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Original Articles

Effects of Air Pollution on the Respiratory Tract of Children

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Summary. The effects of air pollution on pulmonary function and respiratory status was evaluated in 1,626 school aged children from a European Alpine region. Based on measurements of SO_2, NO_2, and O_3 as well as infrared imaging and lichen mapping, three zones of exposure were defined. Results of standardized respiratory questionnaires, medical examinations, and lung function tests were compared among the children in the three different exposure zones. After controlling for age, sex, height, socioeconomic status, and exposure to environmental tobacco smoking, areas of increased SO_2 and NO_2 as well as areas of increased ozone (max. half hourly mean value, 146 ppb) were significantly associated with decrements of forced expiratory volume in 1 s (FEV_1) and flow rates at 50 and 75% of vital capacity (FEF_{50}, FEF_{75}). In addition, areas with increased ozone had a higher prevalence of asthma In all regions, maternal smoking was associated with reduced expiratory flow rates and increased prevalence of asthma. The results provide evidence that outdoor pollution and exposure to passive smoking are risk factors for childhood respiratory health. **Pediatr Pulmonol. 1993; 15:68–74.** © 1993 Wiley-Liss, Inc.

Key words: Respiratory health; pulmonary function; ozone; passive smoking; socioeconomic status; air pollution.

INTRODUCTION

A number of cross-sectional surveys have demonstrated that air pollution affects the respiratory status of children. Most studies reported an increased frequency of acute and chronic pulmonary problems associated with air pollution.[1–6] Other studies documented a decreased pulmonary function after long-term exposure to air pollution. Zapletal[7] observed a significant reduction of forced expiratory flow at 50% (FEF_{50}) and 75% of vital capacity (FEF_{75}) in school children living in an area with increased concentrations of SO_2 and dust particles. In the Chattanooga Study, Shy et al.[8] found a weak association between decrements in forced expiratory volume at 0.75 seconds ($FEV_{0.75}$) and residence in regions with higher NO_2 pollution. Summer camp studies reported statistically significant correlations between a decline in various lung function parameters and moderately elevated ozone concentrations.[9,10] Kinney et al. in Six Cities Study[11] found decrements in FVC and FEF_{75} in connection with relatively low levels of O_3 (78 ppb).

This study in Austria was designed to determine the effects of air pollution on the respiratory tract of school children in three well-defined environments. Possible confounders such as environmental tobacco smoking (ETS) and socioeconomic status (SES), which may affect the association, were taken into account. Measurements of exposure variables included biological indicators (infrared imaging and lichen mapping) in addition to the standard assessment of air pollution. We tried to test the hypothesis of an association of lung disease and chronic air pollution, mainly by measuring the lung function.

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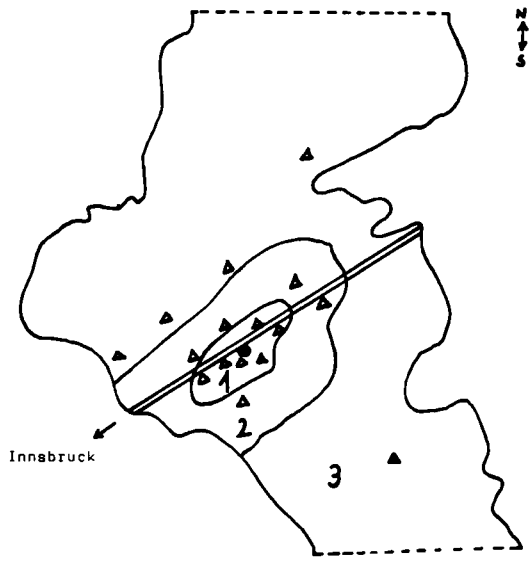

Fig. 1. Geographic location of the different zones. =, highway; △, emittent; •, monitoring station.

MATERIALS AND METHODS

Measurements were made in the Austrian Tyrolian Alps from January to April 1989. The area under investigation is at an altitude between 500 and 1,200 m. It is an area of 1,200 km^2 with different levels of exposure to air pollutants. Therefore the area is divided into three different study zones (Fig. 1).

Zone 1 has a copper recycling plant that is responsible for most of the emissions. In recent years atmospheric sulfur, heavy metals, and dioxin were excessive. After the installation of a washer in 1986, SO_2 was reduced by 0.07 mg/m^3 to 0.02 mg/m^3 annually. SO_2 was monitored using a UV Fluorescence Analyser Monitor Lab (ML) 8850. In Zone 1 a higher concentration of NO_2 adds to the pollution mentioned above. Ambient NO_2 levels were measured by passive samplers (Palmes tubes). Zone 3, at a higher altitude, is characterized by high ozone levels with peaks of 146 ppb and with an over-the-year average of 52 ppb. Ozone was measured by the chemiluminescence method (ML 8410 Bendix 8002). In Table 1 levels of SO_2, NO_2, and O_3 are presented. Mean ozone levels for different altitudes are shown in Figure 2. Furthermore, infrared enhanced color photographs obtained by aeroplane, and lichen mapping[12] methods considered to be indicative of effects of air pollution on vegetation showed a high degree of dead or dying needle trees in Zone 1, whereas vegetation in Zones 2 and 3 was less damaged.

School children, 6 to 15 years of age were enrolled, excluding those having lived for less than 3 years in a given area. Respiratory health status was assessed by means of a questionnaire, medical examination, and pulmonary function tests. The questionnaires completed by the parents were used to identify respiratory tract symptoms and respiratory diseases in the subject's medical history. Concerning asthma, the diagnosis had to be confirmed by a physician; medication and attack rates in the preceding 12 months were evaluated.

Efforts were made to document the degree to which each individual had been exposed to indoor pollution by assessing the ETS (number of smokers, the smokers' relation to the child, and the daily number of cigarettes the child is exposed to) and the type of heating system (central heating, type of stove). Questions about the level of education of the parents assessed the SES. Children were given a medical examination including lung auscultation, palpation of cervical lymph nodes, and visualization of the tonsils. Lymph nodes exceeding 5 mm and tonsils exceeding the pharyngeal ring were classified as enlarged. Methods of examination were standardized and the reliability of the clinical evaluations was established during a pilot study. Children who had respiratory tract infections within 3 weeks of examination were excluded from the study. The child's smoking history was elicited. Flow–volume curves were obtained using a computerized pneumotachograph (Multi Spiro PC Medical Equipment Designs, Inc). By means of a special software permitting the display of a birthday cake with burning candles on the computer screen (the stronger the expiration, the easier it was for the child to blow out the candles) good cooperation could be obtained. Measurements were performed by trained physicians. We used the testing standards of the American Thoracic Society.[13]

Statistical Methods

For statistical analysis the computer packages SPSS[14] and SAS[15] were used. Raw lung function data were transformed into values standardized by sex and height, using the regression formulas of Zapletal.[16] Analysis of covariance[17] was used to test the effect of air pollution on lung function in the three zones adjusting for age, sex, ETS, SES, and type of heating. P values less than 0.05 were regarded as statistically significant. The effect of the differently polluted zones on the prevalence of low FEF_{75} values, respiratory symptoms, and diseases was tested using the logistic regression method,[18] adjusting for the same confounders as described above. The prevalence ratio[19] between zones was determined using adjusted prevalences obtained from logistic regression.

RESULTS

The 1,626 children included in the survey represented a response rate of 88.4%. We noted no differences of response rate between zones; 50 children were excluded because they had been living in one zone for less than 3 years; 174 were excluded from the evaluation because

TABLE 1—Levels of SO_2, NO_2, and O_3 in 1988/89[a]

	Zone 1	Zone 2	Zone 3	National standard
SO_2 (mg/m^3)				
max. HMV	0.66	0.15	0.11	0.20
AMV	0.020	0.014	0.012	
NO_2 (ppb)[b]				
MMV	17.4	13.0	7.8	
O_3 (ppb)				
max. HMV	102	112	146	60
AMV	15	26	52	

[a]HMV, half-hour mean value; AMV, annual mean value; MMV, monthly mean value.
[b]During mos. 1–4, 1989.

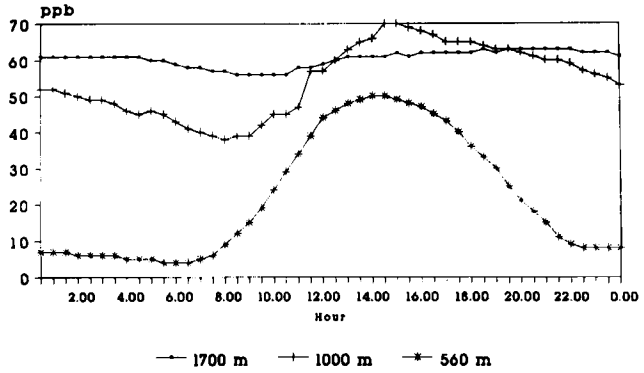

Fig. 2. The daily pattern of mean ozone levels during the summer of 1988 at different altitudes.

lung function tests could not be performed during a respiratory infection. Eighty-two children (5.2%) had invalid lung function tests. Exclusion rate was not larger in any particular zone; 1,320 children had pulmonary function tests that could be evaluated.

Mean values of pulmonary function tests in the three study zones are presented in Table 2. All parameters were better in the least polluted Zone 2. Comparing the flow rates at mid- to low lung volumes, there was a significant difference at $P = 0.029$ for FEF_{50} and at $P = 0.040$ for FEF_{75} between Zone 2 and Zone 1, the zone with the highest concentrations of SO_2 and NO_2. Lung function also declined pronouncedly in Zone 3 (with higher ozone levels). Children living in this area also frequently had values less than 70% predicted. The prevalence of distinctly reduced FEV_1 values (less than 80% predicted) was less than 1%, with no differences between zones.

The questionnaire results are presented in Table 3. The prevalence of coughing was increased in the highly polluted Zone 1, although the difference from the other zones was not statistically significant. Croup was also more common in Zone 1.

Prevalence of asthma was increased in the more polluted zones; in the zone of elevated ozone it was highest, with a P value of 0.004. The overall asthma prevalence was 4.1%, similar to other studies reported in the literature.[20] Higher prevalence of asthma among boys was confirmed in our data as well. To eliminate false-negative and false-positive reporting of allergy, children with discrepancies in the questionnaire concerning drug treatment and their reported asthma and pollinosis (defined as seasonal appearing of conjunctivitis and rhinitis) were RAST tested. Only one false-negative case was identified. Other potential indicators of morbidity from the questionnaire data were hours of school absence and the use of medical drugs, but these did not differ between zones. According to our medical evaluations, children in the most polluted area tended to have enlarged cervical lymph nodes.

Passive smoking was assumed to be one of the important indoor air pollution factors. A significant correlation between passive smoke exposure and respiratory morbidity (Table 4) and decreased lung function (Table 5) was noted in the case of maternal smoking. Multiple logistic regression also confirmed the increased risk of asthma with a prevalence ratio of 2.07 if the child's mother was a smoker. The prevalence of reported active smoking was too low (2%) to be evaluated. No association was found between pulmonary function and the type of heating. Our study population, however, used gas stoves, which are very rarely considered a risk factor for childhood respiratory disease.[21]

DISCUSSION

Evidence had been presented that maximal expiratory flow volume curves appear to be most valuable for assessing early effects of air pollution on respiratory function.[7,22,23] Our data demonstrate a clear relationship between the impairment of air flow rate at mid- to low lung volumes and residence in areas with elevated levels of airborne pollutants (SO_2, NO_2, O_3). Using the cut-off point established by Hibbert,[24] we assumed that flow rates lower than 70% of the predicted value document a clinically relevant impairment of lung function.

The lack of agreement between results of lung function and reported symptoms and diseases is remarkable. This

TABLE 2—Lung Function (mean ± SD, % of Predicted) in the Three Different Zones (Adjusted for ETS, SES, Heating, Age, and Sex)

	Zone			Significance	
	1 (n = 164)	2 (n = 526)	3 (n = 630)	*P* 1 vs. 2	*P* 2 vs. 3
FVC	99.5 ± 10.5	102.0 ± 10.1	101.4 ± 10.6	0.007	n.s.
FEV_1	105.8 ± 11.0	108.9 ± 11.1	107.2 ± 11.4	0.002	0.012
PEF	107.0 ± 15.9	109.8 ± 16.3	107.9 ± 16.5	n.s. (0.06)	n.s. (0.06)
FEF_{50}	101.6 ± 20.0	105.9 ± 22.3	102.3 ± 21.1	0.029	0.001
FEF_{75}	94.7 ± 25.0	100.7 ± 28.8	94.9 ± 26.0	0.040	0.001
FEF_{75}[a] (< 70% pred.)	13.9	10.5	15.8	n.s.	0.012

[a]The numbers for FEF_{75} values below 70% predicted, under the columns for Zone 1, 2, and 3 indicate prevalence %.

TABLE 3—Prevalences of Respiratory Diseases and Symptoms in the Three Zones (Adjusted for ETS, SES, Heating, Age, and Sex)

	Prevalence (%)			Prevalence ratio (*P* value)	
	Zone 1	Zone 2	Zone 3	1 vs. 2	3 vs. 2
Asthma	4.8	2.7	6.4	1.79 (n.s.)	2.36 (*P* = 0.004)
Male	7.4	4.4	8.5	1.69	1.93
Female	1.8	1.0	4.0	1.77	3.95
Pollinosis	2.5	2.1	2.6	1.18 (n.s.)	1.26 (n.s.)
Croup	13.3	10.8	11.1	1.22 (n.s.)	1.03 (n.s.)
Pneumonia	7.8	9.6	9.0	0.81 (n.s.)	0.94 (n.s.)
Chronic bronchitis	7.5	9.3	6.6	0.80 (n.s.)	0.71 (n.s.)
Coughing	13.6	12.0	9.3	1.13 (n.s.)	0.78 (n.s.)
Rhinitis	19.1	21.2	17.3	0.90 (n.s.)	0.82 (n.s.)

is consistent with results of Detels et al.[23] from the large cohort study in Los Angeles. These authors documented significantly worse lung function test results especially related to small airway function in a more polluted area, but were unable to differentiate between the cohorts on the basis of clinical symptoms.

Zapletal[7] had argued that functional changes in the small airways of "asymptomatic healthy" children can play an important role in development of chronic lung diseases. We speculate that the small (but statistically significant) decrements of flow rates at low lung volumes in our study population may be of prognostic relevance and may predispose these children to develop respiratory tract illness in the future. To prove this hypothesis we plan to continue the analysis in a longitudinal study.

Ozone, a major component of photochemical air pollution is currently under study in rural and alpine regions. These regions seem to have higher ozone levels, mainly as a result of less scavenging of ozone by nitric oxide originating from highway traffic.[25] The pattern of ozone levels during summer (Fig. 2) documents that at an altitude of 1,000 m ozone levels up to 60 ppb occur over 24 hours; due to relatively high annual mean values of 52 ppb in this region (Table 1), episodes of recovery are rare.

Our data indicate that residence in the area of elevated ozone increases the risk (prevalence ratio, 1.50) of low small airway-related lung function. Although there is some evidence of adaptive response to repeated ozone exposure in adults,[26] we assume that chronic exposure to moderate levels of ozone as well as short exposure to more elevated levels (higher than 120 ppb) explain the decrement in the children's lung function. The hypothesis of the risk associated with chronic ozone exposure is supported by recent findings of Schwartz,[27] who reported a highly significant negative correlation between annual ozone (and NO_2) concentrations and pulmonary function.

Although one would expect a lower prevalence of asthma at higher altitudes, due to less exposure to house

TABLE 4—Combined Effect of Maternal Smoking and Pollution on Respiratory Diseases (Adjusted for SES, Heating, Age, and Sex)[a]

	Mother non-smoker	Mother smoker	Prevalence ratio	Significance, *P* value
Asthma (%)	3.4	6.9	2.07	0.008
Zone 1	4.7	6.0	1.26	n.s.
Zone 2	2.1	4.9	2.32	0.08
Zone 3	4.9	11.1	2.25	0.022
URI (%)	17.5	21.4	1.21	n.s.
Zone 1	17.5	23.5	1.34	n.s.
Zone 2	20.5	23.2	1.13	n.s.
Zone 3	16.3	20.3	1.25	n.s.
LRI (%)	25.6	31.1	1.22	0.014
Zone 1	25.4	28.9	1.14	n.s.
Zone 2	27.0	32.9	1.22	n.s.
Zone 3	24.8	31.2	1.26	0.08

[a]URI, upper respiratory infection; LRI, lower respiratory infection.

TABLE 5—Combined Effect of Maternal Smoking and Pollution on Lung Function (Adjusted for SES, Heating, Age, and Sex)

	Mother non-smoker	Mother smoker	Prevalence ratio	Significance *P* value
Reduced FEF_{75}[a] (% of subjects)	12.9	14.3	1.11	n.s.
Zone 1	14.3	13.6	0.95	
Zone 2	9.8	12.7	1.30	
Zone 3	15.3	16.9	1.11	
FEF_{75} (mean % of predicted)	99.2	92.6		0.001
Zone 1	94.5	98.5		
Zone 2	103.2	93.9		
Zone 3	96.6	89.1		

[a]$FEF_{75} < 70\%$ of predicted.

dust mites,[28] we found an increased prevalence of asthma in this area. We assume, as documented previously,[29,30] that the higher ozone level increases bronchial hyperreactivity and is therefore responsible for the higher asthma rate. In Zone 1, SO_2 and NO_2 should also be considered as possible causes of bronchial hyperreactivity.[31]

The effects of exposure to parental smoking on children are particularly important. High frequency of respiratory infections and reduced pulmonary function related to ETS have been reported.[32–35] In other studies FEV_1 was significantly lower in children living in homes of smokers. Our results showing reduced FEF_{50} and FEF_{75}, which might indicate damage of small airways, are consistent with the data reported by Vedal[36] and recent investigations by Strachan.[37] The higher impact of maternal compared with paternal smoking has been previously reported.[33,36,38] Our findings of an increased risk of childhood asthma related to maternal smoking also correspond to other reports.[38,39] Hasselblad[40] showed a dose–response relationship between impairment of FEV_{75} and maternal smoking. We did not find that the daily number of cigarettes consumed by the parents had an influence on lung function or symptoms in the children under investigation. In more polluted areas there was no additional effect of passive smoking. A similar pattern has recently been observed by Teculescu,[41] who reported an influence of passive smoking in the nonpolluted zone. Unlike other investigators who found a negative correlation between lower SES and lung function,[36,42] we saw no such effect. In our study a positive association existed between higher SES and more frequent reports of respiratory symptoms and diseases. This might be explained by a higher reporting rate by better educated parents, something noted in a recent epidemiologic study on croup syndrome in 8,420 German children.[43]

In conclusion, we have found a significant association between the exposure to air pollution and decrements in lung function in a large cross-sectional study. Self-reported prevalence of asthma was associated with air pollution. Although reports of other respiratory symptoms and respiratory diseases failed to show this pattern, the evidence that chronic exposure to airborne pollutants may

result in decrements of lung function deserves serious consideration in the context of preventing airway diseases in adults.

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