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Locally-generated particulate pollution and respiratory symptoms in young children.

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Abstract

Background: Particulate matter <10μm (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: We aimed to define the association between primary PM_{10} (particles directly emitted from local sources) on prevalence and incidence of respiratory symptoms in a random sample cohort of 4,400 Leicestershire children aged 1 to 5 years who were 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 (OR) and 95% confidence intervals calculated for each $\mu g/m^3$ increase.

Results: Exposure to primary PM_{10} was associated with 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 to1.58) respectively. In addition, there was 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, we found a consistent association between locally-generated primary PM_{10} and prevalence and incidence of cough without a cold, and incidence of wheeze, which was independent of potential confounders.

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Introduction

There is now a consensus that exposure to particulate matter from the combustion of fossil fuels with a 50% cut-off aerodynamic diameter of 10μm (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 focussed on young children. First, Braun-Fahrländer *et al* [5] reported 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. Second, in a cohort study, Brauer *et al* [6] estimated exposure in the home to $PM_{2.5}$ in children aged 2 years of age, and found a positive, but non significant, association with wheeze and dry cough at night. Third, Gehring *et al* [7], using the same methodology, found no association between PM_{10} and parent-reported wheeze in children 1 and 2 years of age. 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 (e.g. congestion charging) in this potentially vulnerable group, are therefore unclear.

PM_{10} may be categorised by either chemical composition, or 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 emissions of PM_{10} from local traffic is 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 road does not take into account prevailing wind direction, or differences in the mix and density of vehicles on main roads. Recently, dispersion models have been developed that 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 cites 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 prevalence and incidence of parent-reported respiratory symptoms in young children. To achieve this aim, we linked respiratory symptom data from a cohort of preschool children surveyed in 1998 and again in 2001, to modelled exposure to locally-generated primary PM_{10} at their home addresses, and sought evidence for a dose-response relationship after adjusting for a number of potential confounding factors.

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Methods

Study population

A cohort of 4,400 children between 1 and 5 years of age was recruited in 1998 from a random sample of the Leicestershire Health Authority Child Health Database. Parents or guardians were sent respiratory symptom questionnaires in 1998 and in 2001. On each occasion, two repeat mailings of non-responders were subsequently sent out at six-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" PM_{10} [20]. To calculate annual "total" PM_{10}, the concentration of locally-emitted "primary" PM_{10} is first calculated for 50 x 50m grids. A uniform concentration, representing "secondary" and "coarse" PM_{10} imported from other counties (e.g. 9.28 $\mu g/m^3$ for 2001), is then added to the primary PM_{10} output. In this study, the "primary" output of the model was used, since it reflects the variation in PM_{10} exposure within Leicestershire. The model does not use actual PM_{10} measurements, rather it models primary PM_{10} emissions for roads using traffic flow data, 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 > 3,500 stretches between main junctions [20]. Airviro calculates the concentration of primary PM_{10} 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 PM_{10} ($\mu g/m^3$), we entered the home co-ordinates (Address-point database, Ordnance Survey, Southampton, UK) into the model, and obtained 8,760 hourly data points for the relevant 50 x 50m 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 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. These were: "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]; and “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] (table 1). Since the spatial

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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 (e.g. Townsend score) in the analysis, but adjusted instead for non-spatial individual measures of socio-economic status, including maternal and paternal education, overcrowding and single parenthood.

Statistical analysis.

Questionnaire data was 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, Wash, USA). Binomial Generalized Linear Models with the logistic link were used in the model examining the association between the primary response variables and local PM_{10}. Exposures were entered both as categorical and linear or terms into the model and we also tested quadratic and cubic terms. 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 per $\mu g/m^3$ increase in local primary PM_{10}. Stratified models and interaction tests were used to assess if the effect of PM_{10} was stronger in children not going to nursery or daycare, and in children not exposed to environmental tobacco smoke.

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Results

The response rate from parents in 1998 was 77.7% (3410/4400), and in 2001 was 60.8% (2580/4245). 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 PM_{10} 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 given in table 1. The output of Airviro confirmed that primary PM_{10} was increased along local emission sources such as main roads (figure 1). Overall, the annual mean (25th to 75th percentile) primary PM_{10} concentration for the cohort was 1.47 (0.73 to 1.93) $\mu g/m^3$ in 1998, and 1.33 (0.8 to 1.84) $\mu g/m^3$ in 2001.

After adjusting for confounders, exposure to locally-generated PM_{10} 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 (figure 2). For prevalence of night-time cough, the ORs were slightly lower in both surveys. Current wheeze was not associated with PM_{10} before adjusting for confounders (table 2). After adjustment, there was evidence for a positive association only in 2001 (table 2). The effect of PM_{10} on health outcomes did not depend 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 PM_{10} between 1998 and 2001, and incident symptoms in children who were initially asymptomatic (table 3). We found a strong association between PM_{10} 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 to 2.99 years, and 1.71 (1.26 to 2.31) in children aged 3 to 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 to1.55) respectively, and for wheeze 1.43 (0.91 to 2.26) and 1.39 (0.86 to 2.25). We found no association between PM_{10} 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 symptoms were: n=406 for cough without a cold, n=466 for night cough and n=221 for wheeze).

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Discussion

Using a dispersion model to estimate differences in exposure of the homes of young children to locally-generated primary PM_{10}, we found a strong association between exposure and 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 PM_{10} 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 for children at 1 year of age, and reported ORs of 1.43 for cough without infection, and 1.39 for dry cough at night for each 1.5 $\mu g/m^3$ increase in $PM_{2..5}$ [7]. Our study extends these data by showing that this association extends 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 PM_{10} and cough is conflicting. The 12 Community Southern Californian cohort study [23] found no association between PM_{10} and cough. In contrast, Braun-Fahrländer *et al* [24] reported a strong significant association between PM_{10} and both chronic and nocturnal dry cough in a cross-sectional survey of 4,470 Swiss children aged 6 to 15 years of age. Further surveys of the Leicester cohort should help to clarify whether this association continues throughout childhood.

Locally-generated primary PM_{10} was associated with prevalence of cough without a cold, and 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.

We found that the association between modelled PM_{10} exposure and wheeze was inconsistent between surveys: i.e. 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 PM_{10} and prevalence of wheezing disorders in children. On one hand, Nicolai *et al* [26] reported an increased prevalence of current wheeze (adjusted OR 1.66) in children aged 9 to 11 years living within 50m of roads with high traffic flows, and Venn *et al* [12] found an increased wheeze prevalence in a subgroup of a UK cohort of children aged 4 to 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 PM_{10} on current wheeze [6;23;24;27]. Indeed, the most recent study of young school-age children [28] 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 PM_{10} may not reflect the size or composition of particles that upregulate cellular mechanisms associated with wheeze. However, we did find an association between primary PM_{10} and new-onset (incident) wheeze in 2001, which supports the speculation that early exposure to PM_{10} 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

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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 defense [30].

There are important limitations to our study. Although PM_{10} 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 μm^2) in alveolar macrophages from healthy infants and children living in Leicestershire [32]. There is debate about the size fraction of PM_{10} responsible for heath effects, with speculation that nanoparticles are the most damaging [31]. In European cities where traffic is the major source of PM_{10}, there is a close correlation between nanoparticle number and PM_{10} concentration [33]. It is therefore likely that modelled primary PM_{10} 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 study limitation, is that there may be an unrecognised confounding variable with a high spatial correlation with traffic-pollution, especially one associated with poor socio-economic status. Indeed, compatible with UK data [22], we found a significant correlation between modelled exposure to primary PM_{10} 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 PM_{10}. Indeed, the association between PM_{10} exposure and cough without a cold in 2001 fell from 1.56 in 2001 to 1.42 (1.18 to1.72) when Townsend score was included as a confounding variable. Third, we did not estimate the effect of "imported" PM_{10} 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. Fourth, although Airviro performs well in modelling the spatial distribution of traffic-associated carbon monoxide [16], which in turn is a valid marker for traffic-associated PM_{10} [34], we did not compare the modeled data by direct measurement. Finally, any estimate of PM_{10} 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 PM_{10} 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 to 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 PM_{10} and 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 PM_{10} 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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Table 1: Prevalence of selected characteristics of the study population surveyed in 1998 and in 2001.

Variable	n*	Prevalence (%)
Age in 1998 survey[†]		
1.0 to1.99 yrs	1085	25
2.0 to 2.99 yrs	1099	25
3.0 to 3.99 yrs	1113	25
4.0 to 4.99 yrs	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 4,400 children were selected for the survey in 1998. Other co-variates 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.

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Table 2: The association between mean annual exposure of the home address to locally-generated primary PM_{10} 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 to1.36	2567	1.21	1.07 to1.38	2164
2001	1.46	1.27 to1.68	2301	1.56	1.32 to1.84	1756
Night-time cough						
1998	1.11	1.01 to1.23	2579	1.06	0.94 to1.19	2174
2001	1.25	1.09 to1.43	2318	1.25	1.06 to1.47	1771
Current wheeze						
1998	0.99	0.89 to1.10	2584	0.99	0.88 to1.12	2175
2001	1.09	0.93 to1.30	2331	1.28	1.04 to1.58	1774

OR, odds ratio; CI, confidence interval

*adjusting for confounding variables in table 1. [†] per $\mu g/m^3$ increase in locally-generated primary PM_{10}, [‡] number of responses

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Table 3: The association between exposure of the home address to locally-generated primary PM_{10} and incident cough and wheeze

	Unadjusted			Adjusted*		
	OR[†]	95% CI	n[‡]	OR[†]	95% CI	n[‡]
Cough without a cold	1.68	1.39 to2.03	1479	1.62	1.31 to 2.00	1287
Night-time cough	1.21	1.00 to1.46	1382	1.19	0.96 to1.47	1191
Wheeze	1.22	0.92 to1.62	1533	1.42	1.02 to1.97	1319

OR, odds ratio; CI, confidence interval. Incident symptoms are defined as those not present in the 1998 and present in 2001 *vs* no symptoms in both surveys.
*adjusting for confounding variables in table 1. [†] per $\mu g/m^3$ increase in locally-generated primary PM_{10}, [‡] number of responses

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Legend for figure 1

Map of annual mean total PM_{10} 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 PM_{10} Areas of high exposure track with heavily used roads. The black line represents the city boundary. The PM_{10} map is © Crown copyright Ordnance Survey, all rights reserved (NC/01/504).

Legend for figure 2

Scatterplots of the relationship between annual exposure to locally-generated PM_{10} at the home address expressed as $\mu g/m^3$, and prevalence of cough without a cold in two repeated surveys of a random stratified-sample of 1 to 5 year olds in Leicestershire (UK). Each data point (•) represents five centiles of unadjusted data sorted by exposure (n=128 in 1998, and n=115 in 2001).

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FIGURE 1

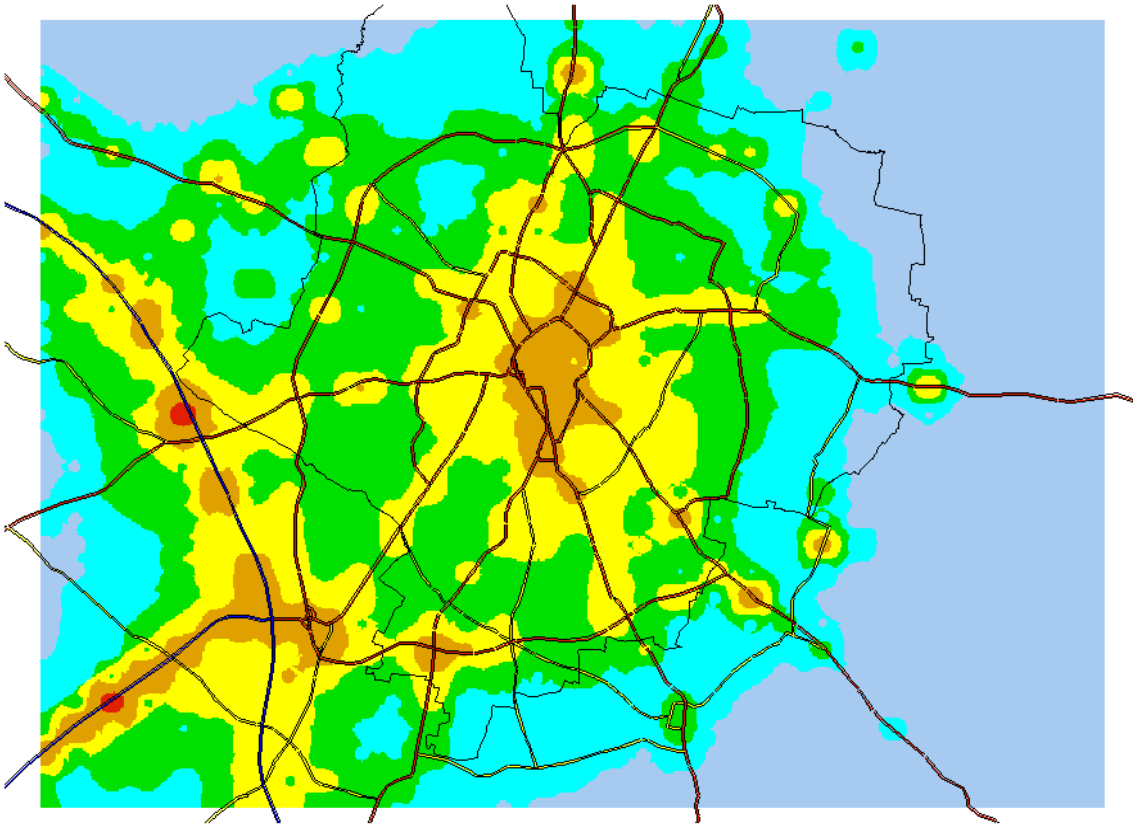

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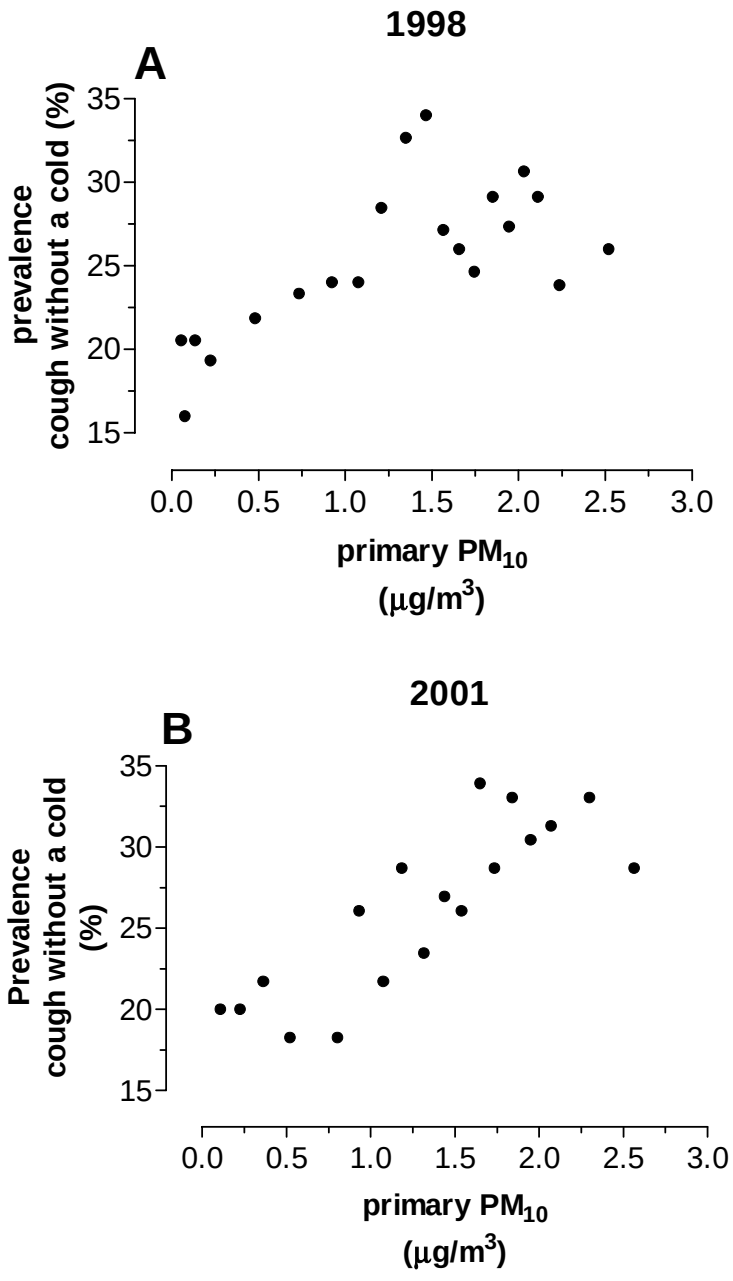
FIGURE 2



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