ACUTE EFFECTS OF SO_2 AND PARTICLES FROM A POWER PLANT ON RESPIRATORY SYMPTOMS OF CHILDREN, THAILAND

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Abstract. Epidemiological studies to evaluate the acute effects of ambient SO_2 on the respiratory health of children provide inconclusive results. A panel study to examine the association of short-term exposure to ambient SO_2 and respiratory symptoms of 196 children for a period of 107 days was conducted in Thailand. Generalized Estimating Equations were used to examine the association of daily variation of air pollution with daily respiratory symptoms. During the study period, SO_2 was not associated with respiratory symptoms in either asthmatics or non-asthmatics, whereas a 10 $\mu g/m^3$ increase in PM_{10} was modestly associated with increases of lower respiratory symptom incidence (OR=1.03, 95%CI=0.98, 1.09) and cough (OR=1.04, 95% CI=1.00, 1.08) in asthmatics. At the low ambient air pollution concentrations observed, particulate matter rather than SO_2 was associated on a $\mu g/m^3$ basis with acute daily respiratory symptoms.

INTRODUCTION

The health effects of exposure to sulfur dioxide (SO_2) and particulate matter are often evaluated jointly in epidemiological studies because these pollutants are the primary products of the fossil fuel combustion process and are usually present together as components of a complex mixture. The epidemiological association of short-term exposure to ambient SO_2 and particulate matter with respiratory symptoms has been investigated in children in many studies; however, these studies provide inconclusive results for the effects of ambient SO_2. Some of these studies demonstrated an increase in daily respiratory symptoms associated with SO_2 (Vedal *et al*, 1987; Roemer *et al*, 1993; Hoek *et al*, 1994; Neas *et al*, 1995) whereas other studies failed to show adverse effects (Braun-Fahrlander *et al*, 1992; Hoek *et al*, 1993, Peters *et al*, 1997; Roemer *et al*, 1998). Epidemiological studies on the independent relationship between sulfur dioxide and respiratory effects are limited by high correlations between SO_2 and particulate air pollution (Koenig, 2000). Most of the previous panel studies on the acute effect of short-term air pollution exposure on respiratory health were conducted in cold weather countries and were subject to some degree of limitation on exposure assessment. In winter, children usually spend a large portion of their time indoors, and the individual exposure estimation based on outdoor fixed-site ambient air concentrations might overestimate personal exposures. Studies of the short-term adverse effects of air pollution in tropical countries where the climate, population characteristics and environmental conditions differ from western countries are still limited.

Other studies reported that people with pre-existing respiratory illnesses were at risk for the adverse effects from exposure to air pollution resulting in hospitalization or death (Schwartz *et al*, 1992; Burnett *et al*, 1995). Asthmatic and symptomatic children are considered to be a group susceptible to the adverse effects of air pollution (Pope and Dockery, 1992). It is still not clear whether non-asthmatic children are also susceptible to ambient SO_2 and particulate matter.

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The present study was designed to evaluate the association between short-term exposure to daily variation in SO_2 and particulate air pollution with respiratory symptoms among asthmatic and non-asthmatic children in Mae Mo, Thailand. The study area is a community where the population is continually exposed to air pollution from a lignite-coal power plant and residential wood burning, where ambient SO_2 and particulate matter appear to be weakly correlated. Thus, the study area provided a good opportunity to evaluate the effects of ambient SO_2 separately from particulate matter.

MATERIALS AND METHODS

Study site and population

Mae Mo, a district in the Lampang Province in north Thailand, comprises a population of 30,000 in 4 sub-districts with 26 villages. Sixty percent of families work in agriculture. A lignite-coal power plant is located in the center of the district, and there are no other industries causing air contamination in the district and other districts nearby. Other sources of ambient air pollutants are from the burning of wood and dry plants for agriculture among villagers and from a lignite mine adjacent to the power plant. The study area included four adjacent villages, approximately 7 km to the south (Sob Pad, Sob Tern, Suan Pa Mae Jang, and Sob Mo) and one village 8 km to the north (Hau Fai) of the power plant. The study population was recruited from a total of 706 school children ages 6-14 years old who resided in the study area. General information on characteristics of the study population was obtained by interviewing the parents of the school children. The initial visit provided information on family background, medical history and respiratory problems including asthma. The parents were asked to complete a respiratory symptom questionnaire modified from the World Health Organization (WHO) questionnaire for children (Florey and Leeder, 1982). The children were considered suspected cases of asthma if they reported positive response to the following question: "Has your child had attacks of shortness of breath or wheezing during the past year?" The suspected asthmatic children were then given a physical examination by a local physician. The criterion that the physician used to designate asthmatic children was based on the history that the asthma symptom was relieved by taking a bronchodilator. Of 706 schoolchildren in the study area, 98 asthmatic children were identified and recruited, and 98 non-asthmatic children were randomly selected from the rest of the children who had no history of chronic diseases.

Acute respiratory symptoms

We recruited local nurses as our field staff to perform interviews. At the meeting place in each village between 15.00-17.00 hours, the nurses interviewed each child about his or her respiratory symptoms in the past 24 hours. The data collection period of 107 days in the winter season was conducted from October 1, 1997 to January 15, 1998. The two-page interviewer-administered questionnaire on the history of respiratory symptoms in the past 24 hours was modified from a questionnaire used in other studies (Ostro *et al*, 1993). The questionnaire included questions on cough, sore throat, wheezing, shortness of breath, phlegm, headache, runny nose, eye irritation, fever, absence from school, medication used in case of asthma attack and daily activities. The questionnaire was pre-tested prior to the study and modifications were made based on feedback by the interviewers.

Two combinations of symptoms used in the analyses were defined according to the definition modified from other studies (Schwartz *et al*, 1994). An upper respiratory symptom (URS) was defined as the presence of runny nose, stuffy nose or sore throat. A lower respiratory symptom (LRS) was defined as the presence of wheeze, chest tightness, shortness of breath, cough or phlegm production. Cough can be considered as either an upper or a lower respiratory tract symptom and was analyzed separately. An incident case was defined as a person who had a symptom on the current day but was symptom-free on the previous day. Daily incidence density rate was defined as the number of children with a new occurrence of symptoms on a given day divided by the number of children at risk on that day. The children at risk were those who were symptom-free on the previous day.

Air pollution and weather data

Daily outdoor air pollution data were obtained from the air-monitoring network in the area

around the power plant. SO_2 data were obtained from the air-monitoring network operated by the Electric Generating Authority of Thailand. Three air pollution-monitoring sites centrally located in the study areas were used (Sob Pad, Sob Mo and Hau Fai). The 24-hr average SO_2 concentrations were measured by a model 43 pulsed fluorescent SO_2 analyzer at all monitoring stations. $PM_{2.5}$ is of particular interest; however, there were numerous missing values for PM_{10} and $PM_{2.5}$ measured by the dichotomous samplers at the study site. We used multiple imputation techniques to estimate the missing values for $PM_{2.5}$ (Heitjan, 1997). We estimated the missing PM_{10} and PM 2.5 values from linear regression models using PM 2.5 as a dependent variable regressed on PM_{10} from beta-gauge and PM_{10} from Hi-volume for Sob Pad and on PM_{10} measured by Hi-volume for Hau Fai. The $PM_{10-2.5}$ were generated by subtracting the daily $PM_{2.5}$ from PM_{10}. The multiple imputation yielded five air pollution data sets for $PM_{2.5}$ and PM_{10}. The final statistical analysis of the relation between respiratory symptoms and air pollution was conducted for each imputed air pollution data set, which yielded a set of five coefficients and variances for each regression model. We combined these five estimates and variances into a summary point estimate and variance using Rubin's formulas (Rubin, 1987). These summary coefficients and variances were then used for calculation of the odds ratio and its 95% CI.

Meteorological data including temperature, wind speed and wind direction were obtained from each monitoring station. For wind direction, a variable indicating the number of hours in a day that the residences were downwind from the power plant was also created. Daily relative humidity and dew point were obtained from measurements at the airport which is located approximately 30 km to the southwest of the study area.

Statistical methods

Descriptive statistics were used for all variables, including correlations between SO_2, particulate air pollution, temperature and relative humidity. We used smoothed plots from generalized additive models (GAM) to examine the relation of the respiratory symptoms against explanatory variables of time and temperature. We used logistic regression to analyze the association between the binary response of respiratory symptoms and air pollution data. Individual observations on each day were used as the unit of analysis. Generalized Estimating Equations (GEE) were used to accommodate potential serial correlation. Sensitivity analysis for choices of working correlation matrix was examined using Akaike's Information Criterion (AIC). Consequently, a first-order autocorrelation (AR1) working correlation matrix was chosen.

We developed base models for URS, LRS and cough incidence. To control for time trend, weekday or weekend, temperature and relative humidity, base models containing these potential confounding factors were created. After comparing the fit of the base models, we included only time variables in most of the base models; temperature variables were not included because they were not significant in improving the models and not a significant predictor of respiratory symptoms. Variables for wind speed and wind direction were also evaluated but did not behave as important predictors of respiratory symptoms. We entered one air pollution variable at a time into the base model initially, then entered SO_2 and particulate air pollutants in the same model. We examined daily 24-hr SO_2 concentrations on the same day, as well as lagged measures up to three days and 3-day (defined as a mean of the concurrent day and previous 2 days) to 5-day averages. We included air pollution as a linear term, and the results of the analysis present the odds ratio with a 95% CI for respiratory symptoms in relation to a 10 $\mu g/m^3$ increase in air pollution. To examine the exposure-response relationship of air pollution and respiratory symptoms on a continuous scale, GAM models with a non-parametric smoothing spline function of air pollutants were used to plot the logit of respiratory symptoms against air pollution concentrations controlling for smooth functions of time and temperature.

Stratified analysis was used to evaluate the relationship of respiratory symptoms and air pollution in asthmatics and non-asthmatics. To compare the effect between both groups, we analyzed the data including both groups in the same models in GEE with interaction terms of air pollution and group variables. The GEE analyses was conducted using PROC GENMOD in SAS (version

8.1) and the GAM was implemented with S-plus 2000.

RESULTS

Air pollution

Table 1 shows the distribution of air pollution over 107 days during the study period. There was a period of low SO_2 and a few days with peak SO_2 levels but the daily average did not exceed 128 μg/m^3. The 24-hr average SO_2 levels were below the Thai Ambient Air Standard (300 μg/m^3). The 24-hr average PM_{10} levels were also below the Thai Ambient Air Standard (120 μg/m^3) on most days. During the study period, there were low correlations between SO_2 and particulate air pollution (r=0.08). Temperature was negatively correlated with particulate air pollution.

The study cohort

There were 48 girls and 50 boys in the non-asthmatic and 54 girls and 44 boys in the asthmatic group. The age distribution of children was similar in both groups with a mean of 11 years (range from 6 to 14 years). These children provided for a total of 19,873 child-days of observation over 107 days of the study or 93.8% of the total possible child-days of observation. Missing child-days were due to no data collection during the New Year period (December 31-January 2) for 588 child-days and to failure of children to participate for 615 child-days. The analysis was restricted to 18,519 child-days, because of missing air pollution data for 948 child-days and excluding children who went out of the study area for more than 4 hours on a day (406 child-days).

Respiratory symptoms

The incidence rates of symptoms per 100 children at-risk varied over time during the study period. In general, daily symptom incidence in asthmatics were higher than those in non-asthmatics, with the median incidence of URS, 33.3 *vs* 23.7; LRI, 5.5 *vs* 2.2; and cough, 8.7 *vs* 4.5 per 100 at risk children.

SO_2 and respiratory symptoms

The incidence of URS, in the one-pollutant and two-pollutant models, was not associated with SO_2 (Table 2). No positive association between lagged exposure to SO_2 and incidence of URS was observed in asthmatics. For non-asthmatics, there was a small positive association of lagged exposure to SO_2 with the incidence of URS (Table 3), but the small association disappeared in the two-pollutant models after controlling for PM_{10}.

In asthmatic children, an increase of 10 μg/m^3 in SO_2 was, however, weakly associated with

Table 1
Percentile distribution of 24-hr mean air pollution, October 1, 1997-January 15, 1998, Mae Mo, Thailand.

	Days (no.)	10%	25%	50%	75%	95%	Max	Min	Mean
Sob Pad									
SO_2 (μg/m^3)	107	2.61	2.76	3.93	14.18	58.66	99.01	1.85	14.05
PM_{10} (μg/m^3)	104	13.03	18.78	28.40	41.74	55.06	153.25	6.63	31.92
$PM_{2.5}$ (μg/m^3)	104	9.0	13.04	21.94	33.39	43.64	119.21	4.52	24.77
Temperature (°C)	107	21.84	22.69	24.35	25.43	27.22	27.91	18.90	24.24
Sob Mo									
SO_2 (μg/m^3)	105	0.24	2.07	5.23	21.26	52.12	128.01	0	16.99
PM_{10} (μg/m^3)	104	12.79	18.62	28.90	44.15	65.63	121.80	4.23	33.64
$PM_{2.5}$ (μg/m^3)	104	9.46	13.78	21.38	32.67	48.57	90.13	3.13	24.89
Temperature (°C)	107	22.6	23.4	24.6	26.5	27.7	28.9	20.3	24.99
Hua Fai									
SO_2 (μg/m^3)	106	5.23	8.18	17.77	32.6	50.6	120.6	2.62	24.72
PM_{10} (μg/m^3)	97	13.34	20.15	28.38	44.52	99.95	113.33	6.98	37.45
$PM_{2.5}$ (μg/m^3)	97	8.95	13.80	20.96	31.66	60.59	106.86	3.67	26.27
Temperature (°C)	106	21.46	22.22	23.57	24.84	26.12	26.94	19.39	23.63

LRS. The association decreased slightly for LRS incidence (OR=1.01) in the two-pollutant models after controlling for particulate air pollution (Table 2). The small positive association of LRS and cough incidence was also found with lagged exposure to SO_2 on 2-day to 5-day averages, all the 95% CIs also included the null value, however. For non-asthmatics, there was negative association between same-day or lagged exposure to SO_2 and LRS incidence. Exposure to SO_2 was not associated with cough in either asthmatics or non-asthmatics (Table 2).

The results of the GAM fitting of the logit of respiratory symptoms against SO_2 concentrations showed that most of the plots exhibited no upward exposure response relationship between SO_2 and respiratory symptom incidence. In both asthmatics and non-asthmatics, the effects on URS and cough incidence did not increase, except for LRS incidence in asthmatics, which tended to increase at SO_2 concentrations >100 μg/m^3, the 95%CI was wide, however, and included the null value (Fig 1).

Association between particulate air pollution and respiratory symptoms

For asthmatic children, a 10 μg/m^3 increment in PM_{10} was associated with a 3% increase (OR 1.03, 95% CI= 0.98, 1.09) of LRS incidence after adjusting for SO_2. Daily 24-hr average PM_{10} was also associated with a 4% increase in incidence (OR 1.04, 95%CI=1.00, 1.08) of cough after adjusting for SO_2 (Table 2). Both $PM_{10-2.5}$ and $PM_{2.5}$ appear to have effects on incidence of both LRS and cough. An increase of 10 μg/m^3 PM_{10} was associated with a 3% increase (OR 1.03,

Table 2
Odds ratios for respiratory symptoms associated with 10 μg/m^3 increase in air pollution on same day among asthmatic and non-asthmatic children, adjusted for time and humidity.

	Asthmatic			Non-asthmatic		
	URS OR(95% CI)	LRS OR(95% CI)	Cough OR(95% CI)	URS OR(95% CI)	LRS OR(95% CI)	Cough OR(95% CI)
One pollutant models						
SO_2	0.99 (0.96, 1.02)	1.02 (0.98, 1.06)	1.00 (0.97, 1.04)	1.01 (0.98, 1.05)	1.0 (0.94, 1.06)	0.99 (0.95, 1.03)
PM_{10}	1.03 (0.99, 1.07)	1.04 (0.99, 1.09)	1.04 (1.00, 1.07)	1.04 (0.99, 1.08)	1.0 (0.93, 1.07)	0.99 (0.94, 1.05)
$PM_{10-2.5}$	1.04 (0.93, 1.17)	1.09 (0.95, 1.26)	1.08 (0.96, 1.21)	1.05 (0.99, 1.19)	0.90 (0.72, 1.11)	0.95 (0.81, 1.11)
$PM_{2.5}$	1.04 (0.99, 1.09)	1.05 (0.98, 1.2)	1.05 (0.99, 1.10)	1.03 (0.96, 1.09)	1.02 (0.93, 1.10)	1.00 (0.93, 1.07)
Two pollutant models						
SO_2	0.98 (0.95, 1.02)	1.01 (0.97, 1.06)	1.00 (0.96, 1.03)	1.01 (0.97, 1.05)	0.99 (0.93, 1.05)	0.98 (0.94, 1.03)
PM_{10}	1.03 (0.99, 1.07)	1.03 (0.98, 1.09)	1.04 (1.00, 1.08)	1.04 (0.99, 1.08)	1.0 (0.93, 1.07)	1.00 (0.95, 1.05)
Two pollutant models						
SO_2	0.98 (0.95, 1.02)	1.01 (0.97, 1.06)	1.00 (0.96, 1.03)	1.00 (0.96, 1.04)	1.0 (0.94, 1.06)	0.98 (0.94, 1.03)
$PM_{10-2.5}$	1.05 (0.93, 1.18)	1.08 (0.94, 1.25)	1.08 (0.95, 1.22)	1.06 (0.98, 1.19)	0.89 (0.72, 1.11)	0.96 (0.82, 1.12)
Two pollutant models						
SO_2	0.98 (0.95, 1.02)	1.01 (0.97, 1.06)	1.00 (0.97, 1.04)	1.01 (0.97, 1.05)	0.99 (0.93, 1.05)	0.98 (0.94, 1.03)
$PM_{2.5}$	1.04 (0.99, 1.10)	1.05 (0.98, 1.10)	1.05 (0.99, 1.11)	1.03 (0.97, 1.09)	1.02 (0.93, 1.11)	1.00 (0.93, 1.07)

Table 3
Odds ratios for respiratory symptoms associated with 10 μg/m³ increase in lagged exposure to SO_2, among asthmatic and non-asthmatic children, adjusted for time and humidity.

	Asthmatic			Non-asthmatic		
	URS OR(95% CI)	LRS OR(95% CI)	Cough OR(95% CI)	URS OR(95% CI)	LRS OR(95% CI)	Cough OR(95% CI)
SO_2						
1-day lag	0.98 (0.95, 1.01)	1.00 (0.96, 1.05)	1.01 (0.97, 1.04)	1.02 (0.98, 1.06)	0.90 (0.83, 0.97)	0.96 (0.92, 1.01)
2-day lag	1.00 (0.97, 1.03)	1.03 (0.99, 1.07)	1.02 (0.98, 1.05)	1.00 (0.96, 1.04)	0.96 (0.90, 1.02)	0.98 (0.93, 1.02)
3-day lag	0.99 (0.96, 1.03)	1.02 (0.98, 1.06)	1.02 (0.98, 1.05)	1.01 (0.98, 1.05)	0.98 (0.92, 1.04)	1.00 (0.96, 1.04)
3-day aver	0.98 (0.94, 1.03)	1.03 (0.97, 1.09)	1.01 (0.97, 1.06)	1.02 (0.97, 1.07)	0.91 (0.83, 1.00)	0.95 (0.89, 1.01)
4-day aver	0.97 (0.93, 1.03)	1.04 (0.97, 1.11)	1.02 (0.97, 1.08)	1.02 (0.96, 1.09)	0.90 (0.81, 1.00)	0.95 (0.88, 1.02)
5-day aver	0.96 (0.91, 1.02)	1.05 (0.97, 1.13)	1.03 (0.97, 1.09)	1.04 (0.98, 1.11)	0.89 (0.80, 1.00)	0.94 (0.87, 1.02)

95% CI= 0.99, 1.07) in URS incidence.

For non-asthmatic children, PM_{10} was positively associated with URS incidence; however, no consistent association of PM_{10} with LRS and cough was observed among non-asthmatic children.

Smoothed plots of the incidence of respiratory symptoms versus particulate air pollution did not show a clear exposure-response relationship for $PM_{10-2.5}$ in either asthmatics or non-asthmatics. For effects of $PM_{2.5}$ on asthmatics, incidence of LRS and cough slightly increased and became flat after $PM_{2.5}$ > 80 μg/m³; however, the confidence intervals are wide due to small numbers of observations at these levels (Fig 2).

Additional analyses were conducted to examine the sensitivity of the effect of model specification and outliers. The sensitivity analysis was done for LRS in the asthmatic group. A model including temperature showed similar results to models without temperature. This procedure indicated that including or excluding temperature in the models did not affect the estimates. Similar results were obtained with nearly identical estimates and statistical confidence levels for SO_2 obtained from the analysis using each day of observation as unit of analysis. Analysis of a model with a different definition for incidence, defined as a child having two prior symptom-free days, (Hoek and Brunekreef, 1993) did not change the effect estimates for particulate air pollution. To evaluate the effect of a period of low SO_2 concentrations on the association between SO_2 and respiratory symptoms, days with $SO_2 \leq 2.62$ were excluded in a fifth model. The results showed similar estimates for SO_2 to the original model (Table 2), suggesting the results were not influenced by days with extremely low SO_2 values.

DISCUSSION

The present study shows no clear association of short-term exposure to ambient SO_2 with respiratory symptoms in either asthmatic or non-asthmatic children. For asthmatic children, particulate air pollution appears to have a small acute effect in increasing respiratory symptoms, especially LRS and cough. A 10 μg/m³ increase in PM_{10} was modestly associated with increases in the incidence of lower respiratory symptoms in asthmatic children. Asthmatic children were more likely to be susceptible to particulate matter than non-asthmatic children were. For non-asthmatic children, there was no consistent evidence for respiratory symptoms associated with increased ambient SO_2 or particle concentrations, and some of

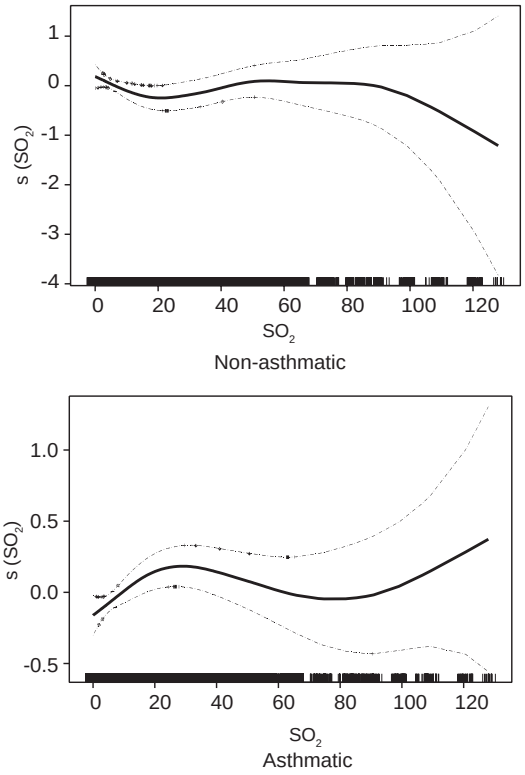

Fig 1–Logit of incidence of LRS *vs* SO_2.

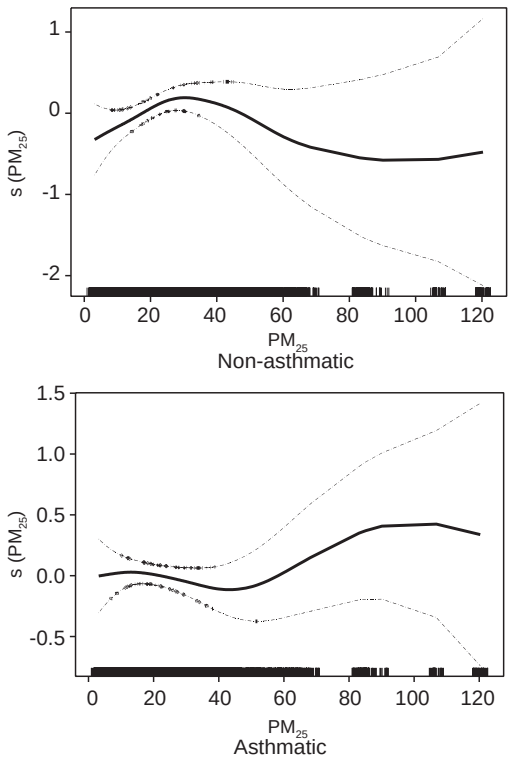

Fig 2–Logit of incidence of cough *vs* PM_{25}.

the associations were opposite in direction to those hypothesized. The results of the present study appear to be robust, as sensitivity analyses of different models yielded similar results.

Epidemiological studies of associations between short-term exposure to daily ambient SO_2 and respiratory symptoms have been conducted in the US and Europe with inconsistent results, which may be due in part to the relatively low levels of SO_2 in the study area. This might suggest that the concentrations were below a threshold effect. Chamber studies showed that asthmatics were more sensitive at lower levels of SO_2 than non-asthmatics (Sheppard *et al*, 1981; Hosrtman *et al*, 1988). In the symptomatic asthmatics, however, bronchoconstriction develops after exposure to SO_2 at 0.25 ppm (715 mg/m^3). This concentration is far higher than that observed in ambient air. Another factor related to the lack of association might be that the asthmatic children included in the present study might have had only mild asthmatic conditions. Because the definition of asthma in the present study was based on reporting a history of asthma during the past 12 months, this procedure might include a large number of children with mild asthma who were less susceptible to air pollution than clinically asthmatic children. Previous studies (Roemer *et al*, 1993; Peters *et al*, 1997) also reported that children with underlying chronic respiratory symptoms were more susceptible to low levels of SO_2.

The modest association between particulate air pollution and respiratory symptoms observed in the present study is consistent with other studies (Dockery and Pope, 1994). In asthmatic children, a 10 μg/m^3 increase in PM_{10} was associated with a 3% increase in LRS (OR=1.03, relative effect=0.03) and a 4% increase in cough incidence (OR=1.04, relative effect=0.04) which translated into absolute risks of 0.16% and 0.35% respectively. It should be noted that the incidence of URS in the study population was relatively high (median daily incidence was 33% in asthmatics

and 24% in non-asthmatics), thus the odds ratios derived from logistic regression models may overestimate the true relative risk (RR). Given the average incidence density rates of URS, we could estimate the RR from OR (Walter, 2000) to be 1.02. Dockery and Pope (1994) combined weighted averages from several studies in the Netherlands, six US cities and southern California. The estimated effect of a 10 $\mu g/m^3$ increase in daily mean PM_{10} concentration was a 3.0% increase in LRS (relative effects) and a 0.7% increase in URS incidence. Studies in the Netherlands (Roemer *et al*, 1993), two Swiss cities (Braun-Fahrlander *et al*,1992), and Uniontown, Pennsylvania (Neas *et al*, 1995) also gave a weighted mean effect of 1.2% increase in cough associated with 10 $\mu g/m^3$ increase in PM_{10}. In animal studies, exposure to particle air pollution irritates the respiratory tract and causes inflammation of the epithelial surface of the tracheobronchial tree, resulting in an increased severity of respiratory infections (Kodavanti and Costa, 1999; Thomas and Zelikoff, 1999). Sustained exposure to air pollution might produce chronic inflammation, and the infection might become more severe because of further damage to the inflamed airways.

The weak association between air pollution and respiratory symptom incidence may reflect that at the low concentration levels, the effects were small. The effects of air pollution also had to compete with other risks such as respiratory infection. The present study observed a relatively higher incidence of respiratory symptoms especially URS relative to other studies in the US and Europe. Respiratory illness is one of the most common causes of morbidity among Thai children, but there was not any report of respiratory outbreak during the study period. Thus, for the population in the present study, the effects of air pollution might be small when compared to other causes such as infection. Further study is needed to examine this issue.

The weak association was not likely to be influenced by temperature. Because the study area had moderate temperature, the average daily temperature and temperature within a day did not vary much during the study period (20°-29°C), therefore having little effect on respiratory health. When we analyzed the data with temperature in the models, the results from the analysis did not materially change the effect estimates.

There were some potential limitations to the present study. First, the levels of ambient air monitoring might not represent the individual exposures, because the use of a few monitoring sites may not adequately reflect the variability between individuals. However, since the area in the present study is relatively small and participants did not reside far from the monitoring stations, these factors would reduce the spatial variability. Using fixed-station daily average should be appropriate for estimating average exposure at the individual level. Another limitation is related to missing data for PM_{10} and $PM_{2.5}$. This missing data is likely to be at random; however, the imputation values for $PM_{2.5}$ and $PM_{10\text{-}2.5}$ possibly have some degree of error. This exposure assignment error might be a possible source of bias toward the null.

Misclassification of reporting daily respiratory symptoms in children is also possible. It could be biased if the reporting of symptoms were associated with air pollution concentrations. It is not likely, however, that the participants were aware of the air pollution levels. If there were some misclassification, it would be at random and would bias the results toward the null. The results of the study also were not likely to be influenced by the interviewer teams for children in different villages. In the data collection procedure, all the interviewers were trained and asked to follow the protocol strictly. Nevertheless, a variable for village, indicating a group of children with the same interviewer, was included in the models. There was no significant difference in effect among the villages.

The strength of this study is that the study area offered a good opportunity to evaluate the effect of SO_2 and particulate matter, because there were no other known pollutants to confound the study. Nitrogen oxide, which mainly comes from motor vehicle exhaust, was barely detected, because the study area is rural with little traffic. Ozone concentrations were also reported as very low. In addition, SO_2 and particulate air pollution were not strongly correlated. Therefore, the main known pollutants in the study, SO_2 and particles appear to be appropriate for evaluation of their effects.

Conclusion

At low levels of daily ambient SO_2 in the study area, SO_2 was not clearly associated with the daily incidence of respiratory symptoms. Conversely, on a microgram per cubic meter basis, particulate air pollution was found to be modestly associated with the incidence of lower respiratory symptoms and cough.

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