸⁴ and described in Ref.⁷² They also used a Bosch GLM 15 Laser Measure (Robert Bosch Tool Corp.) to measure the sleeping area volume. When the technicians visited the homes to pick up equipment on the last day of air sampling, they briefly interviewed caregivers on IAQ-related characteristics and activities during the previous 24 h. Specifically, they asked about the number of windows open, any cooking or burning food activity, sweeping and vacuuming activity, and A/C and candle/incense use. Additionally, HEPA families were asked whether they turned either air cleaner off in the past month, if yes, for how many days approximately, and the speed at which they usually run the two units during the day and at night.

While conducting the study, we became aware of a community weatherization program that was also offered by the Yakima Valley Farm Workers Clinic, which provided HEPA furnace filters and HEPA vacuum cleaners to some families and conducted more substantial household retrofits (eg, insulation, air and/or duct sealing, roof repair, exhaust fan replacement, carpet replacement with laminated wood flooring) in select cases. We identified HAPI families participating in this program before or during final air sampling in our study.

2.4 | Data processing and statistical analyses

A detailed description of how we processed the MicroPEM data is provided in Ref.⁷² We used R (versions 3.1.3 and 3.6.1) and a statistical significance criterion of $p = 0.05$ for all statistical analyses. Chi-square and Wilcoxon rank-sum tests were used to compare demographic, residential proximity, and IAQ characteristics between HEPA and control families, to evaluate the success of the randomization. We used natural log transformed measurements for all calculations since histograms of the raw data appeared log normal and Pearson correlation coefficients to assess bivariate associations between baseline and follow-up levels. We fit ANCOVA (analysis of covariance) models comparing the effect of the HEPA cleaners on reducing endotoxin loadings, and PM_{10} and $\text{PM}_{10-2.5}$ concentrations in the sleeping area, adjusting for baseline levels. Diagnostics included residuals and leverage plots, and potential outliers/leverage points were investigated by fitting the models with and without these observations and comparing results. We also tested for homogenous

vs. heterogeneous slopes in the fitted regressions for each group by examining the 95% confidence intervals (CIs) of a group $\times$ baseline interaction term.

We explored various factors potentially affecting the precision of the model results by adding them individually to the main model and comparing results. These included indoor sources and characteristics/activities affecting ventilation/air exchange, specifically occupancy (number of adults, number of children), current pet ownership (dogs/cats), moving between sampling visits, weatherization program participation, room volume, RH, window opening (usual), have A/C, used A/C last 24 h of air sampling, use humidifier (usual), cleaning activities (ie, have working vacuum with HEPA filter, cleaned for visit, number of times vacuumed sleeping area during air sampling, number of times dusted the sleeping area, vacuumed last 24 h of air sampling (yes/no), swept last 24 h of air sampling (yes/no)), visitor habits (people remove shoes when they visit, people wipe feet when they visit), and technician-observed level of dust on surfaces in sleeping area. Additional variables tested in the endotoxin model included residence location (farm, rural, town), proximity (<800 m) to dairy/cattle farming, furry/feathered pets (indoor or outdoor; including dogs and/or cats), furry/feathered pets allowed in child's sleeping area, household pests (seen cockroaches past year, problems with mice or rats past year), have doormat at every outside door, sleeping area flooring type (carpeting vs. wood/tile/vinyl/concrete), evidence of water damage in sleeping area room/closet, and evidence of mold/mildew in sleeping area room/closet. The last two variables were technician-observed while the rest were self-reported. Additional variables tested in the PM models included type of home heating (electric vs. gas/heating oil/wood), candle/incense use (usual), and cooking activity last 24 h of air sampling (food burning, stove use, oven use).

3 | RESULTS

3.1 | Demographic, home characteristics, and IAQ-related behaviors of the cohort

Demographic characteristics of the 71 families completing the study through the one-year follow-up sampling (89% retention) are described in Ref.⁷² Briefly, approximately half lived in a rural vs. in town setting, with approximately half of the rural homes located on farms. All were within 800 m of crop agriculture (a criterion for study inclusion), while approximately a third were also within 800 m of dairy or beef cattle operations. Two-thirds lived in detached single-family homes while one-third lived in apartments/duplexes or trailers. Most had lived at their current address for several years or more, but seven families moved between baseline and follow-up sampling. Most reported an annual total household income <\$60 000 (with 58% reporting <\$30 000) and nearly all children in the study (96%) were on public health insurance. The HEPA and control families did not differ significantly with respect to any of these demographic or home siting characteristics.

Table 1 summarizes selected IAQ-related characteristics at baseline of the HEPA vs. control homes. Both groups had an average of two adults and three children living in the home while more than half reported having dogs and/or cats or other furry or feathered pets. Most had air conditioning, approximately a quarter used a humidifier, and a third reported regular candle/incense use. A few families reported seeing cockroaches in the past year while approximately 40% said they had problems with mice or rats in the past year. Most

reported cleaning for the sampling visit and vacuuming and/or dusting several times during air sampling. Most said visitors wiped their feet when they came to inside while fewer families required visitors to remove their shoes. In the child's sleeping area, approximately half the families had carpeting (wall-to-wall or area rugs) while three quarters reported always or sometimes opening windows to ventilate when the weather allows. Technicians observed moderate levels of dust in most of the sleeping areas and evidence of water damage or mold/mildew in less than a quarter of homes. HEPA and control families were not statistically significantly different with respect to any of these key IAQ characteristics. Nine families (six HEPA, three control) participated in the YVFWC weatherization program during the study.

3.2 | Baseline and follow-up endotoxin loadings, and PM₁₀, and PM_{10–2.5} concentrations in the child's sleeping area

The HEPA and control families did not differ significantly with respect to season, year, or proportion of weekdays of IAQ sampling. Table 2 summarizes the endotoxin loadings and PM₁₀ and PM_{10–2.5} concentrations in the child's sleeping area. Baseline endotoxin loadings were similar between the HEPA and control groups with geometric means (geometric SD) of 1565 (6.3) EU/m² and 2110 (4.9) EU/m², respectively. At follow-up, geometric mean endotoxin loadings dropped 20% (to 1259 EU/m²) in the HEPA group and 29% (1493 EU/m²) in the control group. Within household baseline and follow-up, endotoxin loading measurements were correlated (Pearson $\rho = 0.55$, $p < 0.0001$, $n = 68$).

Baseline PM₁₀ and PM_{10–2.5} concentrations were also similar between groups with geometric mean concentrations of 22.5 (1.8) $\mu\text{g}/\text{m}^3$ and 9.5 (2.9) $\mu\text{g}/\text{m}^3$, respectively, in the HEPA group and 19.8 (1.8) $\mu\text{g}/\text{m}^3$ and 7.7 (2.0) $\mu\text{g}/\text{m}^3$, respectively, in the control group. At follow-up, the geometric mean PM₁₀ and PM_{10–2.5} concentrations decreased 55% and 45%, respectively, in the HEPA group vs. 14% and 22%, respectively, in the control group. Within household baseline and follow-up, PM₁₀ and PM_{10–2.5} measurements were correlated (Pearson ρ 's: PM₁₀ 0.50, $p < 0.0001$, $n = 62$; PM_{10–2.5} 0.55, $p < 0.0001$, $n = 57$). The strong correlations between baseline and follow-up measurements for all three contaminants indicated the importance of controlling for baseline levels in the ANCOVA models.

Nine HAPI families (six HEPA, three control) participated in the YVFWC weatherization program during the study. Geometric mean endotoxin loadings did not differ significantly between weatherization and non-weatherization families at baseline—2162 (6.1) EU/m² ($n = 9$) vs. 1768 (5.5) EU/m² ($n = 62$), respectively (Wilcoxon rank-sum $p = 0.56$)—or follow-up—879 (6.5) EU/m² ($n = 9$) vs. 1466 (4.8) EU/m² ($n = 59$), respectively (Wilcoxon rank-sum $p = 0.48$). Similarly, geometric mean PM₁₀ and PM_{10–2.5} concentrations did not differ significantly between weatherization and non-weatherization families at either baseline or follow-up. At baseline, the geometric mean PM₁₀ was 22.9 (1.7) $\mu\text{g}/\text{m}^3$ vs. 20.9 (1.8) $\mu\text{g}/\text{m}^3$ (Wilcoxon rank-sum $p = 0.57$) for weatherization ($n = 9$) and non-weatherization ($n = 62$) families, respectively, while geometric mean PM_{10–2.5} was 12.2 (1.7) $\mu\text{g}/\text{m}^3$ vs. 8.4 (2.5) $\mu\text{g}/\text{m}^3$ (Wilcoxon rank-sum $p = 0.12$) for weatherization ($n = 7$) and non-weatherization ($n = 58$) families. At follow-up, geometric mean PM₁₀ was 10.6 (1.8) $\mu\text{g}/\text{m}^3$ ($n = 7$) vs. 13.6 (1.9)

$\mu\text{g}/\text{m}^3$ ($n = 55$) (Wilcoxon rank-sum $p = 0.39$) and geometric mean $\text{PM}_{10-2.5}$ was 5.1 (2.6) $\mu\text{g}/\text{m}^3$ ($n = 5$) vs. 5.7 (2.3) $\mu\text{g}/\text{m}^3$ ($n = 52$) (Wilcoxon rank-sum $p = 0.94$) for weatherization and non-weatherization families, respectively.

3.3 | HEPA cleaner effects

In the ANCOVA analysis, HEPA families had 4% lower endotoxin loadings at follow-up on average compared with control families, adjusting for baseline loadings, but the difference was not statistically significant (95% CI, -87% to 50%; multiple $R^2 = 0.30$) (Figure 1). The results were robust to outliers/influential points. Several IAQ-related variables were significantly associated with follow-up endotoxin loadings when tested in sensitivity analyses (ie, residence location, number of children living in the home, cleaned for visit, seen cockroaches the past year) but did not change the overall conclusion of no statistically significant intervention effect (see Table S1).

In the PM_{10} ANCOVA model, HEPA families had 46% lower (95% CI, 31%–57%; multiple $R^2 = 0.47$) PM_{10} at follow-up, on average, than control families, adjusting for baseline concentrations (Figure 2). Results were robust to outliers/influential points. Of the IAQ variables tested in sensitivity analyses, only vacuuming the child's sleeping area on the last day of air sampling was significantly associated with follow-up PM_{10} concentrations; including this covariate in the model increased the HEPA effect by 0.4% (see Table S2).

For $\text{PM}_{10-2.5}$, we fit a model with heterogeneous slopes since the regression parameter for the group $\times$ baseline concentration interaction was statistically significant and evaluated the HEPA effect at selected percentiles of the baseline concentrations (Figure 3; multiple $R^2 = 0.45$). At the 50th and 75th percentile baseline $\text{PM}_{10-2.5}$ concentrations, respectively, HEPA families had 49% (95% CI, 6%–110%) and 89% lower (95% CI, 28%–177%) $\text{PM}_{10-2.5}$ at follow-up compared with control families. Similar to the PM_{10} results, the $\text{PM}_{10-2.5}$ results were robust to outliers/influential points. Of the covariates tested in sensitivity analyses, none were statistically significant predictors of follow-up $\text{PM}_{10-2.5}$ concentrations or changed the HEPA effect at either the 50th or 75th percentile baseline concentrations (see Table S3).

4 | DISCUSSION

In this randomized trial of the effectiveness of home HEPA cleaners in a rural agricultural setting, we previously reported a 60% reduction in $\text{PM}_{2.5}$ in the child's sleeping area in HEPA vs. control homes.⁷² Although the trial was powered to detect 30–50% reductions in $\text{PM}_{2.5}$ (based on achievements of earlier trials in urban cohorts)^{85,86} and not other contaminants, we also found significant reductions in PM_{10} and $\text{PM}_{10-2.5}$. Geometric mean sleeping area PM_{10} and $\text{PM}_{10-2.5}$ concentrations decreased 55% and 45%, respectively, in HEPA homes vs. 14% and 22%, respectively, in control homes. All participants received information and supplies for green cleaning and education on reducing asthma triggers in the home, including sources and activities contributing to PM and endotoxin. Families randomized to also receive portable HEPA cleaners for the sleeping and main living areas showed larger reductions in PM_{10} and $\text{PM}_{10-2.5}$ compared with control families, similar to the $\text{PM}_{2.5}$ effect we observed. This was expected since the HEPA units are designed to filter

a wide range of particle sizes, and because the PM_{2.5} size fraction accounted for a large proportion of PM₁₀ in the HAPI homes. On average, the sleeping area PM_{2.5} concentrations were approximately half the magnitude of the PM₁₀ measurements: the mean (SD) PM_{2.5} to PM₁₀ ratio was 0.52 (0.20) while the mean PM_{10-2.5} to PM₁₀ ratio was 0.48 (0.20), among 122 collocated PM_{2.5} and PM₁₀ measurements.

In general, the PM₁₀ levels we measured in HAPI homes were similar to^{64,87-90} or lower than⁹¹⁻⁹⁵ those reported for other homes (non-smoking, non-wood burning) with children in urban/suburban areas. The PM_{10-2.5} levels were also similar to^{34,37,62,64,96,97} or slightly higher than^{32,33,35,98} other studies reporting indoor residential measurements.

The magnitude of the HEPA cleaner effect we observed for PM₁₀ and PM_{10-2.5} was comparable to those observed in previous trials measuring these specific size fractions, although the number of studies is limited. Eggleston et al.⁸⁵ conducted a randomized trial of asthma education plus HEPA cleaners and other allergen reduction measures (eg, pest extermination) in the homes of 100 children with asthma in urban Baltimore, USA. They observed 30% and 39% reductions in median bedroom PM₁₀ at 6 and 12 months, respectively, in the intervention homes—reductions they attributed primarily to the HEPA cleaners—while median levels increased in the control homes.⁸⁵ A similar 3-arm randomized trial of HEPA cleaner effects on sleeping area PM₁₀ and PM_{2.5} in urban Baltimore homes of children with asthma reported a mean baseline to follow-up (6 months) PM_{10-2.5} difference of -9.6 (16.0) µg/m³ in the two intervention groups combined (HEPA cleaners, HEPA cleaners plus health coach), which differed significantly from the control group (delayed HEPA cleaners) (Mann-Whitney $p = 0.009$).⁸⁶ The majority of homes in both Baltimore trials had smokers, with baseline PM₁₀ and PM_{10-2.5} concentrations approximately double the HAPI baseline on average.^{85,86}

McNamara et al.⁹⁹ examined HEPA cleaner effects on winter-time living area PM in 48 woodstove homes of children with asthma in Montana, Idaho and Alaska (USA), with baseline PM_{10-2.5} levels similar to HAPI. The primary outcome was PM_{2.5}, and they indeed found significant reductions in PM_{2.5}. However, the difference in PM_{10-2.5} between the intervention homes, who received HEPA cleaners with high (ie, 13) MERV (Minimum Efficiency Reporting Value) rated filters, vs. control homes who received low MERV (ie, 2, eg, low-efficiency furnace filters) filters, was not statistically significant and levels decreased in both groups.⁹⁹

Three recent studies from East Asia, although not focused explicitly on homes of children with asthma, provide additional evidence on the effectiveness of portable HEPA cleaners on indoor concentrations of these particle size fractions in non-smoking homes. In a crossover trial conducted at several urban locations in Japan, two groups of 16 healthy adults were randomly assigned to either HEPA or sham filtration (in the living room) for 4 weeks followed by the alternate treatment with a 4 weeks washout period in between.⁹⁸ Mean PM_{10-2.5} and PM_{2.5} (PM₁₀ was not reported) decreased slightly during true compared with sham filtration but the differences were not statistically significant in a two-sample t test.⁹⁸ Mean PM_{10-2.5} levels in this study were generally low (~3 µg/m³) compared with what we and others have measured. A double-blind, placebo-controlled trial of HEPA cleaners

in bedrooms and living rooms of 44 adults with allergic rhinitis in two cities in South Korea found 53.2% and 30.7% decreases on average in bedroom and living room PM₁₀, respectively, using a repeated measures approach.¹⁰⁰ Mean baseline PM₁₀ in this study was 33.4 (5.6) µg/m³ in the bedrooms and 15.5 (2.1) µg/m³ in the living rooms, similar to what we observed in HAPI. A single-blind 4-day crossover trial of HEPA cleaners in 20 urban apartments in Chongqing, China reported significant reductions in both PM₁₀ and PM_{10-2.5} compared with sham filtration.¹⁰¹ Mean PM₁₀ and PM_{10-2.5} concentrations during sham filtration were higher than the HAPI homes—56.1 (35.8) µg/m³ and 51.2 (35.9) µg/m³, respectively—dropping to 36.5 (20.8) µg/m³ and 34.1 (19.8) µg/m³, respectively, during true filtration.¹⁰¹ Together, these trials as well as the HAPI study demonstrate that PM₁₀ and PM_{10-2.5} reductions of approximately one-third to one-half may be achievable in a variety of real-world settings, at a range of baseline concentrations.

We observed no meaningful change in endotoxin loadings in homes assigned to the HEPA vs. control families. From baseline to follow-up, geometric mean EDC endotoxin dropped 20% in HEPA homes and 29% in control homes, but the HEPA vs. control group difference was not statistically significant. We examined multiple potential likely sources of endotoxin in sensitivity analyses and found a few significant influences on follow-up loadings, that is, residence location, number of children in the home, cleaning for the sampling visit, seen cockroaches in the past year (Table S3); however, none changed the overall conclusion of no statistically significant HEPA cleaner effect. Our finding of no intervention effect could also be due in part to unmeasured confounding.

The geometric mean endotoxin loadings we measured in HAPI homes were similar to those of other studies using EDCs with two-week deployments, including studies of farm homes in Iowa, USA,^{75,77} homes near livestock farms in dense agricultural regions of the Netherlands,⁴⁴ and non-smoking urban apartments and homes in Denmark,^{43,102} and the Netherlands,¹⁰³ and slightly higher than those in 60 homes of children in Wellington, NZ using four-week EDC deployments.¹⁰⁴ One EDC study found higher endotoxin loadings in farm vs. non-farm homes.¹⁰⁵ Geometric mean loadings among HAPI farm homes were slightly higher than those in rural/non-farm or in-town settings, but the differences were not statistically significant at baseline or follow-up (analysis of variance $p = 0.9044$ baseline, $p = 0.0977$ follow-up).

We found no prior published studies of HEPA cleaner effectiveness on endotoxin in settled or airborne PM in residential settings. One experimental study in Beijing, China, evaluated the potential for portable HEPA cleaners to increase airborne endotoxin by measuring endotoxin on new HEPA filters in units operated in a room for a week, on days with different ambient air quality.¹⁰⁶ They measured endotoxin, viable, dead, and free bacterial DNA on five used filters, as well as the effect of an existing HEPA cleaner on airborne endotoxin using gravimetric air samples and particle number counts 30 cm downstream from the unit as it was turned on and off for 3 h.¹⁰⁶ The authors reported a 10-fold increase in PM-mass normalized airborne endotoxin in filtered vs. unfiltered air from the existing unit, but a statistically significant decrease using the new HEPA filter units.¹⁰⁶ This demonstrates the potential need to regularly replace filters, to avoid unintentionally increasing instead of decreasing airborne endotoxin. In PM size-resolved analyses, the authors showed that the

clean filters effectively removed airborne endotoxin associated with the larger size fractions (eg, $>2.1\ \mu\text{m}$) while endotoxin associated with smaller fractions, especially $1.1\text{--}2.1\ \mu\text{m}$, increased.¹⁰⁶ They also found strong correlations between free but not dead bacterial DNA and endotoxin loadings (EU/cm^2) on the routinely used filters, suggesting that both the endotoxin and free DNA potentially originated from bacteria accumulated on the filter, later degraded by the mechanical stresses of air flow across the filter surface, and subsequently entrained in the outgoing air flow.¹⁰⁶

Although not a HEPA cleaner trial, the Louisa Environmental Intervention Project (LEIP) compared the effectiveness of an extensive household intervention that included a complete professional house cleaning including draperies, carpets, mattresses, bedding, and HVAC systems plus provision of a high efficiency vacuum cleaner and education to an educational intervention alone.¹⁰⁷ The LEIP study assessed exposures to airborne endotoxin and reservoir dust endotoxin across four time points in four household locations in 89 rural Iowa homes. The investigators reported significant reductions in airborne and reservoir dust endotoxin that were most pronounced in farm homes. The extensive intervention compared with the educational intervention was associated with reduced reservoir dust endotoxin in farm homes. The LEIP study showed that effective reduction in endotoxin levels mainly in the bedroom was associated with a reduction in nighttime respiratory symptoms in asthmatic children.

We measured endotoxin as a secondary outcome in the HAPI trial because of its importance as an asthma trigger,¹⁵ and chose the EDC method for its convenience, minimal intrusiveness, and low maintenance requirements during long (ie, 14 days) deployment periods.^{75–77} It is unclear whether our finding of no HEPA effect on endotoxin loadings is attributable to the air cleaners themselves, high dependence on use by participants (ie, more so than required to observe an effect on PM), a difference in the settled dust size fractions sampled by the EDCs vs. those filtered by the HEPA units, a combination of all three, or some other unmeasured factor. We previously reported that we were unable to objectively measure air cleaner use in our study because of battery and other failures of the HOBO on/off data loggers we installed in the units to measure compliance.⁷² The few loggers that did provide complete data showed that not all families used their HEPA cleaners consistently, similar to what prior HEPA trials report.^{85,99,108–110} Motivating compliant use is important for optimal performance with respect to airborne PM¹¹¹ but it is unclear what effect HEPA cleaners have on settled dust loadings at any compliance level.

In indoor residential settings, endotoxin has been found on both airborne fine and coarse PM^{32–37,112–115} as well as in settled dust measured via EDC.^{43,44,75,105,116} While toxicological investigations have long established that inhalation of endotoxin-containing dust, even at low environmental levels (eg, ng/m^3), triggers the secretion of inflammatory cytokines and subsequent airway inflammatory responses, consensus is lacking on which size fractions are most important.^{2,78} The EDC method was developed as a cost-effective alternative to active sampling and a more efficient alternative to passive methods (eg, petri dish dustfall collection), which require long deployments to collect sufficient volumes.¹⁰⁵ The EDC's electrostatic fibers attract and capture airborne dust more rapidly than a truly passive device, and one validation study in nine farm and seven non-farm homes

showed EDC endotoxin loadings more closely correlated with concentrations in airborne PM₁₀ samples than concentrations in vacuumed floor dust.¹⁰⁵ Another study in 10 farm homes showed a strong correlation (ie, Pearson $\rho = 0.70$, $p < 0.001$) between 7-day endotoxin measurements from collocated EDCs and button samplers designed to sample 95% of airborne 1 μm particles and 50% of airborne particles up to 100 μm .⁷⁵ In this study, optical particle counters were also deployed during the winter sampling season to estimate the PM size fractions associated with each type of endotoxin measurement. Notably, winter EDC endotoxin loadings were strongly correlated with PM_{2.5} but not PM_{10-2.5}, while button sampler endotoxin concentrations were strongly correlated (ie, $\rho > 0.90$) with both size fractions.⁷⁵ This suggests that the dust size fractions captured by the EDCs might not represent the full range of endotoxin-containing fine and coarse PM size fractions present in indoor air. Since endotoxin is generally stable/long-lived in indoor residential environments,^{117,118} and since settled dust serves as a reservoir for airborne PM,¹¹⁹ controlling both airborne and settled dust endotoxin may be important for reducing asthma exacerbation risks. Despite our finding of no HEPA effect on EDC endotoxin, our findings that the HEPA cleaners effectively lowered both fine and coarse airborne PM in the children's bedrooms is encouraging, considering that both size fractions likely contained endotoxin although we did not measure this directly. Additional studies are needed to confirm whether or not HEPA cleaners are effective at directly lowering airborne endotoxin in homes of children with asthma.

The main strengths of this study are the assessment of both endotoxin and PM₁₀/PM_{10-2.5}, consideration of multiple household factors potentially influencing levels of these contaminants, and the rural, agricultural setting. Limitations are our inability to objectively measure compliance and the lack of blinding to intervention group. Our findings were nonetheless robust in a variety of sensitivity analyses and show that HEPA cleaners can effectively reduce by approximately half PM₁₀ and PM_{10-2.5} in the bedrooms of children with asthma.

5 | CONCLUSIONS

The HAPI study contributes to a growing body of evidence that portable HEPA cleaners are effective not only at lowering indoor PM_{2.5} concentrations but also at lowering levels of the larger particle size fractions (ie, PM₁₀, PM_{10-2.5}) in a variety of real-world residential settings. Portable HEPA cleaners, combined with asthma education, indoor source control and household ventilation, may help reduce pediatric asthma morbidity. Access to these solutions should be promoted particularly in low-income rural agricultural communities with high pediatric asthma burdens. Further studies are needed to evaluate the effect of HEPA cleaners on airborne endotoxin in homes and to elucidate optimal use and maintenance scenarios in which portable HEPA cleaners can reduce rather than contribute to children's indoor endotoxin exposures.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Practical Implications

- In this randomized trial of rural agricultural households, HEPA cleaners effectively reduced PM_{10} in the child's bedroom 46% compared with asthma education alone; meaningful reductions in endotoxin loadings were not observed.
- The HEPA cleaners also reduced $PM_{10-2.5}$ by differing amounts depending on baseline levels, that is, 49% at 50th percentile baseline $PM_{10-2.5}$ and 89% at 75th percentile baseline $PM_{10-2.5}$;
- These PM_{10} and $PM_{10-2.5}$ reductions are similar to those found in trials of HEPA cleaners in other residential settings, although the number of studies is limited.
- This trial adds further evidence that portable HEPA air cleaners can complement community health worker asthma education programs and be part of an effective overall strategy to reduce asthma triggers in the bedrooms of children with asthma.

FIGURE 1.

Scatter plot of baseline vs. follow-up ln-transformed endotoxin loading (EU/m²) in the child's sleeping area. Fitted lines represent the ANCOVA model equations for the HEPA (darker solid line) and control group (lighter solid line); the vertical difference between solid lines represents the intervention effect in the sleeping area; the lines are nearly indistinguishable in this figure, with a difference of -0.0368 which corresponds to a percent difference of $100\% \times (1 - \exp(\text{diff}))$ or more specifically in this case, $100\% \times (1 - \exp(-0.0368)) = 3.6\%$, between the HEPA and control groups; note that the axes are logarithmically scaled to correspond with the statistical analysis of log concentrations

FIGURE 2.

Scatter plot of baseline vs. follow-up PM₁₀ in the child's sleeping area. Fitted lines represent the ANCOVA model equations for the HEPA (darker line) and control group (lighter line); the vertical difference between lines (-0.6077 , corresponding to a percent difference of $100\% \times (1 - \exp(\text{diff}))$, or more specifically in this case, $100\% \times (1 - \exp(-0.6077)) = 45.5\%$) represents the intervention effect; note that the axes are logarithmically scaled to correspond with the statistical analysis of log concentrations

FIGURE 3.

Scatter plot of baseline vs. follow-up $PM_{10-2.5}$ in the child's sleeping area. Fitted lines represent the model equations with different slopes for the HEPA (darker line) and control group (lighter line); gray vertical lines represent (from left to right) the 25th, 50th, 75th and 90th percentiles of baseline $PM_{10-2.5}$ concentrations and the vertical difference between lines represents the intervention effect; letting δ be the difference between the two lines at these percentiles, the percent differences, $100\% \times (1 - \exp(\delta))$, are -12.9%, 49.2%, 88.8%, and 159.5%, respectively; note that the axes are logarithmically scaled to correspond with the statistical analysis of log concentrations

Selected characteristics of participant homes at baseline that may affect indoor air quality ($N = 70$)^a, HEPA vs. control households

TABLE 1

Characteristic/Activity	HEPA ($n = 35$)		Control ($n = 35$)		Difference <i>p</i> -value ^b
	Mean (SD)	Percent ($n = 36$) ^c	Mean (SD)	Percent	
Residence location					
On a farm		22.2		28.6	0.83
Rural, not on a farm		22.2		20.0	
Town		55.6		51.4	
Within 800 m of dairy and/or cattle operations		22.2 ($n = 36$) ^c		40.0	0.22
Occupancy					
		($n = 36$) ^c			
Number adults	2.3 (0.7)		2.5 (1.0)		0.60
Number children	3.0 (1.4)		2.9 (1.7)		0.52
Pets					
Have dog(s)		62.9		57.1	0.63
Have cat(s)		20.0		17.1	0.76
Have any furry/feathered pets allowed indoors		52.8		31.4	0.07
Have furry/feathered outdoor pets (eg, chicken, goat, cow, horse)		11.1		5.7	0.41
Type of heat used (more than one type possible)					
Electric (furnace/baseboard/space heaters)		77.1		74.3	1.0
Gas/heating oil/wood		8.6		8.6	1.0
Have air conditioner		91.4		97.1	0.30
Use humidifier (usual)		28.6		23.5 ^d	0.63
Use candles/incense (usual)		37.1		34.3	1.0
Seen cockroaches in home past year		14.3		5.7	0.23
Problems with mice or rats past year		37.1		45.7	0.63
Have working vacuum with HEPA filter		37.1		40.0	0.81
Cleaned for visit		88.6		74.3	0.12
People remove shoes when they visit the house		17.1		25.7	0.38
People wipe feet when they visit the house		80.0		68.6	0.27
Have door mats at every outside door		34.3		37.1	0.40

Characteristic/Activity	HEPA (<i>n</i> = 35)		Control (<i>n</i> = 35)		Difference <i>b</i> <i>p</i> -value
	Mean (SD)	Percent	Mean (SD)	Percent	
Child's sleeping area					
Relative humidity	51.4 (6.4) (<i>n</i> = 36) ^c		53.6 (9.3)		0.27
Room volume (m ³)	26.7 (7.8) (<i>n</i> = 36) ^c		27.4 (10.4)		0.85
Flooring type					
Carpeting		62.9		51.4	0.33
Wood/tile/vinyl/concrete		37.1		48.6	
# times vacuumed past 14 days	5.8 (5.0)		8.1 (5.4)		0.09
# times dusted past 14 days	4.1 (4.8)		3.7 (4.9)		0.61
Evidence of water/water damage in room/closet ^e		11.4		5.7	0.39
Evidence of mold/mildew in room/closet ^e		17.1		25.7	0.38
Open windows to ventilate					
Always/sometimes		77.1		77.1	1.0
Never/no windows that open		22.9		22.9	
Level of dust on surfaces ^e					
Heavy		14.3		0.0	0.06
Moderate		77.1		85.7	
None		8.6		14.3	

Abbreviation: SD, standard deviation.

^aOne family in HEPA group refused the HEC interview.

^bHEPA and control groups compared using chi-square tests for categorical variables and Wilcoxon rank-sum tests for non-normally distributed continuous variables.

^cAll HEPA families completed the baseline questionnaire and have baseline relative humidity and room volume measurements.

^dOne control family missing response to humidifier question at baseline (answered "No" at follow-up).

^eTechnician observed.

TABLE 2

Baseline and follow-up endotoxin loading and PM₁₀ and coarse PM (PM_{10-2.5}) concentrations in the child's sleeping area^a

	<i>n</i>	HEPA (<i>N</i> = 36)			<i>n</i>	Control (<i>N</i> = 35)		
		GM (GSD)	Min	Max		GM (GSD)	Min	Max
Endotoxin (EU/m ²) ^a								
Baseline	36	1565 (6.3)	35.1	44 779	35	2110 (4.9)	56.6	76 035
Follow-up	35 ^b	1259 (5.3)	29.8	66 615	33 ^c	1493 (4.8)	82.9	80 130
PM ₁₀ (µg/m ³)								
Baseline	36	22.5 (1.8)	9.2	94.5	35	19.8 (1.8)	7.4	63.9
Follow-up	30 ^d	10.2 (1.7)	4.1	28.5	32 ^e	17.0 (2.0)	4.9	72.1
PM ₁₀₋₂₅ (µg/m ³)								
Baseline	33 ^f	9.5 (2.9)	0.2 ^g	89.3	33 ^h	7.7 (2.0)	2.2	28.3
Follow-up	28 ⁱ	5.2 (1.8)	1.0	14.3	32 ^j	6.0 (2.6)	0.6	45.1

Abbreviations: *N*, sample size at baseline; GM, geometric mean; GSD, geometric standard deviation; EU, endotoxin units.

^aThe room the child primarily slept in was designated as the child's sleeping area. There were some instances when the child did not sleep in their bedroom or they did not have a room, so samplers were set up in the room where the child usually slept.

^bOne follow-up sample from a HEPA family excluded because ice packs leaked during transport.

^cTwo follow-up samples from control families were excluded one because the child tampered with it, the other because field technicians ran out of samplers.

^dSix follow-up samples from HEPA families were excluded, three because they had large negative masses, one because the MicroPEM timestamp data were missing so a sample duration could not be calculated, one because the MicroPEM was not deployed due to a missing cable that could not be replaced, and one because the family switched the sleeping and living area MicroPEMs on an unknown day.

^eThree follow-up samples from control families were excluded, one because the post sampling flow rate was >20% from the target rate, one because of uncertainty about the MicroPEM inlet size, and one because the family played with the MicroPEM.

^fOne baseline PM_{2.5} sample from a HEPA family was excluded because sampling duration was under 24 h, so PM_{10-2.5} could not be calculated, and two baseline PM_{10-2.5} observations from HEPA families were excluded because they had large negative values.

^gOne baseline PM_{10-2.5} observation from a HEPA family had a low negative value (-1.2 µg/m³) and was replaced with the PM_{10-2.5} limit of detection, 0.2 µg/m³.

^hTwo baseline observations from control families were missing a PM_{2.5} sample (one had sample duration <24 h; this other had PM₁₀ inlet mislabeled as PM_{2.5} inlet), so PM_{10-2.5} could not be calculated.

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i_j In addition to the missing PM_{10} observations, one follow-up observation from a HEPA family was missing a $PM_{2.5}$ sample (sample duration <24 h), so $PM_{10-2.5}$ could not be calculated, and one follow-up $PM_{10-2.5}$ observation from a HEPA family was excluded because it had a large negative value.

i_j In addition to the missing PM_{10} observations, one follow-up $PM_{2.5}$ sample from a control family was excluded, because it had a large negative mass, so $PM_{10-2.5}$ could not be calculated.