Maternal Smoking during Pregnancy and Postnatal Exposure to Environmental Tobacco Smoke as Predisposition Factors to Acute Respiratory Infections

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This study compared susceptibility to respiratory morbidity in a cohort of 9-year-old children exposed congenitally and postnatally to environmental tobacco smoke (ETS) to susceptibility in a cohort of unexposed children. The epidemiologic study included 1129 children: 594 boys and 535 girls attending the second grade of grammar schools in Kraków, Poland. We found strong evidence that children exposed to ETS in their homes were more susceptible to acute respiratory tract illnesses than unexposed children. A dose–response relationship between degree of exposure [for lower ETS exposure, odds ratio (OR) = 1.32; for higher ETS exposure, OR = 1.74] supports a causal explanation for the association observed. The significant trend of increased risk of respiratory infections due to ETS level in nonatopic children whose mothers did not smoke cigarettes during pregnancy suggests a direct effect of ETS exposure on the child's respiratory health. ETS combined with allergy nearly tripled the risk of acute respiratory tract illness (OR = 3.39; 95% CI, 1.93–5.93), and maternal smoking during pregnancy had a modifying effect on the risk of respiratory illnesses due to ETS after accounting for atopy. The stronger effect of ETS in atopic children and in those whose mothers smoked during pregnancy may be a result of biologic interaction of endogenous and environmental factors. The results of this study are of relevance to public health policy, as children with higher risk of respiratory infections may be more susceptible to environmental hazards later in adolescence or in adulthood. Respiratory infections also increase demands for medical interventions in terms of outpatient services and hospital admissions. In addition, respiratory illnesses cause missed school days, and caring for a sick child may lead to absenteeism from work. *Key words:* acute respiratory infections, atopy, children, environmental tobacco smoke, epidemiology, maternal smoking, predisposition. *Environ Health Perspect* 105:302–306 (1997)

Maternal smoking during pregnancy has significant effects on fetal growth and development and may affect lung growth as well as the immune system (*1,2*). Postnatal exposure to environmental tobacco smoke (ETS) has been incriminated as a major factor adversely influencing the respiratory health of young children (*3–24*). All the studies that have examined the incidence of respiratory illnesses and exposure to ETS in children under the age of 2 years have shown a positive association between such illnesses and exposure to ETS. It has been shown that lower respiratory tract illnesses occurred more often during the first year of life in children who have one or more parents who smoke than in children of nonsmokers, and this pattern was evident in studies conducted with different methodologies and in different geographic regions (*25*). These studies also proved that younger children are more susceptible to the adverse effects of ETS than older children.

The evidence for an effect of ETS was weaker and less convincing for school-age children, although the trends were the same as those reported for younger children. This may be due to a decrease in illness frequency, to physiological development of the respiratory tract or immune system with age, or to decreased contact between smoking parents and the child with age. However, the literature contains a remarkable lack of information on the long-term consequences of maternal smoking during pregnancy in terms of respiratory morbidity in older children after accounting for current ETS status and atopy, which is the major predisposing factor to acute episodes of respiratory illness.

The present study had three specific objectives. First, we aimed to compare the susceptibility to respiratory morbidity over the last 12 months in a cohort of 9-year-old children exposed postnatally to ETS with that of an unexposed cohort. Second, we wanted to test the hypothesis that susceptibility to respiratory infections in older children is the consequence of both maternal cigarette smoking during pregnancy and postnatal ETS exposure. Another objective was to test the hypothesis that atopy significantly modifies the susceptibility of children to acute respiratory infections induced by ETS.

Materials and Methods

The study was carried out in the course of the epidemiologic research project on childrens' health in Kraków, Poland. The field study was conducted in 1995 among 9-year-old schoolchildren attending 14 schools located in different areas of Kraków. The study sample included 1,129 subjects; 594 boys and 535 girls attending the 2nd grade of grammar schools in the city. The data on respiratory infections were available for 97% (1,116) of the defined sample.

Trained health professionals interviewed the mothers at schools or at homes and gathered standardized information about social background, family and child's respiratory health, and data on episodes of respiratory infections over the last 12 months. Respiratory infections of the upper (tonsillitis) and lower respiratory tract (laryngitis/tracheitis and bronchitis) that occurred in the last year before the interview and that were diagnosed by a doctor were the subject of the analysis. No data on severity of respiratory illness or the kind of medical interventions were collected.

At the interview, data were also collected about the mother's smoking habit during pregnancy, the duration and average number of cigarettes smoked daily, and the time of giving up smoking if the mother was previously a smoker. Mothers were also asked whether the child had been exposed to ETS at home. ETS exposure was categorized as 1) no lifetime exposure (ETS0), 2) 9 or fewer cigarettes smoked daily in the household over the child's lifetime (ETS1), or 3) 10 or more cigarettes smoked daily in the household over the child's lifetime (ETS2). In the category ETS1 we also included subjects reporting smoking more than 9 cigarettes daily within the household but irregularly and not over the child's lifetime. The weighted mean of the number of cigarettes smoked daily in this group was 7; in category ETS2 the estimated mean of cigarettes smoked was 17.

To allow for factors that could influence both smoking and respiratory illnesses, allergy and the education level of parents as a social index of the family were included in the multivariate analysis. The child was defined as allergic or atopic if the mother reported that allergy had been diagnosed by a doctor, irrespective of the allergy type and diagnostic methods used by the doctor.

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Predisposition (susceptibility) to acute respiratory infections in a child was defined as frequent episodes (three or more) of respiratory infections over the last 12 months preceding the interview.

The study sample did not contain children for whom the data on respiratory infections in the last year were not available, and 31 children with low birthweight (≤2000 g) were also excluded.

The preliminary analysis consisted of univariate descriptive statistics, two-way cross-tabulations and chi-square calculations. For multivariate analysis of the predisposing factors associated with the high occurrence of respiratory infections, logistic models were introduced to allow for the assessment of individual factors after simultaneous adjustment for confounders in the model.

Results

On average, there were 1.8 (SD = 2.9) episodes of respiratory infections in children in the last 12 months preceding the interview, and about 23% of children experienced an excess of infections (three or more) in the last year. Generally, there were lower rates of respiratory infections in girls than in boys, but the difference was not statistically significant. In the allergic children and in those who came from families with lower social status, the number of infections was significantly higher than in children from families with higher social status (Table 1).

In the sample of 9-year-old schoolchildren, about 60% of mothers neither smoked cigarettes during pregnancy nor after the delivery of the child; however, 15.6% of mothers smoked cigarettes during the entire pregnancy. There was an overall high collinearity between maternal smoking during pregnancy, postnatal maternal smoking, and ETS exposure of children. About 90% of women who smoked during pregnancy were still smoking when the child was 9 years old (Fig. 1). Moreover, the children of mothers who confirmed active cigarette smoking during pregnancy were exposed postnatally to a much higher degree of ETS within the household (Fig. 2). In the group of children whose mothers did not report during pregnancy, 41% were not exposed to ETS in the household; in the group of children whose mothers smoked during pregnancy, only 4% were not exposed to ETS at home. Children from the families with lower educational status were more often exposed to ETS and their mothers more often smoked cigarettes during pregnancy (Fig. 3).

Figure 4 shows the prevalence of children predisposed to acute respiratory infections according to mothers' smoking habits during pregnancy, atopy in the child, and

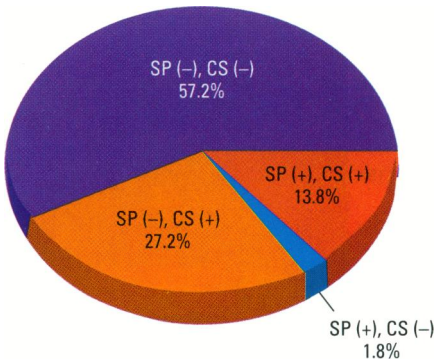

Figure 1. Mothers' smoking status during pregnancy (SP) and their current smoking habit (CS).

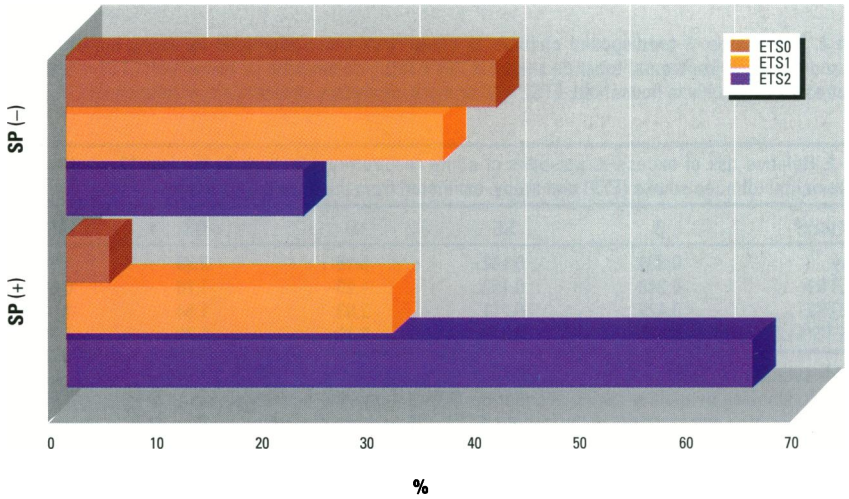

Figure 2. Smoking during pregnancy (SP) and environmental tobacco smoke (ETS). ETS0 = no smoking in household; ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked daily in household.

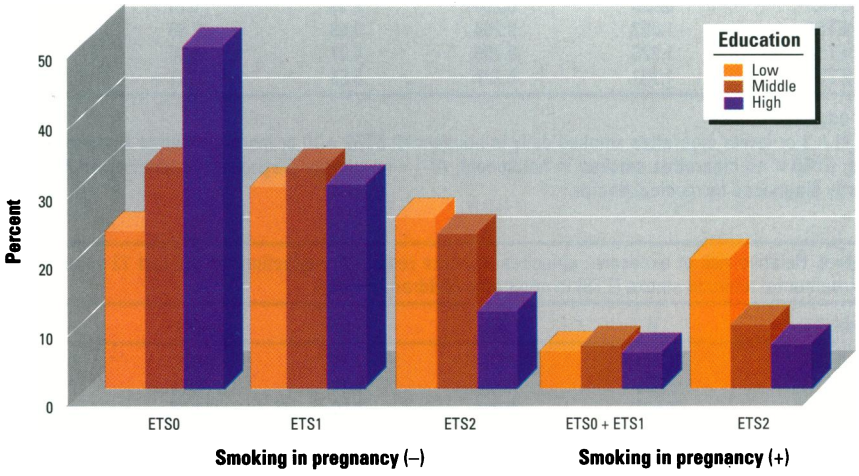

Figure 3. Smoking by mothers during pregnancy (SP) and environmental tobacco smoke (ETS) by education level of parents. ETS0 = no smoking in household; ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked daily in household.

Table 1. Average number of respiratory infections in the last 12 months and prevalence of children susceptible to acute respiratory infections

	n	Mean (SD)	Susceptible %[a]
Child's gender			
M	589	1.8 (3.1)	23.1
F	527	1.7 (2.8)	22.8 $p = 0.962$
Parents' education			
Low	294	2.1 (3.4)	26.5
Middle	471	1.8 (3.1)	23.6 χ^2(trend) = 5.02
High	350	1.4 (2.4)	19.1 df1, $p = 0.025$
Allergy diagnosed by medical doctor			
No	864	1.5 (2.5)	19.1
Yes	252	2.8 (3.9)	36.1 $p = 0.000$
Smoking during pregnancy			
No	935	1.7 (2.9)	21.7
Yes	179	2.2 (3.1)	29.6 $p = 0.139$
Environmental tobacco smoke (ETS)[b]			
ETS0	387	1.6 (3.0)	18.6
ETS1	393	1.8 (2.9)	23.4 χ^2(trend) = 7.89
ETS2	332	2.0 (3.0)	27.4 df1, $p = 0.005$

[a]Susceptible children: 3 or more spells of respiratory infections over the last 12 months.
[b]ETS0 = no cigarettes smoked in household; ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked daily in household.

postnatal ETS exposure in the household. ETS affected susceptibility to respiratory infections to a smaller degree than smoking by mothers during pregnancy. Allergic children had a significantly higher rate of respiratory infections compared to nonallergic children, and about twice that proportion of children had an excess of acute respiratory infections in the last year. There was a clear and significant trend for rate of infections in nonatopic children with reported ETS exposure, and the prevalence of children predisposed to acute respiratory infections increased with the number of cigarettes smoked daily in the household.

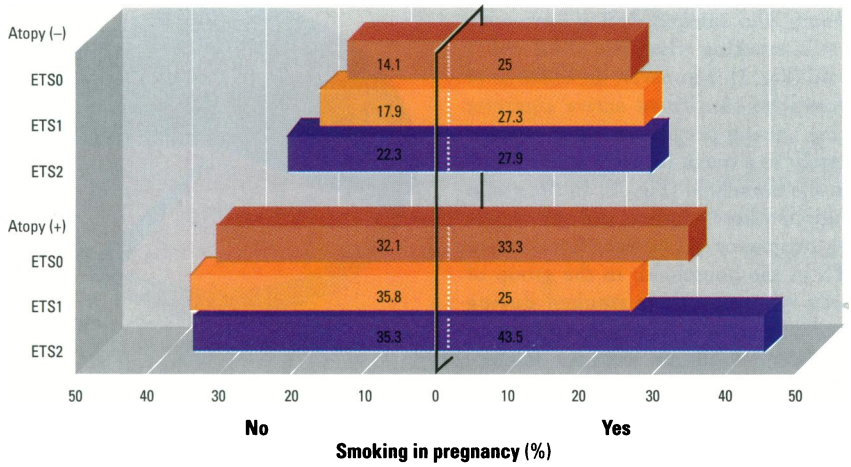

Figure 4. Prevalence of predisposed children to acute respiratory infections by atopy, smoking during pregnancy, and environmental tobacco smoke (ETS). ETS0 = no smoking in household; ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked daily in household.

Table 2. Relative risk of excessive episodes of acute respiratory infections in the last 12 months due to environmental tobacco smoke (ETS) and atopy, estimated from the logistic regression

Predictors[a]	β	SE	*t*	OR	95% CI
Allergy	0.834	0.162	5.15	2.30	1.68–3.17
SP(-)ETS1	0.248	0.192	1.29	1.28	0.88–1.87
SP(-)ETS2	0.429	0.211	2.03	1.54	1.01–2.32
SP(+)ETS(1+2)	0.689	0.219	3.15	1.99	1.30–3.06

OR, odds ratio.
[a]SP(-) = no smoking by mother during pregnancy; SP(+) = smoking by mother during pregnancy; ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked daily in household.

Table 3. Relative risk of excessive episodes of acute respiratory infections in the last 12 months due to environmental tobacco smoke (ETS) and atopy in children without *in utero* exposure to maternal smoke (*n* = 910)

Predictors[a]	β	SE	*t*	OR	95% CI
A(-)ETS1	0.280	0.235	1.19	1.32	0.83–2.10
A(-)ETS2	0.555	0.254	2.19	1.74	1.06–2.87
A(+)ETS0	1.052	0.294	3.58	2.86	1.61–5.10
A(+)ETS1	1.220	0.285	4.27	3.39	1.93–5.93
A(+)ETS2	1.197	0.337	3.55	3.31	1.71–6.42

OR, odds ratio.
[a]ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked in household; ETS0 = no cigarettes smoked in household; A(-) = no allergy diagnosed by medical doctor; A(+) = allergy diagnosed by medical doctor.

Table 4. Relative risk of excessive episodes of acute respiratory infections in the last 12 months due to environmental tobacco smoke (ETS) in nonatopic children (*n* = 833)

Predictors[a]	β	SE	*t*	OR	95% CI
SP(-)ETS1	0.280	0.235	1.19	1.32	0.83–2.10
SP(-)ETS2	0.555	0.254	2.19	1.74	1.06–2.87
SP(+)ETS1	0.842	0.366	2.30	2.32	1.13–4.76
SP(+)ETS2	0.855	0.292	2.92	2.35	1.32–4.17

OR, odds ratio.
[a]ETS1 = 9 or fewer cigarettes smoked daily in household; ETS2 = 10 or more cigarettes smoked in household; SP(-) = no by mother smoking during pregnancy; SP(+) = by mother smoking during pregnancy.

Relative risk estimates for the occurrence of susceptibility to respiratory infections were estimated using multiple logistic models in which, in addition to allergy and smoking during pregnancy, the effect of ETS was considered at two levels (Table 2). The data showed that allergy in children was the strongest factor in terms of odds ratios (ORs) (OR = 2.3; 95% CI, 1.68–3.17); however, the effect of ETS was only significant at the higher level of exposure (OR = 1.54; 95% CI, 1.01–2.32). The same statistical model applied to the group of children without *in utero* exposure to tobacco smoke showed that the presence of allergy significantly strengthens the effect of ETS (Table 3). While for infections and ETS1 with the absence of allergy, the OR was 1.32 (95% CI, 0.83–2.10), ETS combined with allergy nearly tripled the risk (OR = 3.39; 95% CI, 1.93–5.93). Neither in this model nor in the subsequent multivariate logistic analysis was the social index of family based on educational level of parents significant if the ETS variable was introduced.

Table 4 presents the results of the analysis in the group of children without atopy. The model confirmed that without prenatal exposure to smoke, the effect of ETS was significant only for higher ETS exposure (OR = 1.74; 95% CI, 1.06–2.87), when combined with prenatal exposure to smoke, it became significant both at the lower ETS level (OR = 2.32; 95% CI, 1.13–4.76) and at the higher ETS level (OR = 2.35; 95% CI, 1.32–4.17). Comparing ORs related to smoking status of mothers during pregnancy with that of postnatal exposure to ETS, one can see that ETS exposure alone was weaker than the combined effect of congenital exposure to tobacco smoke and ETS.

Discussion

Our study provides strong evidence that older children exposed to ETS in their homes are considerably more susceptible to acute respiratory tract illnesses than unexposed children. A dose–response relationship between degree of exposure (as measured by number of cigarettes smoked in the household) and the excess rates of respiratory episodes supports a causal explanation for the association observed. The significant trend of increased susceptibility to respiratory infections with the level of ETS exposure, after adjusting for allergy and smoking during pregnancy, suggests the existence of a direct effect of ETS exposure on the child's respiratory health. A stronger effect of ETS in atopic children and in

those whose mothers smoked during pregnancy may be explained by the fact that their increased susceptibility resulted from biologic interaction of endogenous and environmental factors.

Our study shows that maternal smoking during pregnancy has a modifying effect on the risk of respiratory illnesses due to ETS after accounting for atopy. We suggest that smoking during pregnancy has long-lasting health consequences in terms of prolonged lung predisposition to acute respiratory illnesses during childhood. However, we could not measure precisely the congenital effects of tobacco smoke because most of the mothers who smoked during pregnancy continued to smoke after the child was delivered and thus the child was exposed postnatally to ETS. Only seven mothers who smoked during pregnancy stopped smoking after delivery of the child, and the size of the sample was too small to allow meaningful statistical analysis.

This study is in good agreement with numerous studies dealing with the incidence of respiratory illnesses and exposure to ETS in small children that have shown a positive association between such illnesses and exposure to ETS. Bronchitis, pneumonia, and other lower respiratory tract illnesses occur up to twice as often during the first 2 years of life in children who have one or more parents who smoke than in children of nonsmokers (*1,2,25*).

However, the results of our study are at variance with results obtained by Taylor and Wadsworth (*26*) in a large cohort of 5-year-old children. The authors said that maternal, but not paternal, smoking had a significant influence on the reported incidence of bronchitis and hospital admission for lower respiratory tract illness in the first 5 years of life. Reported rates of admissions to the hospital for lower respiratory tract diseases were higher in children of mothers that smoked during pregnancy, but the rates were just as high in children born to mothers who started smoking only postnatally and in children of mothers who remained nonsmokers. The authors concluded that maternal smoking affects the incidence of respiratory illnesses in children mainly through a congenital effect and only to a lesser extent through passive exposure after birth. Discrepancies between the Taylor and Wadsworth study and our study may be due to differences in ETS definition. In their study (*26*), ETS exposure was based on maternal and paternal smoking habit reported in the interview. This may be a source of bias in estimating ETS exposure level, as a number of cigarettes smoked daily by parents may not reflect the real level of ETS exposure in the household. In our study ETS exposure level was estimated from the number of cigarettes smoked daily in the household irrespective of parents' smoking habit. Another source of discrepancy may have been the definition of health outcome, as we did not analyze severity of infections or hospital admissions.

Most studies have found that the ETS effect is stronger among children whose mothers smoke than among those whose fathers smoke. This finding is often used to support the hypothesis that *in utero* exposure to maternal smoke has a stronger effect than exposure to ETS. Our study provides fairly convincing evidence that the increased incidence of acute respiratory diseases cannot be attributed exclusively to *in utero* exposure to maternal smoke. In fact, Chen (*27*) and co-workers (*28,29*) reported an increased risk of acute respiratory illnesses in Chinese children living with smoking fathers in the absence of smoking mothers. Furthermore, Woodward and coworkers (*30*) found that children of smoking mothers were significantly more susceptible to acute respiratory illnesses even after mothers who smoked during pregnancy were excluded from the analysis.

Exposure measurement bias may have influenced the results of our study. Epidemiologic studies rely mostly on questionnaires or interviews to assess exposure. However, unvalidated self-reports of cigarette smoking may bias true estimates of relative risk of smoking-related health outcomes (*31–34*). A comparison of smoking habit status assessed by questionnaire with plasma cotinine levels at delivery carried out recently by Jedrychowski et al. (in preparation) shows that questionnaire data regarding smoking habit during pregnancy are of low sensitivity (52%) but of high specificity (98%). If one applies these validity estimates of tobacco smoking during pregnancy to respiratory infections in children, the observed risk is underestimated by the exposure misclassification error (*35,36*). The uncorrected OR for increased incidence of respiratory infections and smoking during pregnancy was 1.8, but after adjustment to exposure misclassification error, the OR appeared to be 2.3. One can assume that estimated effects of passive smoking are also biased in the same direction.

We may conclude that ETS exposure is an important factor increasing susceptibility of older children to respiratory infections, and this effect is independent from the atopic constitution of the child and eventual congenital effect of smoking. This issue is of great relevance for public health, as children with higher risk of respiratory infections may be more susceptible in adolescence or in adulthood to the effects of passive or active smoking and possibly also to other environmental hazards. Respiratory infections also increase demands for medical interventions in terms of outpatient services and hospital admissions. In addition, these illnesses cause missed days of school, and caring for a sick child may lead to absenteeism from work.

References

1. National Research Council. Environmental tobacco smoke: measuring exposures and assessing health effects. Washington, DC:National Academy Press, 1986.
2. U.S. DHHS. The health consequences of involuntary smoking. A report of the Surgeon General. (PHS) 87–8398. Washington, DC:Department of Health and Human Services, 1986.
3. Bland M, Bewley BR, Pollard V, Banks MH. Effect of children's and parents' smoking on respiratory symptoms. Arch Dis Child 53:100–105 (1978).
4. Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins IT. Passive smoking in childhood. Respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis 133:966–973 (1986).
5. Cameron P, Kostin JS, Zaks JM. The health of smokers' and nonsmokers' children. J Allergy Clin Immunol 43:336–341 (1969).
6. Colley JTR, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. Lancet 2:1031–1034 (1974).
7. Ekwo EE, Weinberger MM, Lachenbruch PA, Huntley WH. Relationship of parental smoking and gas cooking to respiratory disease in children. Chest 84:662–668 (1983).
8. Forastiere F, Corbo GM, Michelozzi P, Pistelli R, Agabiti N, Brancato G, Ciappi G, Perucci CA. Effect of environment and passive smoking on the respiratory health of children. Int J Epidemiol 21:66–73 (1992).
9. Lebowitz MD, Burrows B. Respiratory symptoms related to smoking habits of family adults. Chest 69:48–50 (1976).
10. Leeder SR, Corkhill RT, Irwig IM. Influence of family factors on asthma and wheezing during the first five years of life. Br J Prev Soc Med 30:213–218 (1976).
11. Martinez FD, Antognoni G, Macri F. Parental smoking enhances bronchial responsiveness in nine-year-old children. Am Rev Respir Dis 138:518–523 (1988).
12. Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. Pediatrics 89:21–26 (1992).
13. Monto AS. Acute respiratory infection in children in developing countries: challenge of the 1990s. Rev Infect Dis 11:498–505 (1989).
14. Murray AB, Morrison BJ. The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma. J Allergy Clin Immunol 77:575–581 (1986).
15. O'Connor GT, Weiss ST, Tager IB. The effect of passive smoking on pulmonary function and nonspecific bronchial responsiveness in a population-based sample of children and young

adults. Am Rev Respir Dis 135:800–804 (1987).
16. Pandey MR, Boleij JSM, Smith KR. Indoor air pollution in developing countries and acute respiratory infection in children. Lancet 1:427–429 (1989).
17. Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease: the effect of host factors and home environmental exposures. Am Rev Respir Dis 128:1038–1043 (1983).
18. Stoddard JJ, Miller T. Impact of parental smoking on the prevalence of wheezing respiratory illness in children. Am J Epidemiol 141:96–102 (1995).
19. Tager IB, Weiss ST, Rosner B, Speizer FE, Spengler JD, Ferris BG. Respiratory illness in children exposed to ambient air pollution, cigarette smoke, and gas stoves in six cities. Am Rev Respir Dis 110:15–26 (1979).
20. Victoria CG, Smith PG, Barros FC, Vaughan JP, Fuchs SC. Risk factors for deaths due to respiratory infections among Brazilian infants. Int J Epidemiol 18:918–925 (1989).
21. Ware JH, Dockery DW, Spiro A, Speizer FE, Ferris BG. Passive smoking, gas cooking, and respiratory health of children living in six cities. Am Rev Respir Dis 129:366–374 (1984).
22. Weiss ST, Tager IB, Speizer FE, Rosner B. Persistent wheeze: its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. Am Rev Respir Dis 122:697–707 (1980).
23. Weiss ST, Tager IB, Schenker M, Speizer E. State of Art. The health effects of involuntary smoking. Am Rev Respir Dis 128:933–942 (1983).
24. Weitzmann M, Gortmaker S, Walker DK. Maternal smoking and childhood asthma. Pediatrics 85:505–511 (1990).
25. U.S. EPA. Respiratory health effects of passive smoking: lung cancer and other disorders. EPA/600/6–90/006F. Washington, DC: Environmental Protection Agency, 1992.
26. Taylor B, Wadsworth J. Maternal smoking during pregnancy and lower repiratory tract illness early in life. Arch Dis Child 62:786–789 (1987).
27. Chen Y. Synergistic effect of passive smoking and artificial feeding on the hospitalization for respiratory illness in early childhood. Chest 95:1004–1007 (1989).
28. Chen Y, Li W, Yu S. Influence of passive smoking on admissions for respiratory illness in early childhood. Br Med J 293:303–306 (1986).
29. Chen Y, Li W, Yu S, Qian W. Chang-Ning epidemiological study of children's health: I. Passive smoking and children's respiratory diseases. Int J Epidemiol 17:348–355 (1988).
30. Woodward A, Douglas RM, Graham NMH, Miles H. Acute respiratory illness in Adelaide children: breastfeeding modifies the effect of passive smoking. J Epidemiol Commun Health 44:224–230 (1990).
31. Coultas DB, Howard CA, Peake GT, Skipper BJ, Samet JM. Discrepancies between self-reported and validated cigarette smoking in a community survey of New Mexico Hispanics. Am Rev Respir Dis 137:810–814 (1988).
32. Haddow JE, Knight GJ, Palomaki GE, McCarthy JE. Second-trimester serum nicotine levels in nonsmokers in relation to birth weight. Am J Obstet Gynecol 159:481–484 (1988).
33. Idle JR. Titrating exposure to tobacco smoke using cotinine—a minefield of misunderstanding. Commentary. J Clin Epidemiol 43: 313–317 (1990).
34. Perez-Stable EJ, Marin G, Marin BV, Benowitz NL. Misclassification of smoking status by self-reported cigarette consumption. Am Rev Respir Dis 145:53–57 (1992)
35. Barron BA. The effects of misclassification on the estimation of relative risk. Biometrics 33:414–418 (1977).
36. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic research. Principles and quantitative methods. New York:Van Nostrand Reinhold, 1982;220–241.