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COMBINED EFFECTS OF OUTDOOR AND INDOOR AIR POLLUTION ON LUNG FUNCTIONS OF SCHOOL CHILDREN

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Abstract

To assess the impact of chronic exposure to air pollutants on lung function growth we calculated outdoor exposure at 87 elementary and high schools from meteorology and concentrations monitored at 9 stations for 5 years (SO_2 38 ± 20 , TSP 58 ± 19 , NO_2 $51 \pm 10 \mu\text{g}/\text{m}^3$). From 18106 pupils examined at school by methods standardized (ATS) 7561 boys and 7484 girls aged 6-16 (9.3 ± 2.6) years were selected who were non-smokers and free of respiratory infections or relevant disease at medical checkup. Passive smoking at home was assessed by parent's questionnaire and interview of child. Simultaneous regression analysis showed decreases of FEV_1 , PEF, MEF_{75} ($p < 0.001$) FVC, MMEF, MEF_{25} ($p < 0.01$) with SO_2 of preceeding winter season (half year mean and 98. percentile). It was not possible to separate the effects of TSP from SO_2 . Partial correlations with NO_2 were found significantly negative for MEF_{25} ($p < 0.001$) and MEF_{50} ($p < 0.01$). No effect was apparent for O_3 . Passive smoking was related to lower $\text{MEF}_{50,25}$, MMEF ($p < 0.001$) and MEF_{75} ($p < 0.05$); gas-cooking to MEF_{75} and PEF ($p < 0.05$). Canonical correlation analysis put more weight to TSP as indicator of outdoor pollution and passive smoking as indicator of indoor pollution. There were no significant interactions of outdoor and indoor pollution but rather additive effects on lung function. The subtle effects of air pollution may disappear over time with the decrease of outdoor pollutants, however, passive smoking is still a matter of concern because of signs of small airway dysfunction which may indicate a risk for chronic obstructive pulmonary disease later in life.

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Key words: Air pollution — Lung function — Passive smoking — School children — Spirometry

Introduction

The impact of chronic exposure to air pollutants on lung function growth of elementary and high school students has been investigated by cross-sectional studies on selected groups by different spirometric methods (Colley and Brasser 1981, WHO-CC 1990), but recent achievements in quality assurance and standardization of flow-volume-measurements (Aigner and Schindl 1986, ATS 1987, ATS 1991, Quanjer et al 1993), and thereby improved reference values (Neuberger et al 1994) enabled large-scaled screening for combined effects of outdoor and indoor air pollution which are more difficult to detect than the effects of higher outdoor pollution in earlier years.

Material and methods

In Linz (county capital of Upper Austria) two mobile teams made lung function tests in all elementary and high schools during the period from 1985 to 1990. To exclude influences from seasonal variations, each school was visited in winter as well as in summer half year. Informed consent by parents was obtained from 85% of the school-children. The examination consisted of an anamnesis, auscultation of the lung and lung function measurements including determination of flow-volume curves. Lung function measurements were done by standardized methods (Aigner and Schindl 1986, ATS 1987). Information on smoking and other sources of indoor pollution in

the child's home was obtained by parent's questionnaire and interview of the child.

To assess outdoor pollution, data from 9 stations monitoring total suspended particulates (TSP), sulphur dioxide (SO₂), nitrogen dioxide (NO₂) and ozone (O₃) were available. Each child was assigned the average value of each air pollutant obtained from the concentration isoline cutting the location of the school for the winter half year preceeding the spirogram.

From a total of 18106 pupils aged 6 to 16 years who were examined in 87 schools 7561 boys and 7484 girls were selected who were non-smokers, free of acute respiratory infections and other relevant acute diseases and who were assigned good compliance in lung function assessment by the physician (age $9,3 \pm 2,6$ years).

From three or more expirations at each test vital capacity (FVC), forced expiratory volume in the first second (FEV₁), peak expiratory flow (PEF), maximal expiratory flow at 75% of FVC (MEF₇₅), at 50% of FVC (MEF₅₀), at 25% of FVC (MEF₂₅) and the mid maximal expiratory flow (MMEF) were recorded.

Since for volume data (FVC and FEV₁) about 90% of the variance and for flow data (PEF, MEF₇₅, MEF₅₀, MEF₂₅ and MMEF) between 40% and 70% of the variance could be explained by anthropometric data (standing height, weight and age) the study of pollution effects has to take this predominant source of variation into account. This was accomplished by relating the measured values to reference values. The reference functions for volume measurements were power functions and for flow measurements exponential functions of height, weight and age separate for each sex (Neuberger et al 1994). Since the ratio of the lung function measurements and reference values were found to be log-normally distributed, the logarithm of this ratio was used as a metamer of the deviation from normal lung function.

These data together with indicators for indoor pollution (passive smoking and gas cooking) and the assigned outdoor pollution indicators were subjected to standard multivariate linear regression analyses with a tolerance value set to 0.01. Additionally, regression analyses including interaction terms for within and between indoor and outdoor pollution indicators were performed. Since none of these analyses yielded significant weights for the interaction

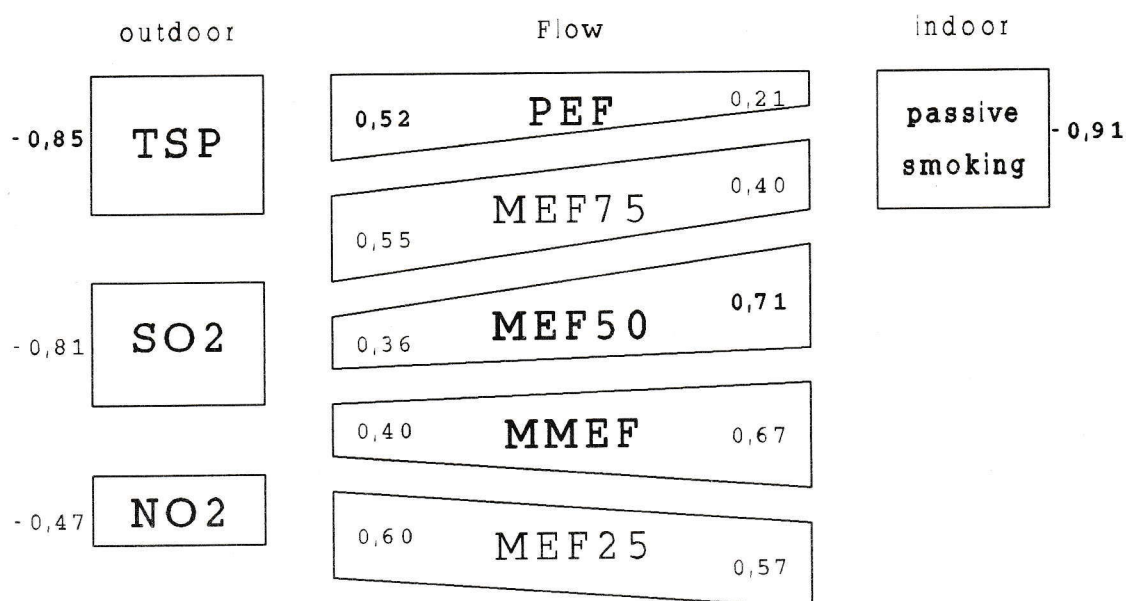
terms only the results from the standard linear regression are reported here.

Furthermore, a canonical correlation analysis was performed for the set of flow volume data (logarithms of the measurement-reference ratio) and the set of out- and indoor pollution indicators.

Results

The long term guideline values of 50 µg/m³ for TSP and SO₂ (WHO 1987) have both been exceeded at the most polluted school areas where at the beginning of the observation period half year means of 100 µg/m³ have been reached during winter (November to April). In winter pollution was generally higher than in summer except to ozone. For the latter and for summer air pollution in general no effects on lung function could be found. NO₂ showed (traffic-related) distributions different from (industry related) TSP and SO₂ and less decrease over time. Means and standard deviations for all schools and 5 winters were (in µg/m³): 58,5±19,23 for TSP, 38,3±20,02 for SO₂ and 50,7±10,48 for NO₂.

Table 1 gives results of regression analysis with best (ATS 1987) lung function test and pollution of preceeding winter half year. Decrease of FEV₁, PEF, MEF₇₅ ($p < 0,001$) and FVC, MMEF, MEF₂₅ ($p < 0,01$) correlated with SO₂ and decrease of PEF ($p < 0,01$) and MEF₂₅ ($p < 0,05$) with TSP, however, it was not possible to separate effects of SO₂ and TSP because of high correlation ($r^2 [0,53]$) of these pollutants in winter. Partial correlations with NO₂ were found significantly negative for MEF₂₅ ($p < 0,001$) and MEF₅₀ ($p < 0,01$). Passive smoking correlated significantly ($p < 0,001$) with all indicators of small airway dysfunction (MEF₂₅, MMEF, MEF₅₀) and with MEF₇₅ ($p < 0,05$). Cooking with gas correlated with MEF₇₅ ($p < 0,01$) and PEF ($p < 0,05$). Results with 98-percentiles of outdoor pollutants were very similar, because the latter correlated highly with half year means in winter. 1st root in canonical correlation analysis gave results which were redundant with multiple regression analysis, but 2nd and 3rd root revealed additional information (Figure 1). Among outdoor pollutants TSP was given the highest weight. Passive smoking as the most important indoor pollution proofed to be an independent influence. There were no significant interactions of outdoor and indoor pollutants.

Fig. 1. Weights for 2nd and 3rd root in canonical correlation analysis between indicators of air pollution and flow rates**Table 1.** Significant partial correlations between indicators of air pollution, forced expiratory volumes (FVC, FEV₁) and flows (PEF, MEF). Percentage of explained lung function variance (R²)

<i>r</i> _{part}	SO ₂	TSP	NO ₂	passive smoking	gas stove	R ² (%)
FVC	-0.027	-	-	-	-	0.2
FEV ₁	-0.036	-	-	-	-	0.3
PEF	-0.084	-0.036	-	-0.023	-0.020	1.6
MEF75	-0.051	-	-	-0.023	-0.029	0.7
MEF50	-	-	-0.026	-0.052	-	0.5
MMEF	-0.023	-	-	-0.044	-	0.5
MEF25	-0.026	-0.022	-0.039	-0.040	-	0.8

$p < 0.05, 0.01, 0.001$ (insignificant but negative correlations are indicated by minus sign)

From the weights given to the flow rates in Figure 1 it can be seen that outdoor pollution effects predominantly early components of the flow volume curves (PEF, MEF₇₅) while indoor pollution from sidestream smoke effects predominantly late components (MEF₅₀, MMEF, MEF₂₅). This is in accordance with results of multiple regression analysis showing passive smoking as an important factor of small airway dysfunction. The decrease in MEF₅₀ with increasing SO₂ was slightly more pronounced in passive smokers and in schools with high TSP, but none of the interactions tested was significant. By example the effect of NO₂ on MEF₂₅ was concentration dependent in areas with low SO₂ and TSP and more pronounced in passive smokers, but in areas with high SO₂ and TSP no dependency of the (lower) MEF₂₅ on NO₂ concentration could be found.

Discussion

Spirometric population studies usually test lung volumes (Lebowitz et al 1992, Qing et al 1993, Wang et al 1993), but Schwartz (1989) already suspected that PEF is a more sensitive indicator of the effects of air pollution. The late components of the flow volume curve could be even more important for the assessment of air pollution effects (Neuberger et al 1994), because they show not only acute, reversible declines (Dassen et al 1986) but also chronic effects of irritants such as NO₂, O₃ or sidestream smoke. Our study found small but significant impairments of these late components of the flow volume curve in healthy school children related to passive smoking and some components of outdoor pollution such as (traffic-related) NO₂.

We cannot exclude that the lack of interaction of pollutants on lung functions is due to the rough classification of exposure. Passive smoking was encoded primarily from child's interview, without consideration of room sizes, ventilation, etc. Lung function decrease was less pronounced in children living with 1 smoker than in children living with 2 or more smokers, however, misclassification might have flattened dose response relationships. For outdoor exposure misclassification could have been even more important, though most children lived within 1500 m of their schools, but their time spent outside was not registered and we have to assume that

misclassification of exposure led to a decrease of observed lung function effects, so that small interactions could not be proven any more, not even in this large sample. On the other hand large populations can only be surveilled by air monitoring networks and not by personal air samplers. Therefore medical studies should use the same air monitoring data which are used for air quality standards and alarm plans.

The subtle effects of outdoor air pollution which we found on lung function may disappear over time if TSP, SO₂ and NO₂ continues to decrease and summer O₃ stays low. Traffic related components are still a matter of concern, especially fine aerosols and NO₂. The decrease of NO₂ was much less pronounced than the decrease of SO₂ and because of its lower solubility in upper airways NO₂ penetrating to the deep lung may contribute to small airway disease as indicated by the correlations which we found with impaired MEF₂₅ and MEF₅₀. Most concerned we are about the effects of passive smoking on the late components of the flow volume curve. Even though these effects are small at school age they could be early symptoms of chronic inflammatory changes in small airways and indicate risk for later chronic obstructive pulmonary disease. 58 % of children examined have to live with one or more smokers and this percentage did not decrease during the observation period. Part of the urban-rural differences in lung function of school children could be due to passive smoking which has been registered in 47 % of Austrian households at the microcensus of 1986.

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