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Domestic airborne fine particulate matter exposure and asthma control among children receiving inhaled steroid treatment

K. E. Woods¹, A. Apsley², S. Semple² and S. W. Turner¹

Abstract

The aims of the present study were to measure domestic PM_{2.5} exposure and explore relationships with indices of asthma severity and control in children prescribed inhaled corticosteroids (ICSs). Disease severity was determined by questionnaire and spirometry. Asthma control was assessed by 5-day peak flow variability (PFV) and children's asthma control test (CACT) on the 1st and 5th day of peak flow testing. Concentrations of PM_{2.5} were measured over a 24-h period. Twenty-two children were recruited, mean age 11.0 years. Across the 22 homes the median time weighted average (TWA) PM_{2.5} concentration (range) was 7.4 µg/m³ (2.0–150.0) and was significantly higher in the seven homes where smoking was reported (24.0 µg/m³) than non-smoking homes (6.0 µg/m³), $p=0.001$. There was a positive association between TWA PM_{2.5} and PFV ($\rho=0.55$, $p=0.015$, $n=19$) and a negative association between TWA PM_{2.5} and CACT ($\rho=-0.56$, $p=0.010$, $n=20$). TWA PM_{2.5} exposure was not related to indices of asthma severity. Peak PM_{2.5} concentration was not associated with any outcome. This exploratory study suggests that even at relatively low concentrations, there is an exposure–response relationship between increasing indoor air PM_{2.5} concentrations and poorer asthma control in children prescribed ICSs.

Keywords

Air pollution indoor, Asthma, Child, Respiratory function testing

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Introduction

Asthma is a common childhood respiratory condition where symptoms can deteriorate in association with changes in air quality. Increases in outdoor air concentrations of fine particulate matter, with a diameter of less than 2.5 µm (PM_{2.5}), have been associated with increased asthma admissions,¹ increased need for medication to relieve acute asthma symptoms,² worsening lung function³ and indirect evidence of increased allergic airway inflammation.^{2,4,5}

Exposure to outdoor air PM_{2.5} is clearly important to childhood asthma but this age group spends approximately 90% of time life indoors⁶ and therefore indoor air PM_{2.5} exposures are likely more relevant to asthma symptoms. Results from outdoor air studies cannot be necessarily extrapolated to those of indoor air and there is evidence that outdoor PM_{2.5} have a stronger influence on biomarkers for asthma than indoor PM_{2.5}.^{5,7} There is

also evidence that indoor air PM_{2.5} concentrations can be higher than outdoors^{8–10} most likely reflecting indoor activities such as cooking,^{8,11} sweeping^{8,11} and smoking.^{11,12} A number of studies have related indoor PM_{2.5} concentrations to asthma outcomes in children and provide evidence of association which may be modified by treatment with inhaled corticosteroids (ICSs).^{5,7,10,13} Two studies, where median indoor air PM_{2.5} concentrations were relatively low (9.5⁵ and 7.6¹³ µg/m³), found ICS treatment apparently protects

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against any harmful effect of PM_{2.5} exposure in children with asthma.^{5,13} In contrast, one study of children treated with ICS who were exposed to median PM_{2.5} concentration of 15.3 µg/m³ reported a positive association between PM_{2.5} exposure and adverse asthma outcomes.⁷

The question as to whether ICS treatment protects children from the possible harmful effects of PM_{2.5} exposure is important to management of the relationship between environmental exposures and asthma symptoms. In the presence of uncertainty in a small literature, this exploratory study was designed to test the hypothesis that among children with asthma treated with ICS, there is a positive relationship between indoor air PM_{2.5} concentration and indices of asthma severity and asthma control.

Materials

PM_{2.5} measurements

The DustTrak (Model 8520, manufacturer TSI Inc, Shoreview, MN, USA) was used to record the concentration of PM_{2.5} in the main living room and bedroom over 24 h. The living room was defined as the room where the family spent most of their time through the day. A conversion factor of 0.295 was applied to account for the difference in density of generally combustion derived PM_{2.5} compared to the Arizona Road Test Dust standard used for calibration of the DustTrak. The monitors were zero calibrated and set to sample at 1.7 l/min using a Drycal flowmeter before each measuring period in accordance with manufacturer's specifications. Measurements were logged every 1 min over the 24-h sampling period. The measurements of PM_{2.5} of interest were expressed as (i) the time weighted average (TWA), (ii) the maximum concentration and (iii) the proportion of time when the concentration was over the threshold of 35 µg/m³, the concentration at that time which was deemed to be 'unhealthy' for outdoor air PM_{2.5} by the US Environmental Protection Agency (US EPA).¹⁴

Methods

Study design

Children with asthma and prescribed ICS were recruited and PM_{2.5} concentrations measured over a 24-h period in the living room and child's bedroom. Parents answered a home environment questionnaire and provided information on their child's current asthma medication and past medical history. Immediately before PM_{2.5} measurements commenced, the child's height and spirometry were measured and the Children's Asthma Control TestTM (CACT) was

completed.¹⁵ Peak flow variability (PFV) was measured over 5 days, and PM_{2.5} concentrations were measured for the first of these days. In recognition of the time lag between PM_{2.5} exposure and development of asthma symptoms,^{7,13,16} the CACT was also completed at the end of the 5-day PFV measurements. This study was approved by the North of Scotland Research Ethics Committees and the study took place between January and April 2009.

Study subjects

Children with a diagnosis of asthma and treated with ICS were identified from a hospital attendance database. Eligible children were aged between 5 and 16 years old and lived within Aberdeen city or the surrounding area. The age, gender and postcode of all children invited to participate were recorded. The Scottish Index of Multiple Deprivation (SIMD) was derived from the child's postcode. SIMD is a standard index of deprivation using 38 indicators across seven domains (income, employment, health, education, skills and training, housing, geographic access and crime) and expressed as quintiles where a higher score indicates greater affluence.

Pulmonary function testing

A hand-held pneumotachograph (21/20, Vitalograph, Bucks, UK) was used to measure spirometry in accordance with international recommendations¹⁷; forced expiratory volume in 1 s (FEV₁) was the principal outcome. Bronchodilator response was not measured. Peak expiratory flow measurements were made at home using a Wright peak flow meter (Clement Clarke, Essex, UK); the highest of three measurements was recorded every morning and evening for 5 days from when PM_{2.5} measurements commenced and PFV was derived using the following formula¹⁸

$$PFV = \frac{PEF_{\max} - PEF_{\min}}{0.5(PEF_{\max} + PEF_{\min})} \times 100$$

Statistical analysis

The relationship between PM_{2.5} concentrations and exposures to tobacco smoke, mould and pets was examined using Mann-Whitney U test. Relationships between PM_{2.5} concentrations and asthma outcomes were studied using Spearman's rank test, and the nature of significant associations was explored by fitting data to linear and logarithmic models. Standard statistical software was used (SPSS version 17.0.0) and significance was assumed where $p < 0.05$.

Results

Study subjects

There were 48 children invited to participate of which 22 consented. Table 1 demonstrates that there were no differences in age, gender and SIMD of the child's home postcode between the 48 invited to take part and those who participated. The mean age of participants was 11.0 years (range 5.3, 15.6) and 13 were boys. Asthma control and PFV were determined in 20 children and % predicted FEV₁ in 19. Living room PM_{2.5} concentrations were lost due to equipment failure in one household. At the time of testing, ICS treatment had been discontinued in three cases. Participant's asthma outcomes and domestic exposures are presented in Table 1.

Relationship between PM_{2.5} concentration and asthma outcomes

The median (interquartile range, IQR) TWA PM_{2.5} concentrations in the living room and bedroom were 7.4 µg/m³ (4.9, 11.5) and 5.6 µg/m³ (3.1, 11.1), respectively (Table 2). Peak PM_{2.5} concentrations and percentage time when concentrations exceeded 35 µg/m³ in the living room and bedroom are presented in Table 2. The TWA PM_{2.5} concentration in the living room was

positively correlated with PFV ($\rho = 0.55$, $p = 0.015$, $n = 19$), as shown in Figure 1, and negatively correlated with CACT on day 1 of peak flow measurements ($\rho = -0.56$, $p = 0.010$, $n = 20$) and also day 5 ($\rho = -0.60$, $p = 0.005$, $n = 20$), shown in Figure 2. Among the 15 non-smoking households, there were inverse relationships between TWA PM_{2.5} concentration in the living room and CACT on day 1 ($\rho = -0.73$, $p = 0.003$) and day 5 ($\rho = -0.68$, $p = 0.008$) but not with PFV. There was a trend for a positive relationship between percentage of time PM_{2.5} exceeded 35 µg/m³ in the living room and PFV ($\rho = 0.44$, $p = 0.053$, $n = 19$). There were no correlations between peak PM_{2.5} concentrations in living room and asthma outcomes, nor between any index of bedroom PM_{2.5} exposure and any asthma outcome. There were no associations between TWA PM_{2.5} concentrations in living room or bedroom and British Thoracic Society (BTS) treatment step, % predicted FEV₁ and number of hospitalisations.

Factors associated with particulate concentrations

Concentrations of TWA PM_{2.5} and the percentage of time where concentrations exceeded 35 µg/m³ were greater in the living room compared to bedroom

Table 1. Details of participants, their asthma outcomes and environmental exposures.

	Participants n = 22	All individuals invited to participate ^a n = 48
Mean age (SD), years	11.0 (3.3)	11.2 (3.4)
Male gender (%)	13/22 (59%)	33/48 (69%)
Median Scottish Index of Multiple Deprivation quintile (range)	3 (1–5)	2 (1–5)
Atopic (%)	17/22 (77%)	
British Thoracic Society Treatment, Step 1	3 (14%)	
Step 2	6 (27%)	
Step 3	11 (50%)	
Step 4	1 (4.5%)	
Step 5	1 (4.5%)	
Mean % predicted FEV ₁ (SD)	80% (19)	
Median CACT score, day 1 (IQR)	21 (18, 23.5)	
Median number of hospitalisations in previous year (IQR)	1 (0, 5)	
Mean % peak flow variability (SD)	27% (15)	
Type of accommodation, House	15 (68%)	
Flat	5 (23%)	
Bungalow	2 (9%)	
Number of smokers at home, None	15 (68%)	
One	4 (18%)	
Two	3 (14%)	
Visible damp at home (%)	7/22 (32%)	
Cat or dog at home (%)	8/22 (36%)	

^aThere were no differences between those invited to take part and those who participated.

(Table 2), Wilcoxon’s test $p=0.044$ and 0.010 , respectively. Concentrations of $PM_{2.5}$ did not exceed $35\mu g/m^3$ in the living room in 11 households and child’s bedroom in 12 households. The median (standard error of mean, SEM) bedroom TWA $PM_{2.5}$ concentrations were $11.0\mu g/m^3$ (14.2) in flats and $4.3\mu g/m^3$ (2.5) in houses, $p=0.014$; corresponding figures for living rooms were $11.0\mu g/m^3$ (27.9) and 6.0 (5.7) $\mu g/m^3$, $p=0.173$. The relationship between exposures to tobacco smoke, mould and pet and living room and bedroom TWA $PM_{2.5}$ concentration is presented in Tables 3 and 4, respectively. Living room and bedroom TWA $PM_{2.5}$ concentrations were higher in households where at least one parent smoked ($p=0.001$ and $p=0.039$) respectively. There was no association

between living in a flat and smoke exposure. There was no difference in living room or bedroom TWA $PM_{2.5}$ concentrations in households where mould was and was not visible and in households where cats and/or dogs were and were not present.

Discussion

This study was designed to better understand the relationship between indoor air $PM_{2.5}$ exposure and asthma symptoms in children treated with ICS. The novel finding was that study reported associations between increasing indoor air $PM_{2.5}$ concentrations and adverse asthma outcomes in children treated with ICS but at lower $PM_{2.5}$ concentrations than

Table 2. Median and interquartile ranges (IQR) of fine particulate ($PM_{2.5}$) exposures in living room and children’s bedrooms.

	Living room n = 21	Bedroom n = 22	p value ^a
Median TWA $PM_{2.5}$ concentrations ($\mu g/m^3$) (interquartile range, IQR)	7.4 [4.9, 11.5]	5.6 [3.1, 11.1]	0.044
Median of peak $PM_{2.5}$ concentrations ($\mu g/m^3$) (IQR)	37.7 [17.5, 110.6]	55.8 [17.5, 74.6]	0.614
Median % total time where $PM_{2.5}$ concentration exceeded $35\mu g/m^3$ (IQR), %	0.05 [0, 5.6]	0 [0, 0.5]	0.010

^aWilcoxon signed ranks test.

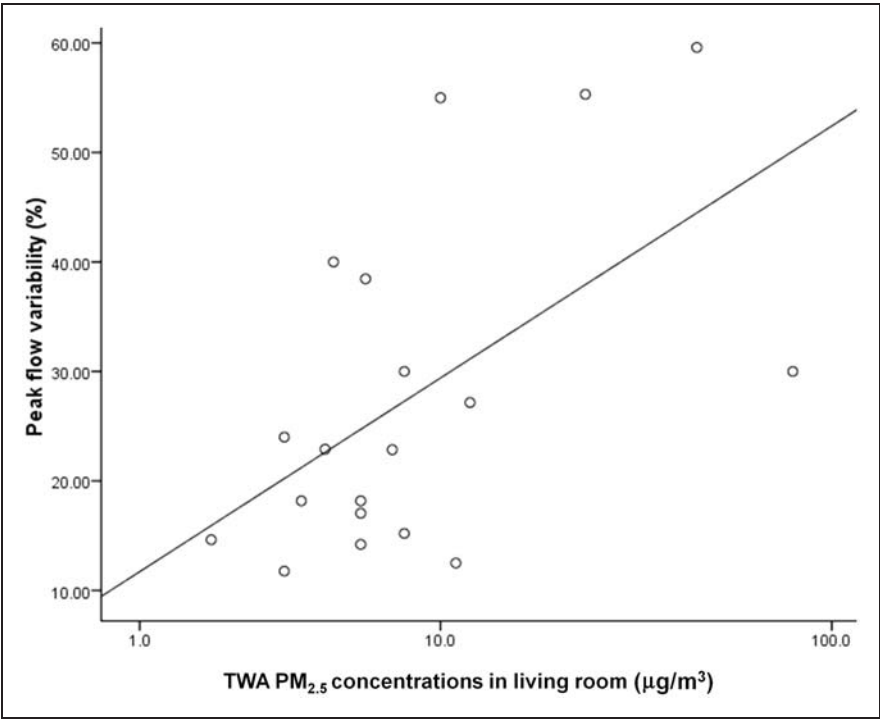


Figure 1. Scatter plot comparing median fine particulate concentrations ($PM_{2.5}$) in the living room over 24 h and peak flow variability over 5 days. $n = 19$, Spearman’s $\rho = 0.55$, $p = 0.015$.

previously reported (7.4 versus $15.3 \mu\text{g}/\text{m}^3$).⁷ Additionally we have described how $\text{PM}_{2.5}$ concentrations differed between bedroom and lounge and we confirm that concentrations are increased in smoking households. Our results need replication in larger

populations but the evidence presented here indicates that even 'low' indoor air $\text{PM}_{2.5}$ exposures, where there is no reported smoking taking place, have significant implications for asthma outcomes in children despite treatment with ICS.

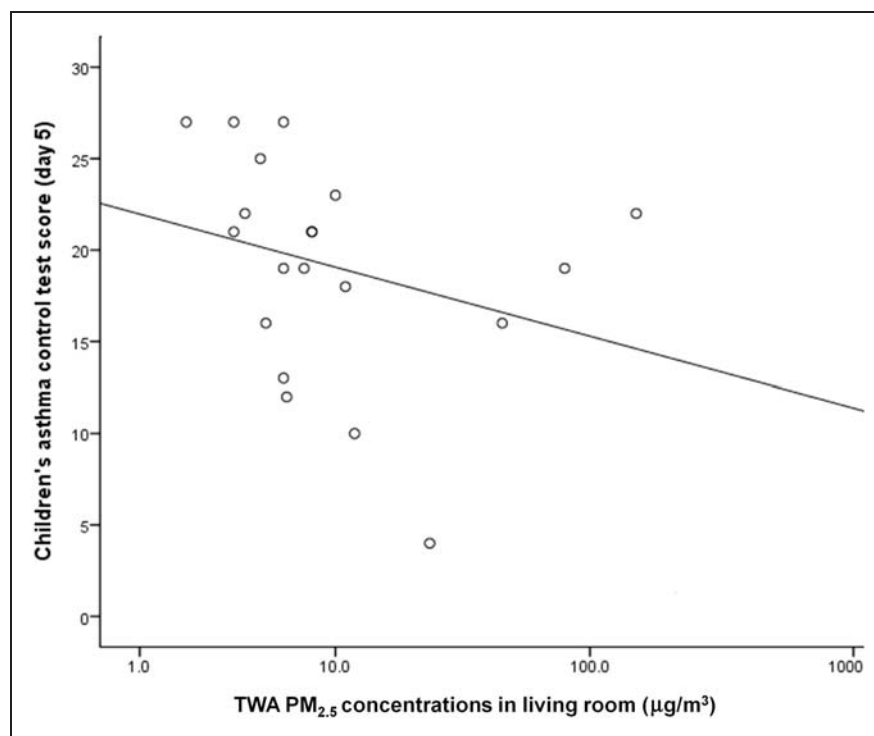


Figure 2. Scatter plot comparing median fine particulate concentrations ($\text{PM}_{2.5}$) in the living room over 24 h and the Children's Asthma Control Test score 5 days after $\text{PM}_{2.5}$ measurements commenced. $n = 20$, Spearman's $\rho = -0.60$, $p = 0.005$.

Table 3. Median living room fine particulate concentrations ($\text{PM}_{2.5}$) in households where exposure did and did not occur to smoking, visible mould and pets.

TWA living room, $\mu\text{g}/\text{m}^3$ (SEM)	Exposed	Not exposed	p value ^a
Median living room $\text{PM}_{2.5}$ concentration in smoking households	24.0 (19.9) $n = 7$	6.0 (0.7) $n = 14$	0.001
Median living room $\text{PM}_{2.5}$ concentration in households with visible mould	9.6 (23.4) $n = 6$	6.2 (5.5) $n = 15$	0.205
Median living room $\text{PM}_{2.5}$ concentration in households with cat and/or dog	7.8 (17.9) $n = 8$	6.2 (5.7) $n = 13$	0.645

^aMann-Whitney U test.

Table 4. Median bedroom fine particulate concentrations ($\text{PM}_{2.5}$) in households where exposure did and did not occur to smoking, visible mould and pets.

TWA bedroom, $\mu\text{g}/\text{m}^3$ (SEM)	Exposed	Not exposed	p value ^a
Median bedroom $\text{PM}_{2.5}$ concentration in smoking households	20.0 (10.4) $n = 7$	5.0 (0.8) $n = 15$	0.039
Median bedroom $\text{PM}_{2.5}$ concentration in bedrooms with visible mould	23.5 (17.5) $n = 4$	5.1 (2.1) $n = 18$	0.098
Median bedroom $\text{PM}_{2.5}$ concentration in households with cat and/or dog	5.5 (9.2) $n = 8$	5.6 (3.4) $n = 14$	0.714

^aMann-Whitney U test.

Our results are consistent with those of a Japanese study which reports associations between increasing $PM_{2.5}$ concentrations and worsening peak flow measurements and increased wheezing among children recruited from the hospital setting and treated with ICS.⁷ Our results extend the findings of Ma et al.⁷ by replicating the findings but at a considerably lower exposure; exposures in the present study were approximately half those experienced by the Japanese children. A second study has also described a positive association between indoor air $PM_{2.5}$ concentrations (mean concentration $40 \mu\text{g}/\text{m}^3$) and asthma symptoms in pre-school children where details of ICS treatment were not presented.¹⁰ At $PM_{2.5}$ concentrations in the magnitude observed in the present report, two studies have found no evidence of association between $PM_{2.5}$ exposure and changes in biomarkers for asthma.^{5,13} One explanation for these apparently inconsistent results is that associations between $PM_{2.5}$ exposure and asthma outcomes are subsumed by ICS treatment but only at 'low' concentrations; the results from the present study do not support this hypothesis. The relationship between $PM_{2.5}$ exposure and asthma outcomes in children appears complex and better understanding is required to inform intervention studies aimed at improving asthma control by modifying $PM_{2.5}$ exposure. Since our study was undertaken, the US EPA has revised the primary annual $PM_{2.5}$ standard concentration to $12 \mu\text{g}/\text{m}^3$ (<http://www.epa.gov/pm/2012/decfs-standards.pdf>) but our small study suggests that this standard may permit exposures which may be disadvantageous for children with asthma.

The observation that $PM_{2.5}$ concentrations were associated with indices of asthma control but not severity is consistent with the dissociation seen between these two asthma outcomes.¹⁹ Asthma severity may be determined early in the development of the disease and there is evidence that reduced lung function, a key index of severity, is determined by factors acting shortly after birth²⁰ or even as early as 10 week's gestation.²¹ In a context where asthma severity is determined at an early stage of development and often persists over periods of many years,²² a poor relationship between $PM_{2.5}$ concentrations and asthma severity might be expected. In contrast, asthma control and $PM_{2.5}$ concentrations vary on a day-to-day basis and changes in the former are plausibly influenced, within a matter of days, by changes in the latter.

There are a number of factors which should be considered when interpreting these results. First, our household $PM_{2.5}$ data are only proxies for personal exposures. Children's personal exposure to $PM_{2.5}$ will be influenced by concentrations experienced at school and other settings. Second, although the number of children studied was comparable with many other

studies^{5,7,13} the number of participants was relatively small and these findings need to be replicated in a larger population. Third, a number of analyses were undertaken and it is possible that the significant relationships reported arose by chance; however, we believe this to be unlikely given the consistency between our findings and those elsewhere. Finally, peak flow measurements are inherently variable in children²³ and this is likely to create false negative results, but despite this we were able to detect a positive association between variability and $PM_{2.5}$.

The relationship between $PM_{2.5}$ exposure and childhood asthma is complex. Important questions include: what is a safe $PM_{2.5}$ concentration; are peak or mean exposures more harmful; what is the delay between exposure to increased $PM_{2.5}$ concentrations and exacerbation. The response to the first of these questions is that although there is no direct limit value for domestic air quality, the USA EPA¹⁴ previously suggested that outdoor air $PM_{2.5}$ concentrations over 24-h periods should not exceed $35 \mu\text{g}/\text{m}^3$, recently revising this down to $12 \mu\text{g}/\text{m}^3$, and the WHO outdoor air guidance value is $15 \mu\text{g}/\text{m}^3$. For the second question, peak and not average outdoor $PM_{2.5}$ exposures have been previously associated with asthma outcomes^{2,16}; other studies of indoor air $PM_{2.5}$ have not reported associations between peak concentrations and outcomes but limited analyses to mean concentrations^{5,7,10,13}; the issue of relevance of peak versus mean exposure for indoor air is unresolved although the present study found no association between peak exposures and outcomes. The nature of the temporal relationship between rising $PM_{2.5}$ and asthma outcomes is reported in several studies and the consensus is that the respiratory sequelae of rising $PM_{2.5}$ exposure are detectable after an interval of 1–3 days in children.^{7,13,16}

Conclusion

This small study finds that at relatively low indoor air $PM_{2.5}$ concentrations in mostly non-smoking households, asthma symptoms and pulmonary function are related to exposure in school-aged children treated with inhaled steroids. Given that there are more than one million children with asthma in the UK²⁴ and many more worldwide, the relationship between indoor air $PM_{2.5}$ exposure and asthma outcomes merits further urgent investigation.

Authors' contribution

ST wrote the first draft of the manuscript and undertook the analysis. All authors made meaningful contributions during the preparation of the manuscript.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Supplementary details

Data can be accessed by contacting s.w.turner@abdn.ac.uk.

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