Table 1: Characteristics of children.

	2012	2013
Number	399	384
Gender (male/female)	205/194	194/190
Height (cm)	132.3 ± 5.9	137.7 ± 7.0
Male	132.2 ± 5.5	136.9 ± 6.3
Female	132.4 ± 6.4	138.5 ± 7.7
Weight (kg)	29.5 ± 5.8	32.4 ± 6.6
Male	29.6 ± 6.2	32.3 ± 6.8
Female	29.3 ± 5.4	32.6 ± 6.4
Allergic disease		
Asthma	38	45
Allergic rhinitis	78	74
Allergic conjunctivitis	8	15
Atopic dermatitis	44	36
Food allergy	19	20

Data are shown as the mean ± S.D.

for ith child and are assumed to be $b_{0,i} \sim N(0, \sigma_b^2)$. ε_{ij} is the error term, $\varepsilon_{ij} \sim N(0, \sigma^2)$. In addition, effects on PEF were measured from the day of ADS exposure until 3 days after exposure because a dust effect on PEF can persist for up to 3 days [10]. Differences in PEF between the 2012 and 2013 results were also evaluated. The two-pollutant model was applied to different combinations of pollutants to assess the stability of the effects of ADS on PEF after adjustment for individual characteristics (age, gender, height, weight, and presence of asthma, allergic rhinitis, allergic conjunctivitis, atopic dermatitis, and food allergies) and meteorological variables (temperature, humidity, and atmospheric pressure). R version 3.0.3 (R Foundation for Statistical Computing, Vienna, Austria) was used for statistical analysis of PEF values and ADS exposure. Differences of nSLO-LA of THP-G8 cells were analyzed by ANOVA using SPSS Statistics (Japanese version 21.0 for Windows, IBM Japan, Tokyo, Japan). All quoted P values are two-sided and the significance level was set to 0.05.

3. Results

3.1. Profile of the Children. The characteristics of the children in the 2012 and 2013 studies are shown in Table 1.

3.2. Air Pollution Levels and Weather Information on ADS Days and Non-ADS Days. In 2012, April 23 and 24 were identified as ADS days. In 2013, March 8 to 10 and 19 and 20 were similarly identified as ADS days. Non-ADS days were defined as all other days from April 1 to May 31, 2012, and from March 1 to May 31, 2013. Daily levels of mineral dust particles (airborne sand dust particles) and suspended particulate matter (SPM) are shown in each period in Figure 2. The levels of air pollutants and weather during the study periods are shown in Table 2.

3.3. PEF. Changes in PEF after exposure to ADS are shown in Figure 3. In order to show the post-ADS-exposure effects,

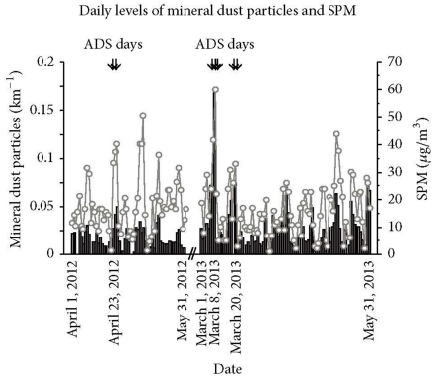


Figure 2: Daily levels of mineral dust particles (airborne sand dust particles) (bar graph) and SPM (line graph). Arrows indicate ADS days.

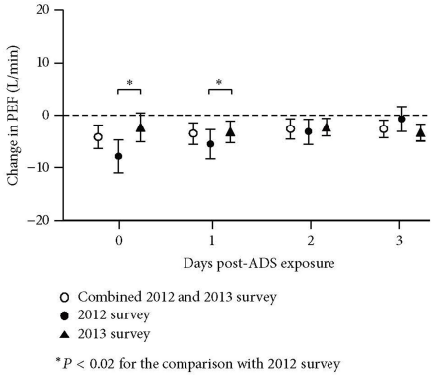


Figure 3: PEF changes caused by an ADS event from 0 (ADS day) to 3 days after ADS exposure in combined 2012 and 2013 (open circles), 2012 (black circles), and 2013 (triangles), with 95% confidence intervals (error bars). Data are controlled for age, gender, height, weight, and presence of asthma, allergic rhinitis, allergic conjunctivitis, atopic dermatitis, and food allergies; meteorological variables such as daily temperature, humidity, and atmospheric pressure; and the linear time trend. There are significant differences in the decrement of PEF on days 0 and 1 between 2012 and 2013 (*$P < 0.02$).

these changes are shown from 0 (ADS day) to 3 days after ADS exposure. In combining 2012 and 2013, the changes in PEF after exposure to ADS exposure were −4.16 L/min (95% CI, −6.33 to −1.99) on day 0, −2.97 L/min (−5.03 to −0.91) on day 1, −2.23 L/min (−4.12 to −0.35) on day 2, and −2.57 L/min (−4.29 to −0.86) on day 3 after ADS. There were significant decreases in PEF from day 0 to day 3 after ADS exposure. In 2012, the changes in PEF were −7.82 L/min (−10.93 to −4.71)

Table 2: Daily air pollutant levels and weather information on ADS days and non-ADS days in 2012 and 2013.

(a)

Measurement	ADS days		
	April 23 and 24, 2012	March 8 to 10, 2013	March 19 and 20, 2013
Daily average temperature, °C	17.6 ± 1.1	13.6 ± 3.8	12.4 ± 1.4
Daily maximum temperature, °C	23.3 ± 3.0	20.4 ± 0.9	18.6 ± 0.0
Daily minimum temperature, °C	13.1 ± 0.7	8.0 ± 5.8	7.9 ± 4.5
Daily average relative humidity, %	70.0 ± 5.7	64.7 ± 7.5	79.5 ± 3.5
Daily minimum relative humidity, %	48.0 ± 12.7	29.3 ± 10.7	52.0 ± 0.0
Daily average atmospheric pressure, hPa	1010.8 ± 1.0	1008.6 ± 3.1	1007.4 ± 3.0
Daily average mineral dust particles, km^{-1}	0.046 ± 0.006	0.084 ± 0.077	0.075 ± 0.001
Daily average nonmineral dust particles, km^{-1}	0.148 ± 0.097	0.138 ± 0.050	0.097 ± 0.001
Daily average SPM, $\mu g/m^3$	39.5 ± 2.1	44.3 ± 19.0	32.5 ± 5.0
Daily average $PM_{2.5}$, $\mu g/m^3$	17.2 ± 1.3	37.3 ± 16.7	37.8 ± 4.5
Daily average SO_2, ppb	1.3 ± 0.6	2.0 ± 1.1	1.9 ± 1.3
Daily average NO_2, ppb	1.5 ± 0.4	3.4 ± 1.8	3.6 ± 0.6
Daily average O_x, ppb	55.9 ± 5.7	63.1 ± 10.5	49.4 ± 5.3

(b)

Measurement	Non-ADS days	
	2012	2013
Daily average temperature, °C	15.7 ± 3.5	13.2 ± 5.0
Daily maximum temperature, °C	20.9 ± 4.5	18.7 ± 5.7
Daily minimum temperature, °C	10.9 ± 4.0	8.1 ± 5.0
Daily average relative humidity, %	70.4 ± 9.3	69.8 ± 9.8
Daily minimum relative humidity, %	44.9 ± 15.4	42.8 ± 13.6
Daily average atmospheric pressure, hPa	1010.0 ± 5.7	1011.3 ± 5.9
Daily average mineral dust particles, km^{-1}	0.016 ± 0.010	0.024 ± 0.017
Daily average non-mineral dust particles, km^{-1}	0.042 ± 0.037	0.073 ± 0.049
Daily average SPM, $\mu g/m^3$	17.7 ± 10.1	17.8 ± 8.9
Daily average $PM_{2.5}$, $\mu g/m^3$	10.3 ± 5.4	17.5 ± 7.3
Daily average SO_2, ppb	0.9 ± 0.6	1.0 ± 0.8
Daily average NO_2, ppb	2.6 ± 1.2	2.75 ± 1.2
Daily average O_x, ppb	50.0 ± 7.5	50.2 ± 8.4

Data are presented as the mean ± S.D., Non-ADS days were all other days except for ADS days from April 1 to May 31, 2012, and March 1 to May 31, 2013.

on day 0, −5.49 L/min (−8.14 to −2.85) on day 1, −3.15 L/min (−5.54 to −0.75) on day 2, and −0.72 L/min (−3.03 to 1.59) on day 3 after ADS. A significant decrease in PEF persisted for 2 days after ADS exposure in 2012. In 2013, the changes in PEF were −2.33 L/min (−5.09 to 0.44) on day 0, −2.72 L/min (−4.80 to −0.64) on day 1, −2.26 L/min (−3.98 to −0.53) on day 2, and −3.04 L/min (−4.68 to −1.40) on day 3 after ADS. A significant decrease in PEF continued from days 1 to 3 after ADS exposure. On days 0 and 1, the decrease in PEF after exposure to ADS in 2012 was significantly higher than that in 2013. In addition, the 2012 and 2013 forest plots indicate clear differences. Significant differences were observed in PEF on days 0 and 1, and the decrement of PEF in 2012 was higher than that in 2013. In a two-pollutant model adjusted for SPM, $PM_{2.5}$, SO_2, NO_2, and O_x, an ADS event in 2012 alone was significantly associated with a decrease of PEF (Table 3). In contrast, in 2013, a similar model gave no significant relationship between ADS events and PEF in children.

3.4. IL-8 Transcriptional Activity and IL-8 Secretion in THP-G8 Cells. In THP-G8 cells stimulated for 5 h with various LPS concentrations, nSLO-LA (a measure of IL-8 transcriptional activity) reached a plateau at 100 ng/mL LPS (Figure 4(a)). Maximum induction of nSLO-LA by LPS (100 ng/mL) occurred between 4 and 6 h (Figure 4(b)). Based on these results, we subsequently used stimulation for 5 h to investigate the effect of ADS airborne particles on IL-8 transcriptional activity. The concentrations of IL-8 in supernatants of THP-G8 cells stimulated with vehicle, LPS (n = 6, 1 ng/mL), and LPS (n = 6, 100 ng/mL) were 1.2 ± 0.2, 26.6 ± 6.2, and 77.4 ± 10.9 $\mu g/mL$, respectively (Figure 4(c)). This increase in IL-8 secretion is in agreement with the augmentation of nSLO in THP-G8 cells.

The pH values of ADS airborne particles (1 mg/mL) collected on April 23 and 24, 2012; March 8 to 10, 2013; and March 19 and 20, 2013 were 7.9, 7.6, and 7.6, respectively. The nSLO-LA values (IL-8 transcriptional activity) of THP-G8

Table 3: Estimated effects of ADS events on PEF in two-pollutant model after adjustment for SPM, $PM_{2.5}$, NO_2, O_x, and SO_2.

Year	Adjustment	Change in PEF	95% CI	P value
2012 and 2013	Adjusted for SPM	−3.00	−5.31, −0.68	0.011
	Adjusted for $PM_{2.5}$	−3.60	−5.94, −1.27	0.002
	Adjusted for SO_2	−2.14	−4.43, 0.15	0.059
	Adjusted for O_x	−3.49	−5.70, −1.28	0.002
	Adjusted for NO_2	−4.20	−6.37, −2.03	0.001
2012	Adjusted for SPM	−6.04	−9.44, −2.64	0.001
	Adjusted for $PM_{2.5}$	−6.48	−9.78, −3.18	0.001
	Adjusted for SO_2	−7.41	−10.69, −4.13	0.001
	Adjusted for O_x	−3.93	−7.25, −0.62	0.019
	Adjusted for NO_2	−10.04	−13.42, −6.67	0.001
2013	Adjusted for SPM	−1.57	−4.56, 1.43	0.306
	Adjusted for $PM_{2.5}$	−1.97	−5.10, 1.15	0.216
	Adjusted for SO_2	−2.19	−5.02, 0.63	0.128
	Adjusted for O_x	0.19	−2.79, 3.18	0.900
	Adjusted for NO_2	−2.45	−4.38, 1.49	0.085

Calculated for an interquartile by ADS and adjusted for individual characteristics (age, gender, height, weight, and presence of asthma, allergic rhinitis, allergic conjunctivitis, atopic dermatitis, and food allergies) and meteorological variables (temperature, humidity, and atmospheric pressure).
ADS: Asian dust storm, PEF: peak expiratory flow, SPM ($\mu g/m^3$): suspended particle matter, $PM_{2.5}$ ($\mu g/m^3$): particulate matter smaller than 2.5 μm in diameter, NO_2 (ppb): nitrogen dioxide, O_x (ppb): photochemical oxidants, SO_2 (ppb): sulfur dioxide, and CI: confidence interval.

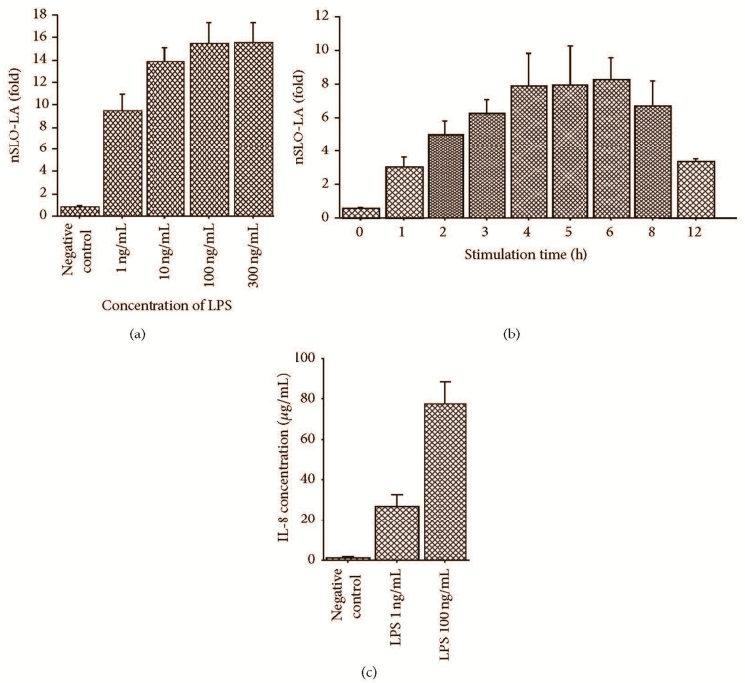
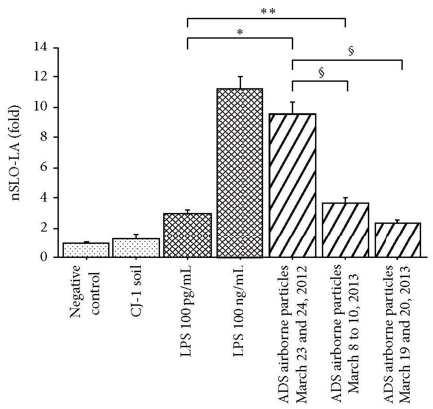


Figure 4: (a) IL-8 transcriptional activity in THP-G8 cells stimulated with LPS at various concentrations ($n = 6$) for 5 h. IL-8 transcriptional activity is based on normalized SLO luciferase activity (nSLO-LA), which was calculated as SLO-LA divided by SLR-LA. The fold induction of nSLO-LA was calculated as the nSLO-LA of treated cells divided by that of untreated cells [18]. (b) IL-8 transcriptional activity in THP-G8 cells stimulated with 100 ng/mL LPS ($n = 6$) for various time periods. (c) Concentrations of IL-8 in supernatants of a stable THP-1-derived IL-8 reporter cell line stimulated with solvent only (negative control), LPS ($n = 6$, 1 ng/mL), and LPS ($n = 6$, 100 ng/mL). The IL-8 concentration was measured using an ELISA kit. Samples were run in triplicate. The assay range was 31.2 to 2000 pg/mL.



*$P < 0.01$ for the comparison with LPS at 100 pg/mL

**$P < 0.02$ for the comparison with LPS at 100 pg/mL

§$P < 0.01$ for the comparison with ADS airborne particles in April 23 and 24, 2012

FIGURE 5: IL-8 transcriptional activity measured using an IL-8 luciferase assay in a stable THP-1-derived IL-8 reporter cell line. Cells were treated with solvent only ($n = 6$, negative control), LPS ($n = 6$, 100 pg/mL, positive control), LPS ($n = 6$, 100 ng/mL, positive control), and ADS airborne particles collected on April 23 and 24, 2012 ($n = 6$, 1 mg/mL), March 8 to 10, 2013 ($n = 6$, 1 mg/mL), and March 19 and 20, 2013 ($n = 6$, 1 mg/mL). *$P < 0.01$ versus LPS at 100 pg/mL, **$P < 0.02$ versus LPS at 100 pg/mL, and §$P < 0.01$ versus ADS airborne particles from April 23 to April 24, 2012.

cells (Figure 5) changed by 0.95 ± 0.09-fold (vehicle, $n = 6$), 2.87 ± 0.28-fold (LPS, $n = 6$, 100 pg/mL), 11.21 ± 0.28-fold (LPS, $n = 6$, 100 ng/mL), 9.56 ± 0.80-fold (ADS particles from April 23 to 24, 2012, $n = 6$, 1 mg/mL), 3.65 ± 0.36-fold (ADS particles from March 8 to 10, 2013, $n = 6$, 1 mg/mL), and 2.33 ± 0.24-fold (ADS particles from March 19 to 20, 2013, $n = 6$, 1 mg/mL).

The pH value of CJ-1 soil was constant at 8.4 for each event. The pH values of ADS airborne particles (1 mg/mL) collected on April 23 and 24, 2012; March 8 to 10, 2013; and March 19 and 20, 2013 were 7.9, 7.6, and 7.6, respectively. THP-G8 cells were stimulated with CJ-1 soil after adjusting the pH of the soil to 7.8 with 0.1N sodium hydroxide. The nSLO-LA values (IL-8 transcriptional activity) of THP-G8 cells (Figure 5) changed by 0.95 ± 0.09-fold (vehicle, $n = 6$), 1.48 ± 0.27-fold (CJ-1 soil, $n = 6$, 1 mg/mL), 2.87 ± 0.28-fold (LPS, $n = 6$, 100 pg/mL), 11.21 ± 0.28-fold (LPS, $n = 6$, 100 ng/mL), 9.56 ± 0.80-fold (ADS particles from April 23 to 24, 2012, $n = 6$, 1 mg/mL), 3.65 ± 0.36-fold (ADS particles from March 8 to 10, 2013, $n = 6$, 1 mg/mL), and 2.33 ± 0.24-fold (ADS particles from March 19 to 20, 2013, $n = 6$, 1 mg/mL). nSLO-LA values in THP-G8 cells stimulated by ADS airborne particles differed significantly from those of controls and cells stimulated with 100 pg/mL LPS. nSLO-LA values also differed significantly for each pairwise comparison of airborne particles collected in the three ADS periods.

3.5. Endotoxin Concentration in Airborne Particles Collected on ADS Days. The endotoxin levels in ADS airborne particles (1 mg/mL) collected on April 23 and 24, 2012; March 8 to 10, 2013; and March 19 and 20, 2013 were 0.19, 0.08, and 0.07 EU/mL, respectively. These values were all lower than the level of 0.89 EU/mL found in 100 pg/mL LPS. The endotoxin concentration in 100 ng/mL LPS was out of the range of the assay.

3.6. Concentration of Metal Elements in CJ-1 Soil and Airborne Particles Collected on ADS Days. The concentrations of metal elements in CJ-1 soil and ADS airborne particles collected on April 23 and 24, 2012; March 8 to 10, 2013; and March 19 and 20, 2013 are shown in Table 4.

4. Discussion

To investigate the effect of ADS on pulmonary function, we monitored daily PEF in school children from April to May 2012 and March to May 2013 and found a significant correlation between exposure to an ADS and pulmonary function. The 2012 survey alone also showed this relationship. When differences in PEF between the 2012 and 2013 results were evaluated, the same relationship of PEF with ADS events was not found in 2013, despite the study being conducted in the same children. The decline of PEF upon ADS exposure in 2012 was also significantly higher than that in 2013, and IL-8 transcriptional activity in THP-G8 cells induced by ADS airborne particles collected in 2012 was also significantly higher than that induced by ADS airborne particles collected in 2013. These results suggest that the effect of ADS on pulmonary function in children is associated with enhanced airway inflammation mediated by elevation of IL-8.

Desert sand can reduce pulmonary function in patients with asthma after exposure at a level of PM_{10} ranging from 1500 to 2000 $\mu g/m^3$/hour [25]. This level of PM_{10} is 10 to 20 times higher than that during an ADS event in Japan. In the current study, the level of mineral dust particles on ADS events on March 8 to 10, 2013, and March 19 and 20, 2013 was about twice as high as that in 2012. However, the decrease of PEF after exposure to ADS in 2012 was higher than that in 2013. These results suggest that the decline in pulmonary function of school children during an ADS event has little connection to mineral dust particles (sand dust particles). Nonmineral dust particles were similar on ADS events in 2012 and 2013. ADS events in 2012 had higher counts of air pollution aerosols than those in 2013. Based on Onishi's criteria [4], the ADS event in 2012 can be classified as Type 1 and those in 2013 as type 2. The air pollution aerosols during ADS can be considered a cause of the significant difference in the effect of the ADS on pulmonary function of school children between 2012 and 2013.

The ADS days in 2013 were defined as moderate, while, on April 23 and 24, 2012, the mean daily average concentration of mineral dust particles was 0.046 ± 0.006 km^{-1} in Matsue

Table 4: Concentration of metal elements in CJ-1 soil and airborne particles collected on ADS days.

Metals (μg/mg)	CJ-1 soil	ADS airborne particles in April 23 and 24, 2012	ADS airborne particles in March 8 to 10, 2013	ADS airborne particles in March 19 and 20, 2013
Al	68.00	28.80	22.40	14.80
As	ND	ND	ND	ND
Ba	0.44	0.12	0.18	0.10
Ca	68.00	29.60	44.00	31.20
Cd	ND	ND	ND	ND
Co	0.01	ND	ND	ND
Cr	0.05	ND	ND	ND
Cu	0.03	0.12	ND	0.07
Fe	26.00	22.00	20.80	14.40
Hg	ND	ND	ND	ND
K	0.19	0.38	0.33	0.30
La	0.03	ND	ND	ND
Mg	18.00	14.00	16.80	14.40
Mn	0.70	0.52	0.56	0.40
Na	0.44	33.60	56.00	64.00
Ni	0.03	0.14	0.15	0.11
P	0.68	ND	ND	ND
Pb	0.02	0.06	0.12	0.06
Si	260.00	140.00	108.00	72.00
Sr	0.26	0.16	0.22	0.16
Ti	2.40	0.96	0.92	0.52
Zn	0.07	0.52	0.64	0.60

ADS: Asian dust storm, CJ-1 soil: soil from the China Loess Plateau, the original ADS soil in the Tengger Desert and Huining located in Gansu Province, and ND: not detected.

City. These values are lower than the thresholds defined in previous studies [9, 21]. However, the daily average level of mineral dust particles on ADS days was higher than that on non-ADS days, as required for definition of an ADS day in the Japan Meteorological Agency criteria used in this study.

Many studies have shown that children are susceptible to air pollution such as NO_2, O_x, and SO_2 [18, 26]. Therefore, we used a linear mixed model and a two-pollutant model to adjust for the effects of NO_2, O_x, and SO_2 on pulmonary function. In both models, ADS in 2012 remained significant after inclusion of NO_2, O_x, and SO_2. However, in the 2013 survey, we were not able to find a significant association with ADS and pulmonary function. These results suggest that airborne particles during ADS decrease pulmonary function irrespective of NO_2, O_x, and SO_2.

IL-8 is a key cytokine in air pollutant-induced airway inflammation [15, 16]. Components that adhered to ADS particles can increase release of IL-6 and IL-8 from airway epithelial cells [27]. We showed that ADS airborne particles promote transcriptional activity and production of IL-8 in THP-G8 cells, with a significant increase in IL-8 transcriptional activity in THP-G8 cells treated with ADS airborne particles compared to those treated with original ADS soil (CJ-1 soil). Additionally, we measured the difference in production of IL-8 by particles collected during each ADS event. The IL-8 transcriptional activity of ADS airborne particles collected in 2012 was significantly higher than that for particles collected in 2013. This difference in production of IL-8 by ADS airborne particles may account for the different effects on pulmonary function in school children in 2012 and 2013.

The production of IL-8 induced by ADS airborne particles in 2012 had a significant difference compared to 2013. However, there was no difference between the two ADS airborne particles in 2013. The ADS events in 2013 had happened close together, and we suspect that the route and composition of the ADS airborne particles in the two events were similar. In fact, when we analyzed the effect on PEF between two ADS events in 2013 separately, the decreases in PEF after exposure to ADS were −4.1 L/min (95% CI, −10.6 to 2.4, $P = 0.21$) in March 8 to 10 and −3.3 L/min (95% CI, −10.8 to 4.1, $P = 0.37$) in March 19 and 20. In both ADS events in 2013, there was not a significant decrease of the effects on PEF. Therefore, we presented the combined results in the main analysis. The differences in the substances and the levels of those substances attached to desert sand dusts depend on the route along which desert sand dusts pass and may play an important role in the effect of ADS on pulmonary function in children.

According to the analysis of the concentrations of metal elements, ADS airborne particles in 2012 had more Al, Cu, Fe, K, and Ti compared to those on March 8 to 10 and March 19 and 20, 2013. The amounts of Al, Fe, and Ti in ADS airborne particles in 2012 were lower than CJ-1. Cu and K may play

a causative role in the difference of production of IL-8 induced by ADS airborne particles. However, Kumar et al. indicated that Cu was not a cause of the production of CXCL1 (a mouse functional homologue of IL-8) and IL-6 induced by ambient and traffic-derived particulate matter, but it did indicate that Fe content of airborne particulate matter may be more important in mouse airway epithelial injury [28]. Metal components attached to ADS airborne particles may be one of the causes of the difference between 2012 and 2013, but further study is needed to determine a role of metal components attached to ADS on the effect of production of proinflammatory cytokines.

Ogino et al. found that some proteins contained in ambient particulate matter are important environmental factors that aggravate airway hyperresponsiveness and airway inflammation in mice [29]. An ADS contains different amounts of β-glucan, which can induce airway inflammation [30, 31]. Thus, in addition to chemical substances, anthropogenic metal components, and sulfate, some proteins and β-glucan attached to ADS airborne particles may play important roles in the reduction of pulmonary function during ADS events.

Inhaled LPS is associated with airway neutrophil inflammation in patients with asthma and in healthy subjects [32–34]. Our results show that ADS airborne particles contain endotoxin, and the endotoxin concentration of ADS airborne particle was lower than that in LPS at 100 pg/mL. However, the IL-8 transcriptional activity induced by ADS airborne particle collected on April 23 and 24, 2012 and March 8 to 10, 2013 was significantly higher than that induced by LPS at 100 pg/mL. Endotoxin may augment IL-8 transcriptional activity in THP-G8 cells, in addition to other substances on ADS airborne particles that may induce IL-8.

Park et al. [10] and Yoo et al. [35] found a relationship between ADS events and PEF in Korean children with asthma, while Hong et al. did not find a significant relationship between ADS events and PEF in children without asthma [36]. Patients with allergic diseases may also be more sensitive to air pollution [37–39]. Therefore, in this study, we analyzed the data after adjustment for allergic diseases. This analysis showed that there was a significant decrease of PEF on ADS days in 2012 compared to 2013, regardless of the presence of allergic diseases. However, the number of subjects with each disease was too small to investigate the association of PEF with ADS. Further studies are needed to define the relationship between ADS and PEF in children with allergic diseases.

In this study, children recorded their PEF value after arriving at school but did not record their PEF value on weekends and public holidays. The ADS days March 9, 10, and 20, 2013 lacked PEF data because they were holidays. However, this intermittent missing data is statistically independent of the ADS events. Thus, it would not cause any serious bias in the results. Although it would raise a reduction of statistical power, the significant associations were still observed in the primary analyses.

There are several limitations in the study. First, we did not investigate diseases other than asthma, allergic rhinitis, allergic conjunctivitis, atopic dermatitis, and food allergies. Second, we were unable to diagnose asthma based on airway hyperresponsiveness to methacholine and reversible airflow limitation. In this study, some children were considered to have asthma, when in fact their wheezing may have been caused by respiratory tract infection or other diseases. However, wheezing caused by respiratory tract infection and other diseases is more common in children under 6 years old, and that is younger than those in our study [40]. Additionally, it is difficult to distinguish asthma and reactive airway disease based on the present diagnostic criteria. Third, we were unable to measure the individual amount of exposure to ADS. Fourth, we did not analyze the composition of the ADS airborne particles. Therefore, this study was not able to investigate which components of ADS airborne particles played important roles in reduction of pulmonary function during the ADS and which components induce IL-8. Further studies are needed to define these components.

5. Conclusion

We conclude that the effect of exposure to ADS on pulmonary function in school children differed among ADS events, and that enhancement of IL-8 transcriptional activity also differed among ADS airborne particles collected during the respective events. These findings suggest that substances attached to ADS airborne particles exacerbate pulmonary function of school children. Further studies are needed to identify the substances attached to the ADS airborne particles that play key roles in exacerbation of pulmonary function.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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