

ENVIRONMENTAL EXPOSURE

Locally generated particulate pollution and respiratory symptoms in young children

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Background: Particulate matter $<10\ \mu\text{m}$ (PM_{10}) from fossil fuel combustion is associated with an increased prevalence of respiratory symptoms in children and adolescents. However, the effect of PM_{10} on respiratory symptoms in young children is unclear.

Methods: The association between primary PM_{10} (particles directly emitted from local sources) and the prevalence and incidence of respiratory symptoms was studied in a random sample cohort of 4400 Leicestershire children aged 1–5 years surveyed in 1998 and again in 2001. Annual exposure to primary PM_{10} was calculated for the home address using the Airviro dispersion model and adjusted odds ratios (ORs) and 95% confidence intervals were calculated for each $\mu\text{g}/\text{m}^3$ increase.

Results: Exposure to primary PM_{10} was associated with the prevalence of cough without a cold in both 1998 and 2001, with adjusted ORs of 1.21 (1.07 to 1.38) and 1.56 (1.32 to 1.84) respectively. For night time cough the ORs were 1.06 (0.94 to 1.19) and 1.25 (1.06 to 1.47), and for current wheeze 0.99 (0.88 to 1.12) and 1.28 (1.04 to 1.58), respectively. There was also an association between primary PM_{10} and new onset symptoms. The ORs for incident symptoms were 1.62 (1.31 to 2.00) for cough without a cold and 1.42 (1.02 to 1.97) for wheeze.

Conclusion: In young children there was a consistent association between locally generated primary PM_{10} and the prevalence and incidence of cough without a cold and the incidence of wheeze which was independent of potential confounders.

There is now a consensus that exposure to particulate matter from the combustion of fossil fuels with a 50% cut off aerodynamic diameter of $10\ \mu\text{m}$ (PM_{10}) exacerbates a range of respiratory conditions in children.^{1–2} Young children may be especially vulnerable to adverse effects of PM_{10} since they have a higher minute ventilation relative to lung size,¹ a higher prevalence of respiratory symptoms,³ and exhibit qualitative differences in lung growth.⁴ Indeed, associations between PM_{10} and respiratory symptoms have been observed in the few studies that have focused on young children. Braun-Fahrländer *et al*⁵ were the first to report an association between the 6 week average concentration of total suspended particulate matter and an increased incidence of coughing episodes in a panel study of preschool children. In a cohort study, Brauer *et al*⁶ estimated exposure in the home to $\text{PM}_{2.5}$ in children aged 2 years of age and found a positive but non-significant association with wheeze and dry cough at night. Using the same methodology, Gehring *et al*⁷ found no association between PM_{10} and parent reported wheeze in children aged 1 and 2 years. To date, no cohort study has examined the association between locally generated PM_{10} and respiratory symptoms over the whole preschool age range. The benefits of local initiatives aimed at reducing emissions (such as congestion charging) in this vulnerable group are therefore unclear.

PM_{10} may be categorised either by chemical composition, size (ultrafine to coarse), or origin, with the latter classifying “primary” PM_{10} as particles emitted directly from combustion sources and “secondary” PM_{10} as particles formed from the oxidation of sulphur and nitrogen dioxides in the atmosphere.⁸ In UK cities, direct primary emission of PM_{10} from local traffic is the most important determinant of variations in individual exposure.^{9–11} Indeed, the proximity of the home to main roads has been used as a marker of individual exposure to the complex mix of gases, volatile organic compounds, and particles emitted by traffic.^{12–15}

However, distance from the road does not take into account prevailing wind direction or differences in the mix and density of vehicles on main roads. In contrast, dispersion models calculate both the generation of primary PM_{10} from local sources and its dispersion into adjacent areas, adjusting for wind direction and other meteorological parameters.^{16–18} For example, Leicester City Council (UK) along with several other European cities has, since 1998, used dispersion modelling for traffic planning.^{19–20}

In this study we aimed to determine the association between locally generated primary PM_{10} , calculated using a dispersion model, and the prevalence and incidence of parent reported respiratory symptoms in young children. Respiratory symptom data from a cohort of preschool children surveyed in 1998 and again in 2001 were linked to modelled exposure to locally generated primary PM_{10} at their home addresses, and evidence for a dose-response relationship was sought after adjusting for a number of potential confounding factors.

METHODS

Study population

A cohort of 4400 children aged 1–5 years was recruited in 1998 from a random sample of the Leicestershire Health Authority Child Health Database. Parents or guardians were sent a respiratory symptom questionnaire in 1998 and again in 2001. On each occasion, two repeat mailings of non-responders were subsequently sent out at 6 week intervals. The study was approved by the Leicestershire Health Authority ethics committee. Data from a subgroup of children surveyed in 1998 have previously been used in a study of the changing prevalence of preschool wheeze.³

Exposure assessment

Exposure to locally generated primary PM_{10} was assessed using the Indic-Airviro dispersion model Version 2.2

(Swedish Meteorological and Hydrological Institute, Norrköping, Sweden). The Leicester City Council's pollution control group uses Airviro to calculate spatial variations in "total" PM₁₀.²⁰ To calculate annual "total" PM₁₀, the concentration of locally emitted "primary" PM₁₀ is first calculated for 50×50 m grids. A uniform concentration, representing "secondary" and "coarse" PM₁₀ imported from other counties (for example, 9.28 µg/m³ for 2001), is then added to the primary PM₁₀ output. In the present study the "primary" output of the model was used since it reflects the variation in PM₁₀ exposure within Leicestershire. The model does not use actual PM₁₀ measurements; rather, it models primary PM₁₀ emissions for roads using traffic flow data and then applies real time wind speed/direction to these data to calculate how these emissions are blown into neighbouring areas. For road emissions the model divides roads into >3500 stretches between main junctions.²⁰ Airviro calculates the concentration of primary PM₁₀ emitted from each road by drawing on a database of updated information on the type of vehicle journeys, average daily traffic flows, speeds, and vehicular mix. Dispersion of emissions is calculated using data of the actual meteorological conditions present at the time.²⁰ To calculate annual exposure of the home address to locally generated primary PM₁₀ (µg/m³), we entered the home coordinates (Address-point database, Ordnance Survey, Southampton, UK) into the model and obtained 8760 hourly data points for the relevant 50×50 metre grid. The 1998 output was further adjusted to take into account vehicle emission factors updated in 1999. Change of home address during the survey period was identified using the Leicestershire Health Authority Child Health Database which included both the date of move and the new address. Since the Airviro provided hourly concentrations, we could adjust for the date of the move. The model could not be used for the edges of Leicestershire since the number and type of cars on roads in neighbouring counties was not available. For longitudinal exposure assessment, the mean of the 1998 and 2001 exposures was used.

Questionnaire data

Three questions were chosen a priori to derive the primary outcome variables:

Table 1 Prevalence of selected characteristics of the study population surveyed in 1998 and in 2001

Variable	n*	Prevalence (%)
Age in 1998 survey (years)†		
1.0–1.99	1085	25
2.0–2.99	1099	25
3.0–3.99	1113	25
4.0–4.99	1102	25
Boys	2304	52
Girls	2095	48
Mother has asthma	554 (3203)	17
Coal heating in the home		
1998	203 (3410)	6
2001	199 (2735)	7
Smoking by household member in the home		
1998	1144 (3382)	34
2001	793 (2543)	31
Either parent continued education past 16 years of age	1986 (3012)	66

*Number of children (total of replies in each category).

†A total of 4400 children were selected for the survey in 1998. Other covariates examined were preterm birth, breast feeding, father with asthma, gas cooking, presence of pets, number of cigarettes smoked by mother, overcrowding, single parenthood, and diet.

- “Did your child usually have a cough apart from colds in the last 12 months?” (cough without a cold);²¹
- “In the last 12 months has your child had a dry cough at night, apart from a cough associated with a cold or a chest infection?” (night time cough);³
- “Has your child had wheezing or whistling in the chest in the last 12 months?” (current wheeze).³

Covariates were selected from the same questionnaire, either because they were considered to be risk factors of lower respiratory symptoms in children or because they could influence the association between respiratory symptoms and pollution exposure within the cohort.⁶ Since the spatial distribution of social deprivation and levels of air pollution are closely correlated in the UK,²² we decided a priori not to include a spatially associated measure of deprivation (such as the Townsend score) in the analysis, but adjusted instead for non-spatial individual measures of socioeconomic status including maternal and paternal education, overcrowding, and single parenthood.

Statistical analysis

The questionnaire data were double entered into EpiInfo software (Version 6.04b, US Centre for Disease Control and Prevention, Atlanta, GA, USA). Subsequent analyses were carried out using SAS Version 8.2 for Windows (SAS Institute, Cary, NC, USA) and S-plus Version 6.1 (Seattle, WA, USA). Binomial generalised linear models with the logistic link were used in the model examining the association between the primary response variables and local PM₁₀. Exposures were entered both as categorical and linear terms into the model and quadratic and cubic terms were also tested. Using likelihood ratio tests to compare the fit of these different models, none of the alternative models performed better than a linear model. Furthermore, as spatial correlation is a concern in this type of analysis, variograms were used to check both for the responses themselves and the residuals from the models for spatial correlation. Odds ratios (ORs) and 95% confidence intervals (CI) were calculated for each µg/m³ increase in local primary PM₁₀. Stratified models and interaction tests were used to assess if the effect of PM₁₀ was stronger in children not going to

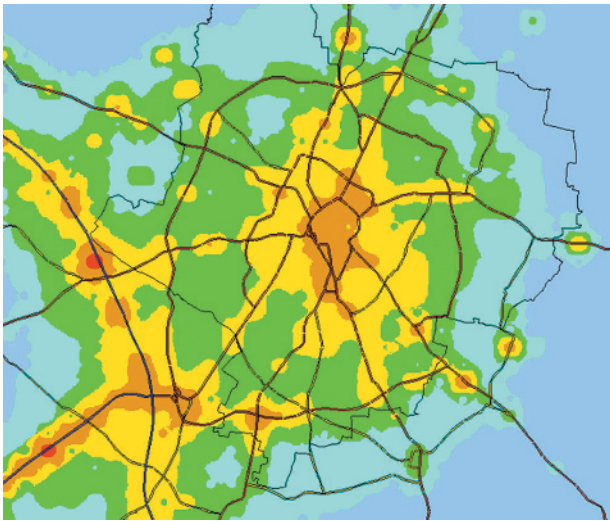


Figure 1 Map of annual mean total PM₁₀ for Leicester calculated using the Airviro dispersion model. The spectrum ranges from blue (low) to green, yellow, orange and red (high). These spatial differences are due to differences in locally generated primary PM₁₀. Areas of high exposure and heavily used roads. © Crown copyright Ordnance Survey, all rights reserved (NC/01/504).

nursery or daycare centres, and in children not exposed to environmental tobacco smoke.

RESULTS

The response rate from parents was 77.7% (3410/4400) in 1998 and 60.8% (2580/4245) in 2001. Between April 1997 and April 2001, 1265 children had moved address once, 438 twice, and 230 more than twice. The mean annual exposure to locally generated PM₁₀ was calculated for 3045 children whose parents responded in 1998, and for 2303 in 2001. Both surveys showed a high prevalence of parent reported respiratory symptoms: for 1998 and 2001, respectively, the prevalence of cough without a cold was 25% and 25%, night time cough 31% and 29%, and current wheeze 25% and 14%. The prevalence of selected characteristics of the study group is shown in table 1.

The output of Airviro confirmed that primary PM₁₀ was increased along local emission sources such as main roads (fig 1). Overall, the annual mean (25–75th percentile) primary PM₁₀ concentration for the cohort was 1.47 (0.73–1.93) µg/m³ in 1998 and 1.33 (0.8–1.84) µg/m³ in 2001.

After adjusting for confounders, exposure to locally generated PM₁₀ was associated with an increased prevalence of cough without a cold in both the 1998 and 2001 surveys (table 2), with evidence of a dose-response effect (fig 2). For prevalence of night time cough the ORs were slightly lower in both surveys. Current wheeze was not associated with PM₁₀ before adjusting for confounders (table 2). After adjustment there was a positive association in 2001 (table 2). The effect of PM₁₀ on health outcomes did not depend on whether or not children were exposed to environmental tobacco smoke or went to nursery care (all interaction tests with p>0.1).

We analysed the association between mean exposure to PM₁₀ from 1998 to 2001 and incident symptoms in children who were initially asymptomatic (table 3). There was a strong association between PM₁₀ and adjusted incident cough without a cold, and somewhat weaker associations with incident wheeze and incident night cough (table 3). Analysis by age did not show evidence for an effect modification, with adjusted ORs for cough without a cold of 1.51 (1.12 to 2.04) in children aged 1–2.99 years and 1.71 (1.26 to 2.31) in children aged 3–4.99 years. For night cough, the ORs for younger and older children were 1.24 (0.94 to 1.66) and 1.14 (0.83 to 1.55) respectively, and for wheeze 1.43 (0.91 to 2.26) and 1.39 (0.86 to 2.25). We found no association between PM₁₀ and persistence of symptoms in children who were symptomatic in 1998 (data not shown), but statistical power for this analysis was very low (numbers of children in the adjusted models for persistence of

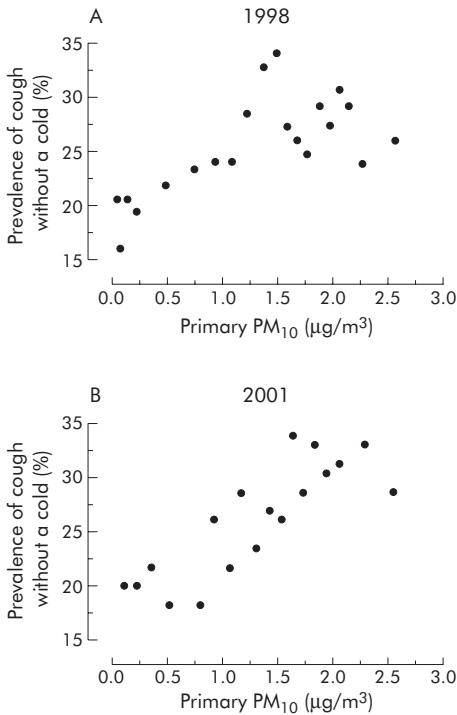


Figure 2 Scatterplots of the relationship between annual exposure to locally generated PM₁₀ at the home address expressed as µg/m³ and prevalence of cough without a cold surveys undertaken in (A) 1998 and (B) 2001 of a random stratified sample of 1–5 year old children in Leicestershire, UK. Each data point represents five centiles of unadjusted data sorted by exposure (n=128 in 1998 and n=115 in 2001).

symptoms were n = 406 for cough without a cold, n = 466 for night cough, and n = 221 for wheeze).

DISCUSSION

Using a dispersion model to estimate differences in exposure of homes of young children to locally generated primary PM₁₀, we found a strong association between exposure and the prevalence and incidence of cough without a cold and night cough which was independent of potential confounders. Furthermore, there was clear evidence for a dose-response relationship. The evidence for an association between primary PM₁₀ and the prevalence and incidence of current wheeze was less consistent. These data are compatible with a German cohort study which estimated PM exposure at the home address of children at 1 year of age and

Table 2 Association between mean annual exposure of the home address to locally generated primary PM₁₀ and prevalence of respiratory symptoms in young children

	Unadjusted			Adjusted*		
	OR†	95% CI	n‡	OR†	95% CI	n‡
Cough without a cold						
1998	1.22	1.10 to 1.36	2567	1.21	1.07 to 1.38	2164
2001	1.46	1.27 to 1.68	2301	1.56	1.32 to 1.84	1756
Night time cough						
1998	1.11	1.01 to 1.23	2579	1.06	0.94 to 1.19	2174
2001	1.25	1.09 to 1.43	2318	1.25	1.06 to 1.47	1771
Current wheeze						
1998	0.99	0.89 to 1.10	2584	0.99	0.88 to 1.12	2175
2001	1.09	0.93 to 1.30	2331	1.28	1.04 to 1.58	1774

OR, odds ratio; CI, confidence interval.
*Adjusted for confounding variables in table 1.
†Per µg/m³ increase in locally generated primary PM₁₀.
‡Number of responses.

Table 3 Association between exposure of the home address to locally generated primary PM₁₀ and incident cough and wheeze (defined as not present in the 1998 survey and present in the 2001 survey versus no symptoms in both surveys)

	Unadjusted			Adjusted*		
	OR†	95% CI	n‡	OR†	95% CI	n‡
Cough without a cold	1.68	1.39 to 2.03	1479	1.62	1.31 to 2.00	1287
Night time cough	1.21	1.00 to 1.46	1382	1.19	0.96 to 1.47	1191
Wheeze	1.22	0.92 to 1.62	1533	1.42	1.02 to 1.97	1319

OR, odds ratio; CI, confidence interval.
*Adjusted for confounding variables in table 1.
†Per µg/m³ increase in locally generated primary PM₁₀.
‡Number of responses.

reported ORs of 1.43 for cough without infection and 1.39 for dry cough at night for each 1.5 µg/m³ increase in PM_{2.5}.⁷ Our study extends these data by showing that the association with cough is present across the preschool age range. By 2001 some of the children in our cohort had reached school age. In this older age group the published evidence for an association between PM₁₀ and cough is conflicting. The 12 Community Southern California cohort study²³ found no association between PM₁₀ and cough. In contrast, Braun-Fahrlander *et al*²⁴ reported a strong significant association between PM₁₀ and both chronic and nocturnal dry cough in a cross sectional survey of 4470 Swiss children aged 6–15 years. Further surveys of the Leicester cohort should help to clarify whether this association continues throughout childhood.

Although we found that PM₁₀ was associated with the prevalence of cough without a cold, and there was a strong association with incident cough in children who were asymptomatic in the 1998 survey, we did not assess whether this type of cough affected the quality of life of children and their parents. However, preschool cough is not necessarily a trivial condition, as indicated by a recent study of Leicestershire general practitioners which reported that preschool children attending with “non-asthmatic” cough suffered significant sleep disruption and decreased activity levels.²⁵ Unfortunately, we did not have the information to quantify children with cough into groups of different severity.

The association between modelled PM₁₀ exposure and wheeze was inconsistent between surveys—that is, while there was no association with prevalent wheeze in 1998, there was evidence for an association with prevalent wheeze in 2001. Similarly, there is no consistency in the published studies on PM₁₀ and prevalence of wheezing disorders in children. On the one hand, Nicolai *et al*²⁶ reported an increased prevalence of current wheeze (adjusted OR 1.66) in children aged 9–11 years living within 50 metres of roads with high traffic flows, and Venn *et al*¹² found an increased prevalence of wheeze in a subgroup of a UK cohort of children aged 4–11 years living within 150 metres of a main road. On the other hand, other population based cohort studies have found no significant effect of PM₁₀ on current wheeze.^{6, 23, 24, 27} Indeed, the most recent study of young school age children²⁸ found no association between living near a main road and the prevalence of asthma. In preschool children, Edwards *et al*²⁹ reported that children admitted to hospital with asthma were more likely to live in areas of high traffic flow (compared with those admitted for non-respiratory reasons), and it is possible that parent reported wheeze is an imprecise descriptor of preschool asthma. Alternatively, modelled local primary PM₁₀ may not reflect the size or composition of particles that upregulate cellular mechanisms associated with wheeze. However, we did find an association between primary PM₁₀ and new onset (incident) wheeze in 2001, which supports the speculation

that early exposure to PM₁₀ may play a causal role in the development of asthma, especially in children with a genetic predisposition to attenuated antioxidant defences.³⁰ Further study of our cohort will be required to establish if new onset wheezing in the 2001 survey is atopic asthma, and if it is associated with mutations in the genes involved in the induction of pulmonary antioxidant defence.³⁰

There are important limitations to our study. Although PM₁₀ is a biologically plausible mediator of health effects,³¹ causation cannot be assumed. Fossil fuel particles do, however, penetrate into the airways of children. In a previous study we found aggregates of carbonaceous nanoparticles (<0.01 µm²) in alveolar macrophages from healthy infants and children living in Leicestershire.³² There is debate about the size fraction of PM₁₀ responsible for health effects, with speculation that nanoparticles are the most damaging.³¹ In European cities where traffic is the major source of PM₁₀, there is a close correlation between nanoparticle number and PM₁₀ concentration.³³ It is therefore likely that modelled primary PM₁₀ reflects exposure to traffic associated nanoparticles, but not necessary to larger “coarse” particles (PM_{2.5–10}) which are mostly derived from soil and sea salts.³⁰

A second limitation of the study is that there may be an unrecognised confounding variable with a high spatial correlation with traffic pollution, especially one associated with poor socioeconomic status. Indeed, compatible with UK data,²² we found a significant correlation between modelled exposure to primary PM₁₀ and Townsend score (*r* = 0.41 in 1998, and *r* = 0.45 in 2001, *p* < 0.0001). Thus, adjusting our data for a spatial measure of deprivation would have resulted in an underestimation of the effect of PM₁₀. For example, the association between PM₁₀ exposure and cough without a cold in 2001 fell from 1.56 to 1.42 (1.18 to 1.72) when the Townsend score was included as a confounding variable.

Thirdly, we did not estimate the effect of “imported” PM₁₀ blown into Leicestershire from other counties and countries. Imported particles may also affect respiratory health, but we could not detect this since concentrations would be close to uniform over the spatial area of the cohort over a 12 month period. Fourthly, although Airviro performs well in modelling the spatial distribution of traffic associated carbon monoxide¹⁶ which in turn is a valid marker for traffic associated PM₁₀,³⁴ we did not validate modelled data by direct measurement. Finally, any estimate of PM₁₀ at the home address can only approximate individual exposure. We did not record time-activity data, but had recorded whether children attended a nursery. The strength of association between PM₁₀ and the health outcomes did not differ whether or not the children went to nursery care. One explanation is that the total time spent in nursery per week is negligible compared with the time spent at or around home in this age group.

In summary, in a cohort of young children we found a consistent association between exposure to locally emitted

primary PM₁₀ and the prevalence and incidence of cough without a cold and night time cough, and incidence of wheeze. We conclude that a reduction in locally generated primary PM₁₀ may have significant health benefits in young children, and that linking paediatric cohort data to pollution dispersion models may help in planning local air quality initiatives.

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