Final Report for TIE-Funded Project: Efficacy of in-home air filtration on reducing ultrafine particles of outdoor origin and impacts on cardiovascular disease risk factors in Puerto Rican adults

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INTRODUCTION

Ultrafine particles (UFP; <100 nanometers diameter) are ubiquitous in the urban environment. UFP originate from combustion sources, such as gasoline- and diesel-powered engines[1] (outdoors) and cooking and smoking (indoors), and have been shown to be toxic to humans.[2,3] Their extremely small size allows them to lodge deep within the lungs and cross biological barriers, which may increase their potential for causing lasting physiological harm.[4,5] Additionally, UFP may be more harmful than their larger counterparts due to their high surface area (per unit mass), allowing them to transfer more toxic chemicals and metals into humans.[2,6] Exposure to UFP may contribute to health effects caused by combustion-related air pollution, such as cardiovascular and respiratory diseases.[2,7]

To date, relatively little work has been done to quantify the extent UFP of outdoor origin are causing health effects in indoor environments due to the challenges inherent in accurately predicting exposure. Accurate exposure assessment is important because misclassification lowers the accuracy of correlations with health effects and could thereby bias results, usually toward null associations. While indoor sources make up a significant portion of the indoor exposure,[8] outdoor-sourced UFP exposure indoors is substantial and, in the absence of smoking, usually larger than indoor sources.[9] Being able to estimate the outdoor-sourced UFP contribution to indoor levels would provide an opportunity to separate its role in overall health from indoor sources. Studies have been able to reduce indoor exposure by using high-efficiency particulate arrestance (HEPA) filters,[10] but it remains unclear how well these filters perform in the real world in reducing outdoor-sourced UFP and what the impact on health may be.

OBJECTIVES

Our objectives were to (1) estimate outdoor-sourced UFP indoors and (2) provide evidence that in-home (real-world) HEPA filtration interventions are capable of reducing markers of cardiovascular disease (CVD) risk. We investigated these aims by conducting a secondary analysis on previously collected pollution and health data from the Boston Puerto Rican Health Study cohort in Boston and Chelsea.[11]

METHODOLOGY

Particle number concentration (PNC; a proxy for UFP) was measured both indoors and outdoors at homes within both Boston and Chelsea. Data collection occurred between May 2012 and December 2014. All monitoring locations used condensation particle counters (CPC; TSI model 3783) at an averaging interval of 30 seconds, except prior to May 2013 when PNC was collected at one-minute intervals. Twenty-four non-smoking homes throughout Boston and Chelsea were selected for six continuous weeks of indoor and outdoor monitoring, each. Participants were dispersed over all four seasons. For three of the six weeks a HEPA filter (MERV 17) was installed to lower indoor particulates, while a sham filter was used for the other three weeks. It was randomized whether the HEPA or sham filter was used first in each home. Blood samples were taken from participants at Week 0, Week 3, and Week 6 of the intervention, and analyzed for markers of CVD risk (i.e., hsCRP, IL-6, and TNFRII).

To remove indoor sources of PNC from the data set, we assumed spikes were from indoor activities (e.g., cooking, cleaning, burning candles). We calculated the 6-hour moving median for indoor measurements and considered spikes to be any indoor measurement that was more than two standard

deviations above this median. Spikes were replaced with the last indoor measurement that was not considered a spike. Statistical analyses were conducted in Stata 14 and R (version 3.2).

RESULTS

Mean PNC were generally higher than median values, due to the presence of very large spikes. Spikes in the data ranged from 0-9%, likely attributable to major indoor sources. HEPA filtration did result in lower measured PNC values than sham filtration (typically 50-85% reduction), but large spikes did not appear to be impacted significantly by either filtration method. Table 1 summarizes the data from all homes. Home 9 was removed from analysis due to equipment failure.

Table 1. PNC exposure and percent of indoor spikes calculated for each home.

			HEPA		*Sham*		*% Indoor Spikes*		
Participant ID	**Order**	**Exposure**	**Median PNC**	**Mean PNC**	**Median PNC**	**Mean PNC**	**Total**	**Sham**	**HEPA**
PR01	Hepa-1st	Indoor	4500	14000	7900	20000	15.86%	17.76%	13.88%
		Outdoor	12000	13000	9000	11000			
PR02	Sham-1st	Indoor	1300	17000	9100	34000	9.01%	9.98%	7.74%
		Outdoor	7900	9400	11000	13000			
PR03	Hepa-1st	Indoor	3700	11000	8400	31000	10.33%	12.19%	7.88%
		Outdoor	7800	9100	9600	12000			
PR04	Sham-1st	Indoor	2000	7200	5600	12000	5.69%	8.08%	2.84%
		Outdoor	7400	8600	9000	11000			
PR05	Hepa-1st	Indoor	1800	13000	7200	32000	11.36%	10.70%	12.28%
		Outdoor	10000	14000	12000	17000			
PR06	Sham-1st	Indoor	3700	38000	12000	68000	6.35%	8.72%	4.08%
		Outdoor	18000	20000	14000	17000			
PR07	Sham-1st	Indoor	7600	16000	15000	25000	8.27%	10.27%	6.81%
		Outdoor	8100	13000	15000	19000			
PR08	Sham-1st	Indoor	3600	9800	7900	17000	6.58%	5.81%	7.37%
		Outdoor	16000	19000	19000	22000			
PR10	Hepa-1st	Indoor	3400	11000	5800	16000	10.79%	11.41%	10.08%
		Outdoor	12000	15000	9400	13000			
PR11	Hepa-1st	Indoor	4500	17000	4300	10000	16.66%	17.01%	16.37%
		Outdoor	7500	9200	7100	9300			
PR12	Sham-1st	Indoor	1400	5900	3200	10000	15.05%	14.20%	16.06%
		Outdoor	9400	13000	7900	11000			
PR13	Hepa-1st	Indoor	1500	24000	6500	40000	11.56%	12.52%	10.09%
		Outdoor	11000	12000	9900	12000			
PR14	Sham-1st	Indoor	2600	11000	5400	19000	10.94%	10.21%	11.79%
		Outdoor	15000	17000	15000	18000			
PR15	Hepa-1st	Indoor	1700	2600	5500	7300	9.75%	8.81%	10.93%
		Outdoor	15000	16000	13000	15000			
PR16	Sham-1st	Indoor	2200	12000	12000	19000	8.17%	8.25%	8.08%
		Outdoor	17000	20000	16000	22000			
PR17	Sham-1st	Indoor	950	3200	3900	7100	8.29%	10.40%	6.22%
		Outdoor	9400	13000	11000	15000			
PR18	Hepa-1st	Indoor	980	11000	3500	13000	14.05%	13.14%	14.97%
		Outdoor	13000	18000	10000	12000			
PR19	Sham-1st	Indoor	2000	15000	4900	14000	7.80%	10.47%	5.16%
		Outdoor	9000	12000	11000	14000			
PR20	Sham-1st	Indoor	460	4000	3300	16000	8.62%	9.20%	8.04%
		Outdoor	6000	7600	11000	14000			
PR21	Hepa-1st	Indoor	1000	8900	4300	17000	11.93%	10.26%	13.43%
		Outdoor	8500	12000	8000	11000			
PR22	Hepa-1st	Indoor	3600	9000	5400	8700	8.67%	7.64%	9.64%
		Outdoor	14000	19000	11000	15000			
PR23	Sham-1st	Indoor	1900	7800	4700	16000	9.13%	9.81%	8.54%
		Outdoor	11000	14000	9600	14000			
PR24	Hepa-1st	Indoor	3500	9500	5600	16000	12.12%	11.16%	13.05%
		Outdoor	14000	18000	13000	16000			

Biomarkers measured in this study tended to be high at baseline, and across visits, which showed participant health overall to be somewhat poor. Changing the PNC exposure (total mean PNC vs. baseline mean PNC, with baseline meaning spikes have been removed) did change the associations of PNC with CVD risk biomarkers, but the change was opposite our expectations (Table 2). Associations became more negative, meaning higher PNC values result in lower CVD biomarker values.

Table 2. Association of PNC with biomarkers

		Beta*	P-value (CI)
hsCRP	Ln mean baseline PNC	-0.253	0.236 (-0.672; 0.166)
	Ln mean total PNC	-0.396	0.222 (-1.030; 0.239)
IL-6	Ln mean baseline PNC	-0.055	0.586 (-0.253; 0.143)
	Ln mean total PNC	-0.121	0.367 (-0.388; 0.147)
TNFRII	Ln mean baseline PNC	-0.049	0.210 (-0.125; 0.027)
	Ln mean total PNC	-0.068	0.180 (-0.168; 0.031)

* Beta is negative for lower blood biomarker values with higher PNC.

DISCUSSION

Although we were able to separate the total measured indoor PNC into outdoor-sourced PNC and indoor-sourced PNC, it did not change the CVD biomarker associations in the expected way. Restricting analysis to just the outdoor-sourced PNC resulted in stronger negative associations, although none of the associations were significant (p-value < 0.05). This trend was observed for all three biomarkers analyzed, which was quite surprising. While this may raise the question as to the toxicity of particulate matter, this seems unlikely as there are convincing arguments to the contrary.[12] It is possible there are other pollutants, which are inversely associated with PNC such as ozone, that are impacting the associations. Ozone is higher in summer and lower in winter. Unfortunately, this project did not look at ozone (nor were ozone or other pollutants measured at the homes) and we were unable to correct for this and other possible similar confounders.

The original design of the study (not the reanalysis) may be one of the reasons for the outcome of this reanalysis project. The location of CPCs may not have provided the best estimate of exposure for the participants. Participants may instead be spending a majority of their time outside of the home, where they could have received higher or lower exposures depending on their location. If participants had received much lower or higher doses of PNC than what we estimated, it could have had a major impact on the biomarker associations. Exposure misclassification is a major concern, and quite possible with this study design. Adjusting for time activity has been shown to impact associations between PNC and inflammatory and coagulation biomarkers.[13] Had we had time-activity data from the participants, we may have been able to adjust the exposures accordingly.

Additionally, there was no wash-out period between the two filtration interventions. It is possible that one or more of the biomarkers did in fact improve with HEPA filtration, but was a delayed effect and did not show up until the sham filtration period (when PNC exposure was generally higher). During the times when HEPA filtration was second, the effect would not have been measured at all. Adding a wash-out period between the two filtration interventions with blood draws at the end of each wash-out period would confirm this suspicion.

CONCLUSIONS

We were able to estimate outdoor-sourced PNC indoors by removing indoor spikes and show that HEPA filtration does substantially decrease outdoor-sourced PNC indoors, but more work is needed to understand the impacts of a real-world HEPA filtration intervention on CVD risk factors. Lessons learned from this reanalysis will benefit future work by informing better study designs to possibly be able to see how real-world interventions can improve cardiovascular health.

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