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Health effects of passive smoking in adolescent children

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Objective and design. To study the effects of passive smoking on health in adolescent schoolchildren by questionnaire, spirometry and laboratory investigations.

Setting. Two schools in the Vanderbijlpark area.

Participants. Seven hundred and twenty-six high-school children of average age 16 years.

Outcome measures. Lung function, serological abnormality or historical (i.e. questionnaire) evidence of ill health.

Results. The prevalence of respiratory illness before and after 2 years, respiratory symptoms, earache over the past year, low birth weight and learning difficulties were found to be significantly increased in the children exposed to parental smoke in the home, especially those exposed to maternal smoking. Spirometric and laboratory parameters, however, were not affected by passive smoking.

Conclusion. This study highlights the harmful health effects of passive smoking in the home.

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In children, passive smoking is known to increase both the occurrence of lower respiratory tract illnesses such as pneumonia and bronchitis, particularly early in life, and the frequency of chronic respiratory symptoms.[1-4] These latter effects are enhanced by simultaneous exposure to other air pollutants.[3] Children exposed to parental smoking also show a small reduction in lung function and in the rate of lung growth during childhood and *in utero*.[5-7] In older children and young adults (5 - 20 years), the prevalence of chronic cough, sputum production and/or persistent wheeze is also positively linked to parental smoking,[1,8] while the prevalence of asthma, atopy, increased serum levels of IgE, and the frequency of middle-ear diseases have also been described in some, but not all, studies.[8-11] Birth weight in the newborn is also negatively affected by maternal smoking, with documented reductions of up to 200 g.[12,13] Children who actively smoke have a reduction in the expected increase of forced expiratory volume in 1 second (FEV_1), and an increased prevalence of cough and breathlessness.[14]

These well-described adverse effects of passive smoking may be related to sensitisation of the migratory and oxidant-generating activities of circulating neutrophils.[15] Phagocyte-derived reactive oxidants (ROs) are immunosuppressive,[16] cytotoxic[17] and potentially carcinogenic[18] and are released in excess in some passive smokers.[15] The finding that lower serum levels of β-carotene and vitamin C (important anti-oxidant nutrients) occur in passive smokers supports this, and they may be indicators of excessive oxidative stress.[19,20] It is possible that industrial pollutants, which themselves often have oxidising potential, may also activate circulating neutrophils and cause harm in a similar fashion.[21]

In this study we investigated the effects of passive exposure to cigarette smoke in the home on the levels of two plasma anti-oxidative nutrients, vitamins C and E, and the development of smoke-mediated, pulmonary immunological or haematological abnormalities. In addition, we utilised data derived from a questionnaire and related this to domestic smoke exposure.

Study population and methods

This study was approved by the Ethics Committee of the University of the Witwatersrand and informed consent was obtained from the Transvaal Education Department, parents and children. The project formed part of the Vaal Triangle Air Pollution Health Study (VAPS).[4]

Study population. The total study population consisted of 726 children, 227 boys and 499 girls, aged 16.3 ± 0.1 (range 14 - 18) years, attending 2 different schools in Vanderbijlpark in the Vaal Triangle. Because parental consent was required, blood and urine samples were obtained from only 406 children. Lung functions were considered to be reproducible and accurate in 395 children. Socio-economic status was defined by the father's education, occupation and domestic income. If these data were unavailable, then those of the mother were used.

Questionnaire. A health questionnaire based on the Harvard Six Cities and Canadian Health and Welfare questionnaires was designed.[4] These were distributed following pilot testing, and prior to the biological sampling and lung function testing. Distribution was to all parents, regardless of whether they had agreed to blood testing or not, and more than 90% of questionnaires were returned. Children were also stratified according to exposure to environmental tobacco smoke.

Laboratory investigations

Luminol-enhanced chemiluminescence (LECL) was used to measure reactive oxidant generation by blood phagocytes;[22] vitamin C[23] and urinary cotinine levels[24] were measured using standard colorimetric procedures, while vitamin E levels were measured by high-performance liquid chromatography.[25] Leucocyte counts and measurement of serum IgE and salivary IgA were performed using standard haematological, radio-immunoassay and rocket electrophoresis procedures respectively.

Lung functions. These were performed with wedge bellows spirometers (Vitallograph: Buckingham, England) loaned by Medical Specialities (RSA). The tests were performed by medical doctors specially trained in the techniques of lung functions. At least two manoeuvres were performed with reproducibility criteria as per the recommendation of the American Thoracic Society.[26] The equipment was calibrated prior to use and daily thereafter. The children's height and weight were measured without shoes, jacket or jersey and recorded to the nearest 0.5 cm and 0.1 kg respectively by trained nursing sisters using an anthropometer. Predicted values were calculated according to the method of Knudsen *et al.*[27]

Statistics

Lung functions. The children were grouped according to parental smoking status and the results analysed using one-way analysis of variance.

Immunological and haematological data. The results of each series of investigations are expressed as the mean value with SEM in parenthesis for the total group of children. Since one of the schools had only a small number of boys, its data were omitted from the multivariate analysis (general linear models (GLMs)). Data on vitamin E levels were limited (N = 63 in the whole group) and likewise excluded from the latter. GLM analysis was performed as described previously.[28] Active smokers were excluded from the aforementioned investigations and studied separately in a matched case-control fashion[29] in which non-smoking controls were matched with the actively smoking children with reference to gender, age, socio-economic status and school.

Questionnaire data. In order to evaluate dependence in a two-way contingency table, Fisher's exact test (2 x 2 table) and the chi-squared test (2 x k table) were employed. Logistic regression analysis was used to investigate systematically the relationship between binary response variables and relevant combinations of independent variables.[30] To illustrate these relationships we utilised odds ratios (ORs), i.e. the ratio of the odds of having a certain symptom in the risk group with the odds of having the same symptom in the non-risk group. For each binary variable the appropriate model was established using stepwise logistic regression analysis; only statistically significant independent variables were used in the model. Since the one school had only a small number of boys, logistic regression analysis was only performed on the data from pupils of the other school. Actively smoking children were investigated via a matched case control study as above. McNemar's test of symmetry was used for these matched pairs to detect a change on the two opposite sides of the diagonal for the respiratory illness parameters.[31]

Results

Frequency of parental smoke exposure. The frequency of exposure to parental smoke in a population of 726 schoolchildren is shown in Table I. More than 60% of the study population were exposed, with 33.7% of the mothers and 48% of the fathers being current smokers. Of these, more than 99% smoked cigarettes. In the group where only the mother smoked, 19% were widowed and 29% divorced, separated or unmarried. Single-parent families were therefore more common in this group.

Table I. The frequency of parental smoke exposure in a population of 726 schoolchildren

	No.	%
Currently		
Parents and other smokers at home (total)	488	67.2
Mother only	95	13.0
Father only	198	27.3
Both parents	150	20.7
Other smokers at home	45	6.2
During pregnancy		
Mother	177	24.4

Lung functions. The spirometric values obtained in the present study did not differ significantly between control children and those passively exposed to cigarette smoke (data not shown). The mean values for FEV, and forced mid-expiratory flow rate (FEF_{25-75}) in the control, non-exposed children (147 in the group) were 106.7 ± 10.4 and 114.7 ± 23.8 respectively (mean ± SD). All measured parameters including FEV, and FEF_{25-75} were within normal limits for the whole group.

Laboratory investigations

Cotinine levels. The urinary cotinine levels in non-exposed children and those passively exposed to parental smoking were 2.3 ± 0.1 µM and 2.9 ± 0.1 µM respectively (P = 0.0007). With histories and urinary cotinine levels of more than 10 µM as markers of active smoking, 43 of 406 (10.5%) schoolchildren were identified as active smokers. The average cotinine level in this group was 51 ± 4 µM. Of the children who actively smoked, 82% came from households in which one or both parents smoked.

Immunological and haematological data. The results of the haematological and selected immunological investigations are shown in Table II. The mean values for all the tests fell within the normal ranges for 16-year-old children. (These results exclude those of active smokers.)

Table II. The immunological and haematological profiles of and cotinine levels in 363 non-smoking teenage school children in Vanderbijlpark

Parameters	Schoolchildren	Normal range
Neutrophil LECL (mV^{-1})	273 (11)*	94 - 441 (271)†
Total leucocyte count (cells x 10^6/ml)	7.3 (0.1)	4.9 - 9.0 (7.0)
Neutrophil count (cells x 10^6/ml)	4.1 (0.1)	1.4 - 6.9 (4.0)
Eosinophil count (cells x 10^6/ml)	0.19 (0.01)	0 - 0.8 (0.17)
Salivary IgA (mg/dl)	6.98 (0.2)	2 - 10
Plasma IgE (IU/ml)	78 (7.8)	12 - 132
Plasma vitamin C (µg/ml)	9.7 (0.1)	8.6 - 12 (10.0)
Plasma vitamin E (µg/ml) ($N = 63$)	5.5 (0.2)	4.4 - 10.5 (6.8)
Urinary cotinine (µM)	2.7 (0.01)	0 - 7 (< 3)
Haemoglobin (g/dl)	14.6 (0.05)	12.0 - 16.0

* Results are expressed as the mean with the SEM in parenthesis.
† Values for non-smoking young adults according to the Departments of Immunology or Haematology, University of Pretoria, expressed as normal range with the mean values, where available, in brackets.

Influence of passive smoking, gender and age on the immunological profiles. The influence of passive smoking on the immunological profiles of children from one school was studied using general linear models with age, gender and passive smoking as independent variables. Passive smoking did not affect these significantly. Vitamin C levels were likewise not significantly different, with levels of 9.5 ± 0.2 and 9.8 ± 0.2 ($P = 0.1010$) in exposed and non-exposed children respectively. Neutrophil LECL, total leucocyte count and plasma levels of vitamin C were significantly higher in girls, and RO generation (LECL) was higher in the older age group (not shown).

Influence of active smoking on the immunological profile. Investigations were performed by means of a case control study. Forty-three schoolchildren who actively smoked were matched with 43 non-exposed children as described in the Methods section. Immunological and haematological parameters were not significantly different from those of the non-smokers (results not shown.) IgE levels were 111 ± 30 v. 60 ± 18 ($P = 0.1842$) and vitamin C levels 9.1 ± 0.3 v. 9.8 ± 0.3 ($P = 0.1537$) in the exposed group v. the matched non-exposed group, respectively.

Questionnaire data

Prevalence of symptoms, respiratory illnesses, low birth weight and learning difficulties in passive smokers. These were compared in the non-exposed and exposed groups using univariate analysis and the data are shown in Table III. The prevalences of respiratory illnesses before and after 2 years, of cough, phlegm, earache over the past year, low birth weight and learning difficulties were significantly increased in the exposed group.

Table III. Prevalence of respiratory illness and symptoms, earache, low birth weight and learning difficulties in adolescent schoolchildren exposed to parental smoking in comparison with those not exposed*

Parameter	Non-smokers (not exposed)	Passive smokers (exposed)	*P*-value
Low birth weight (less than 2.3 kg)	18 (39)†	27 (111)†	0.007
Respiratory illness before 2 years	10 (23)	16 (77)	0.017
Respiratory illness after 2 years	15 (34)	22 (106)	0.010
Cough, first thing in the morning	7 (17)	12 (56)	0.040
Phlegm	22 (51)	27 (130)	0.057
Earache past year	28 (65)	36 (173)	0.014
Learning difficulties	14 (32)	20 (92)	0.031

* The category 'other smokers at home' was excluded.
† Results are shown as the percentage of the total group with absolute numbers in brackets.

ORs for the association of passive smoking and health parameters. These were calculated by logistic regression analysis of data from one school. The significant associations are shown in Table IV. Maternal smoking, in particular, was negatively associated with many of the health indices, with a lesser contribution from low socio-economic status. Paternal smoking was not significantly associated with any of the parameters. A cumulative effect of smoking by both parents on these health parameters was not evident, except in the case of croup for which the OR was 2.55 ($P = 0.0387$).

Table IV. Logistic regression analysis of relationships between parental smoking and low birth weight, respiratory illness and symptoms, earache and learning difficulties

Parameters	Odds ratio	95% CI	*P*-value
Low birth weight			
Maternal smoking during pregnancy	2.63*	1.61 - 4.31	0.0001
Low socio-economic status	1.98	1.25 - 3.13	0.0037
Respiratory illness before 2 years			
Maternal smoking and maternal smoking during pregnancy	2.18	1.25 - 3.78	0.0057
Respiratory illness after 2 years			
Maternal smoking	3.62	2.30 - 5.70	0.0001
Maternal v. paternal smoking	3.07	1.60 - 5.89	0.0009
Pneumonia ever			
Maternal smoking	3.23	1.54 - 6.80	0.0135
Maternal v. paternal smoking	4.36	1.72 - 11.0	0.0066
Croup ever			
Maternal smoking	4.68	2.58 - 8.5	0.0010
Maternal v. paternal smoking	3.88	1.55 - 9.71	0.0041
Cough first thing in the morning			
Maternal smoking	2.95	1.44 - 6.03	0.0045
Maternal v. paternal smoking	5.79	2.37 - 14.14	0.0017
Low socio-economic status	2.27	1.14 - 4.54	0.0204
Earache the past year			
Maternal smoking	2.07	1.39 - 3.07	0.0217
Girls	2.86	1.82 - 4.50	0.0001
Learning difficulties			
Maternal smoking during pregnancy	2.08	1.21 - 3.56	0.0001
Younger than 16 years	1.77	1.08 - 2.92	0.0247

* These analyses were performed on the data from only one school. The independent variables considered in the models for the above outcomes are: maternal smoking during pregnancy (nos 1 - 8); paternal smoking during pregnancy (1); maternal or paternal smoking from birth to 2 years (2); current maternal or paternal or both parents smoking (3 - 8). The category 'other smokers at home' was omitted from the analysis.

Passive smoking was also associated with a previous history of sinus problems ($P = 0.0321$), sinusitis during the past year ($P = 0.0157$) and a combination of lower respiratory tract illnesses or symptoms (i.e. bronchitis, pneumonia, cough, phlegm and wheeze; $P = 0.0537$). These were not significantly associated with smoking in either parent. In addition, odds of there being lower respiratory tract illness or symptoms were higher in girls (OR = 1.76; $P = 0.0093$) and in children from a lower socio-economic group (OR = 1.52; $P = 0.0398$).

Historical evidence of allergy or bronchial asthma was not significantly associated with parental smoking. Meaningful conclusions, however, could not be made with regard to asthma, since it was reported in only 5% of the total study population.

Comparison of respiratory illnesses and symptoms in children who actively smoked and non-exposed children. Because of the small number of children who actively smoked ($N = 43$), a matched case control study was undertaken to compare respiratory illnesses and symptoms in this group with non-exposed children. Using McNemar's test of symmetry, no significant trends in occurrences of the reported illnesses were detected. However, when a combination of lower respiratory tract illnesses and symptoms was compared, such a trend was evident ($P = 0.0218$) with significantly more cases in the actively smoking group (17 cases in the latter group v. 6 cases in the non-exposed group).

Discussion

In this study we investigated certain health parameters in a group of schoolchildren resident in the Vaal Triangle, an area known to have relatively high levels of atmospheric pollutants, particularly suspended particulate matter.[4] Pulmonary functions and selected immunological investigations, including measurement of reactive oxidant generation, circulating leucocyte counts, plasma IgE and salivary IgA levels, as well as serum vitamin C concentrations were all found to be within the ranges considered normal for subjects not exposed to significant atmospheric pollution. These observations demonstrate that pulmonary and immune functions are not affected by the levels of industrial atmospheric pollution prevalent in the Vaal Triangle. The pulmonary function data are in agreement with those previously reported by Zwi *et al.*[32]

Pulmonary functions, leucocyte counts, oxidant generation by circulating neutrophils, plasma vitamin C levels and immunological parameters were also within normal limits in the subgroup of children passively exposed to cigarette smoke in the home. We have previously reported that acute experimental exposure to high levels of sidestream tobacco smoke is accompanied by increases in both the number and pro-oxidative activity of circulating leucocytes,[15] while plasma vitamin C levels are significantly reduced in passively smoking adults.[19-20] The absence of detectable effects on these systemic parameters of smoking-related pro-inflammatory activity indicates a relatively low intensity and/or duration of exposure to sidestream smoke in our study group. However, we did observe that current maternal smoking strongly and negatively influenced most of the historical evidence of respiratory illness and symptoms. In addition, we confirmed previous findings showing that maternal smoking during pregnancy is associated with low birth weight[12,13] and learning difficulties[33-36] (low socio-economic status was also associated with low birth weight and cough). These effects of maternal smoking may be due to the fact that children are exposed for longer periods to their mothers, but may also relate to the fact that respiratory damage is initiated *in utero*.[7] On a cautionary note, however, there was a greater frequency of single parents in the current maternal smoking group and the possible influence of emotional stress cannot be excluded. This point will be addressed in an ongoing study (VAPS) involving a much larger group of children.

Finally, this study has highlighted the detrimental effects of parental smoking, especially maternal smoking. Our previous data[4] have shown that although the Vaal Triangle has heavy atmospheric pollution, the present levels have only a trivial impact on the health of that population. At present, the most important preventive strategy with regard to atmospheric pollution is to dissuade parents from smoking within the vicinity of their children.

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