Smoking behaviours and indoor air quality: a comparative analysis of smoking-permitted versus smoke-free homes in Dhaka, Bangladesh

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ABSTRACT

Introduction Exposure to secondhand smoke (SHS) is a health risk to non-smokers. Indoor particulate matter ($PM_{2.5}$) is associated with SHS exposure and is used as a proxy measure. However, $PM_{2.5}$ is non-specific and influenced by a number of environmental factors, which are subject to geographical variation. The nature of association between SHS exposure and indoor $PM_{2.5}$—studied primarily in high-income countries (HICs) context—may not be globally applicable. We set out to explore this association in a low/middle-income country setting, Dhaka, Bangladesh.

Methods A cross-sectional study was conducted among households with at least one resident smoker. We inquired whether smoking was permitted inside the home (smoking-permitted homes, SPH) or not (smoke-free homes, SFH), and measured indoor $PM_{2.5}$ concentrations using a low-cost instrument (Dylos DC1700) for at least 22 hours. We describe and compare SPH and SFH and use multiple linear regression to evaluate which variables are associated with $PM_{2.5}$ level among all households.

Results We surveyed 1746 households between April and August 2018; 967 (55%) were SPH and 779 (45%) were SFH. The difference between $PM_{2.5}$ values for SFH (median 27 μg/m^3, IQR 25) and SPH (median 32 μg/m^3, IQR 31) was 5 μg/m^3 (p<0.001). Lead participant's education level, being a non-smoker, having outdoor space and smoke-free rule at home and not using kerosene oil for cooking were significantly associated with lower $PM_{2.5}$.

Conclusions We found a small but significant difference between $PM_{2.5}$ concentrations in SPH compared with SFH in Dhaka, Bangladesh—a value much lower than observed in HICs.

INTRODUCTION

Exposure to secondhand smoke (SHS) is a serious health hazard among non-smoking adults and children.[1] The harmful consequences of SHS mostly affect women and children by causing lower respiratory infections,[2] middle ear disease,[3] tuberculosis,[4] chronic obstructive pulmonary disease and exacerbation of asthma.[2] Globally, every year, around 890 000 lives and 10.9 million disability-adjusted life years are lost because of SHS exposure.[5]

There are several well-accepted objective methods to assess SHS exposure such as measurement of nicotine or particulate matter ($PM_{2.5}$) in the air or cotinine in body fluids as a biomarker of inhaled nicotine.[6] While nicotine and cotinine are tobacco specific, airborne $PM_{2.5}$ is not. Despite this, indoor $PM_{2.5}$ concentration has been widely used as a marker of SHS and is a useful indicator for evaluating indoor smoke-free policies.[7–9] $PM_{2.5}$ concentrations have also been shown to be several times higher in smoking-permitted homes (SPH) compared with smoke-free homes (SFH).[10–13] Moreover, factors such as the presence and number of smokers living in the home, the number of smokers who smoke on a daily basis, number of cigarettes smoked, location of smoking inside the home (bedrooms, kitchen), types of tobacco products used and household ventilation influence the concentration of indoor $PM_{2.5}$.[13 14] However, the nature of association between $PM_{2.5}$ and SHS is based on data collected from high-income countries (HICs) and little is known about the presence and magnitude of these associations in the rest of the world. We aimed to study the association between indoor $PM_{2.5}$ levels and SHS in a lower/middle-income country (LMIC) setting, where most of the previous studies have looked at $PM_{2.5}$ in relation to stoves or cooking fuels. This is important for two reasons. First, the number of smokers is rising in LMICs[15] with an accompanying increase in SHS exposure.[16] Second, $PM_{2.5}$ is influenced by a wide range of outdoor (eg, motor vehicles, construction, industrial processes, desert dust) and indoor (eg, cooking and biomass fuel combustion) sources,[17 18] which are likely to differ between HIC and LMIC contexts. For example, there is higher ambient air pollution, use of a range of domestic fuels and greater air exchange rates through ventilation in many LMIC settings compared with HICs. As low-cost $PM_{2.5}$ counting instruments such as the Dylos DC1700 are becoming increasingly accessible in LMIC,[19] it is timely to assess if $PM_{2.5}$ can be used as a marker of SHS[6] in such settings.

In this paper, we compare indoor $PM_{2.5}$ concentrations between SFH and SPH, and identify other factors that may be associated with differences in concentrations.

METHODOLOGY

Study design and settings

This cross-sectional design used baseline data collected as part of a cluster randomised controlled trial (cRCT) called Muslim Communities Learning About Second-hand Smoke in Bangladesh (MCLASS

II).[20] This cRCT aimed to evaluate the effectiveness and cost-effectiveness of a community-based intervention to improve domestic indoor air quality in Dhaka, Bangladesh. We included 1746 recruited households.

Study participants

We recruited households with at least one adult resident who smoked cigarettes or other forms of tobacco (eg, bidi, shisha) regularly (at least 25 out of 30 days/month) and at least one other non-smoking resident. We excluded households that used coal or biomass fuel for cooking or other domestic purposes since we expected that such combustion sources would mask the effect of smoking on indoor $PM_{2.5}$ concentrations. Each household nominated a 'lead participant' who consented and completed the household questionnaire. There were no restrictions on which member of the household the lead participant could be, provided they were an adult resident; they could be either a smoker or a non-smoker.

Data

Data were collected by trained field investigators. A structured questionnaire was used to collect household-level data including: number of adults, children, smokers and bedrooms; presence of adjacent outdoor spaces (such as garden, yard, balcony or veranda); type of fuel used for cooking (electric/liquefied petroleum gas (LPG)/natural gas/biogas, kerosene); indoor smoking rules for residents and visitors; and asset index value. Since household income is a fluctuating variable and often subject to measurement bias, asset index was used as a proxy measure for household wealth and hence their long-run economic status.[21] Questions on access to electricity, flush toilet, fixed telephone, cell phone, television, radio, refrigerator, car, motorcycle and cattle were asked. First, asset weight for each of the households was calculated following a standard Principal Component Analysis.[21 22] Then, by using this specific weight for a household and its holding status of the aforementioned asset, asset index for each household was calculated.

The following individual-level data for the lead participant were collected: age, sex (male, female), highest education years (completed years of education) and current smoking status (smoker, non-smoker). Where the lead participant was a smoker, the following individual-level data were also collected: use of different tobacco products (eg, cigarette, bidi), number of total tobacco products consumed daily and number of days of smoking in the last 30 days. However, we were unable to assess the dose–response outcome between tobacco smoked (number of tobacco product consumed inside or outside home) and $PM_{2.5}$, as the goal of this trial was to achieve complete abstinence of indoor smoking.

In addition, we used Dylos DC1700 (Dylos, California, USA) machines installed in the households to measure and record indoor airborne particulate concentrations ($PM_{2.5}$) every minute. These devices were placed in the main living room of each home as far as possible from the kitchen and windows. They were plugged in and run from mains electricity; a 400 Bangladeshi taka (equivalent to US$5) incentive was offered to compensate for the increased electricity costs and household members' time spent completing questionnaires. In the case of a power cut, the battery was able to support the device for up to 6 hours. The devices were installed in homes for at least 24 hours, with the aim to record a full 24 hours of data, though data were accepted if no less than 22 hours of recordings were available. If less than 22 hours of data were recorded in the first instance (perhaps due to machine failures or the device being mistakenly switched off by a member of the household), two further attempts were made over the next few days in which the devices were reinstalled in the homes and left for a minimum of 24 hours. Fine particle number concentrations were converted to $PM_{2.5}$ mass concentrations using an established methodology.[19] Each Dylos DC1700 device was calibrated against a factory-calibrated TSI Sidepak AM510 Personal Aerosol Monitor (TSI, Minnesota, USA).

Data analysis

We stratified the households into two groups—those that permitted smoking indoors (SPH), and those that did not (SFH). SFH status was based on responding 'only outside' to two questions: 'Are people who live with you allowed to smoke…?' and 'Are people who visit your house (including family members), allowed to smoke…?'. Homes were considered as SPH if residents or visitors were allowed to smoke in any part of the inside of the home. Household and the lead participant characteristics are presented and compared across the two groups (SFH and SPH) using a t-test for continuous variables (or the Mann-Whitney U test where data were not normally distributed) and the X^2 test for categorical variables. A small number of households allowed visitors to smoke inside the home but not residents, these were classed as SPH in the main analyses, but exploratory descriptive analyses of $PM_{2.5}$ values were conducted with these households reallocated to the SFH group.

Ordinary least squares multiple linear regression was conducted including all households with the outcomes of average and maximum household $PM_{2.5}$ (μg/m^3) values, and household-level and lead participant-level characteristics as independent variables. We specified three different models for each outcome. The first only included lead participant-level independent variables. The second included both lead participant-level and household-level independent variables. The third added SFH/SPH status to the model. Goodness of fit in each model was assessed using the adjusted R^2 statistic. Model assumptions were checked using a QQ plot to assess the normality of residuals, and a scatter plot of fitted values versus residuals, and White's test,[23] to assess heteroscedasticity. Residuals from the models using untransformed outcome data were not normally distributed, so $PM_{2.5}$ data were log-transformed for the final analysis models, which significantly improved model fit (see online supplemental material 1).

For each model, we assessed multicollinearity (using the variance inflation factor (VIF), where a VIF greater than 10 may suggest concerning correlation between the explanatory variables.

Statistical significance was assessed using two-sided tests at the 5% level. We used Stata V.15 for all analysis.

RESULTS

Data were collected between April and August 2018, for 1801 households. Data for 55 households could not be used as at least 22 hours of $PM_{2.5}$ measurements were not achieved. We therefore analysed data from 1746 households; 967 (55%) were SPH and 779 (45%) were SFH (table 1).

SPH tend to have fewer adult residents, fewer bedrooms, not have outdoor space and not use electricity for cooking as compared with SFH. Average household $PM_{2.5}$ concentration was significantly higher among SPH than SFH (mean 45.2 (SD 40.6, median 32.0) compared with 38.4 (SD 34.3, median 27.0); $p<0.001$). 'One-minute household maximum $PM_{2.5}$ concentration' was also significantly higher among SPH than SFH (mean

Table 1 Characteristics of household and ambient air ($PM_{2.5}$) stratified by whether the home is defined as being 'smoke free' or not

Household characteristics	Smoke-free homes (n=779)	Smoke-permitted homes (n=967)	Total (n=1746)	Associated test p value
Number of adults in household				
Mean (SD)	2.5 (0.8)	2.3 (0.7)	2.4 (0.8)	<0.001§
Median (min, max)	2 (1, 6)	2 (1, 6)	2 (1, 6)	
Number of children in household				
Mean (SD)	1.4 (1.1)	1.4 (1.1)	1.4 (1.1)	0.39§
Median (min, max)	1 (0, 7)	1 (0, 6)	1 (0, 7)	
Number of smokers				
Mean (SD)	1.1 (0.3)	1.1 (0.3)	1.1 (0.3)	0.05§
Median (min, max)	1 (1, 3)	1 (1, 3)	1 (1, 3)	
Number of bedrooms				
Mean (SD)	1.5 (0.7)	1.3 (0.6)	1.4 (0.7)	<0.001§
Median (min, max)	1 (0, 6)	1 (1, 5)	1 (0, 6)	
Home has outdoor space, n (%)				
Yes	495 (63.5)	450 (46.5)	945 (54.1)	<0.001‡
No	284 (36.5)	517 (53.5)	801 (45.9)	
Fuel used for cooking, n (%)				
Electricity	77 (9.9)	70 (7.2)	147 (8.4)	0.05‡
LPG/natural gas/biogas	705 (90.5)	902 (93.3)	1607 (92.0)	0.03‡
Kerosene	29 (3.7)	33 (3.4)	62 (3.6)	0.73‡
Asset index				
Mean (SD)	0.4 (4.9)	0.3 (4.4)	0.4 (4.7)	<0.001§
Median (min, max)	−0.5 (−0.9, 44)	−0.3 (−0.8, 44)	−0.3 (−0.9, 44)	
Maximum $PM_{2.5}$ value (µg/m^3)*				
Mean (SD)	309.3 (285.8)	372.6 (290.4)	344.3 (290.0)	<0.001§
Median (min, max)	208.0 (21.0, 1376.0)	286.0 (20.0, 1304.0)	248.5 (20.0, 1376.0)	
IQR	342	419	377	
Mean $PM_{2.5}$ value (µg/m^3)†				
Mean (SD)	38.4 (34.3)	45.2 (40.6)	42.2 (38.0)	<0.001§
Median (min, max)	27.0 (2.0, 290.0)	32.0 (1.0, 422.0)	30.0 (1.0, 422.0)	
IQR	25	31	28	

*Maximum $PM_{2.5}$ value: maximum 1 min derived value for each household.
†Mean $PM_{2.5}$ value: an average of 1440 min of data was collected for each household.
‡X^2 test.
§Mann-Whitney U test.
LPG, liquefied petroleum gas; $PM_{2.5}$, particulate matter.

372.6 (SD 290.4, median 286.0) compared with 309.3 (SD 285.8, median 208.0); p<0.001).

In additional analyses, in which we reallocated SPH homes where smoking was permitted by visitors only (21 households) to the SFH group, the mean and maximum $PM_{2.5}$ values were almost identical to the original categories.

To demonstrate differences in short-term concentrations within homes where smoking did and did not take place, the distribution of minute-by-minute $PM_{2.5}$ concentrations in SPH and SFH is shown in figure 1. SPH were significantly right-shifted compared with SFH indicating more individual minutes at higher concentrations (Kolmogorov-Smirnov test p<0.001). Overall, 5.6% of minutes in SPH had concentrations higher than 150.4µg/m^3 (the US Environmental Protection Agency's threshold for 'very unhealthy' concentrations)[24] compared with 4.2% of minutes in SFH.

Lead participants in SPH were more likely to be men, to have completed fewer years' education and to be a current smoker than in SFH (table 2). In addition, smoker lead participants in SPH tended to smoke more cigarette and have a higher daily consumption compared with SFH.

In the first model for mean $PM_{2.5}$, we observed that years of education (p<0.001) and smoking status (p<0.001) of the lead participant are statistically significantly associated with air quality (table 3). The effect of education is very small; however, for every additional year of education, the average household $PM_{2.5}$ value reduces by a factor of 0.98 (95% CI 0.97–0.99). The mean $PM_{2.5}$ value of the households for which the lead participant is a smoker is, on average, 1.31 times higher (95% CI 1.14–1.51) than households where the lead participant is not a smoker.

In the second model for mean $PM_{2.5}$, education level (p<0.001) and smoking status (p=0.003) of lead participant remain statistically significant correlates, with similar magnitudes of effect. In addition, use of kerosene as a cooking fuel was found to almost double the indoor air pollution (multiplies expected value of mean $PM_{2.5}$ by 1.95, 95% CI 1.65–2.32, p<0.001) relative to households that do not use kerosene, while having an outdoor space is associated with a significant reduction in average $PM_{2.5}$ by a factor of 0.86 (95% CI 0.81–0.92, p<0.001).

In the third model, the magnitude and significance of the covariates affecting the air quality were found to be reasonably consistent with the previous model. In addition, there was evidence that SPH status is predictive of $PM_{2.5}$ (p=0.02), though the difference is very small; the average estimated $PM_{2.5}$ value was 8% higher (95% CI 1%–15%) in SPH relative to SFH.

The VIF ranged from 1.39 to 1.40 for all three models and no evidence of heteroscedasticity was found in any of the models.

As with average $PM_{2.5}$, education level and smoking status of the lead participant were significantly associated with maximum

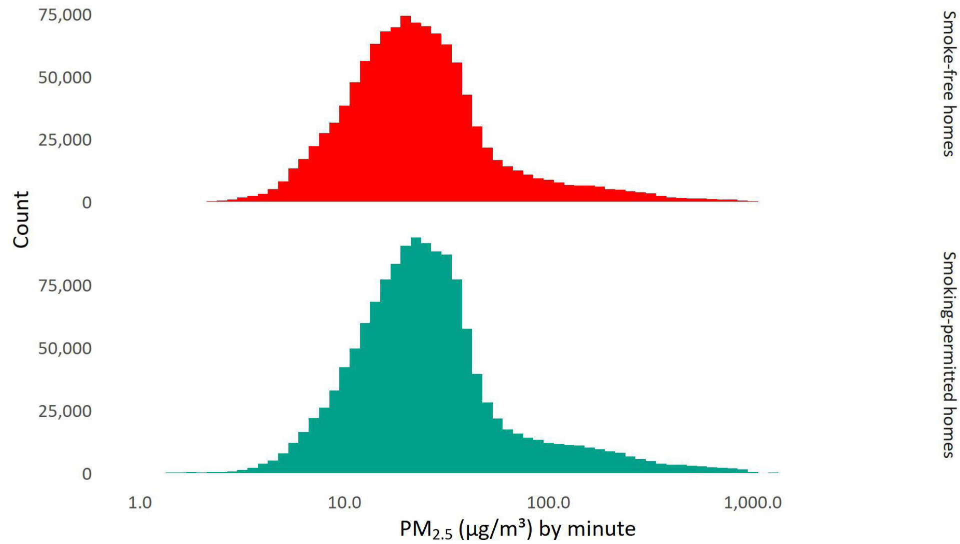

Figure 1 Distribution of $PM_{2.5}$ concentration at each minute by smoking policy in the home. Note the logarithmic scale on the x-axis. $PM_{2.5}$, particulate matter.

$PM_{2.5}$ in all three models (table 4). In models 2 and 3, an increase in household asset index was observed to be associated with a very small but statistically significant decrease in maximum $PM_{2.5}$, as were the lead participant not being a smoker, having outdoor space and not using kerosene for cooking, though these effects were larger. In model 3, age of the lead participant was statistically significant but the magnitude of the effect was negligible. On average, the maximum $PM_{2.5}$ was 1.17 times higher than in SFH (95% CI 1.06–1.28, p=0.001).

DISCUSSION

In this cross-sectional analysis, we used baseline data collected as part of the MCLASS II cRCT. This study of 1746 households is the largest of its kind to measure $PM_{2.5}$ over a whole 24-hour period using low-cost air particle monitors in an LMIC setting, and the first to specifically investigate the use of such devices to assess SHS concentrations in homes in an LMIC setting. We show that it is feasible to conduct a large scale, population-based

Table 2 Characteristics of household lead stratified by whether the home is defined as being 'smoke free' or not

Lead participant's characteristics	Smoke free homes (n=779)	Smoke-permitted homes (n=967)	Total (n=1746)	Associated test p value
Age (years)				
Mean (SD)	39.5 (12.5)	40.5 (12.5)	40.1 (12.5)	0.12*
Gender, n (%)				
Male	724 (92.9)	939 (97.1)	1663 (95.2)	<0.001†
Female	55 (7.1)	28 (2.9)	83 (4.8)	
Highest education years, n (%)				
Mean (SD)	6.4 (5.0)	4.1 (4.2)	5.1 (4.7)	<0.001*
Current smoking status, n (%)				
Non-smoker	133 (17.1)	42 (4.3)	175 (10.0)	<0.001†
Smoker	646 (82.9)	925 (95.7)	1571 (90.0)	
Among the smoker lead participants	**n (%)=646 (41.1)**	**n (%)=925 (58.9)**	**n (%)=1571 (100.0)**	
Only cigarette smoker, n (%)				
Cigarette	645 (99.8)	895 (96.8)	1540 (98.0)	<0.001†
Other	1 (0.2)	30 (3.2)	31 (1.9)	
Only bidi smoker, n (%)				
Bidi	7 (1.1)	70 (7.6)	77 (4.9)	<0.001†
Other	639 (98.9)	855 (92.4)	1494 (95.1)	
Median (min, max)	10 (3, 20)	10 (4, 40)	10 (3, 40)	
Total tobacco product (cig/bidi) consumed/day				
Mean (SD)	10.3 (5.5)	14.4 (7.9)	12.7 (7.3)	<0.001*
Number of days smoked any tobacco product in last 30 days				
Mean (SD)	29.6 (1.3)	29.9 (0.6)	29.8 (0.9)	<0.001‡
Median (min, max)	30 (25, 30)	30 (20, 30)	30 (20, 30)	

*T-test.
†X^2 test.
‡Mann-Whitney U test.

Table 3 What factors predict average PM value? Three multiple linear regression models with log-transformed mean $PM_{2.5}$ value as the outcome

Variable (reference variable)	Model 1 (characteristics of lead participant) Coefficient (95% CI)	P value	Model 2 (characteristics of lead participant and household) Coefficient (95% CI)	P value	Model 3 (characteristics of lead participant and household and their indoor smoking policy at home) Coefficient (95% CI)	P value
Age	−0.001 (−0.004 to 0.001)	0.32	−0.001 (−0.004 to 0.002)	0.43	−0.001 (−0.004 to 0.001)	0.30
Gender (male)						
Female	0.12 (−0.06 to 0.31)	0.19	0.10 (−0.08 to 0.29)	0.28	0.11 (−0.08 to 0.29)	0.26
Highest education grade	−0.02 (−0.03 to 0.01)	**<0.001*****	−0.02 (−0.03 to 0.01)	**<0.001*****	−0.02 (−0.03 to 0.01)	**<0.001*****
Lead participant smoking status (non-smoker)						
Smoker	0.27 (0.13 to 0.41)	**<0.001*****	0.22 (0.07 to 0.36)	**0.003****	0.19 (0.05 to 0.34)	**0.009****
Asset index	—	—	−0.002 (−0.01 to 0.00)	0.51	−0.002 (−0.01 to 0.00)	0.47
Number of adults in household	—	—	−0.04 (−0.09 to 0.02)	0.17	−0.03 (−0.09 to 0.02)	0.24
Number of children in household	—	—	0.02 (−0.01 to 0.05)	0.17	0.02 (−0.01 to 0.05)	0.12
Number of smokers	—	—	0.01 (−0.11 to 0.13)	0.90	−0.003 (−0.13 to 0.12)	0.97
Number of bedrooms	—	—	0.05 (−0.00 to 0.11)	0.06	0.05 (−0.00 to 0.11)	0.06
Home has outdoor space (no)						
Yes	—	—	−0.15 (−0.21 to 0.08)	**<0.001*****	−0.14 (−0.20 to 0.07)	**<0.001*****
Fuel used for cooking (electricity/LPG/natural gas/biogas)						
Kerosene	—	—	0.67 (0.50 to 0.84)	**<0.001*****	0.67 (0.51 to 0.84)	**<0.001*****
Indoor smoking policy of home (smoke-free home)						
Smoke-permitted home	—	—	—	—	0.08 (0.01 to 0.14)	**0.02****
Total observations	1746		1746		1746	
F (probability)	18.8 (**<0.001*****)		14.6 (**<0.001*****)		13.9 (**<0.001*****)	
Adjusted R^2	0.039		0.079		0.081	

P value significance level: 10% (*), 5% (**), 1% (***).
LPG, liquefied petroleum gas; $PM_{2.5}$, particulate matter.

indoor air quality study in an LMIC. Just over half of our studied households allowed smoking in the home. These households tended to have fewer adult residents and bedrooms, access to outdoor space and use natural gas/LPG for cooking in comparison to SFH. The difference in median daily household $PM_{2.5}$ concentrations was only 5 µg/m^3 (higher in SPH), much lower than estimates from HICs. For example, in the UK, one study found the difference to be 28 µg/m^3 between SPH (median 31 µg/m^3, similar to than seen in our study of 32 µg/m^3) and SFH (median 3 µg/m^3).[25] Multiple factors can plausibly explain this difference. Outdoor $PM_{2.5}$ values are much higher in Dhaka than London or other cities in HICs. We purposefully took the indoor $PM_{2.5}$ measurements during the rainy season in Bangladesh to minimise the impact of outdoor air pollution, as $PM_{2.5}$ concentrations are highly seasonal in Dhaka.[26] Furthermore, our study site, Mirpur, is in one of the most populated areas in Dhaka, and two large construction activities (metro rail and a flyover) were conducted in the last few years in this area, which might have elevated the background $PM_{2.5}$ level. However, our study is limited by a lack of data on outdoor $PM_{2.5}$ concentration over the course of the baseline measurement period. Continuous ambient air pollution data are available only from one monitor at the US Embassy in central Dhaka, far from Mirpur, which (given the local sources previously identified) is unlikely to represent local ambient $PM_{2.5}$. Second, many of our study households were living in slums where housing is generally poorly constructed and likely to have high levels of outdoor to indoor air exchange. Another possible explanation of the high $PM_{2.5}$ concentrations measured in homes could be the use of insecticide mosquito coils that were burned inside some homes due to a dengue fever outbreak in Dhaka during our study period. Previous work has suggested that burning mosquito coils can generate $PM_{2.5}$ mass that is equivalent to several cigarettes.[27] Other plausible reasons for a smaller difference in $PM_{2.5}$ concentrations between SPH and SFH could be related to the behaviour of household members. For instance, a possible Hawthorne effect[28] may exist, which may influence participants' awareness and behaviour during the 24-hour $PM_{2.5}$ concentration measurement as devices were visibly present. Measuring for a longer period could be a possible solution to avoid this Hawthorne effect, however, a study in the UK[29] showed that the average of a full 6-day measurement provided similar results to the first 24 hours. Moreover, in SPH it is possible that smokers prefer to smoke outside the home when they are with friends, colleagues or common peer groups and, consequently, smokers in Dhaka city may smoke less frequently inside the home compared with smokers who smoke at home in the UK. This may also support our finding that having outdoor space can reduce the $PM_{2.5}$ concentrations. Importantly, presence of a private outdoor space is a crucial factor for designing an intervention to reduce indoor SHS as this is an 'opportunity' in the COM-B model[30] (a behavioural system that connects three essential conditions: capability, opportunity and motivation).

Table 4 What factors predict maximum PM value? Three multiple linear regression models with log-transformed maximum $PM_{2.5}$ value as the outcome

Variable (reference variable)	Model 1 (characteristics of lead participant)		Model 2 (characteristics of lead participant and household)		Model 3 (characteristics of lead participant and household and their indoor smoking policy at home)	
	Coefficient (95% CI)	P value	Coefficient (95% CI)	P value	Coefficient (95% CI)	P value
Age	−0.003 (−0.01 to 0.00)	**0.07***	−0.004 (−0.01 to 0.00)	**0.09***	−0.004 (−0.01 to 0.00)	**0.04****
Gender (male)						
Female	−0.005 (−0.27 to 0.26)	0.97	−0.04 (−0.30 to 0.23)	0.79	−0.03 (−0.29 to 0.23)	0.83
Highest education grade	−0.04 (−0.05 to 0.03)	**<0.001*****	−0.03 (−0.04 to 0.02)	**<0.001*****	−0.03 (−0.04 to 0.02)	**<0.001*****
Lead participant smoking status (non-smoker)						
Smoker	0.32 (0.12 to 0.51)	**0.001****	0.25 (0.04 to 0.45)	**0.02****	0.20 (−0.00 to 0.41)	**0.05***
Asset index	—	—	−0.01 (−0.02 to 0.00)	**0.02****	−0.01 (−0.02 to 0.00)	**0.02****
Number of adults in household	—	—	−0.05 (−0.13 to 0.03)	0.19	−0.04 (−0.12 to 0.04)	0.30
Number of children in household	—	—	0.02 (−0.02 to 0.06)	0.39	0.02 (−0.02 to 0.07)	0.27
Number of smokers	—	—	−0.01 (−0.18 to 0.17)	0.92	−0.03 (−0.21 to 0.14)	0.74
Number of bedrooms	—	—	0.11 (0.03 to 0.19)	**0.008****	0.11 (0.03 to 0.19)	**0.007****
Home has outdoor space (no)						
Yes	—	—	−0.22 (−0.31 to 0.13)	**<0.001*****	−0.21 (−0.29 to 0.11)	**<0.001*****
Fuel used for cooking (electricity/LPG/natural gas/biogas)						
Kerosene	—	—	0.83 (0.59 to 1.07)	**<0.001*****	0.84 (0.61 to 1.08)	**<0.001*****
Indoor smoking policy of home (smoke-free home)						
Smoke-permitted home	—	—	—	—	0.16 (0.06 to 0.25)	**0.001****
Total observations	1746		1746		1746	
F (probability)	22.7 (**<0.001*****)		15.4 (**<0.001*****)		15.1 (**<0.001*****)	
Adjusted R^2	0.047		0.083		0.089	

P value significance level: 10% (*), 5% (**), 1% (***).
LPG, liquefied petroleum gas; $PM_{2.5}$, particulate matter.

According to this model, presence of this 'opportunity' can potentially influence the 'motivational' behaviour for keeping the home smoke free.

We found that use of kerosene as a cooking fuel was a strong predictors of indoor $PM_{2.5}$ concentrations. Other studies in LMICs have also found a similar association. For instance, in India, the $PM_{2.5}$ value was doubled among kerosene users compared with LPG users (mean 109, SD 14 µg/m^3 vs 57, 7 µg/m^3), and in Nepal the $PM_{2.5}$ value was increased about 146% (95% CI 103%–200%) in kerosene users compared with households that used electricity as a cooking fuel.[31 32]

Our study also found that the daily household $PM_{2.5}$ concentrations decreased with an increase of each education year of the household lead participant. This is consistent with the literature that the education level of the household head is an important factor for keeping the household SHS free. For instance, a study in Greece[33] showed that SHS exposure significantly declined with increase of education level. Furthermore, one study among women in Bangladesh showed the same association between SHS exposure and education level[34] as our study; however, another study in China[35] did not show any association.

Our regression results show a low adjusted R^2 value (0.039–0.081 and 0.047–0.089), which explains that there are many possible explanatory variables that should be considered, given that different behavioural effects and lack of outdoor air pollution data are involved, as discussed earlier.

A likely potential driver for reductions in smoking prevalence in HICs is the implementation of smoke-free legislation in restaurants, bars and other public, indoor premises.[9] However, in many LMICs such legislation is often poorly implemented or enforced[36] and the social norms in the majority of homes in LMICs therefore still permit smoking indoors. Importantly, many smokers are unable to smell tobacco smoke, which makes them unaware of the level of tobacco-related exposure.[37] Considering these, an objective measurement is required to measure the concentration of SHS and to promote a smoke-free environment.[19 38] In this study we measured the concentration of indoor SHS ($PM_{2.5}$) and found that $PM_{2.5}$ is generally higher in SPH compared with SFH. Although this study showed the feasibility of implementing a large-scale indoor air quality study in LMIC settings, the small difference of $PM_{2.5}$ between SPH and SFP indicates to use $PM_{2.5}$ as a marker of SHS in such settings is challenging and would require confirmation through further studies in similar context. There are considerable practical challenges using optical particle counters such as the Dylos to measure exposure to SHS in LMIC settings.[39] Temperature and humidity can influence the measurement of $PM_{2.5}$ using these devices, though the effect is likely to have been systematic across both types of households given that we measured during the months when humidity is relatively stable across a 24-hour average.[40] Background or ambient $PM_{2.5}$ concentrations can also make it difficult to detect the additional $PM_{2.5}$ generated by smoking indoors, particularly when smoking is only occasional. To address this we carried out our measurements during months when reference monitors from the US Embassy indicated that ambient $PM_{2.5}$ concentrations were lowest and most stable. Our use of 1-minute time resolved data also facilitated identification of the differences between SFH and SPH as demonstrated in our figure 1.

This study was conducted only in urban setting and due to movement restriction in highly secured areas, the higher-income population could not be included. However, this was a large scale study with a big sample population, therefore, most of the urban features were captured. These findings can be shared with different stakeholders and policymakers in Bangladesh and other LMICs where there are high concentrations of SHS indoors. These data may help develop preventive interventions to encourage household members to reduce smoking indoors. Additionally, more research is required to understand what type of interventions have the potential to be effective in changing the behaviour of those smokers who continue to smoke indoors at home in LMIC settings.

What this paper adds

- Indoor particulate matter ($PM_{2.5}$) concentration is a well-established marker of secondhand smoke (SHS) exposure and has been widely used as an important indicator for evaluating indoor smoke-free policies in high-income countries (HICs).
- Little is known of the association between $PM_{2.5}$ and SHS in low/middle-income countries (LMICs), where other variables such as high levels of ambient air pollution may cause distortion at a scale different from HICs.
- We found that in a typical LMIC urban setting, $PM_{2.5}$ concentration can still differentiate between homes that permit smoking from those that do not but the difference is significantly smaller compared with that observed in HICs.

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Contributors TF cooridinated the study data collection and management, prepared the manuscript, developed the analytical strategy, contributed to the statistical analysis and interpretation of the results, and wrote the first draft of the report. KS and RH conceptualised the study. SS led the design of obtaining $PM_{2.5}$ measurements. CF, RD and SMA contributed to statistical analysis and interpretation of the results. NM and A-MM contributed to the interpretation of results and revision of the report. All authors participated in manuscript revisions, and read and approved the final manuscript.

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Patient consent for publication Not required.

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Data availability statement Data are available upon reasonable request. The data used in this study are the baseline data of a cluster randomised controlled trial (cRCT) study. The protocol manuscript link is given below https://trialsjournal.biomedcentral.com/articles/10.1186/s13063-018-3100-y. The final analysis of the post-intervention data is ongoing. Data could be made available upon reasonable request after completion of all intended publication of this trial. It would be best to contact the trial chief PI (Siddiqi, Kamran, email: kamran.siddiqi@york.ac.ukORCiD ID: 0000-0003-1529-7778) for further queries.

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