



Association between particulate matter concentration and symptoms of atopic dermatitis in children living in an industrial urban area of South Korea

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ABSTRACT

Introduction: Increased exposure to particulate matter (PM) appears to increase the development of atopic diseases and allergic sensitization. This study evaluated the association between daily levels of PM with diameters less than 10 μm (PM₁₀) and PM_{2.5} and symptoms of atopic dermatitis (AD) in children living in an industrial urban area.

Methods: Indoor PM₁₀ and PM_{2.5} concentrations were measured with an optical particle counter in two preschools near large industrial complexes in Ulsan, South Korea during two 6-month periods (May–October of 2012 and 2013). Twenty-one children with AD from these preschools were enrolled and observed daily for AD symptoms during the same periods. Indoor and outdoor PM concentrations were used to estimate PM exposure based on time activity patterns.

Results: Analysis of PM₁₀ and PM_{2.5} concentrations showed that indoor and outdoor PM₁₀ levels varied similarly throughout each 6-month period. In addition, indoor concentration of PM_{2.5} had high correlation with ambient outdoor concentration of PM₁₀. Correlation analysis also indicated a significant positive correlation between the exacerbation of AD symptoms and daily mean exposure to PM₁₀ and PM_{2.5}. Based on a generalized linear mixed model (GLMM), PM exposure was significantly associated with the exacerbation of AD symptoms, with a maximum adjusted odds ratio (aOR) of 1.399 for a 10 $\mu\text{g}/\text{m}^3$ increase of PM_{2.5} (95% CI: 1.216–1.610).

Discussion: Our findings suggest that short-term exposure to PM can exacerbate AD in young children living in an industrial urban area. PM_{2.5} had a stronger effect than PM₁₀ on exacerbation of AD symptoms.

1. Introduction

Particulate matter (PM) is an environmentally ubiquitous air pollutant that is a growing public health concern. Many epidemiological and toxicological studies have found associations between ambient PM concentrations and adverse health outcomes (Kappos et al., 2004; Lippmann et al., 2003; Pope, 2000; Schwarze et al., 2006). PM can increase allergic sensitization and exacerbate symptoms of asthma (De Haar et al., 2006) and may also increase the development and exacerbation of atopic dermatitis (AD), one of the most common allergic disorders in children. Although there are still controversies regarding the health effects of PM, it can clearly induce oxidative stress in the

skin, which leads to skin barrier dysfunction or immune dysregulation (Ahn, 2014). Several recent studies showed that exposure to PM can exacerbate the symptoms of AD in school children (Kim et al., 2013; Song et al., 2011; Morgenstern et al., 2008), but there is only limited direct evidence supporting this association. Further studies are needed to clarify the relationship between PM exposure and AD development, because AD is clearly associated with decreased quality-of-life.

There is growing concern in many countries about the effects of PM on AD in areas with high air pollution due to increases in urbanization and industrialization. High PM concentrations increase PM exposure, and this could increase health problems associated with PM, such as allergic diseases. Hence, it is necessary to investigate the effects of PM

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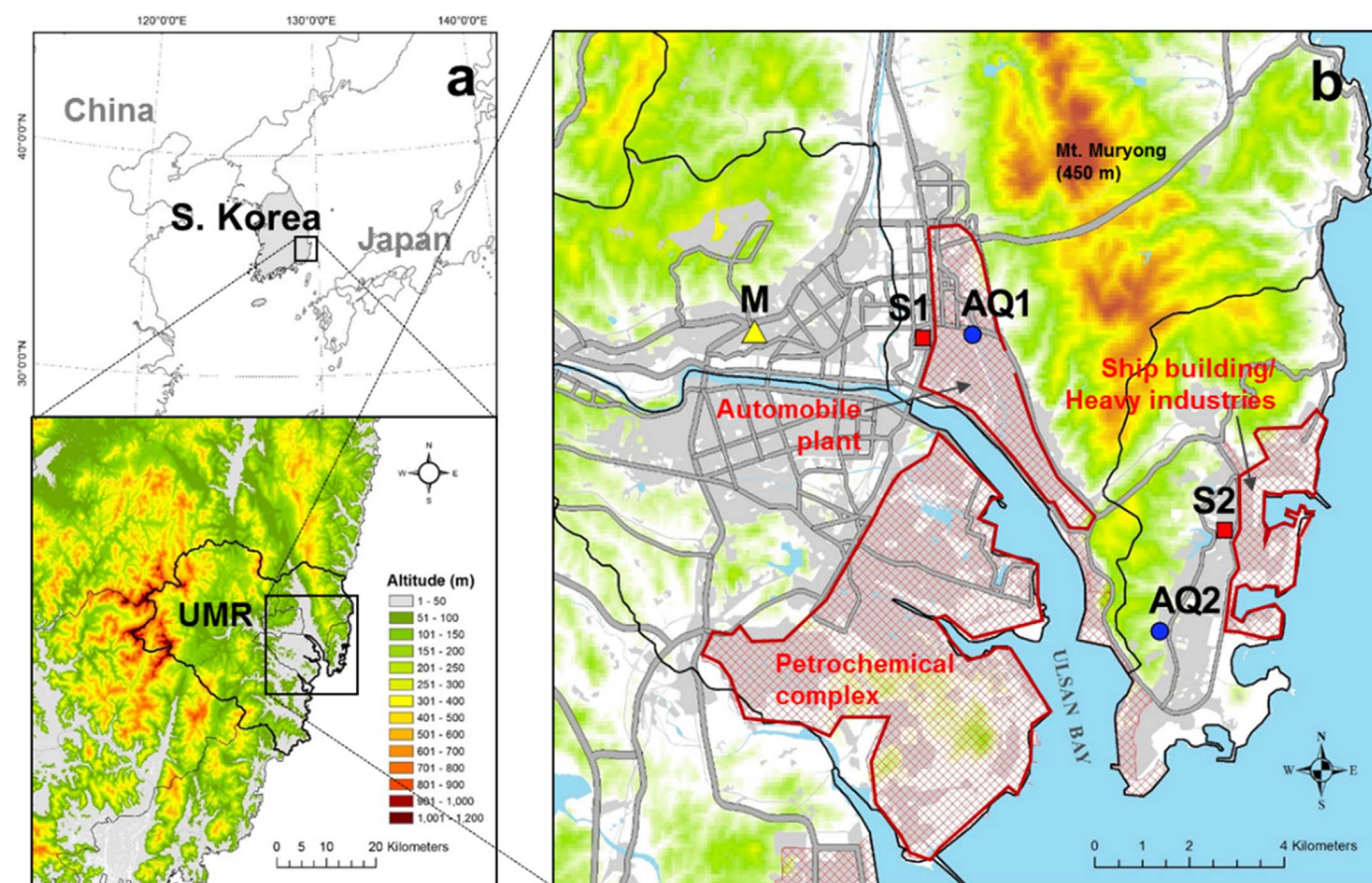


Fig. 1. Map showing (a) location of the UMR in South Korea and (b) urban environments and measurement sites: indoor measurement sites at two preschools (S1 and S2), air quality monitoring sites (AQ1 and AQ2) and meteorological sites (M). Red shaded regions and gray lines indicate industrial areas and main roads, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

on AD, especially in areas with high levels of PM. More specifically, identification of an association between short-term elevations of PM (which is common in many polluted cities) with symptoms of AD is needed to establish a better strategy for prevention and management of AD. However, few studies have examined the quantitative effects of short-term exposure to PM on AD in areas with high pollution, especially urban areas that are near large industrial complexes.

This study used long-term daily monitoring data to evaluate the association between PM exposure and AD symptoms in preschool children who live in industrial urban area of South Korea

2. Material and methods

2.1. Study area

The Ulsan metropolitan region (UMR) is located on the coast of southeastern Korea (Fig. 1). It has a population of 1.2 million, contains a central urban area with high traffic density, and large industrial complexes, including the world's largest automobile assembly plant and a petrochemical complex on the coast. Air pollution in the UMR is mainly due to emissions from these urban transportation and industrial facilities. We selected two preschools (S1 and S2, Fig. 1b) that are near these large industrial complexes for measurements of daily AD symptoms, PM concentrations, and their relationships. Several industrial plants, which are potential PM sources, are close to these preschools and residential areas, and these industrial emissions could have significant effects on indoor and outdoor PM pollution. Two air quality monitoring sites (AQ1 and AQ2, Fig. 1b) that are near the schools and operated by the Korea Ministry of Environment, were selected to obtain

ambient outdoor concentrations of PM with diameters less than $10\ \mu\text{m}$ (PM₁₀).

2.2. Indoor PM measurements

Indoor concentrations of PM₁₀ and PM_{2.5} were continuously measured with the Grimm Series 1.108 Aerosol Spectrometer (Grimm Tech., Inc., Douglasville, GA, USA), a portable optical particle counter, in two preschools (S1 and S2) from May to October of 2012 and 2013, respectively. The spectrometer sensitivity is 1 particle/L or $1\ \mu\text{g}/\text{m}^3$, and instrument reproducibility is $\pm 2\%$. Ambient air is drawn into the unit via an internal volume-controlled pump at a rate of 1.2 L/min. Previous publications provided detailed descriptions of this instrument (Cheng and Lin, 2010; Labortechnik Ltd., 1996). Monitoring was performed inside the classroom on the first floor of each building. The instrument was placed 1.5 m above the floor, and more than 2 m from doors and windows. The logging interval was set as 1 min. All children performed their normal activities and most of windows of the classroom normally remained closed during the entire sampling period. Several windows in each preschool were typically opened for ventilation and doors were frequently opened by entry and exit of persons. The indoor monitoring was not available for a specific period including summer vacation (19% and 11% of 6 months for S1 and S2, respectively).

2.3. Exposure assessment

Individual's total exposure to PM were estimated using indoor and outdoor PM concentrations based on time activities. The daily PM exposure estimates were from the average of indoor concentrations in the

classroom during school hours (9 a.m. to 5 p.m.) and ambient outdoor concentrations at two air quality monitoring sites (AQ1 and AQ2, Fig. 1b) during non-school hours. The AQ1 and AQ2 are operated by the Ministry of Environment (MOE) of Korea and PM10 data were obtained by measurements in every 1-h period for 24 h.

For the estimation of PM2.5 exposure, we used scaled PM10 concentrations at AQ1 and AQ2 based on the average ratio of PM2.5/PM10 of 0.54, which was derived from the monthly average air quality data in Ulsan from National Institute of Environmental Research (NIER, 2015), because there were no PM2.5 measurements at both AQ1 and AQ2 sites.

The ambient PM10 concentrations at AQ1 and AQ2 were measured by β -ray absorption method and were periodically calibrated by reference values generated by gravimetric method. We used the PM10 data finally authorized by National Ambient air quality Monitoring Information System (NAMIS). All study children live within several kilometers of their preschools, and they spend similar times outdoors, so the ambient concentrations at AQ1 and AQ2 reflect individual exposure to PM at each residential environment.

2.4. Symptom diary and AD definition

Twenty-one children (11 boys and 10 girls; 10 at S1 and 11 at S2) with confirmed AD, who were aged 1–5 years, were monitored for daily AD symptoms during the same time as the PM measurements. The presence of AD was confirmed by qualified pediatricians, who examined the neck and the flexural areas of both arms. Parents were instructed to record a symptom diary that describes the extent of itching and sleep disturbance, erythema, dryness, oozing, and edema on a scale of 0–4. The presence of AD was defined as a score of 2 or more on a subjective symptom scale for itching and sleep disturbance, with at least one objective symptom (erythema, dryness, edema, or oozing) on the same day. This definition of AD is similar to that of Kim et al. (2013), although they used stricter criteria for the subjective symptom scale.

2.5. Statistical analysis

The relationship between the indoor and outdoor datasets was evaluated using Pearson's correlation coefficient (r) for each preschool. The t -test was used to compare the distributions of PM concentrations experienced by children with and without AD symptoms. The effects of PM on AD symptoms were evaluated using odds ratios (ORs), calculated by logistic regression. To estimate the overall effect for all subjects, we adopted a generalized linear mixed model (GLMM) with binomially distributed errors, considering the repeated assessment of AD symptoms of each subject. For this model, we treated PM10 and PM2.5 as fixed effects, and each participant as a random effect.

The covariates in our analysis were age, sex, meteorological factors (air temperature and relative humidity), Scoring Atopic Dermatitis (SCORAD) index at the initial visit, and use of topical corticosteroids. We assessed the relationship of AD with these risk factors using a multivariable model, which only included variables that were significant in the univariate analysis. Sex was treated as an *a priori* confounder, and included in the final models regardless of significance. Basic statistical analyses were conducted using the SPSS version 18.0. The GLMM analysis was performed using the “lme4” package (version 3.1.2) of R version 3.0.0 (The Comprehensive R Archive Network, <http://cran.r-project.org>).

3. Results

3.1. Indoor and outdoor PM concentrations

We evaluated distributions of indoor and outdoor PM concentrations during the same periods in which we measured daily AD symptoms. Table 1 shows the monthly average concentrations of indoor

Table 1

Monthly distributions of indoor and ambient outdoor PM concentrations at two preschools (S1 and S2).

Preschool	Month	PM10 and PM2.5 ($\mu\text{g}/\text{m}^3 \pm \text{S.D.}$)			I/O ratio (PM10)
		Indoor PM10	Indoor PM2.5	Outdoor PM10 ^a	
S1	2012.5	36.6 $\pm$ 17.2	24.7 $\pm$ 13.0	68.1 $\pm$ 26.7	0.53
	2012.6	26.6 $\pm$ 10.2	16.4 $\pm$ 6.3	56.0 $\pm$ 13.5	0.47
	2012.7	55.5 $\pm$ 32.5	31.3 $\pm$ 19.8	73.5 $\pm$ 23.7	0.76
	2012.8	56.3 $\pm$ 21.4	27.7 $\pm$ 9.4	59.5 $\pm$ 12.6	0.94
	2012.9	35.3 $\pm$ 19.0	18.8 $\pm$ 8.6	53.9 $\pm$ 12.7	0.65
	2012.10	40.3 $\pm$ 13.6	19.9 $\pm$ 9.6	57.5 $\pm$ 17.4	0.70
	Mean	41.8 $\pm$ 19.0	23.1 $\pm$ 11.1	61.4 $\pm$ 17.8	0.68
S2	2013.5	40.5 $\pm$ 14.4	26.7 $\pm$ 9.9	63.2 $\pm$ 30.4	0.64
	2013.6	22.7 $\pm$ 9.7	15.6 $\pm$ 5.8	44.1 $\pm$ 16.2	0.52
	2013.7	16.4 $\pm$ 8.4	11.7 $\pm$ 7.6	50.2 $\pm$ 19.6	0.33
	2013.8	37.1 $\pm$ 11.5	21.8 $\pm$ 5.7	50.6 $\pm$ 16.7	0.73
	2013.9	24.0 $\pm$ 11.8	16.0 $\pm$ 8.4	28.2 $\pm$ 11.5	0.85
	2013.10	23.6 $\pm$ 12.1	13.3 $\pm$ 6.1	34.5 $\pm$ 14.9	0.68
	Mean	27.4 $\pm$ 11.3	17.4 $\pm$ 7.3	45.1 $\pm$ 18.2	0.61

^a Ambient concentrations monitored at AQ1 and AQ2 near the each school.

PM10 and PM2.5 at the two preschools, and the ambient outdoor PM10 concentrations (at AQ1 and AQ2) near the preschools for 6 months during 2012 and 2013. The average indoor PM10 and PM2.5 levels were higher for S1 than S2 during all months except May and the highest indoor concentrations in S1 were during July and August.

Linear correlation analyses (Table 2) indicate statistically significant positive correlations between indoor and outdoor daily PM levels at both preschools. Interestingly, ambient outdoor PM10 concentration had stronger correlations with indoor PM2.5 concentration (S1: $r = 0.755$, S2: $r = 0.634$) than with indoor PM10 concentration (S1: $r = 0.462$, S2: $r = 0.523$).

Fig. 2 shows time series of daily average indoor and outdoor PM10 concentrations and indoor/outdoor (I/O) ratios which were calculated using the PM10 data measured at the same time. The changes of indoor and outdoor PM10 levels were similar in S1 and S2, as indicated by their significant correlations (Table 2). Notably, the daily I/O ratio of PM10 varied substantially, from 0.13 to 1.98 at S1 and from 0.08 to 3.15 at S2. There were largely higher I/O values in August and September at each preschool, but there were no clear seasonal patterns. The I/O ratios much higher than 1 were also observed for some days at both preschools when ambient outdoor concentrations were relatively quite low compared to indoor ones.

3.2. Association between PM exposure and AD symptoms

Fig. 3 shows the relationship between PM exposure and AD symptoms. The levels of PM10 and PM2.5 exposure were higher on days when children reported AD symptoms in both preschools. An independent sample t -test indicated significant differences ($p < 0.001$) in PM concentrations on days with and without symptoms.

We also analyzed the temporal association between PM exposure and AD symptoms by examining the daily variations of these

Table 2

Linear correlation coefficients among indoor and ambient outdoor PM concentrations.

PM (Unit: $\mu\text{g}/\text{m}^3$)	Indoor PM10 (S1/S2)	Outdoor PM10 (AQ1/AQ2)
Indoor PM10 (S1/S2)	–	–
Outdoor PM10 (AQ1/AQ2)	0.462/0.523 ($n = 142/160$)	–
Indoor PM2.5 (S1/S2)	0.784/0.852 (150/164)	0.755/0.634 (150/162)

All coefficient values are statistically significant ($p < 0.01$).

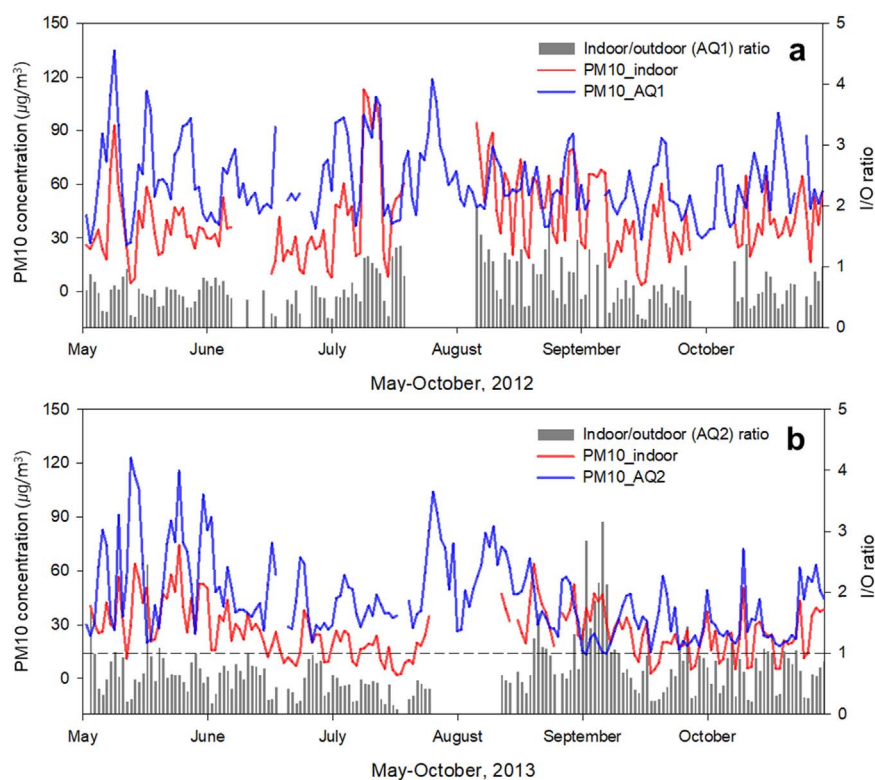


Fig. 2. Daily mean indoor and outdoor PM10 concentrations and their ratios at two preschools for the period May–October in (a) 2012 (S1) and (b) 2013 (S2).

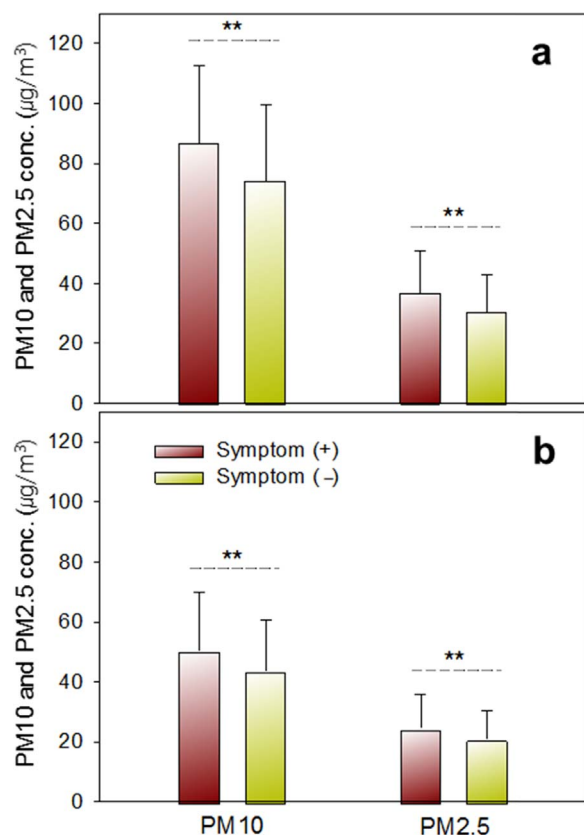


Fig. 3. Comparison of PM concentrations at (a) S1 and (b) S2 preschools between days with and without AD symptoms. Vertical bars represent mean and SD. **: $p < 0.01$.

parameters (Fig. 4). The patterns of daily changes in PM exposure and AD symptoms were similar, as indicated by the significant Pearson correlation coefficients (S1: $r = 0.50$ for PM10, $r = 0.51$ for PM2.5; S2:

$r = 0.56$ for PM10, $r = 0.57$ for PM2.5; $p < 0.01$ for all r values).

Table 3 shows the adjusted odds ratios (aORs) for the effect of PM on AD symptoms based on GLMM analysis. PM10 and PM2.5 were both significantly associated with the exacerbation of AD symptoms (S1: aOR = 1.215 for PM10, aOR = 1.399 for PM2.5; S2: aOR = 1.195 for PM10, aOR = 1.359 for PM2.5; $p < 0.01$ for all aORs associated with increase in $10 \mu\text{g}/\text{m}^3$ of PM10 and PM2.5). Notably, PM2.5 had a stronger effect on AD symptoms than PM10 in both preschools.

We also determined the association of AD symptoms with PM exposure for each of the 21 children using the logistic regression model, after adjustment for sex and other significant covariates (Fig. 5). These results show that PM exposure had a significant positive effect on the exacerbation of AD symptoms in most children, and that the effect seemed to be stronger for PM2.5 than PM10. A maximum OR of 4.44 was observed with a $10 \mu\text{g}/\text{m}^3$ increase of PM2.5. However, the ORs and confidence intervals were quite large for some children, especially in analysis of the effect of PM2.5.

4. Discussion

This 6-month longitudinal study investigated the effects of PM exposure on AD symptoms in young children living in an industrial urban area. The present study evaluated monthly average concentrations of indoor PM10 and PM2.5 at two preschools and ambient outdoor PM10 concentrations at AQ1 and AQ2 (sites near each school) over 6 months of 2012 and 2013. The average indoor PM levels were higher in S1 than S2 during all months except for May. This could be related to the higher outdoor PM10 concentration at S1 in 2012, or to differences in the effects of indoor emission and human activities at these preschools. The highest indoor concentrations of PM were measured during July and August at S1, possibly because of infiltration of outdoor high-level PM into the classroom. High ambient PM concentration in Ulsan normally occurred in summer when southerly winds are prevailed (Oh et al., 2016). Frequent ventilation during the summer months, therefore, could lead to increase of the indoor PM level. Previous studies reported associations between elevated indoor levels of PM and greater use of

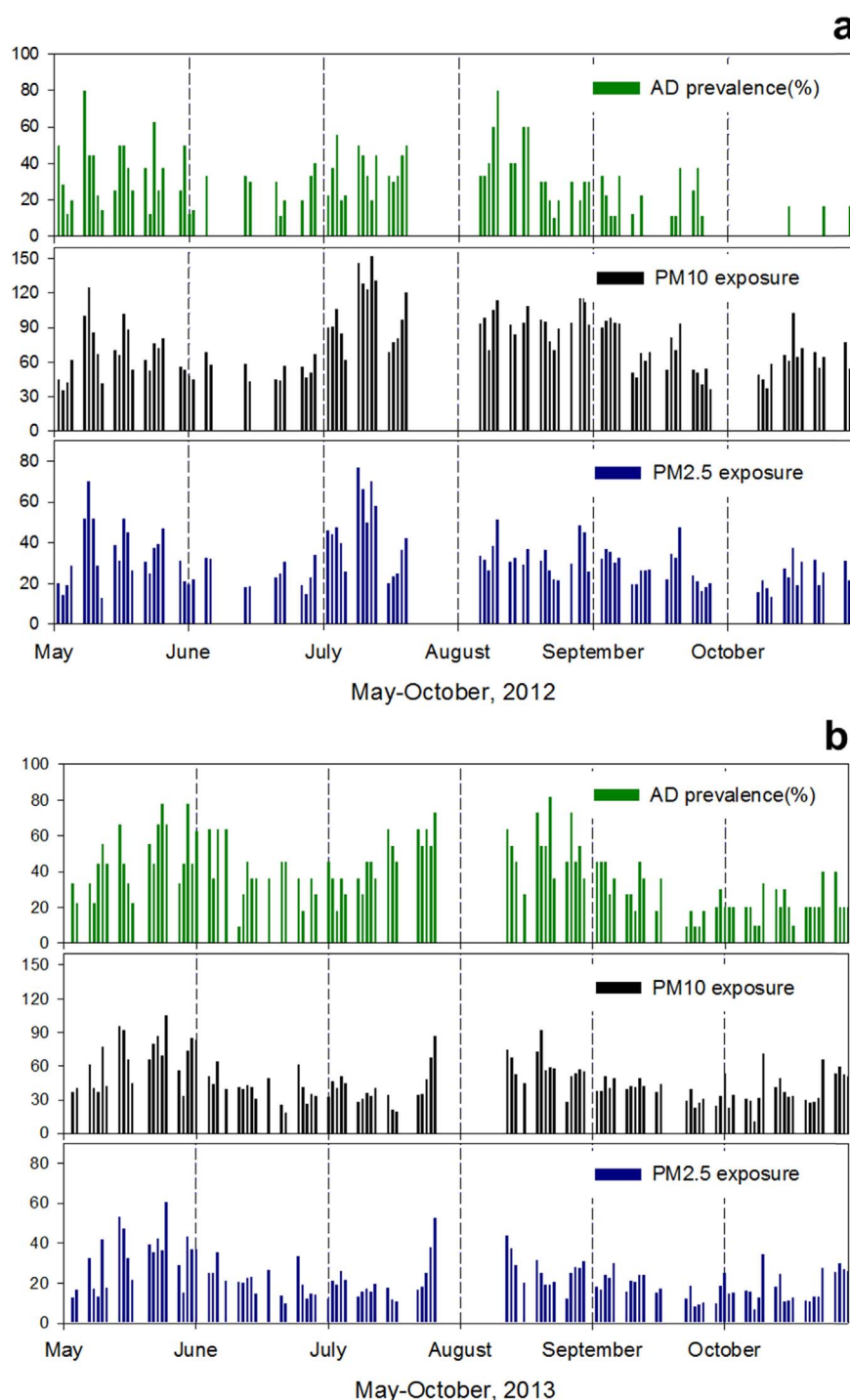


Fig. 4. Variations of daily PM exposure concentrations and the prevalence rate of AD symptom presences for 10 and 11 patients at S1 and S2 preschools, respectively.

Table 3
Effects of PM exposures on AD symptoms presence in preschool children.

Air pollutants	Adjusted OR (95% CI) ^a	
	S1	S2
PM ₁₀	1.215 (1.118, 1.320)	1.195 (1.099, 1.299)
PM _{2.5}	1.399 (1.216, 1.610)	1.359 (1.174, 1.574)

^a ORs were calculated using GLMM for each 10 µg/m³ increase of PM exposure concentration (All P-values < 0.01). Odd ratios were adjusted for age, sex and ambient temperature and relative humidity, SCORAD at enrollment, and the use of topical corticosteroids.

ventilation (Rojas-Bracho et al., 2000; Williams et al., 2003; Pekey et al., 2010).

The findings in this study suggest that short-term exposure to PM could exacerbate AD symptoms. More specifically, our time series data showed a significant association between daily PM exposure and daily AD symptoms. Results from logistic regression analyses also indicated significant effects of short-term exposure to PM₁₀ and PM_{2.5} on the exacerbation of AD symptoms.

Our analysis of all patients taken together and of individual patients showed that PM_{2.5} exposure was a stronger risk factor for AD symptoms than PM₁₀ exposure. This supports the findings of previous studies, that fine particulates exacerbate AD symptoms more than coarse particulates. The stronger effect of PM_{2.5} on exacerbation of AD

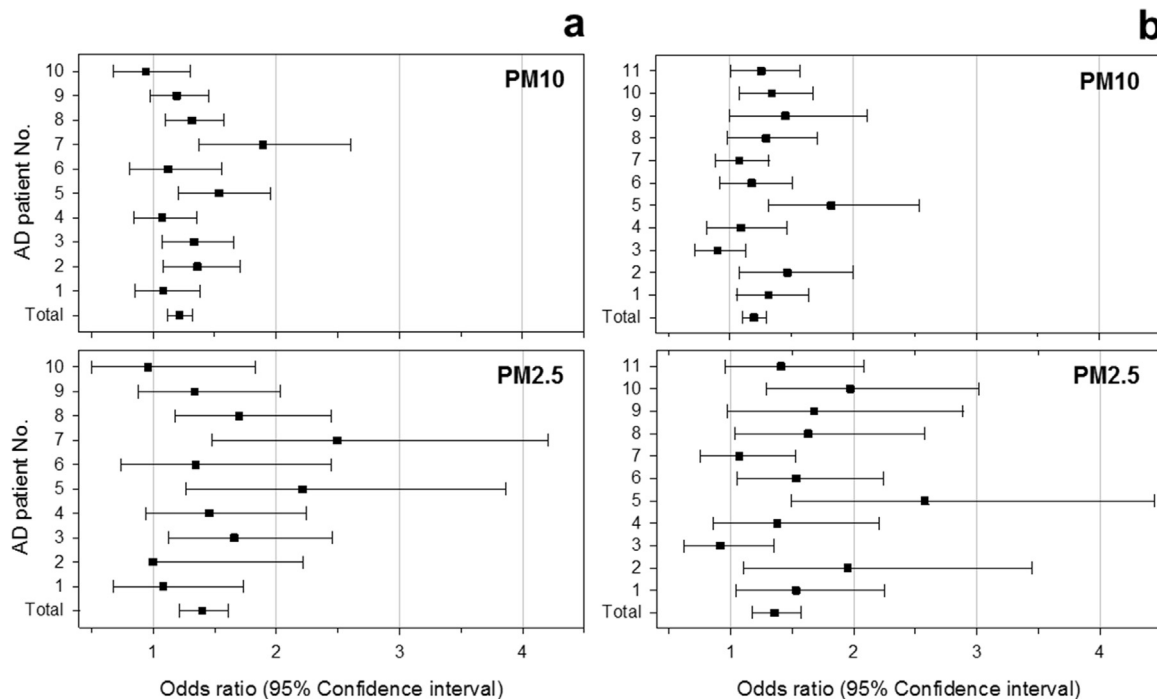


Fig. 5. Association (odds ratios) between PM exposure and AD symptoms by individual patient at preschool (a) S1 and (b) S2 ($n = 10$ for S1, $n = 11$ for S2). Total indicates the result from GLMM fitting for all patient.

symptoms may be because fine particles more easily penetrate the skin (Kimata, 2004). Our results also suggest that PM may exacerbate AD symptoms in children living in industrial urban areas, which have many sources of fine PM and proper condition for chemical production of secondary PMs. Considering the greater indoor penetration of PM_{2.5} (Chen and Zhao, 2011) and the stronger correlation between indoor PM_{2.5} and outdoor PM₁₀ (Table 2), it appears that outdoor PM concentrations in industrial urban areas mainly contribute to indoor exposure to PM_{2.5}, whereas indoor PM₁₀ is more influenced by students' activities. Although there is no information on the composition of PM_{2.5} in our study area, it seems likely that secondary fine PMs formed by chemical reactions of pollutants emitted by industrial facilities and traffic sources near S1 and S2, also have a role in the exacerbation of AD symptoms in our subjects. Recently, several long-term studies showed that significant association between prenatal ambient traffic-related air pollutants (NO₂ and CO) and the development of eczema or AD in preschool children (Deng et al., 2016; Huang et al., 2015; Lu et al., 2017). These previous results are in an agreement with our findings that short-term exposure of PM_{2.5}, which may be a marker of traffic-related air pollutants, may aggravate AD symptoms in preschool children. These results also showed that we should consider the effect of prenatal long-term exposure to traffic-related air pollutants (such as NO₂, CO, and PM_{2.5}) on AD symptoms in preschool children as well when addressing the plausible effect of short term exposure on AD symptoms.

Interestingly, the PM effect sizes for children in S1 and S2 were similar, although average indoor and outdoor PM concentrations of S1 were considerably greater (Table 1). Whether the magnitude of PM change may be more important than the absolute PM concentration in the exacerbation of AD symptoms needs to be clarified in further studies.

The present study has important public health implications. The strong correlation between indoor and outdoor PM concentrations and the significant effect of PM on AD symptoms imply that lowering ambient PM levels may benefit children with pre-existing AD. Our study has several strengths. First, we used a longitudinal study design to evaluate PM and AD symptoms over two 6-month periods. Second, we

improved data accuracy of patient's exposure to PM using both the indoor and outdoor PM concentrations based on averaged time activities. Third, we analyzed a large data set by fitting a GLMM, after controlling for potential confounders, and then were able to obtain more objective and reliable results.

This study also had several limitations. First, the effects of PM on AD are not entirely clear, because there could have been other confounding factors that we did not consider, such as exposure to other pollutants, meteorological conditions, and allergens such as pollen, house dust mites, and various fungal spores. Moreover, we have no measurement data on chemical composition of PM, so there should be uncertainty in clarifying what sources mainly contributed to the PM_{2.5} concentrations which was a stronger risk factor for AD symptoms. Second, our data has uncertainties in the total individual exposure assessment, and this could have led to errors in evaluation of the effects of PM on AD symptoms. The large differences in ORs and confidence intervals for individual children (Fig. 5) may indicate that these uncertainties result from different exposures among individuals. No data on PM concentrations at individual homes, different proximity of children to the preschools or monitoring sites, and different time activities, all of which could have led to errors in our assessment of PM exposure. Third, PM measurements in indoor and outdoor air were performed in different methods described in this manuscript. We had not a specific calibration factor based on measurements using two different methods at the same preschool. Based on an experimental study by Cheng (2008), aerosol Spectrometer can underestimate by approximately 25% actual PM concentration measured by the gravimetric method. However, in our study, possible differences between indoor PM concentrations by the two methods were included uniformly for all individuals to estimate total exposure concentrations. Thus, potential bias in individual's exposure estimates from un-calibrated indoor concentrations would not change the PM effect on AD symptoms. Fourth, we only analyzed data between May and October, because of limitations in daily PM monitoring and in collecting data on AD symptoms. The collection of PM data at the different preschools during different years may also have affected our estimates of the effect of PM on AD symptoms. Finally, the present study lacked number of sample participants (21 children).

In conclusion, we found that short-term exposure to PM exacerbates AD symptoms in young children living in an industrial urban area of South Korea. PM_{2.5} had a stronger effect than PM₁₀ on the exacerbation of AD symptoms. Whether the magnitude of PM change may be more important than the absolute PM concentration in the exacerbation of AD symptoms needs to be clarified in further studies. Further research is needed to better quantify the relationship between exposure to PM and AD in individual patients, and to determine the possible effects of other pollutants and allergens on AD.

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