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PULMONARY DISEASES

Fetal and postnatal exposure to tobacco smoke and respiratory health in children

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Abstract. The aim of this paper was to find out whether fetal exposure to environmental tobacco smoke (ETS), as compared to postnatal ETS exposure, is an independent risk factor for respiratory symptoms and diseases in younger schoolchildren. The cross-sectional epidemiological study comprised population of 1,561 Polish schoolchildren, aged 9–11 years. Information on the exposure to tobacco smoke and other sources of indoor air pollution at home, respiratory and allergic health status, and socio-economic status of the family was obtained by questionnaire survey. The respiratory health status was described by presence of wheezing, attacks of dyspnoea (noted during the last year or ever), bronchitis, wheezy bronchitis and asthma, ever diagnosed by a physician. Multivariate logistic regression analysis with adjustment for age, sex, area of residence, household density, damp and mould stains found at home, use of coal-fired stove, co-habitant pets, mother's education and paternal current and past smoking habit was used to assess the effect of fetal and postnatal exposures on respiratory health outcomes. The results of the multivariate analyses revealed statistically significant associations between fetal exposure to ETS and wheezing ever: log OR = 1.4 (95% CI: 1.0–2.0), attacks of dyspnoea ever: log OR = 1.8 (95% CI: 1.1–2.9), bronchitis: log OR = 2.1 (95% CI: 1.5–2.9), and wheezy bronchitis: log OR = 1.8 (95% CI: 1.1–2.9). The effect of postnatal ETS was statistically significant only for bronchitis: log OR = 1.4 (95% CI: 1.1–1.9). The results of our study showed that fetal exposure to tobacco smoke is an independent risk factor for symptoms of wheeze and wheezy bronchitis in schoolchildren when compared to postnatal ETS exposure.

Key words: Asthma, Children, Fetal, Passive, Postnatal, Smoking

Introduction

Children's exposure to harmful substances of tobacco smoke begins at prenatal period and, if pregnant woman smokes after the delivery, it continues postnatally. Thus, it is difficult to distinguish between health effects linked to either of the two periods of environmental tobacco smoke (ETS) exposure. This methodological problem affects especially cross-sectional epidemiological studies using questionnaire survey to assess the exposure [1]. Definite conclusions concerning the effects of fetal exposure on the respiratory health status of schoolchildren are rare [2, 3]. Using population-based data obtained from a cross-sectional epidemiological study, we attempted to verify the hypothesis that fetal exposure to ETS, as compared to postnatal ETS exposure, is an independent risk factor for respiratory symptoms and diseases occurring in younger schoolchildren.

Material and methods:

Study design and population

The project was performed in 1996–1998 as the cross-sectional epidemiological study, part of the multi-centre Central European Study on Air Pollution and Respiratory Health (CESAR). Details of the study protocol were described previously [4, 5]. The subjects were children aged 9–11 years, attending primary schools in 4 Polish towns: Swietochlowice, Kedzierzyn-Kozle, Pszczyna and Kielce. Their natural or foster parents were invited to take part in the study via schools and submitted their written consent to their participation. Questionnaires were distributed by regular mail and back collected at schools. The response rate reached approximately 80% in all 4 study areas.

Respiratory health outcomes

Information about the presence or absence of respiratory symptoms and diseases was obtained from the Polish version of the CESAR questionnaire. The questionnaire used in CESAR study was designed especially for the purpose of this study and based on the standard epidemiological tools available in the literature, adapted and validated for use in Central Europe [6, 7]. It contained questions on respiratory symptoms and diseases noted in children, details of child's and family health status, information about exposure to 'maternal' and 'paternal' tobacco smoke,

data on housing conditions and socio-economic status of the family. The following respiratory symptoms and diseases were analysed: wheezing occurring ever and wheezing in the last year, attacks of dyspnoea with wheezing occurring ever and attacks of dyspnoea with wheezing in the last year, ever diagnosed by physician: asthma, wheezy bronchitis and bronchitis.

Assessment of exposure to tobacco smoke

Assessment of exposure to tobacco smoke was based on a questionnaire survey. Information was collected concerning smoking habit of mother, father and other dwellers. Fetal and postnatal ETS exposures were defined according to the maternal smoking history: fetal exposure was assumed to be associated with smoking during pregnancy, and postnatal exposure with tobacco smoking during the child's lifetime but not during the pregnancy. The question in the questionnaire concerning smoking during pregnancy was: 'Did mother of the child smoke during being pregnant with this child' and a question related to postnatal exposure was: 'Does mother of the child smoke now or did she smoke in the past and, if she smoked in the past, when did she start and give up smoking?'. The primary exposure of interest was smoking during pregnancy. None of the women smoked during pregnancy only, and the habit reported during pregnancy continued also during the lifetime of a child. Therefore, the effect of fetal exposure was evaluated by comparing the effect in children whose mothers smoked both during and after the pregnancy with that in children exposed to ETS only postnatally. Non-exposed children (no ETS during and/or after pregnancy) were included in the reference group.

Statistical analyses

Statistical analyses were performed using the STATA package [8]. Prevalence of respiratory symptoms and diseases was calculated in unexposed children and in children exposed to tobacco smoke, classified according to the categories of maternal exposure. In order to assess the effects of fetal and postnatal ETS exposure and other potential risk factors for respiratory symptoms and diseases univariate and multivariate logistic regression analyses were used to assess odds ratios with 95% confidence intervals (log OR; 95% CI). Multivariate logistic regression analysis involved adjustment for age, sex, area of residence as an indication of outdoor air pollution, household density, damp and mould stains found at home, use of coal-fired stove, co-habitant pets, and mother's education. The procedure enabled also the control of paternal current and past smoking. Results of univariate and multivariate logistic regression analyses were interpreted according to the values of crude and adjusted for confounders logistic odds ratios and their 95% confidence intervals. Statistical inferences were based on the conventional level of statistical significance ($p < 0.05$).

Results

The study population included 1,561 children: 794 (50.9%) were girls and 767 (49.1%) were boys. Six hundred and fifty four mothers (41.9) had never smoked. Smoking during pregnancy was reported by 389 (24.9%) women, all of whom smoked also after the delivery. Smoking during the lifetime of the child (and prior abstaining from smoking during the pregnancy) was reported by 461 (29.5%) mothers. For 57 (3.7%) women, information concerning smoking was not available. Average number of cigarettes smoked by women reporting current or past smoking during and after pregnancy was 9.0 (SD: ± 6.0 cigarettes), whereas in a group of mothers reporting smoking habit during the lifetime of a child was 4.6 (SD: ± 4.9). The both categories of maternal exposure to tobacco smoke included former smokers: in a group of women who were smoking during and after pregnancy 62 (16.1%) of women gave up smoking and in a group of mothers who were smoking after pregnancy only 184 (39.9) stopped smoking. Among the fathers, 818 (52.4%) were current smokers, 280 (17.9%) were former smokers, and 395 (25.3%) had never smoked. No information on smoking was available for 68 (4.4%) fathers.

Wheezing ever was reported for 319 (20.4%) children, wheezing during the last year occurred in 135 (8.6%) children, whereas attacks of dyspnoea ever were reported in 133 (8.5%) and attacks of dyspnoea during the last year in 60 (3.8%) children. Physician-diagnosed asthma occurred in 31 (2.0%) children, bronchitis ever in 1071 (68.5%) children, and wheezy bronchitis in 153 (9.8%) children.

Prevalence of all analysed symptoms and diseases was higher in children who were fetally and postnatally exposed to maternal tobacco smoke when compared to unexposed children and children exposed postnatally only. An inverse association was observed only for physician-diagnosed asthma (Table 1).

The association between fetal and postnatal exposures and respiratory symptoms/diseases was explored by means of univariate logistic regression analysis. The results of univariate logistic regression revealed statistically significant associations between fetal exposure to ETS and attacks of dyspnoea ever: crude log OR = 1.6 (1.1–2.5) and bronchitis ever: crude OR = 1.7 (1.3–2.2) and between postnatal ETS exposure and bronchitis ever: crude OR = 1.4 (1.1–1.8). Among other potential determinants of respiratory symptoms and diseases exposure to damp and moisture stains at home remained statistically

Table 1. Prevalence of respiratory symptoms and diseases according to categories of maternal exposure to ETS

	No maternal exposure, N = 654		Postnatal exposure to ETS, N = 461		Fetal and postnatal exposure to ETS, N = 389	
Respiratory symptom/disease	N	%	N	%	N	%
Wheezing ever	129	19.7	84	18.2	93	23.9
Wheezing in last year	54	8.3	31	6.7	42	10.8
Attacks of dyspnoea ever	47	7.2	35	7.6	44	11.3
Attacks of dyspnoea in last year	22	3.4	13	2.8	20	5.1
Asthma ever	14	2.1	9	1.9	6	1.5
Bronchitis ever	419	64.1	329	71.4	292	75.1
Wheezy bronchitis ever	57	8.7	46	10.0	45	11.6

significant risk factors for wheezing occurring ever: crude log OR = 1.8 (1.4–2.3) and in last year: crude OR = 1.5 (1.0–2.2) and attacks of dyspnoea occurring ever: crude OR = 1.4 (1.0–2.1), bronchitis: crude OR = 1.5 (1.2–1.9) and wheezy bronchitis: crude OR = 1.6 (1.1–2.2). Coal-fired stove was statistically significant risk factor for wheezing ever: crude log OR = 1.6 (1.0–2.5), wheezing occuring in last year: crude OR = 2.0 (1.1–3.5), attacks of dyspnea occurring ever: crude OR = 1.9 (1.0–3.3). Presence of pets at home was associated with increased risk of attacks of dyspnoea ever: crude OR = 1.6 (1.1–2.3) and attacks of dyspnoea occuring in last year: crude OR = 2.1 (1.2–3.7) as well as for asthma: crude OR = 2.2 (1.0–4.8) and wheezy bronchitis: crude OR = 1.4 (1.0–1.9) – Tables 2 and 3.

The results of multivariate analyses revealed statistically significant associations between fetal exposure to ETS and wheezing ever: adjusted log OR = 1.4 (95% CI: 1.0–2.0), attacks of dyspnoea ever: adjusted log OR = 1.8 (95% CI: 1.1–2.9), physician–diagnosed bronchitis ever: adjusted log OR = 2.1 (95% CI: 1.5–2.9), and wheezy bronchitis: adjusted log OR = 1.8 (95% CI: 1.1–2.9). When analysing the effect of postnatal exposure, statistically significant associations were observed only for bronchitis: adjusted log OR = 1.4 (95% CI: 1.1–1.9). No associations were seen for either postnatal or fetal exposure and 'last year' wheezing, 'last year' attacks of dyspnoea or asthma. Among other analysed determinants of respiratory symptoms and diseases older age (11 years) was a statistically significant risk factor for attacks of dyspnoea occurring ever: adjusted log OR = 1.6 (1.1–2.5) and bronchitis: adjusted OR = 1.7 (1.3–2.2). Exposure to damp and mould stains at home was a significant risk factor for wheezing ever: adjusted log OR = 1.7 (1.3–2.3), bronchitis ever: adjusted log OR = 1.5 (1.1–1.9) and wheezy bronchitis: adjusted log OR = 1.6 (1.1–2.4). Presence of coal-fired stove at home was associated with wheezing occurring in last year: adjusted log OR = 2.2 (1.1–4.2) and attacks of dyspoea occurring ever: adjusted log OR = 2.0 (1.0–3.9). Presence of pets at home was associated with increased risk of attacks of dyspnoea ever: adjusted log OR = 1.6 (1.0–2.3), attacks of dyspnoea occurring in last year: adjusted log OR = 2.4 (1.3–4.5), and asthma: adjusted log OR = 2.3 (1.0–5.3). Tables 4 and 5 show the results of the multivariate analyses in relation to both categories of ETS exposure and other potential determinants of respiratory health outcomes.

A summary of results of multivariate logistic regression analyses are also shown in Figures 1 and 2.

Discussion

Results of our study revealed that respiratory health effects of fetal ETS exposure were different from the effects of postnatal exposure to tobacco smoke in children. Fetal exposure to ETS was associated with an increased risk of wheezing ever and attacks of dyspnoea ever, as well as physician-diagnosed bronchitis and wheezy bronchitis. No effect of fetal exposure was observed for wheezing or attacks of dyspnoea recorded last year or ever physician-diagnosed asthma. Postnatal ETS exposure was a statistically significant risk factor for bronchitis only.

The effect of ETS on the occurrence of respiratory symptoms and diseases in schoolchildren is confirmed by a number of meta-analyses of existing epidemiological studies [9–12]. They report the effect of passive smoking on the occurrence of wheezing (OR = 1.24, 95% CI: 1.10–1.34), breathlessness (OR = 1.31, 95% CI: 1.08–1.59) and asthma. The risk of asthma was related to the smoking habit of either parent (OR = 1.21, 95% CI: 1.10–1.34) and the effect of maternal smoking was greater (OR = 1.36) than the effect of paternal smoking (OR = 1.07). Interesting results are provided by recent studies on asthma and exposure to passive smoking [13, 14]. They suggest that exposure to tobacco smoke early in life and asthmatic heredity may be important co-factors increasing risk for asthma. ETS exposure may interact with genetic susceptibility to asthma and fetal exposure to ETS increase risk for asthma [14]. All those findings suggest that genetic susceptibility to

Table 2. Effect of potential risk factors on respiratory health outcomes: Results of univariate logistic regression analyses

	Age	Male gender	Area[a]	Damp/mould stains at home	Household density	Coal-fired stove at home	Pets	Maternal education[b]	Paternal current and ex-smoking
Wheezing ever	10 yrs: 0.8 (0.6–1.0)[c] 11 yrs: 0.9 (0.6–1.3)	0.9 (0.7–1.1)	S: 1.3 (0.9–1.8) K: 1.4 (0.9–2.0) P: 1.3 (0.9–2.0)	1.8 (1.4–2.3)	1.0 (0.8–1.3)	1.6 (1.0–2.5)	1.1 (0.8–1.4)	S: 0.9 (0.6–1.3) P: 0.8 (0.5–0.7)	1.1 (0.8–1.5)
Wheezing last year	10 yrs: 0.8 (0.5–1.2) 11 yrs: 0.9 (05–1.6)	0.9 (0.6–1.2)	S: 1.1 (0.7–1.9) K: 1.6 (0.9–2.6) P: 1.2 (0.7–2.2)	1.5 (1.0–2.2)	1.1 (0.8–1.6)	2.0 (1.1–3.5)	1.1 (0.7–1.5)	S: 0.7 (0.4–1.3) P: 1.1 (0.6–2.0)	1.0 (0.7–1.6)
Attacks of dyspnoea ever	10 yrs: 0.7 (0.5–1.0) 11 yrs: 0.7 (0.4 –1.2)	1.2 (0.5–1.6)	S: 0.6 (0.4–1.1) K: 0.9 (0.6–1.5) P: 1.0 (0.6–1.6)	1.4 (1.0–2.1)	1.1 (0.8–1.6)	1.9 (1.0–3.3)	1.6 (1.1–2.3)	S: 0.8 (0.5–3.4) P: 1.9 (0.7–5.1)	1.3 (0.8–3.3)
Attacks of dyspnoea last year	10 yrs: 0.9 (0.5–1.5) 11 yrs: 0.7 (0.3–1.6)	0.9 (0.5–1.6)	S: 0.6 (0.2–1.2) K: 1.4 (0.7–2.7) P: 0.8 (0.8–1.8)	1.4 (0.8–2.4)	1.0 (0.6–1.7)	1.2 (0.5–3.2)	2.1 (1.2–3.7)	S: 1.3 (0.5–3.4) P: 1.9 (0.7–5.1)	1.6 (0.8–3.3)
Asthma ever	10 yrs: 0.5 (0.2– 1.0) 11 yrs: 0.3 (00.6–1.2)	1.4 (0.6–2.8)	S: 0.9 (0.3–2.5) K: 0.8 (0.3–2.5) P: 1.4 (0.5–3.8)	1.3 (0.6–2.7)	0.8 (0.4–1.6)	–[d]	2.2 (1.0–4.8)	S: 1.2 (0.4–3.6) P: 0.6 (0.2–2.2)	0.6 (0.3–1.2)
Bronchitis ever	10 yrs: 0.9 (0.7–1.2) 11 yrs: 1.0 (0.7–1.3)	0.8 (0.6–1.0)	S: 1.3 (1.0–1.7) K: 1.5 (1.1–2.0) P: 1.2 (0.8–1.6)	1.5 (1.2–1.9)	0.7 (0.6–0.9)	0.8 (0.5–1.2)	0.8 (0.6–1.0)	S: 0.8 (0.6–1.2) P: 0.5 (0.3–0.7)	0.9 (0.7–1.22)
Wheezy bronchitis ever	10 yrs: 0.9 (0.6 –1.3) 11 yrs: 0.7 (04–1.2)	0.9 (0.6–1.3)	S: 1.1 (0.7–1.8) K: 1.0 (0.6–1.7) P: 1.0 (0.6–1.7)	1.6 (1.1–2.2)	0.9 (0.6–1.2)	0.8 (0.4–1.7)	1.4 (1.0–1.9)	S: 0.7 (0.4–1.1) P: 0.4 (0.2–0.6)	0.8 (0.6–1.2)

[a]Areas of residence: S – Swietochlowice, K – Kedzierzyn-Kozle, P – Pszczyna.
[b]Maternal education: P – primary, S – secondary, Higher as a reference category.
[c]Values of crude odds ratios and in parentheses 95% confidence intervals.
[d]Analysis not performed due to low numbers.

Table 3. Effect of maternal ETS exposure on respiratory health outcomes: Results of univariate logistic regression analyses

	Postnatal ETS exposure	Fetal and postnatal ETS exposure
Wheezing ever	0.9 (0.7–1.2)[a]	1.3 (0.9–1.2)
Wheezing last year	0.8 (0.5–1.3)	1.3 (0.9–2.0)
Attacks of dyspnoea ever	1.1 (0.7–1.7)	1.6 (1.1–2.5)
Attacks of dyspnoea last year	0.8 (0.4–1.7)	1.5 (0.8–2.9)
Asthma ever	0.9 (0.4–2.1)	0.7 (0.3–1.9)
Bronchitis ever	1.4 (1.1–1.8)	1.7 (1.3–2.2)
Wheezy bronchitis ever	1.2 (0.8–1.7)	1.4 (0.9–2.1)

[a]Values of crude odds ratios and in parentheses 95% confidence intervals.

ETS exposure and period of exposure may determine risk for asthma and emphasize a role of gene – environment interactions in aetiology of this disease.

A recent review of the published epidemiological studies showed an apparent impact of ETS exposure on the frequency of infectious diseases [11]. Exposure to tobacco smoke was found to increase the number of 'lower respiratory infections', especially in early life. This meta-analysis of 24 studies revealed a total odds ratio of 1.57 (95% CI: 1.42–1.74) for smoking of either parent and OR = C1.72 (95% CI: 1.55–1.91) for maternal smoking when analysing the effect on 'lower respiratory infections'. The findings are consistent in that the majority of evidence on respiratory effects of passive smoking exposure in children shows the greater effect of maternal smoking and points to the importance of *in utero* exposure.

Results of a recently published review of existing data suggest that fetal and early postnatal periods seem to be a critical window frame for the effects of environmental exposures in child's life, since respiratory system is in the phase of intensive growth and rapid development [15, 16]. Also toxicological differences in terms of route of exposure to tobacco smoke during fetal and postnatal periods potentially imply differences in health effects. *In utero* exposure to tobacco smoke is associated mainly with absorption of harmful substances from tobacco smoke by placenta, whereas postnatal exposure is associated with passive inhalation of tobacco smoke and respiratory route of exposure [17].

However, it is difficult to distinguish between respiratory effects attributable to maternal fetal and postnatal ETS exposures. The situation in which women smoke only in pregnancy is in practice non-existent; the women who smoked during pregnancy continued smoking also after the delivery. A high correlation between fetal and postnatal exposure to tobacco smoke causes that, in cross-sectional studies, it is virtually impossible to clearly distinguish between those two exposures. That is why the issue of the effects of fetal and postnatal ETS exposure is not often addressed in epidemiological studies. Most of the existing studies report the effect of fetal exposure on the lung function in newborns; however, results of some observations suggest that the effect of *in utero* exposure on lung function persists until the school-age and is more pronounced than the effects of postnatal exposure [18, 19]. Longitudinal observations provide interesting findings. The results of a prospective study of 1,000 children in Arizona revealed that fetal exposure was associated with increased occurrence of wheezing early in life [20]. Another study conducted in 24 communities from North America showed that in schoolchildren aged 8–11 years, current ETS exposure at home was associated with increased prevalence of wheezing [21]. Results of another study conducted in 5762 Californian schoolchildren revealed that *in utero* exposure to tobacco smoke was a stronger predictor of current and persistent wheeze and physician-diagnosed asthma than postnatal exposure [22]. Also, results of a large epidemiological study conducted in Italy revealed that *in utero* exposure was a stronger determinant of current wheezing and asthma in 6–7 years old schoolchildren than postnatal exposure to tobacco smoke [23]. Results of our study are consistent with the published evidence. We found the effect of fetal ETS on the frequency of non-specific 'asthmatic' symptoms ever and on the frequency of infectious diseases like bronchitis and bronchitis with an 'asthmatic' ('wheezy') component. We also observed an effect of both fetal and postnatal ETS exposures on the frequency of bronchitis; however, fetal exposure to ETS was a stronger risk factor than postnatal ETS exposure. Effect of *in utero* exposure seem to disappear with age (no effect on current wheeze and current dyspnoea). It is in good agreement with other observations showing the largest effect of fetal ETS exposure at younger ages, with the subsequent magnitude of the effect declining with age [23, 24]. Our observations are also consistent with results of longitudinal studies which revealed statistically significant effect of maternal smoking during pregnancy on wheeze, associated rather with wheezy bronchitis, not asthma [25, 26]. On the other hand, association between fetal and postnatal ETS exposures and respiratory infections was also confirmed by observations from another Polish study of 1,129 children. Results showed an association between fetal and postnatal exposure to ETS and acute respiratory infections, with a stronger effect of fetal exposure [27].

We found no effect of either fetal or postnatal tobacco smoke exposure on the frequency of asthma; however, a non-significant inverse trend for asthma prevalence was observed. This finding is difficult to explain; however, one possible explanation is that mothers of asthmatic children quit smoking. Another problem is associated with the fact, that we collected information on diagnosis of asthma confirmed by a physician from parental questionnaire. Prevalence of

Table 4. Effect of potential risk factors on respiratory health outcomes: Results of multivariate logistic regression analyses

	Age	Male gender	Area[a]	Damp/mould stains at home	Household density	Coal-fired stove at home	Pets	Maternal education[b]	Paternal current and ex-smoking
Wheezing ever	10 yrs: 0.9 (0.7–1.2)[c] 11 yrs: 1.3 (0.9–1.2)	0.7 (0.3–1.7)	S: 0.9 (0.3–2.3) K: 1.1 (0.5–2.5) P: 1.4 (0.9–2.2)	1.7 (1.3–2.3)	0.9 (0.7–1.2)	1.6 (0.9–2.6)	1.1 (0.8–1.4)	S: 0.8 (0.5–1.1) P: 0.6 (0.4–0.9)	1.1 (0.8–1.5)
Wheezing last year	10 yrs: 0.8 (0.5–1.3) 11 yrs: 1.3 (0.9–2.0) 10 yrs: 1.1 (0.7–1.7)	0.8 (0.3–2.5)	S: 0.8 (0.2–2.9) K: 1.3 (0.4–4.1) P: 1.3 (0.7–2.3)	1.4 (0.9–2.1)	1.0 (0.7–1.5)	2.2 (1.1–4.2)	1.1 (0.7–1.6)	S: 0.6 (0.4–1.2) P: 0.8 (0.4–1.5)	0.9 (0.6–1.5)
Attacks of dyspnoea ever	11 yrs: 1.6 (1.1–2.5)	1.0 (0.3–3.4)	S: 0.5 (0.1–2.1) K: 0.8 (0.2–2.9) P: 1.0 (0.5–1.7)	1.2 (0.8–1.8)	1.0 (0.6–1.5)	2.0 (1.0–3.9)	1.6 (1.0–2.3)	S: 0.7 (0.4–1.3) P: 0.6 (0.3–1.2)	1.4 (0.8–2.3)
Attacks of dyspnoea last year	10 yrs: 0.8 (0.4–1.7) 11 yrs: 1.5 (0.8–2.9)	0.4 (0.05–3.4)	S: 0.2 (0.02–1.8) K: 0.5 (0.06–3.8) P: 0.6 (0.2–1.5)	1.3 (0.7–2.3)	0.7 (0.4–1.3)	1.0 (0.3–3.4)	2.4 (1.3–4.5)	S: 1.1 (0.4–3.1) P: 1.6 (0.5–4.5)	1.7 (0.8–3.6)
Asthma ever	10 yrs: 0.9 (0.4–2.1) 11 yrs: 0.7 (0.3–1.9)	2.3 (0.2–22.6)	S: 1.7 (0.1–21.8) K: 1.3 (0.1–11.5) P: 1.3 (0.5–3.7)	1.4 (0.6–3.1)	0.7 (0.3–1.5)	–[d]	2.3 (1.0–5.3)	S: 1.2 (0.4–3.4) P: 0.8 (0.2–2.8)	0.6 (0.3–1.3)
Bronchitis ever	10 yrs: 1.4 (1.1–1.8) 11 yrs: 1.7 (1.3–2.2)	1.1 (0.6– 2.2)	S: 1.5 (0.7–3.4) K: 1.6 (0.8–3.3) P: 1.3 (0.9–1.8)	1.5 (1.1–1.9)	0.8 (0.6–1.1)	0.8 (0.5–1.2)	0.8 (0.7–1.1)	S: 0.8 (0.5–1.2) P: 0.5 (0.3–0.7)	0.7 (0.5–1.0)
Wheezy bronchitis ever	10 yrs: 1.2 (0.8 –1.7) 11 yrs: 1.4 (0.9–2.1)	0.9 (0.3–2.9)	S: 1.2 (0.3–4.1) K: 1.1 (0.4–3.6) P: 1.2 (0.7–2.1)	1.6 (1.1–2.4)	0.8 (0.6–1.2)	0.9 (0.4–1.9)	1.3 (0.9–1.9)	S: 0.7 (0.4–1.2) P: 0.3 (0.2–0.6)	0.8 (0.5–1.2)

[a]Areas of residence: S – Swietochlowice, K – Kedzierzyn-Kozle, P – Pszczyna.
[b]Maternal education: P – primary, S – secondary, Higher as a reference category.
[c]Values of crude odds ratios and in parentheses 95% confidence intervals.
[d]Analysis not performed due to low numbers.

Table 5. Effect of maternal ETS exposure on respiratory health outcomes: results of multivariate logistic regression analyses

	Postnatal ETS exposure[a]	Fetal and postnatal ETS exposure[a]
Wheezing ever	0.8 (0.6–1.2)[b]	1.4 (1.0–2.00), $p = 0.049$
Wheezing last year	0.7 (0.4–1.2)	1.4 (0.9–2.3)
Attacks of dyspnoea ever	0.9 (0.6–1.6)	1.8 (1.1–2.9), $p = 0.02$
Attacks of dyspnoea last year	0.7 (0.3–1.5)	1.5 (0.8–3.1)
Asthma ever	1.1 (0.5–2.8)	1.2 (0.4–3.3)
Bronchitis ever	1.4 (1.1–1.9), $p = 0.01$	2.1 (1.5–2.9), $p = 0.0001$
Wheezy bronchitis ever	1.2 (0.8–1.9)	1.8 (1.1–2.9), $p = 0.02$

[a]Adjusted for age, sex, area of residence, damp/mould stains found at home, household density, coal-fired stove at home, maternal education and paternal current and ex-smoking.[b]Values of odds ratios and, in parentheses, 95% confidence intervals.

physician-diagnosed asthma in examined population was low and when compared to relatively high frequency of 'asthmatic' symptoms may suggest that asthma may be underdiagnosed in examined population. Relying on 'diagnostic labelling' by physicians may result in misclassification of asthma status in child. It could be an explanation of a fact, that our results are not consistent with published evidence in terms of lack an association between ETS exposure and asthma.

Among factors determining respiratory health status in children exposure to tobacco smoke is one of well-documented but effect of other risk factors is also important as suggested by published evidence [13, 26]. We explored the importance and controlled for possible effect of other factors like age, sex, outdoor air pollution, and exposure to other indoor factors. Among analysed risk factors for 'asthmatic' symptoms exposure to damp/moisture at home and presence of coal-fired stove were statistically significant which is consistent with results of other studies confirming a role of factors associated with housing quality [28]. Presence of coal-fired stove doubled risk for 'spastic' symptoms and was larger than the effect

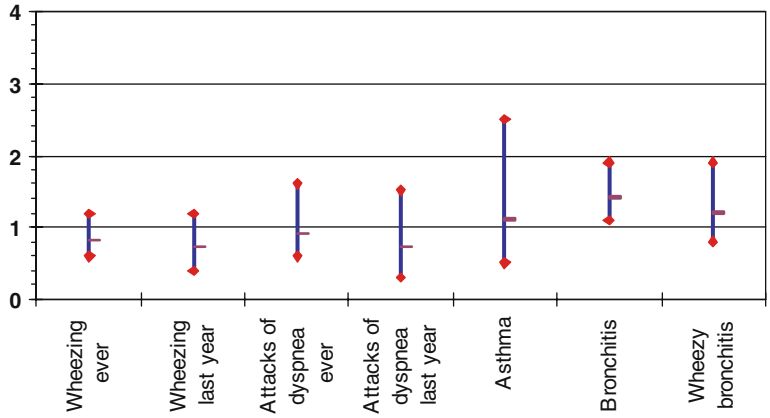

Figure 1. Effect of postnatal ETS exposure on respiratory symptoms and diseases.

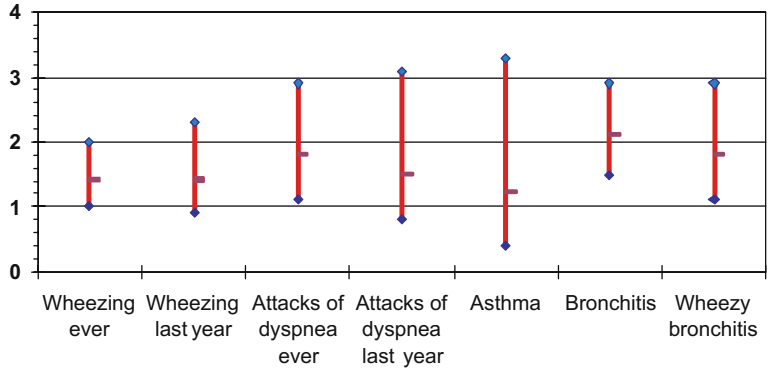

Figure 2. Effect of fetal ETS exposure on respiratory symptoms and diseases.

of fetal ETS. Other significant and stronger than fetal ETS risk factor for attacks of dyspnoea and asthma included exposure to pets which is also well-documented risk factor for asthma and asthma-like symptoms [28]. Other risk factors like age, male gender and outdoor air pollution were not significant. In our study the effects of household density and maternal education as indices of socio-economic status (SES) were not statistically significant. The lack of associations between SES and respiratory health outcomes is consistent with results of recent longitudinal study and results of review performed by German researchers [29, 30]. The studies gave conflicting results, some did not show associations at all. The effect of paternal smoking was not significant what is consistent with literature data reporting larger effect of maternal smoking [9, 10].

Our study has some limitations. The most important is an imperfect assessment of exposure to tobacco smoke. Assessment of exposure in our cross-sectional study was done on the basis of the questionnaire and information concerning parental smoking was collected retrospectively. It could result in a recall bias. Information on exposure obtained from parents was not validated by objective measures in our study. However, exposure to ETS expressed as the rate of parental smoking is a more accurate measure of exposure than number of cigarettes smoked daily, particularly when assessment of exposure is done in a retrospective manner [31]. Another methodological problem was associated with a duration of smoking and proper classification of exposure. Women who were smoking during pregnancy and after delivery and gave up smoking during the lifetime of a child were classified as smokers which could result in a misclassification of exposure. This problem exists in cross-sectional studies and creates a limitation of results also in our study.

Another important problem was associated with a fact, that children's populations classified in our study as exposed to fetal ETS and as exposed to postnatal ETS were not homogenous in terms of exposure. Those classified as exposed to fetal ETS came from families in which mothers smoked almost twice more cigarettes (average number of cigarettes – 9.0) as compared to those children, who were exposed to postnatal ETS and their mothers smoked less (average number of cigarettes – 4.6). Also proportion of mothers who gave up smoking was lower in a group of children who were exposed to fetal ETS when compared to children exposed postnatally (16.1% vs. 39.9%). We could assume, that maternal smoking behaviour influences potential respiratory health effects in both periods of exposure. Women who smoke during pregnancy and after delivery up to schoolage of a child are heavier smokers and they less likely give up smoking when compared to women who smoke only during a postnatal period and create a category of lighter smokers, who are more likely to stop smoking.

Families participating in the project were informed about the main purpose of the study, which was related to air pollution, but no special attention was paid to smoking or environmental tobacco smoke. Blinding of the specific study question was likely to reduce information bias in reporting exposure. Also the questionnaire used in the project was validated and results revealed high reproducibility (91%) of answers to questions on parental smoking [32].

In conclusion, the results of our study has revealed that fetal exposure to tobacco smoke is an independent risk factor for symptoms of wheeze and wheezy bronchitis in schoolchildren when compared to postnatal ETS exposure. No effect of either fetal or postnatal ETS exposures was observed for asthma. However, both categories of ETS exposure (fetal and postnatal) constituted a statistically significant risk factors for increased frequency of bronchitis. The evidence on harmful effects of maternal smoking habit on future status of children's respiratory health should be used to strengthen the effectiveness of ant-smoking campaigns addressed to women entering the reproductive age.

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