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Characterizing peak exposure of secondhand smoke using a real-time PM_{2.5} monitor

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Abstract

Although short-duration elevated exposures (peak exposures) to pollutants may trigger adverse acute effects, epidemiological studies to understand their influence on different health effects are hampered by lack of methods for objectively identifying peaks. Secondhand smoke from cigarettes (SHS) in the residential environment can lead to peak exposures. The aim of this study was to explore whether peaks in continuous PM_{2.5} data can indicate SHS exposure. A total of 41 children (21 with and 20 without SHS exposure based on self-report) from 28 families in New York City (NY, USA) were recruited. Both personal and residential continuous PM_{2.5} monitoring were performed for five consecutive days using MicroPEM sensors (RTI International, USA). A threshold detection method based on cumulative distribution function was developed to identify peaks. When children were home, the mean accumulated peak area (APA) for peak exposures was 297 ± 325 hour* $\mu\text{g}/\text{m}^3$ for children from smoking families, six times that of the APA for children from non-smoking families ($\sim 50 \pm 54$ hour* $\mu\text{g}/\text{m}^3$). Average PM_{2.5} mass concentrations for SHS exposed and unexposed children were 24 ± 15 $\mu\text{g}/\text{m}^3$ and 15 ± 9 $\mu\text{g}/\text{m}^3$, respectively. The average SHS exposure duration represents $\sim 5\%$ of total exposure time, but $\sim 13\%$ of children's total PM_{2.5}

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SUPPORTING INFORMATION

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exposure dose, equivalent to an additional 2.6 $\mu\text{g}/\text{m}^3$ per day. This study demonstrated the feasibility of peak analysis for quantifying SHS exposure. The developed method can be adopted more widely to support epidemiology studies on impacts of short-term exposures.

Keywords

peak analysis; peak exposure; real-time $\text{PM}_{2.5}$; residential environment; secondhand smoke; threshold detection

1 | INTRODUCTION

Secondhand cigarette smoke (SHS) has been a prevalent indoor $\text{PM}_{2.5}$ source that can result in short-term high-level $\text{PM}_{2.5}$ exposure.^{1,2} Though the introduction of smoking regulations in many countries has reduced SHS exposure in certain public locations (e.g., restaurants and schools), SHS is still a major source of exposure in residential environments.^{3–5} Residential SHS exposure is of concern especially for children because they spend a large proportion of time at home.^{6–8} Moreover, childhood is a critical time for lung development and maturation of the immune system.^{9,10} As of 2013, worldwide there were still 4.5% (in Panama) to 79% (in Indonesia) of children under 15 years old exposed to residential tobacco smoke.¹

Lines of evidence have demonstrated that SHS exposure can lead to various adverse health effects.^{5,11} A single SHS exposure event increases the risk for platelet aggregation, decreased levels of serum ascorbic acid, and causes narrowing of blood vessels.^{12–14} Chronic SHS exposure can initiate irreversible damage on cardiovascular and respiratory systems.^{15,16}

Accurately characterizing pollutant exposure is the key factor in understanding the exposure-response relationship in epidemiology studies.^{17,18} For SHS, several exposure assessment methods have been used, including questionnaires, indoor or personal air sampling for nicotine levels, and cotinine levels in biological samples.^{19,20} Each method has strengths and limitations.²¹ Questionnaires have been used for classifying smoking or non-smoking homes but are limited in their capacity for quantifying different levels of exposures due to people having different sensitivities to tobacco smoke²²; nicotine exposure using integrated air samplers is useful for average SHS exposure assessment, but cannot provide exposure details such as the exposure time and frequency.²³ Salivary and urinary cotinine levels are affected by the wide range in the rates of metabolism of nicotine²⁴; cotinine which is a good marker of active smoking vs. non-smoking is plagued in SHS studies by overlapping distributions between SHS exposed and non-exposed individuals, resulting in very different thresholds being used in different studies to assess exposure.^{25–27}

Recently developed miniaturized, “real-time” $\text{PM}_{2.5}$ samplers offer a chance to characterize continuous SHS exposure. On average, the time it takes to smoke a cigarette is 5–7 minutes.²⁸ $\text{PM}_{2.5}$ levels can increase substantially with tobacco combustion in a confined space or within a close distance to the smoker.^{29,30} The effect of cigarette smoke on $\text{PM}_{2.5}$ concentration typically lasts for less than half an hour.^{31,32}

Continuous air sampling data allow us to evaluate the time, location, and level of peak exposures. The objective of this study is to develop an approach to measuring peak exposures to PM_{2.5}, investigate the feasibility of this approach for characterizing SHS exposure, and comparing the difference in SHS exposure between smoking and non-smoking homes. We hypothesize that the difference in PM_{2.5} peak exposures between SHS exposed and unexposed children is more distinct than that in daily or weekly average PM_{2.5} mass concentration.

2 | METHODS AND MATERIALS

2.1 | Study participants and PM sampling

The Environmental Monitoring and Biological Airway Response Study (EMBARs) is a cohort study aiming to field test and validate PM_{2.5} sensors for multiple parameters, including UVPM (ultraviolet active PM), PM_{2.5} peak exposure, and their utility as indicators of personal SHS exposure. In 2013, 21 children from 14 smoking families and 20 from 13 non-smoking families were recruited from Washington Heights in New York City (NYC, New York, USA).

Personal and residential PM_{2.5} monitoring was conducted for five consecutive days for each subject using the MicroPEM miniaturized real-time portable particulate sensor (v3.2, RTI, USA). The MicroPEM monitors real-time PM data via a nephelometer while also collecting an integrative Teflon filter sample, allowing the nephelometer data from each deployment to be gravimetrically calibrated. The gravimetrically weighed filters can also be analyzed for additional elemental and organic pollutants.³³ Physical activity information is also recorded from a built in 3-axis accelerometer, allowing wearing compliance or physiological parameters to be evaluated.³⁴ In addition to the personal monitoring, a second MicroPEM monitor was placed in the living room of the participant's home. To obtain the 5-day run time, the personal monitor was run in a duty cycle mode of 30 seconds on and 30 seconds off and had an additional external battery pack of 3 alkaline AA cells, while the residential monitor had an additional external battery pack of 3 alkaline D cells, which allowed for the pump to be on continuously. More details about sensor settings and filter-weighing processes are described in previous research.³⁵

At the conclusion of sampling, each participant completed a questionnaire including general demographic information, time-activity patterns, and SHS exposure status over the past five days (eg, "In the last 5 days, has anyone (including visitors) smoked in any part of your home?"). To evaluate the influence of ambient PM_{2.5} on indoor levels, concurrent hourly ambient PM_{2.5} data from the closest US Environmental Protection Agency (EPA) monitor were analyzed.³⁶

Continuous PM_{2.5} concentrations were processed to minute average data and then evaluated for baseline adjustment following methods in Zhang et al.³⁷ Only real-time continuous data meeting the "Good" or "Acceptable" standard after calibration were used in further analyses.

2.2 | Peak analysis

In order to characterize PM_{2.5} peaks, we determined thresholds using a cumulative distribution function (CDF) plot, a method that has been used for identifying the influence of emissions from a strong source such as biomass combustion from cookstoves.^{38,39} Figure 1 shows an illustration of our approach for identifying the threshold level, above which would be considered peak concentrations. The x-axis was the cumulative probability with a log-normal distribution scale while the y-axis was PM_{2.5} concentration. Two regression lines could be defined (Figure 1) and the intersection point of these two lines was defined as the threshold during the defined sampling period.

After determining the thresholds for each sampling period, data points above the thresholds were selected and peaks were defined by start and end points. Data points above the threshold were connected as initial peaks. A start or end point of a peak was the nearest inflection point (to an initial peak) based on slopes of the curve. An inflection point was determined based on where slopes between this point and its left and right adjacent points had opposite positive and negative values. To reduce the impact of noise or short-duration events in real-time PM_{2.5} data on the slope calculation, we calculated slopes with two points that were five-minute away from each other instead of two adjacent points. After identifying the start and end points of a peak, the peak was defined by connecting continual data points between them; the baseline was then the straight line between them. Peaks lasting for less than 3 minutes were not considered as real peaks (Figure 2). These spikes can be due to abrupt movement of the unit.

After determination of start and end points of a peak, baseline was defined as the line connecting the start and end points; “peak range” was defined as all the PM_{2.5} data points between these two points; “peak duration” (hr) was defined as the time duration of peak range; while time-weighted peak area (hour*µg/m³) was the product of the excess PM_{2.5} concentrations for peaks (after subtracting the non-peak baseline). The accumulated peak area (APA) is the sum of the time-weighted peak area of all peaks, following the approach in Meijster.⁴⁰

Post-study experiments were carried out in carefully observed residential settings with cigarette smoking. Observations were made on air exchange rates and number and timing of cigarettes being smoked. Observations and data from these post-study experiments were used to inform our analysis and method development here.

The highest point of a peak (peak point) was generated separately using the “findPeaks” function in the “quantmod” package (version: 0.4–13) in R. In the post-study experiments, multiple peak points were observed within one peak range in some cases. Some of these cases were due to indoor air convection while the rest were real peak points from different PM_{2.5} emission events. We manually defined that within 30 minutes, only the peak point with maximum PM_{2.5} value was the real peak point of the peak range while any secondary peaks within this time period were identified as false peaks (Figure 2). Total peak number was the total number of peak points over the deployment time period. Peak number per day was the result of total peak number divided by total sampling days.

To detect the influence from residential SHS, TAD (time-activity diary), questionnaire data were used to determine time periods when the participants were home, and continuous PM_{2.5} data within these periods were extracted from both personal and residential monitors for further peak analysis. Peaks having durations less than or equal to 60 minutes were selected to estimate PM_{2.5} from SHS. The rationale for the 60-minute duration was that the influence of smoking on PM_{2.5} levels from tobacco smoking rarely lasted longer than 1 hour in our post-study experiments and in other studies.³² Depending on parameters such as the ventilation rate and the number of smokers in the home, SHS-derived peak duration varies, but typically lasts less than 60 minutes. Studies also showed that peaks due to family cooking normally lasted for more than 1 hour.^{41,42} Peak indexes like accumulated peak area (APA), number of peaks, peak duration, and average peak area (AvgPA) of all peaks within 60 minutes were summarized for each deployment. In this study, we only consider peaks when participants were at home as possible SHS affected ones, since PM_{2.5} from cigarette smoking can dissipate within seconds to a few minutes in outdoor microenvironments.³⁰

Other indexes such as accumulated background area at home (ABAH or PM_{2.5} exposures due to concentrations below the SHS baselines when children were home), accumulated area at home (AAH), and accumulated area outside home (AAOH) were also calculated for each deployment based on both personal and residential monitors. Inhalation flow rate was used to calculate contribution of SHS to PM_{2.5} exposure. Lacking calibrated information on children's activity level, an average volumetric inhalation rate of 13.6 m³/day was applied to calculate the extra amount of inhaled PM_{2.5} due to SHS exposure (exposure = accumulated area*volumetric inhalation rate*1/24 (day/hour)).⁴³

2.3 | Data analysis

The wearing compliance of each participant was calculated with accelerometer data recorded every 10 seconds by MicroPEM sensors (see Appendix S1 for details). Analysis of variance (ANOVA) was conducted to compare peak indexes among those that reported smoker(s) at home during the monitoring period (SHS_q exposed group) vs. those that did not (Non-SHS_q group). Linear regression modeling was performed to examine the effect of factors like gender, age, and heating season on peak exposure. Because race was not evenly distributed between the two groups (SHS_q and Non-SHS_q), linear regression was conducted solely for the "Mixed" race group that had the most children recruited. All analyses were processed with R (version 3.5.0; R Foundation for Statistical Computing).

3 | RESULTS

3.1 | Descriptive statistics

Among the 41 recruited children, 36 were included for further peak analysis. For the 5 samples that were excluded, one sensor used for residential monitoring stopped working while four personal continuous data failed to meet our validation criteria even after baseline correction and gravimetric normalization.³⁵ Average sampling period for these 36 participants was 4.7 days (95% CI: 4.3–5.1 days). Characteristics of the participants are shown in Table 1. There was no significant difference in gender and age ($P > .5$) between

SHS_q exposed and unexposed groups, whereas significant difference existed in racial distribution ($P < .05$).

Based on self-reported questionnaire data, a total of 18 children should have had residential indoor SHS exposure. In 14 (77.8%) of these SHS_q homes, people also reported smoking out of the window in their home. The average wearing compliance of wearing the personal monitor for all children was 10.1 hours each day (95% CI: 8.95–11.2 h/day), and assuming that the children slept for ~9 hours per day,⁴⁴ this results in average percent wearing compliance during waking hrs of 67%.

3.2 | Thresholds, peak number, and peak duration

Thresholds based on CDF plots of residential and personal samples were between 9–40 $\mu\text{g}/\text{m}^3$, significantly higher ($P < .001$) than concurrent ambient $\text{PM}_{2.5}$ (6–16 $\mu\text{g}/\text{m}^3$) concentrations. Calculated thresholds were similar between residential ($19.8 \pm 5.8 \mu\text{g}/\text{m}^3$, 95% CI: 17.8–21.8 $\mu\text{g}/\text{m}^3$) and personal ($20.8 \pm 6.7 \mu\text{g}/\text{m}^3$, 95% CI: 18.6–23.0 $\mu\text{g}/\text{m}^3$) monitoring data ($P > .05$) but the thresholds for personal samples for the SHS_q group ($23.3 \pm 7.7 \mu\text{g}/\text{m}^3$, 95% CI: 19.5–27.0 $\mu\text{g}/\text{m}^3$) were significantly higher than for Non-SHS_q children ($18.9 \pm 5.4 \mu\text{g}/\text{m}^3$, 95% CI: 16.6–21.2 $\mu\text{g}/\text{m}^3$) ($P < .05$). Because of the lower thresholds, more peaks with relatively low $\text{PM}_{2.5}$ levels were included in data of children living in homes reported to not have active smokers, and the average peak duration are similar among SHS_q and Non-SHS_q groups ($P > .05$).

Total peak durations (TPD) were higher for residential (6.1 hour) and personal (5.6 hour) measurements in smoking families, 1.8 and 1.6 times those from non-smoking families (Table 2), respectively. Total peak number from smoking families (16) was 1.5 times that from non-smoking families (11) for residential deployments and 1.6 times that from non-smoking families for personal deployments ($P < .05$). Similarly, peak number per day was 4.0 for SHS_q exposed children (personal samples), 1.8 times of that of the Non-SHS_q children (2.2, $P < .005$).

3.3 | $\text{PM}_{2.5}$ concentration and accumulated $\text{PM}_{2.5}$ areas

Average personal $\text{PM}_{2.5}$ mass concentration for SHS_q children was $24.3 \pm 15.4 \mu\text{g}/\text{m}^3$, 1.6 times that of Non-SHS_q children ($14.8 \pm 9.2 \mu\text{g}/\text{m}^3$). As for residential measurements, the concentration was $24.8 \pm 12.1 \mu\text{g}/\text{m}^3$ for in SHS_q homes and $17.2 \pm 10.9 \mu\text{g}/\text{m}^3$ for those in Non-SHS_q homes (Figure 3). The y-axis was log(10)-transformed to better display differences between the SHS_q and Non-SHS_q homes.

The statistics derived from the analysis of the continuous data for peak and background areas are presented in Table 2. Differences between residential and personal measurements were negligible for all the various statistics derived, as p -values ranged from 0.11 to 0.97 (last two rows of Table 2). The difference between SHS_q and Non-SHS_q subjects were more distinct. APA for SHS_q exposed children and their families were $297 \pm 325 \text{ hour} \cdot \mu\text{g}/\text{m}^3$ (personal) and $263 \pm 248 \text{ hour} \cdot \mu\text{g}/\text{m}^3$ (residential), 6.0 and 7.3 times the APA for the personal and residential measurements from families in Non-SHS_q homes, respectively (Figure 3).

Almost all African-American children recruited were in the SHS_q group while more children of mixed race were in the Non-SHS_q group (Table 1). Table 3 shows the linear regression analysis for APA, which revealed that African-American children had higher APA than children of mixed race ($P < .05$). Within either African-American children or mixed race children, the SHS_q group had higher APA (based on either residential or personal samples) than the Non-SHS_q group ($P < .001$). After excluding race from the regression model, among various predictors including heating season, gender, and candle/incense burning, SHS_q status turned to be the only variable associated with APA levels ($P < .001$) (Table 3). The regression model also suggests that APA levels of the SHS_q group were 195 hour* $\mu\text{g}/\text{m}^3$ higher than the Non-SHS_q groups ($P < .001$).

Average peak area (AvgPA) showed a similar trend. AvgPA was 22 ± 23 and 21 ± 20 hour* $\mu\text{g}/\text{m}^3$ for personal and residential sampling in the SHS_q group, and were only about 6 hour* $\mu\text{g}/\text{m}^3$ for both personal and residential samples of those from the Non-SHS_q group (Table 2). The only exceptions were two SHS_q participants from the same family who claimed a daily SHS exposure. No peak was detected for the first participant while both personal and residential AvgPA (4.9 and 3.7 hour* $\mu\text{g}/\text{m}^3$ each) for the second participant (the same family) were much lower than average personal and residential levels of the SHS_q exposed group. Measured nicotine levels were also low in this home (data not presented), confirming low SHS exposure.

Accumulated area at home (AAH) and accumulated area outside home (AAOH), including both peak and non-peak exposures, were also summarized (Table 2) to estimate total PM_{2.5} exposure at home and in non-residential environments, respectively. For SHS_q subjects, AAH was 1704 ± 807 hour* $\mu\text{g}/\text{m}^3$ and 1467 ± 919 hour* $\mu\text{g}/\text{m}^3$ for residential and personal measurements, respectively, which were 1.7 and 1.5 times that of Non-SHS_q subjects ($P < .05$). The latter had AAH of 1009 ± 374 and 962 ± 322 hour* $\mu\text{g}/\text{m}^3$ for residential and personal measurements. The ratios of AAH were similar to that of PM_{2.5} mass concentration between SHS_q and Non-SHS_q groups. AAOH of these two groups was similar with a ratio of about 1.0. Accumulated background area at home (ABAH) was used to characterize the portion of residential PM_{2.5} contributed by the outdoor environment,³⁵ and ABAH levels were similar between SHS_q and Non-SHS_q families as well (Table 2).

To delineate the effect of wearing compliance on peak exposure level, we compared personal AvgPA when the sensors were worn to that when not worn. The personal AvgPA was 21.5 hour* $\mu\text{g}/\text{m}^3$ when the sensors were worn, similar to ($P > .5$) the AvgPA level (20.6 hour* $\mu\text{g}/\text{m}^3$) when wearing compliance is close to 0 (ie, sensors were not worn but most likely to be left running at home where the monitors were given to the children).

3.4 | PM_{2.5} inhalation

Assuming an inhalation rate of 13.6 m³/day for these children, extrinhaled PM_{2.5} from personal peak exposure was 168 μg for SHS_q children (calculated by multiplying APA with inhalation rate), representing ~13% of their total exposure over the whole sampling period (Table 4). In comparison, total peak duration for these peaks in the personal samples from SHS_q homes (5.6 hr) only accounted for ~5% of the total sampling time.

For SHS_q and Non-SHS_q children, considerable differences existed in accumulated PM_{2.5} mass for both residential ($P < .005$) and personal ($P < .05$) measurements, whereas inhaled PM_{2.5} from time periods without peaks were indistinguishable between the two groups (Table 4). Accumulated PM_{2.5} mass exposure from non-residential environments (ie, AAOH) was comparable for SHS_q and Non-SHS_q groups as well ($P > .5$).

4 | DISCUSSION

Total PM_{2.5} exposure based on time-integrated filters is a less sensitive marker for characterizing SHS exposure, mainly because it is averaging in all the other PM_{2.5} sources (eg, ambient, cooking) in addition to tobacco smoke.^{21,45} In this study, despite the fact that PM_{2.5} mass concentrations of the SHS_q group were 1.4–1.6 times that of the Non-SHS_q group, there was a large proportion of the distributions in PM_{2.5} concentrations that overlapped between the two groups (Figure 3A), demonstrating the poor specificity of the simple mass concentrations for differentiating SHS exposure. Average PM_{2.5} mass concentration was largely affected by various sources including ambient PM_{2.5} level (estimated as ~87% of the total), and PM_{2.5} from SHS was further diluted by other PM_{2.5} sources²⁹.

As demonstrated in this study, as well as others,^{30,46} cigarette smoking led to a rapid increase in indoor particle levels and a decrease after the smoking event, therefore forming peaks in the PM_{2.5} time series data, verifying the sensitivity of PM_{2.5} to tobacco smoke. The method developed here for identifying and quantifying peaks allowed us to evaluate its utility for characterizing SHS exposure, including the time of exposure, accumulated peak area, and peak exposure levels. Peak exposures lasting for less than an hour can lead to immediate physiological changes, as observed in previous studies.⁴⁷

Thresholds were determined to identify peaks, allowing the elevated concentrations to be distinguished from background levels.⁴⁸ In this study, the peak threshold of each deployment was computed, differing from methods used in most other studies, in which a uniform threshold was generally selected based on certain air quality guidelines.^{49,50} We purposely chose this approach because the indoor background PM_{2.5} level could be affected by several factors including ambient levels, ventilation rates, and indoor activities such as cooking and cleaning.^{51–53} Application of a uniform threshold could result in large errors for background PM_{2.5} levels. The wide range of determined thresholds (9–40 µg/m³) in this study is consistent with there being inherent error in using a uniform threshold. It was also questionable to select a threshold based on certain guidelines given the confirmed health effects of PM_{2.5} below guidance levels.^{54,55} For simplicity, only one threshold was selected for each deployment in this study after determining that background PM_{2.5} baseline levels derived for each day in multi-day deployments were pretty stable. However, we suggest determining daily threshold levels when studies are conducted in locations with large daily changes in PM_{2.5}.

The accumulated PM_{2.5} peak area and average peak area appeared to be reliable markers with both sensitivity and specificity. APA is 6–7 times higher for SHS_q participants compared to Non-SHS_q participants, consistent with Loffredo et al (2016), who reported that

1-hour $PM_{2.5}$ concentrations in a cafeteria with people smoking was 6 times that of non-smoking indoor environments.⁵⁶ Similarly, AvgPA also has a sharp contrast among SHS_q and Non-SHS_q families, with a ratio of ~3.7 between the two groups. The difference in residential AvgPA between SHS_q and Non-SHS_q families could potentially be expected to be even larger if there were no children at home, given the fact that smoking out of the window was observed in 78% of the SHS_q families and presuming that the smokers would be less likely to make the extra effort to smoke out of a window if there were no children present.

The high APA was mainly ascribed to cigarette smoking. As mentioned above, $PM_{2.5}$ peaks associated with family cooking typically lasted for more than 1 hour and since we only considered peaks less than 1 hour for SHS exposure, the peak misclassification between smoking and cooking would be largely reduced. There was no significance difference ($P > .1$) in the accumulated area of either background level at home (ABAH) or total $PM_{2.5}$ level outside home (AAOH). Statistical analysis demonstrated that other potential indoor $PM_{2.5}$ sources, such as heating season and reported candle/incense burning, are not associated with APA. Thus it is reasonable to attribute $PM_{2.5}$ concentration differences between these two groups to smoking.

Total peak number, peak number per day, and total peak duration were also associated with self-reported SHS status but the difference between the two groups was less distinct than with APA and AvgPA. Wilson et al³¹ found that total peak duration for families with smokers was 1.2 times that of families whose members had quit smoking. Peak number has barely been discussed in prior studies. Earlier $PM_{2.5}$ peak studies instead focused more on the maximum $PM_{2.5}$ concentration observed during smoking events.^{31,49} Cho et al reported a significant association between peak $PM_{2.5}$ concentration and smoking frequency even with the coexistence of vehicle emissions.⁴⁹ Compared with either peak $PM_{2.5}$ concentration or peak duration, APA appeared to be a better indicator of SHS in both sensitivity and selectivity; furthermore, it is directly relevant in determining the fraction of $PM_{2.5}$ from SHS exposure.

Risks from tobacco smoke are so significant that cigarette equivalence has long been used to illustrate the hazards of other sources of air pollutants.^{15,57,58} Compared with predicting $PM_{2.5}$ exposure of active smokers, predicting the level of $PM_{2.5}$ obtained from SHS exposure for non-smokers is more challenging due to the variable dilution due to distance between smokers and non-smokers, housing type, and ventilation rates.⁴⁶ Questionnaire-based approaches are also challenged by different perceptions of SHS exposure.^{22,26} Based on APA estimates, $PM_{2.5}$ exposure originating from SHS for children in NYC was equivalent to an additional $2.6 \mu g/m^3$ of $PM_{2.5}$ per day, 31% of the ambient annual average $PM_{2.5}$ concentration in NYC ($8.4 \mu g/m^3$ in 2014).⁵⁹ For reference, a reduction of $0.71 \mu g/m^3$ in annual average $PM_{2.5}$ concentration helps to avoid 290 premature deaths in NYC per year.⁶⁰

On average, children from SHS_q families were exposed to SHS for 1.2 hour per day. With increasing public awareness of tobacco hazards,^{61,62} smokers tend to avoid children when smoking. Furthermore, in NYC smoking prevalence and smoking amount has decreased due

to significantly increased cost from local taxes.⁶³ Despite these factors, our study observed that SHS exposure still represents ~13% of total PM_{2.5} exposure for children in smoking families. In addition, many studies showed a higher toxicity for cigarette smoke compared with ambient PM sources such as dust and traffic.^{64–66} To improve indoor air quality for people especially for vulnerable groups,⁶⁷ SHS is still an important pollutant from a public health perspective.^{1,20}

Peak analysis developed in this study allows us to quantify the contribution of PM_{2.5} from SHS exposure from specific locations and time periods. Data from this study and studies using this approach can inform and shape health policy regulating SHS, especially for children.

Limitations also exist. Even though the SHS contribution to APA is predominant, we cannot completely rule out the influence of other sources such as short cooking events (eg, within 30 minutes) or longer-term resuspension events (eg, vacuuming or sweeping the floor). In addition, we did not randomize the groups (due to the limited number of SHS families); thus race, a possible confounding variable, may have impacted the observed associations. We also assumed there is at most one peak within any 30 minutes since most smokers, under typical conditions, will not light another cigarette immediately after finishing one. This may not be true for heavy smokers or in situations where there are multiple smokers at home. Finally, the primary classification for the comparisons was based on self-reported TAD questionnaires. Subjectivity in questionnaires may lead to bias in classification.^{22,26} An approach for identifying the possible misclassification will be detailed in another publication using five different SHS markers, including the accumulated peak area developed in this study.

5 | CONCLUSIONS

An approach based on the cumulative distribution probability function was successfully developed to calculate a peak threshold of each deployment. Determined threshold values ranged widely (9 to 40 $\mu\text{g}/\text{m}^3$), demonstrating the necessity of determining thresholds for each deployment. The average PM_{2.5} mass concentration distributions between self-reported SHS exposed and unexposed children are slightly different but overlap; whereas accumulated peak area, whose level from self-reported smoking families was 6 times that of children from self-reported non-smoking families, had much better selectivity and sensitivity. For children from smoking families, peak exposure lasted for 5% of their monitoring time, but accounted for 13% of their total exposure. Given the high toxicity of cigarette smoke and susceptibility of children, there is the need for continued efforts in reducing SHS exposure. Finally, advances in identification and quantification of short-term exposure events can foster and support large-scale studies assessing health effects of these short-term exposures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Practical implications

- This study developed a method to analyze continuous PM_{2.5} data to identify short-duration elevated exposures or peak exposures and applied the method for characterizing indoor SHS exposure.
- How to extract useful exposure information from continuous or “real-time” data has been a bottleneck for epidemiological studies using real-time monitors.
- Methods are needed before the epidemiological community can carry out studies to answer the key questions of whether peak exposures are driving certain health outcomes or just chronic average exposures, and if peak exposures matter, then how big do they need to be?
- In this case, the developed method together with time-activity diary and GPS data allowed us to quantify the contribution of PM_{2.5} from SHS exposure with rich temporal and spatial information.
- Data from this study and studies using this approach can inform and shape health policy regulating SHS, especially for children.

FIGURE 1.

Cumulative probability distribution of $PM_{2.5}$ concentration over the course of sampling period from a participant sampler. *Inflection point of two regression lines in the plot is set to be the threshold for that deployment

FIGURE 2.

Real and false peaks identified in personal PM_{2.5} real-time data from a NYC participant with a self-reported smoker at home. (AAOH, Accumulated area outside of home (away from home); ABAH, Accumulated background area at home)

FIGURE3.

Comparison of measured PM_{2.5} concentration and accumulated peak area (APA) indexes among self-reported SHS exposed (SHSq) and non-exposed (Non-SHSq) groups. The y-axis is log-transformed, while yellow dot represents the mean value

TABLE 1

Characteristics of the study population

	SHS _q	Non-SHS _q	Total	P-value
Family	12	13	25	
Participant	18	18	36	
Gender				
Male	8	7	15	.744
Female	10	11	21	
Age (y)	12.6 ± 2.6	12.2 ± 2.4	12.4 ± 2.5	.704
Race				
Latino	2	0	2	.019
Hispanic	1	1	2	
African-American	6	1	7	
Mixed	9	16	25	

Abbreviations: SHS_q, self-reported via questionnaire as home with smoker(s); Non-SHS_q, self-reported via questionnaire as non-smoking home

TABLE 2
Distribution and variance analysis of peak indexes for residential and personal measurements

	Threshold	Total peak number	Peak number per day	Average peak duration	TPD	APA	AvgPA	ABAH	AAH	AAOH
Personal										
SHS _q	23	16	4.0	0.35	5.6	297	22	643	1467	853
Non-SHS _q	19	10	2.2	0.35	3.6	50	6	647	962	949
<i>P</i>	.047*	.037*	.002**	.658	.062	.003***	.012*	.960	.029*	.633
Residential										
SHS _q	21	16	3.2	0.40	6.1	263	21	784	1704	984
Non-SHS _q	19	11	2.3	0.35	3.4	36	6	692	1009	989
<i>P</i>	.473	.039*	.094	.146	.005**	.000***	.002**	.287	.002**	.867
Personal Residential										
SHS _q	1.2	1.6	1.8	1.0	1.6	6.0	3.7	1.0	1.5	0.9
Non-SHS _q	1.1	1.5	1.4	1.2	1.8	7.3	3.7	1.1	1.7	1.0
<i>P</i>(Residential vs Personal)										
SHS _q	0.40	0.94	0.41	0.67	0.77	0.77	0.92	0.14	0.502	0.59
Non-SHS _q	0.82	0.72	0.80	0.11	0.79	0.28	0.87	0.53	0.68	0.97

Abbreviations: SHS_q, self reported via TAD questionnaire as smoking occurred in home during deployment period; Non-SHS_q, self reported via TAD questionnaire as no smoking occurred in home during deployment period; APA, accumulated peak area while at home; ABAH, accumulated background area at home; AAH, accumulated area at home; TPD, total peak duration; AAOH, accumulated area outside of home; AvgPA, average peak area.

* $P < .05$.

** $P < .005$.

*** $P < .001$.

TABLE 3

Results of the linear regression model for accumulated peak area (APA)

	Estimate	SE	<i>t</i> value	<i>P</i> -value
All variables				
SHS _q vs Non-SHS _q	117	53	2.18	.033 [*]
Home vs Personal monitoring	−33	47	−0.72	.48
Candle/Incense Burning vs Non	−47	58	−0.82	.42
Heating vs NonHeating Season	−34	54	−0.64	.53
Age	7	10	0.77	.45
Male vs Female	8	49	0.16	.88
Hispanic vs African Amer.	−190	117	−1.62	.11
Latino vs African Amer.	−3	116	−0.03	.98
Mixed vs African Amer.	−218	70	−3.10	.003 ^{**}
Exclude race				
SHS _q vs Non-SHS _q	195	51	3.86	.000 ^{***}
Home vs Personal monitoring	−31	50	−0.62	.54
Candle/Incense Burning vs Non	−33	55	−0.60	.55
Heating vs NonHeating Season	−53	54	−0.99	.33
Age	10	10	0.99	.33
Male vs Female	55	50	1.11	.27

^{*}
P < .05.^{**}
P < .005.^{***}
P < .001.

TABLE 4

Inhaled PM_{2.5} from different exposure pathways (unit: µg), including contributions to total exposure during the sampling periods and variance analysis comparing SHS_q exposed and Non-SHS_q groups

Sample-type	Self-report category	APA	ABAH	AAOH
Personal	SHS _q	168 (12.8%)	364 (27.7%)	484 (36.8%)
	Non-SHS _q	28.2 (2.5%)	366 (32.9%)	568 (51.0%)
	<i>P</i>	0.003**	0.96	0.63
Residential	SHS _q	149 (9.8%)	444 (29.2%)	558 (36.3%)
	Non-SHS _q	20.3 (1.8%)	392 (34.6%)	561 (49.5)
	<i>P</i>	0.000***	0.29	0.99

Abbreviations: APA, Accumulated Peak Area while at home; ABAH, Accumulated background area at home; AAOH, Accumulated area outside of home (away from home).

** *P* < .005.

*** *P* < .001.