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The feasibility of an air purifier and secondhand smoke education intervention in homes of inner city pregnant women and infants living with a smoker

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Abstract

Objectives—Secondhand smoke (SHS) and other air pollutants adversely affect the health of pregnant women and infants. A feasibility study aimed at reducing air pollution in homes of pregnant women or infants living with a smoker was completed.

Methods—In collaboration with the Baltimore City Health Department, women ≥ 18 years of age and either pregnant nonsmokers, or post-partum (any smoking status) with an infant age 0–12 months were recruited. Homes had at least one smoker. Intervention included two air purifiers and secondhand smoke education. Outcomes included feasibility, change in fine particulate matter ($PM_{2.5}$), air nicotine, and salivary cotinine pre- and post-intervention.

Results—Fifty women were enrolled (mean age 27 years, 92% African American, 71% single, 94% Medicaid eligible, 34% reported smoking) and 86% completed the study. Of the 50 women, 32 had infants and 18 were pregnant at time of enrollment. Post- intervention, 70% of participants reported smokers were less likely to smoke indoors, and 77% had at least one air purifier turned on at the final visit. Participant satisfaction was high (91%) and 98% would recommend air purifiers.

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Conflict of interest

The authors declare that they have no conflict of interest.

Indoor $PM_{2.5}$ was significantly decreased ($P < 0.001$). Salivary cotinine was significantly decreased for non-smoking women ($P < 0.01$) but not infants, and no significant change in air nicotine occurred ($P = 0.6$).

Conclusions—Air purifiers with SHS education is a feasible intervention in homes of women and infants. These data demonstrate reduction in indoor $PM_{2.5}$ and salivary cotinine in non-smoking adults. Air purifiers are not an alternative for smoking cessation and a home/car smoking ban. Smoking cessation should be strongly encouraged for all pregnant women, and nonsmoking mothers with infants should be counseled to completely avoid SHS exposure. This study provides support for a future intervention evaluating clinical endpoints.

Keywords

Fine particulate matter; Air nicotine; Salivary cotinine; Indoor air; Secondhand smoke; Air purifier

1. Introduction

Secondhand tobacco smoke (SHS) and other air pollutants adversely affect the health of pregnant women and infants. According to the Surgeon General, SHS exposure is linked to low birth weight, sudden infant death syndrome, and early childhood respiratory diseases (U.S. Department of Health and Human Services, 2006). Indoor air can be contaminated by various compounds including gases (carbon monoxide, radon and volatile organic chemicals), gas/vapors and particulates from environmental tobacco smoke (ETS), biological contaminants (mold and bacteria), and particulate matter. Particulate matter is suspended in air and originates from indoor (dust, mold, bacteria, tobacco smoke, gas cooking, wood burning fireplaces, cleaning activities) as well as outdoor sources (pollen, combustion from motor vehicles and power plants) (Diette et al., 2008). Particle size determines the location of deposition in the respiratory tract. While particles < 10 μm in diameter (PM_{10}) can be inhaled, fine particles < 2.5 μm ($PM_{2.5}$) reach the alveoli leading to health problems (Diette et al., 2008). ETS contains more than 4000 chemicals, many of which are known or suspected toxic or carcinogenic agents. ETS is a major source of indoor air pollution and 40% of U.S. children are exposed at home where they spend a majority of their time (Centers for Disease Control and Prevention, 2016).

Over the past decade, The Johns Hopkins Center for Childhood Asthma in the Urban Environment has documented that indoor air pollution in inner city Baltimore homes is significantly higher than simultaneously measured ambient and suburban home concentrations, and levels often exceed the annual limits for ambient pollution exposure set by the U.S. Environmental Protection Agency (EPA) (McCormack et al., 2008; Simons et al., 2007).

Education and counseling interventions to reduce SHS exposure have demonstrated varying success (Baxi et al., 2014), and trials utilizing indoor air purifiers have demonstrated their feasibility and sustained effectiveness in reduction of indoor air pollution in homes of children with asthma (Batterman et al., 2012; Du et al., 2011; Eggleston et al., 2005; Lanphear et al., 2011; Butz et al., 2011). Studies evaluating the health effects of air purifiers have shown that they may be beneficial in children with asthma (Eggleston et al., 2005;

Lanphear et al., 2011; Butz et al., 2011) but the feasibility of these interventions in the homes of pregnant women and infants without chronic respiratory illnesses is unknown. Establishing feasibility in this population is important prior to conducting a trial since an intervention aimed at prevention requires individuals to be motivated in the absence of illness (Institute of Medicine, 2001). Additionally, there are known barriers to recruitment and retention in our target population of low income minority women (El-Khorazaty et al., 2007).

A pilot study was conducted to evaluate the feasibility of deploying air purifiers in the homes of pregnant women or infants who live with a smoker. Feasibility was assessed via willingness of women to enroll, participant retention, observed use of the air purifiers, report of smoking behaviors, and satisfaction with the intervention. Secondary objectives included measurement of the change in $PM_{2.5}$, air nicotine, and salivary cotinine from baseline to the fourth week of continuous air purifier use. We hypothesized that the intervention would prove feasible in the identified population, and that four weeks of air purifier use combined with SHS education would lead to significant reduction in indoor air pollution ($PM_{2.5}$, air nicotine) as well as salivary cotinine in pregnant women and infants.

2. Methods

2.1. Study design (Fig. 1)

A single arm, unmasked clinical trial was conducted to evaluate the feasibility and effectiveness of a combined air purifier and SHS education intervention. Participants had four home visits scheduled during the five-week study period. Environmental monitoring occurred at baseline and during the fourth week of air purifier use. Two air purifiers were placed in each home (adult/infant bedroom and the living area) after baseline air monitoring was completed and participants were encouraged to keep air purifiers on during the remainder of the study. Saliva samples were obtained from adult and infant participants during the second and final home visit (pre- and post- intervention) for cotinine analysis as a biomarker of SHS exposure. The Johns Hopkins Medical Institutional Review Board approved the study, and all participants provided written informed consent before beginning the study (Fig. 1).

2.2. Study participants

Women were referred from one of the Baltimore City programs providing services for women and children who are low income and/or at risk for poor birth outcomes or they were directly recruited from local Women, Infants, and Children offices (WIC). Participants were eligible if they met the following criteria: (1) Female ≥ 18 years of age and pregnant at time of enrollment (by report) and a non-smoker (by report) *or* mother/infant dyad: mother ≥ 18 years and infant 0–12 months (mother could be a smoker or nonsmoker by report); (2) Participant in a Baltimore City program; (3) Reported smoker in the home (either mother participant or another household member). Participants were excluded based on the following: (1) Pregnant woman reported being a smoker herself; (2) Post-partum or pregnant woman unwilling or unable to participate; (3) Non-English speaking; (4) Planned to move

out of the Baltimore area in the next 6 months; (5) Transitional housing; (6) No electricity in the home.

2.3. Intervention

Each participant received two air purifiers that provided 1500 square feet of coverage each (HealthMate™ Standard). Air purifiers were donated by Austin Air® (Buffalo, NY). The HEPA air purifier works by removing large particles (dust, hair, and dander) that are suspended in the air, then medium particles such as mold and pollen, are filtered. A carbon filter can remove chemicals, gases, and odors. Finally, the HEPA filter removes 99.97% of particles larger than 0.3 μm. Participants kept the air purifiers at the end of the study. SHS education included discussion and supportive pamphlets by the Maryland Department of Health and Mental Hygiene, EPA, and American Academy of Pediatrics about the adverse health effects of SHS exposure, tips on smoking cessation and how to support smoking cessation, and information about the locally available free quit line services.

2.4. Outcomes

2.4.1. Feasibility—Feasibility was assessed via willingness of women to enroll, participant retention, observed use of the air purifier (study staff documented if the air purifiers were present in the home and turned on at home visits 3 and 4), participant report of smoking behaviors in the home during the study (smoking inside versus outside), and satisfaction with the air purifiers.

2.4.2. Air quality assessment—Air sampling was conducted continuously for approximately one week at baseline and during the fourth week of air purifier use. Monitoring occurred in the living or sleeping area because these are indoor locations where participants would spend the most time. $PM_{2.5}$ samples were collected at a flow rate of 4 L/min using Personal Environmental Monitor impactors (MSP corp, St. Paul, MN) loaded with a 37-mm, 2.0 μm pore-size, polytetrafluoroethylene membrane (Teflo, Pall Laboratory). Sampler flow rates were calibrated at the beginning and end of each sampling period using primary standards (DryCal Bios International Corporation, Butler, NJ). PM gravimetric analysis was conducted on a Mettler T5 microbalance (Mettler Toledo, Inc., Columbus, OH) after filters were equilibrated for 24 h at constant temperature and humidity. For our analysis, post flow rates that could not be calculated or were out of the expected range of 2.8–6.1 L/min were further scrutinized. Indoor temperature and humidity was measured concurrently using a HOBO temperature and humidity data logger (Onset Corporation, Pocasset, MA). We obtained Baltimore City EPA monitoring site data for ambient $PM_{2.5}$ during each week of indoor monitoring. Air nicotine was measured with passive samplers according to standard methods (Hammond et al., 1995). The sampler relies on passive diffusion of nicotine into a filter where it is trapped. The filter was analyzed using gas chromatography with mass spectroscopy with a nitrogen phosphorous detector (Shimadzu, USA). The limit of detection (LOD) for air nicotine was 0.021 μg of nicotine, divided by the sampled volume of air to obtain airborne nicotine concentration in $\mu g/m^3$. Air nicotine concentration greater than the LOD was defined as positive for indoor SHS exposure. For quality control, 10% duplicates and 10% blanks of both $PM_{2.5}$ and air nicotine were collected.

2.4.3. Salivary cotinine—Cotinine is a standard biomarker of environmental tobacco smoke exposure as it is the major metabolite of nicotine with a half-life of 15–19 h, and levels measured in blood, saliva, and urine are highly correlated (Florescu et al., 2009). Salivary cotinine collection and analysis by enzyme-linked immunosorbent assay (ELISA) was carried out per a standard protocol developed by Arizona State University Institute for Interdisciplinary Salivary Bioscience Research and Salimetrics®.

2.5. Statistical analysis

Summary statistics were expressed as means or proportions, as appropriate. Baseline characteristics between participants who completed the full study and those who dropped out were compared using a Fisher's exact test for categorical variables and a Mann-Whitney test for continuous variables, as appropriate. Non-parametric tests were used to analyze $PM_{2.5,}$ air nicotine, and salivary cotinine data since results were not normally distributed at baseline or at follow up. Baseline and post intervention levels of air nicotine, $PM_{2.5}$, and salivary cotinine were compared using a Wilcoxon sign rank test. A Mann-Whitney or Kruskal-Wallis test was used to evaluate for associations between home characteristics and cooking/cleaning activities with baseline $PM_{2.5}$. All analyses were performed with Stata statistical software (version 13.0; StataCorp, College Station, TX).

3. Results

3.1. Baseline characteristics (Tables 1 and 2)

Fifty women were enrolled between April 2014 and March 2016 (Fig. 2). The average age of our adult participants was 27 years (± 5.8 years). Most women were African American (n = 45, 92%), single (n = 35, 70%), and high school graduates (n = 33, 67%). For those reporting an annual income, the majority earned < $10,000 per year and 92% (n = 46) received medical assistance. Most women lived in an attached (e.g. row house), rented home. There were no differences in demographic factors for participants who dropped out compared to those who completed the study. Participants reported spending most of their time (77%) indoors at home. Approximately one third of the participants were pregnant at the time of enrollment. Of the 32 infants, 53% (n = 17) had mothers who were smokers. Infant ages ranged from newborn to 12.8 months and 53% (n = 17) were female. As far as adverse birth outcomes for our infant participants, 19% (n = 6) were born prematurely at less than 37 weeks gestation. One infant was low birth weight at term (< 2500 g). At baseline, most participants reported that there were 1–3 smokers living in the home and one reported that there were eight smokers in the home. Only three participants reported having a smoking ban. The remainder reported that smoking was either permitted anywhere or in some places at sometimes. Smoking occurred throughout the home (kitchen/dining area, bedroom/bathroom, living room, or basement). Most participants reported that the smoker stepped out of the room or went outside if the children were home while they were smoking (Tables 1 and 2).

3.2. Feasibility

Over 2 years, 107 women were screened and 50 were enrolled (Fig. 2). Our retention rate was 86% (43 of 50 women completed the study). Those who did not complete the study

were either: no longer interested (n = 4), moved (n = 1), or could no longer be contacted (n = 2). At follow up, most women (n = 40, 93%) reported using at least one air purifier part of the day, every day. If participants did not run their air purifiers all day every day, they reported that they turned them off: while they were home, if it was too cold, overnight, when they left the home, while the TV was on, and when they needed to use the outlet for something else. Study staff documentation found that 88% (n = 37) of participants had at least one purifier turned on at home visit 3 and 60% (n = 25) had both on. At the end of the study, 40 of the remaining 43 participants had both air purifiers in their home. At home visit 4, 77% (n = 33) had at least one turned on and 58% (n = 25) had both on. During the study, most (70%, n = 30) participants reported that smokers were less likely to smoke indoors, 23% (n = 10) reported there was no difference in their behavior, and 7% (n = 3) reported that the smoker was more likely to smoke inside. Participants reported that none of the smokers quit during the study. Of the 43 participants who completed the study, 91% (n = 39) reported satisfaction with the air purifiers and 98% (n = 42) would recommend their use to family and friends who are exposed to SHS in the home (Table 3).

3.3. $PM_{2.5}$ (Fig. 3, Table 3)

Baseline data was available for 47 homes (two participants dropped out before visit 2 and one monitor was unplugged). There were 40 samples available for follow up analysis (7 participants dropped out before visit 4, 1 monitor failed, and 2 monitors were turned off). Baseline median $PM_{2.5}$ was 31 μg/m3 (IQR 17–63 μg/m 3) and there was no statistically significant difference observed between homes of participants who completed the study versus those who dropped out. After four weeks of air purifier use, the median $PM_{2.5}$ was significantly reduced by 45% to 17 $\mu g/m^3$ (IQR 10–35 $\mu g/m^3$) $P < 0.001$. In a sensitivity analysis using only the $PM_{2.5}$ samples from homes where the air purifiers were observed to be turned on at both home visits 3 and 4 (20 samples) the median decreased by 52% to 14.9 $\mu g/m^3$ (IQR 9.8–25.5 $\mu g/m^3$). No significant associations were identified between baseline $PM_{2.5}$ and various home characteristics (fuel type, stove type, infestations with mice or roaches) or cleaning activities (dusting, sweeping, wet mopping, vacuuming). Report of burning food during the week of baseline monitoring, but not stove, oven, or toaster use was significantly associated with increased baseline $PM_{2.5}$ ($P = 0.001$). Report of burning food was not associated with $PM_{2.5}$ at follow up. Baseline ambient median $PM_{2.5}$ was 9.3 μg/m 3 (IQR 8,12 μg/m 3). There was no significant change in ambient $PM_{2.5}$ post intervention (median 8.5 $\mu g/m^3$ (IQR 6,11 $\mu g/m^3$; $p = 0.08$)). There was a significant difference ($P < 0.001$) between indoor and ambient $PM_{2.5}$ both at baseline and post intervention (Fig. 3).

3.4. Air nicotine (Table 3)

Baseline air nicotine was available for 49 homes and median concentration was 0.15 $\mu g/m^{3,}$ (IQR 0.02–0.51 $\mu g/m^3$). No significant difference was noted in air nicotine at follow up (median 0.32 $\mu g/m^3$, IQR 0.02–0.86 $\mu g/m^3$), $P = 0.6$. At baseline and follow up, 84% (n = 41/49 and n = 36/43 respectively) of homes had an air nicotine level above the level of detection (LOD).

3.5. Salivary cotinine (Table 3)

Baseline median salivary cotinine was 215 ng/mL (IQR 173–597 ng/mL) for reported smokers and 2.1 ng/mL (IQR 0.92–4.3 ng/mL) for reported nonsmokers. There was a significant decrease in salivary cotinine at follow up in nonsmoking women (median post = 1.3 ng/mL, IQR 0.71–2.2 ng/mL), $P < 0.01$. Baseline median infant cotinine was 8 ng/mL. Infants with mothers who reported smoking had higher baseline salivary cotinine than those with nonsmoking mothers (12.9 ng/mL and 3.3 ng/mL respectively). There was no significant change in salivary cotinine for infants post- intervention ($P = 0.4$).

4. Discussion

In this pilot study, we demonstrated the feasibility of an air purifier and SHS education intervention in homes of urban pregnant women and infants who reside with a smoker. We successfully recruited our target population of women who were low income, dependent on public assistance, and at risk for poor birth outcomes. The participants in this study had a higher rate of preterm birth (19%) than the rate in Baltimore City (12.5%) and almost double the National average (9.6%) (March of Dimes Foundation, 2016a; March of Dimes Foundation, 2016, 2016b). Of the 91 eligible women who were screened, 55% agreed to participate and 86% completed the study. This is similar to the rates of recruitment (56–67%), but slightly lower than the retention rates (91–97%) reported in other air purifier intervention studies (Eggleston et al., 2005; Lanphear et al., 2011; Butz et al., 2011). These trials included children with asthma and so parents may have been motivated to improve their child's health. While we did not recruit based on the presence of a chronic illness, women may have been interested in decreasing their infant's exposure to SHS but found it difficult, in particular if the smokers were older family members who owned the home (Hoehn et al., 2016). Multiple barriers to participation and retention exist in research studies among minority populations, including mistrust of medical research, inconvenient study protocols, transportation issues, and lack of access to information about available studies (El-Khorazaty et al., 2007; Nicholson et al., 2011). Intervention studies recruiting from similar populations of inner city women have noted retention rates of 59–89% and emphasized the importance of face-to-face recruiting, clinical staff buy-in, convenient scheduling, frequent contact, and incentives for participants to improve recruitment and retention (El-Khorazaty et al., 2007; Nicholson et al., 2011; Hovell et al., 2000; Collins et al., 2011). We can attribute our success to having the support of several Baltimore City programs including home visiting staff who referred clients as well as local WIC office staff. We found that in-person recruitment at WIC was the most efficient method since these offices were often busy during the day and women could be introduced to the study and screened for eligibility before or after their appointment. Monetary incentives were not provided, but participants kept their air purifiers at the end of the study. The study design allowed for frequent participant contact, and one study coordinator completed all home visits which provided continuity. To decrease participant burden, all study visits were brief (one hour or less) and were completed in the home at a time that was convenient. During the one month intervention, we found that participants were overall adherent with using their air purifiers (77% were observed to be using at least one air purifier at the end of the study). This is similar to the adherence (48–68%) reported by others with a similar intervention but

over longer study periods of 6–12 months (Eggleston et al., 2005; Lanphear et al., 2011; Butz et al., 2011). During the study design phase, there was a concern that having air purifiers in the home may encourage smoking indoors, but most of our participants (93%) reported that the smoker was less likely to smoke inside or that there was no change in smoking behaviors, although a few (7%) did report that the smoker was more likely to smoke inside. Participants who completed the study reported high satisfaction, although we were not able to capture the opinions of the women who dropped out.

Indoor $PM_{2.5}$ in our participant homes (baseline mean = 43 and median = 31 μg/m ^{3}) was similar to other studies in Baltimore City (Eggleston et al., 2005; Butz et al., 2011; McCormack et al., 2009) as well as other U.S. cities that measured $PM_{2.5}$ in homes with a smoker (Du et al., 2011; Hunt et al., 2011; Wallace et al., 2003). Smoking indoors has been associated with $PM_{2.5}$ measurements that are 14–34 μg/m ^{3} higher than in nonsmoking homes (Du et al., 2011; Hunt et al., 2011; Wallace et al., 2003; Breysse et al., 2005), therefore providing a greater potential for exposure reduction among individuals living in smoking homes. The EPA does not have indoor air quality standards, but 45% of our homes had baseline $PM_{2.5}$ concentrations that were higher than the recommended ambient annual mean of 35 μg/m^3 and 60% were higher than 25 μg/m ^{3}, which is the annual indoor standard set by the World Health Organization (WHO) (World Health Organization, 2005). Our participant homes had significantly higher indoor $PM_{2.5}$ when compared to simultaneously measured ambient concentrations. These findings are concerning since prenatal and postnatal $PM_{2.5}$ exposure has been associated with adverse health effects including reduced fetal growth (Jedrychowski et al., 2004, 2013), respiratory related infant mortality (Woodruff et al., 2006), wheezing in infancy (Hunt et al., 2011), and increased asthma symptoms and rescue medication use (McCormack et al., 2009). After the four-week intervention, there was a statistically significant decrease (−14 μg/m ^{3}, 45%) in median $PM_{2.5}$. There was no significant change in ambient $PM_{2.5}$ between baseline and post intervention and since there was no change in air nicotine (suggesting indoor smoking was not decreased), and no smokers reported quitting, we can likely attribute the significant decrease in indoor concentrations to the air purifier intervention. Two previous trials in Baltimore noted mean differences of −20 μg/m^3 (47%) and −14 μg/m ^{3} (37%) for $PM_{2.5}$ at 6 and 12 month follow up respectively (Eggleston et al., 2005; Butz et al., 2011). Similar to other studies using air purifiers, a difference in air nicotine was not detected (Lanphear et al., 2011; Butz et al., 2011). According to a report by the EPA, even air purifiers with a carbon filter generally do not remove all gaseous pollutants and therefore many of the carcinogenic gas phase pollutants from tobacco smoke are left behind (Environmental Protection Agency, 2009). Third hand smoke (THS) is an additional source of involuntary exposure for nonsmokers in the home. THS is the residual of tobacco smoke pollutants that remain on surfaces and in dust for months after a cigarette is smoked and can therefore be inhaled, ingested, or dermally absorbed (Matt et al., 2011). Portable air purifiers are also not as effective at removing larger particles once they have settled on the ground or surfaces in the home (Environmental Protection Agency, 2009). Authors who have shown improvement in asthma symptoms (Eggleston et al., 2005; Butz et al., 2011) or decreased unscheduled asthma visits (Lanphear et al., 2011) associated with decreased levels of $PM_{2.5}$ despite no change in air nicotine or salivary cotinine values, have suggested that it is the PM that plays a major role

in triggering respiratory symptoms (Lanphear et al., 2011; Butz et al., 2011). Future studies will need to address this deficiency as exposure to any compounds in SHS poses a significant environmental health risk for pregnant women, infants, and children.

In our study, 60% of infants had baseline salivary cotinine concentrations above the level used to identify adult smokers (> 3 ng/mL) (Benowitz et al., 2009). Other authors have similarly noted cotinine levels in children exposed to SHS that are as high as adult smokers (Blaakman et al., 2015; Chang et al., 2000). Some authors have documented that the half-life of cotinine is longer in infants than in adults (Florescu et al., 2009) but the literature is not consistent and Dempsey and colleagues observed that infants and children ages 2–84 months had a salivary cotinine half-life that was similar to adults (Dempsey et al., 2013). Potential explanations for elevated cotinine concentrations in infants and young children include increased exposure due to higher minute ventilation, close contact with an active adult smoker (Dempsey et al., 2013), or THS exposure while playing on the floor where they can ingest dust containing nicotine (Matt et al., 2011). Interestingly, there was a significant decrease in salivary cotinine for nonsmoking adults but not for infants. There was actually a significant increase in salivary cotinine for infants of nonsmoking mothers. Perhaps after SHS education, adults were more likely to avoid exposure to SHS inside and outside of the home whereas infants were not able to avoid being around the smoker in the home. Since we were not able to account for SHS exposure outside the home, this may have influenced salivary cotinine levels.

Our study had some limitations. The study was short, so we were not able to show whether the improvement in indoor air quality was sustainable over a longer period. Other studies using indoor air purifiers have demonstrated sustained effects in air quality improvement over 6–12 months (Eggleston et al., 2005; Lanphear et al., 2011; Butz et al., 2011). Air purifier use was measured by self-report and confirmed during two home visits by study staff. In future studies, we would consider using a more objective measurement of longitudinal compliance with the intervention such as a current sensor to monitor air purifier use. Since smoking behaviors may change as a result of air quality monitor placement and completing questionnaires about SHS exposure, salivary cotinine should be measured during the initial home visit prior to any other study procedures. Finally, education occurred with the adult participant who may not have been the smoker in the household. Education was focused on the dangers of SHS exposure, how to reduce SHS exposure, and encouraging smoking cessation. Future studies should consider a multimodal intervention that includes counseling the smoker to quit and enforces a complete home smoking ban. Future studies should also include measures to assess change in smoking behavior in relation to an intervention.

5. Conclusions

In conclusion, our study demonstrated that an indoor air pollution reduction intervention including two air purifiers and SHS education is highly feasible, and to our knowledge, is the first to implement such an intervention in homes of women who are pregnant or have infants and live with a smoker. Our findings confirm that indoor air pollution in inner city Baltimore homes is elevated compared to the standards set by the EPA and WHO and

demonstrates that air purifiers in combination with SHS education are effective in reducing indoor fine PM and a biomarker of SHS exposure in non-smoking adults. Although the air purifiers effectively reduced indoor $PM_{2.5}$ they did not eliminate SHS exposure (air nicotine and salivary cotinine for infants were unchanged), therefore it remains imperative to enforce a home and car smoking ban as well as encourage smoking cessation for all adult smokers in the home. Complete elimination of SHS exposure for infants, pregnant women, and nonsmokers is preferred over any mode of exposure reduction. In addition to source elimination, the EPA recommends ventilating the home with clean outdoor air in order to improve indoor air quality. If these measures are not adequate, an air purifier can be considered (Environmental Protection Agency, 2009). These findings provide support for future multimodal intervention trials examining clinical outcomes, as indoor air pollution and SHS exposure are important modifiable risk factors for adverse birth outcomes and pediatric respiratory disease.

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Abbreviations

SHS	secondhand smoke
$PM_{2.5}$	fine particulate matter
EPA	Environmental Protection Agency
BCHD	Baltimore City Health Department
WIC	Women, Infants, and Children
LOD	limit of detection
WHO	World Health Organization

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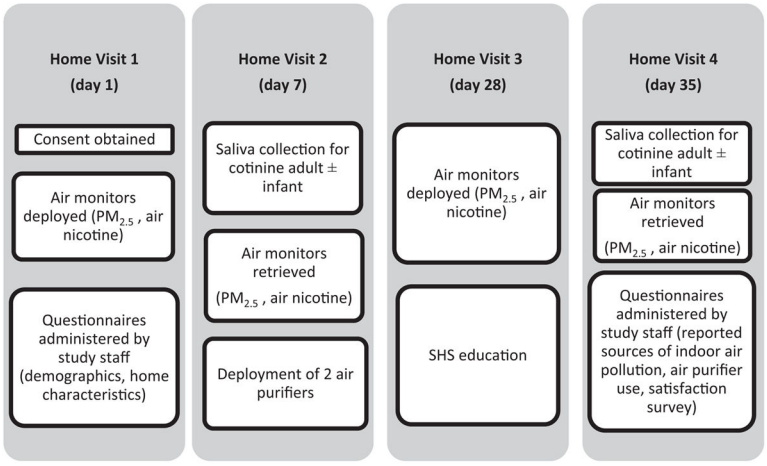

Fig. 1.
Study design.

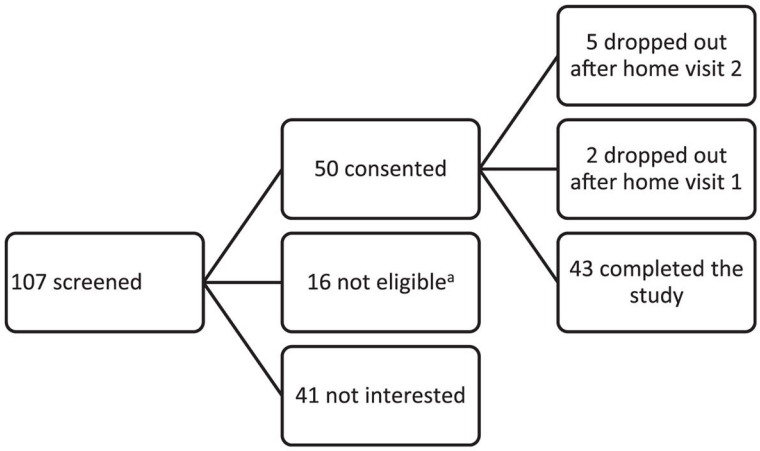

Fig. 2. Study flow diagram

[a]age of infant (n = 3), no smoker in home (n = 5), mom < 18 years (n = 3), pregnant smoker (n = 4), not pregnant or no infant (n = 1).

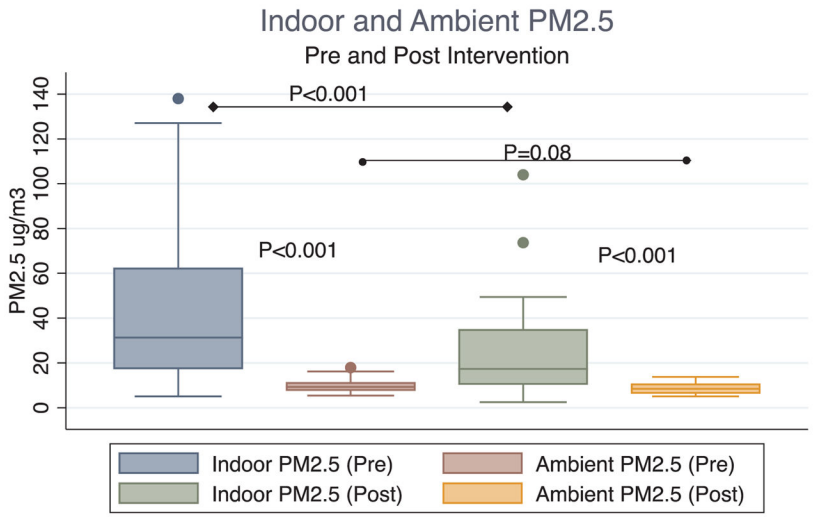

Fig. 3.

Boxplot of indoor and ambient PM2.5, pre and post intervention.

Table 1

Adult Baseline Characteristics (N = 50).

		Pregnant Women (N = 18)	Mothers (N = 32)	Total (N = 50)
Age, mean (range), in years		25.7 (19–36)	27.3 (18–41)	26.7 (18–41)
Smoker, n (%)	Self	0 (0)	17 (53)	17 (34)
	Significant other	6 (33)	11 (34)	17 (34)
	Other adult	13 (72)	14 (44)	27 (54)
Race, n (%)	African American	16 (89)	29 (94)	45 (92)
	White	1 (6)	2 (6)	3 (6)
	Mixed	1 (6)	0 (0)	1 (2)
Income, n (%)	< $10,000	6 (33)	12 (38)	18 (36)
	$10,000–15,000	2 (11)	5 (16)	7 (14)
	$15,000–35,000	1 (6)	6 (18)	7 (14)
	$35,000–50,000	1 (6)	1 (3)	2 (4)
	Unsure/prefer not to answer	8 (44)	8 (25)	16 (32)
Medicaid, n (%)		17 (94)	29 (91)	46 (92)
Marital Status, n (%)	Single	14 (78)	21 (66)	35 (70)
	Married	2 (11)	5 (16)	7 (14)
	Unmarried couple	2 (11)	5 (16)	7 (14)
Maternal Education, n (%)	Grade 9–11	6 (33)	10 (31)	16 (32)
	HS graduate	7 (39)	12 (38)	19 (38)
	Some college	5 (28)	8 (25)	13 (26)
	College graduate	0 (0)	1 (3)	1 (2)
Asthma diagnosis, n (%)		7 (39)	12 (39)	19 (39)
Baseline Salivary cotinine (ng/mL), median (IQR)	Reported smokers	N/A	215 (173,597)	215 (173,597)
	Reported nonsmokers	3 (0.92,4.3)	1.8 (1.1,4.1)	2.1 (0.92,4.3)

Table 2

Infant Baseline Characteristics (N = 32).

Age range	8 days – 12.8 months
Female, n (%)	17 (53)
Preterm birth (< 37 weeks), n (%)	6 (19)
Low birth weight (< 5 lb 8 oz) at term	1 (3)
Ever wheeze, n (%)	8 (25)
Baseline salivary cotinine > 3 ng/mL, n (%)[a]	18 (60)

[a]Optimal serum cotinine cut-point = 3 ng/mL to discriminate smoking from non-smoking adults in the US (sensitivity = 96.3%, specificity = 97.4%) (Benowitz et al., 2009), which is equivalent to 4 ng/mL for salivary cotinine using Salimetrics EIA ®.

Table 3

Secondary outcomes.

	Pre-intervention	Post-intervention	P-value[a]
Environmental Assessments (median, IQR)			
$PM_{2.5}$ (μg/m3)	31 (17,63)	17 (10,35)	**P < 0.001**
Air nicotine (μg/m^3)	0.15 (0.02,0.51)	0.32 (0.02,0.86)	P=0.6
Biomarker of Personal Exposure- salivary cotinine (ng/mL) (median, IQR)			
Adult nonsmoker	2 (0.92,4.3)	1.3 (0.71,2.2)	**P < 0.01**
Adult smoker	215 (173, 597)	290 (178, 649)	P= 0.6
Infants	8 (2.7, 17.9)	7.8 (4.9, 18.7)	P = 0.4
Infant, mom nonsmoker	3.3 (2,8.7)	5 (2.8,12.7)	**P=0.046**
Infant, mom smoker	12.9 (5.8,20.9)	9 (5,21.4)	P=0.9

[a]Wilcoxon signed- rank test.