Parental Smoking and the Risk of Childhood Asthma

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Abstract: Data from two random population surveys are used to assess the relationship between parental smoking and the prevalence of asthma in children aged 0–17. Data from a 1977 Midwestern urbanized county indicate that, if mothers smoked, the prevalence of parent reported asthma increased from 5.0 per cent to 7.7 per cent (estimated relative risk of 1.5), and the prevalence of functionally impairing asthma increased from 1.1 per cent to 2.2 per cent (relative risk of 2.0). In a more rural Eastern county in 1980, a lower overall prevalence of asthma was noted. However, similar estimated relative risks of asthma (1.8) and functionally impairing asthma (2.4) were found to be associated with maternal smoking. Inconsistent relationships were found between the estimated prevalence of asthma and paternal smoking. When multivariate controls were introduced, the relationships between maternal smoking and asthma persisted. Estimated attributable risks indicate that between 18 per cent and 34 per cent of the asthma reported in these samples can be attributed to maternal smoking. Implications of these findings for primary care physicians are discussed. (*Am J Public Health* 1982; 72:574–579.)

Introduction

The health hazards of cigarette smoking have been well documented and widely accepted.[1–4] Recently attention has focused on the relationship between exposure to cigarette smoke and the health of nonsmokers. Several studies [5–7] have noted the substantial effects of cigarette smoke on "indoor pollution." Repace and Lowrey, for example, conclude that "levels of respirable suspended particulates in places where tobacco is smoked greatly exceed levels found in smoke-free environments, outdoors, and vehicles on busy commuter highways."[8] White and Froeb found that nonsmokers who were chronically exposed to cigarette smoke in the workplace had levels of pulmonary function similar to that of light smokers and lower than nonsmokers in a smoke-free environment.[9] Hirayama found a significantly increased risk of lung cancer for nonsmoking wives of heavy smokers.[10]

The present study focuses upon parental smoking and childhood asthma. Asthma is one of the leading causes of chronic illness in children; children with asthma experience a characteristic hyperreactivity of the airways to a variety of environmental factors, including irritants such as tobacco smoke.[11] Studies have noted relationships between air pollution and the onset of asthma attacks,[12,13] but no significant relationships between parental smoking and the prevalence of asthma have been reported. Several studies have noted relationships between parental smoking and acute respiratory illness in children,[14–18] and the results have been fairly consistent. A significant dose-response relationship was found between parental smoking and reported bronchitis and pneumonia in infants by Harlap and Davies[19] and Colley, *et al.*[20] One study found a dose-response relationship between parental smoking and adenoidectomy and tonsillectomy in children,[21] and another demonstrated a significant relationship to pulmonary function in children.[22] O'Connell and Logan examined clinical records of asthmatic and nonasthmatic children; they found only a small difference in the incidence of parental smoking, but a majority of the parents of their asthmatic children reported that cigarette smoke aggravated the asthma, and elimination of smoking generally led to improvement.[23]

The analyses described below report on the results of two population surveys carried out in 1977 and 1980 in two locations across the United States.

Materials and Methods

A random household health survey was conducted in Genesee County, Michigan in 1977. Information upon 3,072 children (aged 0–17) and their households was obtained from an adult family member, usually the mother. The response rate was 81 per cent. The city of Flint, Michigan (population 165,000 in 1977) is an industrial city in the southeastern region of the state of Michigan. Flint and the surrounding Genesee County (total population 450,000) are heavily dependent upon automobile-related industries for employment.

Berkshire County, Massachusetts (population in 1980 of 146,000) is a relatively rural county which forms the western

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boundary of the State of Massachusetts. The largest city is Pittsfield (1980 population of 53,000). A randomly dialed telephone survey of households with children (aged 0–17) was conducted in 1980, using many identical questions from the Genesee County survey. The response rate for this survey was 81 per cent; information upon 894 children was available for analysis.

The smoking habits of mothers and fathers in the households were assessed using identical questions in both the Michigan and Massachusetts surveys; the respondent was asked whether or not each household member smoked cigarettes, and if so, how much. For analysis, mothers and fathers were classified as either smokers or nonsmokers. The samples were too small to reliably estimate relations involving different amounts of smoking.*

The presence of asthma among children was assessed using a 22-item chronic condition checklist in the Genesee County survey, and a 21-item checklist in the Berkshire County survey. In Genesee County, the parents were handed a card listing all of the conditions (Appendix A). The same checklist except for the item "bronchitis," was read to respondents in the Berkshire County survey. The questions asking about asthma, hayfever, and any other kind of allergy were exactly the same as questions asked in earlier surveys in Rochester, New York[24] and in the National Health Examination Survey,[25] and are very similar to checklists used in the National Health Interview Survey.[26]

If the parent (usually the mother) confirmed that the child had a certain condition, they were asked, "Does it affect his/her ability to attend school or do any of the things a child his/her age usually does?" "Yes" responses to this question were coded as indicating a functionally impairing condition. Parents were also asked whether a doctor had been seen about this condition.

A number of studies have indicated that parent reports in general overestimate the prevalence of clinically diagnosed chronic conditions. Others have noted, however, that overreporting tends to decline with the reported severity of the condition, i.e., more severe conditions, which have a greater impact upon the individual, tend to be more completely reported.[24,27]

For example, a household survey in Alamance County, North Carolina resulted in an estimated prevalence of a variety of respiratory conditions in children under age 21 of 16.3 per cent. In contrast, a clinically validated estimate (moderate and severe cases only) of 2.9 per cent was obtained.[28] Another household survey in Rochester, New York (utilizing exactly the same questions as the present study) produced a prevalence estimate of 3.2 per cent of asthma in children 0–17 years of age. Follow-up interviews of children aged 6–17 resulted in a validated estimate of .9 per cent prevalence of asthma.[24] These estimates are also consistent with validated estimates obtained in a population study on the Isle of Wight in England for 10–12 year olds; a prevalence of asthma of 2.3 per cent was estimated; when only chronically handicapping asthma was considered, this estimate was halved.[29]

In summary, significant errors in the reporting of childhood asthma have been found in studies utilizing parent interviews, although there is evidence that these errors tend to decrease if the severity of the condition is taken into account. It is important to note, however, that these errors will not bias relationships estimated between parental smoking and asthma unless asthma misreporting is related to parental smoking.** Tests for such systematic bias are reported below.

Results

Overall, the estimated prevalence of asthma in these samples appears similar to that found in a national sample of children in the 1963–1965 National Health Examination Survey,[32] 5.3 per cent among children aged 6–11. The corresponding estimates in Genesee County (1977) and Berkshire County (1980) are 6.5 per cent and 2.6 per cent respectively. The lower estimate for Berkshire County also corresponds to relationships reported from National Health Interview Survey data in 1970, which noted a decreased prevalence of asthma among children living in rural areas, as opposed to urban areas.[26]

The estimated prevalence of functionally impairing asthma in the present samples (1.5 per cent, .8 per cent) is also comparable to the clinically validated prevalence estimates noted above; again, a higher prevalence is found in the urban sample.

Validation of these parent reports is made possible through use of other questions in the household surveys. In the Michigan survey, if a child was noted as having asthma, parents were asked if they had "been to a doctor about this?" All of the parents who reported a child with asthma said they had been to a doctor about the child's condition. In the Massachusetts survey, another question asked for all children was the following: "Today or yesterday has (child's name) taken or used any medicine, salves, or pills that were prescribed by a doctor?" Of the 29 children in this sample reported by parents as asthmatic, seven were reported to be taking asthma medication regularly; the medication was specified in six of these cases, and no children without asthma were reported as receiving asthma medication. Of the seven children in the sample identified as having functionally impairing asthma, four were reported to be taking medication for the condition. The answers to both of these survey questions suggest that the parent reports are valid

*Of the mothers who smoked, 61 per cent in the Michigan sample and 56 per cent in the Massachusetts sample reported smoking a half to one pack per day.

**In regression analysis, it is well known that random error in the dependent variable will not bias estimates of regression coefficients, although the explained variance will decrease.[30] The analogous corollary for the logistic regressions used later is derived from the well-known fact that odds ratios that characterize a cross-classification are invariant to multiplication of rows or columns by a constant.[31] Thus, if both smoking and nonsmoking mothers overreport asthma by a proportionate amount, estimates of odds ratios will not be affected.

TABLE 1—Estimated Prevalence of Asthma in Children, by Smoking of Mothers; Genesee County, Michigan (1977) and Berkshire County, Massachusetts (1980).

Site/Condition	Maternal Smoking Yes	Maternal Smoking No	Estimated Relative Risk	χ^2	p-value (one-tailed)
Genesee County, MI (N)	(1,255)*	(1,817)			
Asthma	7.7	5.0	1.53	9.1	.001
Functionally Impairing Asthma	2.2	1.1	1.95	5.4	.01
Berkshire County, MA (N)	(330)	(564)			
Asthma	4.5	2.5	1.83	2.8	.05
Functionally Impairing Asthma	1.2	0.5	2.28	1.2	.13

*Sample sizes are given in parentheses.

and again indicate that parent reporting likely increases in accuracy as the severity of the condition increases.

In the Genesee County, Michigan sample, 41 per cent of the children aged 0–17 had mothers who smoked. The estimated prevalence of asthma was 7.7 per cent for children of smoking mothers, and 5 per cent for children of nonsmoking mothers (Table 1). These differences are statistically significant at p = .001*** A similar relationship is also noted in this Table between maternal smoking and the estimated prevalence of functionally impairing asthma, 1.1 per cent versus 2.2 per cent; indicating a doubling in the risk among children of smoking mothers.

Data from the more rural Berkshire County sample produced similar estimates of relative risk, although the significance levels were lower due to the smaller sample size. Thirty-seven per cent of the children aged 0–17 in this sample had a mother who smoked. The estimated prevalence of asthma symptoms increased from 2.5 per cent to 4.5 per cent among children with mothers that smoked, and the estimated prevalence of functionally impairing asthma doubled (from .5 per cent to 1.2 per cent).

No significant differences in the relationships between maternal smoking and asthma, and maternal smoking and functionally impairing asthma were found between the two geographic areas. Log-linear models were fitted, testing for this three-way interaction, and no significant differences emerged. Thus, data from the two sites were pooled, using a variant of the Mantel-Haenszel procedure.[31] This analysis, as expected, results in slightly higher levels of statistical significance of the asthma and smoking relationships.

Other Explanatory Variables

The relationship of paternal smoking to childhood asthma was also explored in both samples, but the results were inconsistent. In Genesee County, Michigan, significant relationships were found between paternal smoking and the prevalence of childhood asthma but not of functionally impairing asthma. In Berkshire County, no significant relationships between paternal smoking and the risk of asthma or functionally impairing asthma were observed.

Parents who smoke could conceivably be over-sensitized to the effects of their smoking, and thus could overreport conditions in their children. This possible bias in symptom reporting was explored by looking at the relationship between maternal and paternal smoking and other chronic conditions‡ for which respiratory symptoms might appear. No significant relationships were found.

Multivariate logistic regressions‡‡ were estimated which predicted asthma and functionally impairing asthma among children from mothers' smoking habits, as well as from the smoking habits of fathers, whether the child had hayfever or any other allergies, and socioeconomic and demographic characteristics of the family and child. The coefficient estimates from these equations estimated from

***All tests of the smoking/asthma relationship will be one-tailed tests; other tests will be two-tailed. It should also be noted that the sample in fact consists of data upon clusters of children; thus, even though households were selected randomly, some design effect is introduced into the sample. We estimate from the present samples that roh,[33] the intraclass correlation coefficient which measures this tendency towards homogeneity within clusters, is .16 for asthma, and .21 for function impairing asthma. The effect of this clustering is to reduce the precision of the survey estimates. For example, in the Genesee County sample a 95 per cent confidence interval around the proportion of children reported as asthmatic by mothers that do not smoke can be estimated at 4–6 per cent if the effects of clustering are ignored. If this design effect is taken into account, the 95 per cent confidence interval becomes 3.8–6.2 per cent. Others have found that design effects are less pronounced for regression coefficients than for estimates of means and proportions.[34] Regressions were run based upon households which predicted the proportion of children in a household with asthma, controlling for family size and, other variables noted later. These results were consistent with results presented later, and thus indicate no significant consequences of this design effect.

‡These conditions included hayfever and any other allergies.

‡‡These logistic regressions estimate linear associations between the predictor variables and the logarithm of the odds for asthma. They were estimated using MMLA, a computer program which produces maximum likelihood estimates, written by W. W. Hauck, Illinois Cancer Council, Chicago, Illinois.

TABLE 2—Coefficient Estimates from Logistic Regressions Predicting Asthma and Functionally Impairing Asthma in Children 0–17, Genesee County, MI, 1977

Variable	Equation Predicting Asthma			Equation Predicting Function-Impairing Asthma		
	Coefficient Estimate	t-Statistic	Odds-ratio	Coefficient Estimate	t-Statistic	Odds-ratio
Mother Smokes (Yes = 1)†	.401	2.45**	1.49	.613	1.94*	1.85
Father Smokes (Yes = 1)	.398	2.40*	1.49	.076	.24	1.08
Age of Child (1 ≤ 5)	−.358	−1.79	.70	−.299	−.78	.74
Sex of Child (1 = male)	.115	.73	1.12	−.236	−.79	.79
Income of Family (1 = poverty; 2 = near poverty; 3 = higher)	.028	.20	1.03	−.077	−.33	.93
Mother's Education	−.226	−2.32*	.80	−.488	−2.44*	.61
Child has Allergies (Yes = 1)	1.169	6.44**	3.22	1.230	3.70**	3.42
Child has Hayfever (Yes = 1)	1.686	8.51**	5.40	1.585	4.36**	4.88

*significant at p = .05
**significant at p = .01
†Other responses coded as "0" if first category is "1"

the Genesee County data are displayed in Table 2 above. A variety of other variables are not included in this Table because they did not add any explanatory power to these equations. These include the number of persons in the household, race, environmental deficiencies observed near the home (available only in the Genesee County sample), number of rooms in the house, and mother's work status.

The most important result of this analysis is that the addition of all of these control variables did not substantially alter the estimated relationships between maternal smoking and the prevalence of asthma and functionally impairing asthma. The estimated odds-ratios which describe these relationships, controlling for the variables, are only attenuated slightly from "unadjusted" estimated odds ratios derived from Table 1.‡‡‡ For example, the odds-ratio relating asthma and maternal smoking changes from 1.57 to 1.49; the corresponding change for the functionally impairing asthma odds ratio is from 1.98 to 1.85. Similar equations were estimated for the Berkshire County sample, resulting in similar estimates of the relationship between maternal smoking and asthma. The smaller sample size from this site, however, resulted in lower levels of statistical significance. These analyses also confirmed the fact that paternal smoking did not in general predict chronic respiratory problems once maternal smoking was controlled.

Bronchitis was included as one of the items on the chronic condition checklist in the Genesee County sample, and significant relationships were found between the prevalence of bronchitis and maternal smoking ($p < .001$; estimated relative risk of 1.5). However, no association with functionally impairing bronchitis was observed, and no significant association between bronchitis and paternal smoking was found. These relationships suggest another effect of maternal smoking, similar to the known effects of individual smoking upon the production of bronchitis in the smoker.[35] This relationship may also reflect the fact that some children with asthma are misdiagnosed as having chronic bronchitis.[36] The converse, however, could also be true. We reestimated the equations described in Table 2 including an additional control variable indicating whether the child had bronchitis or not. While the bronchitis variable was significant in these equations, maternal smoking was still significantly related to both asthma and function impairing asthma, and the coefficient estimates were only slightly attenuated (to 1.42 and 1.70).

One other variable which could have important implications is the presence of asthma in parents, but we believe that this variable could have an attenuating effect upon the present relationships. We assume that parents with a history of asthma have a smaller probability of taking up smoking. Thus, there could be a selection of "non-asthmatic" parents into the group of smokers, and subsequently a selection of "non-asthmatic" children to smoking parents, a situation that would result in an attenuated relationship between parental smoking and childhood asthma.

Estimates of Attributable Risk

The significance of these estimated relationships for both medical practice and public health are strongly conditioned by the proportion of mothers that smoke; because more than one-third of mothers in both of these samples smoke, the estimated attributable risks[37] associated with maternal smoking are substantial.* The estimates of relative risk in Table 1 indicate that 18 per cent and 23 per cent respectively of asthma in children in the two sites may be

‡‡‡In the present situation, the odds ratio closely approximates the estimated relative risk.

*Attributable risk can be defined as the "maximum proportion of a disease that can be attributed to a characteristic or etiological factor."[37]

attributed to maternal smoking, and 28 per cent and 34 per cent respectively of functionally impairing asthma in the two populations can be attributed to maternal smoking.

We have already indicated above that the estimated relative risks associated with maternal smoking are somewhat attenuated as control variables are introduced into the logistic regressions; if these estimates are used in the calculations, slightly smaller attributable risks result (e.g., 17 per cent and 26 per cent rather than 18 per cent and 28 per cent).

Because of the small numbers of conditions reported, the 95 per cent confidence intervals[38] around these estimates are necessarily large; for example, the confidence interval around the estimated percentage of asthma in Genesee County, Michigan, attributable to maternal smoking ranges from 5 per cent to 29 per cent. In spite of this imprecision, the similarity of the estimated attributable risks in the two different populations tends to validate the estimates. Other validations are still needed, however, ideally using more refined measures of both the presence of asthma, the level of functional impairment involved, and the actual air pollution which may be attributed to maternal and paternal smoking.

Smoking and Disability Days

Because other studies have reported relationships between acute illness and parental smoking[14–18] and substantial relationships are reported here between asthma and maternal smoking, an obvious question to be addressed is the extent to which the relationships between acute illness and maternal smoking may be due in part or in whole to the relationships estimated here between chronic illnesses such as asthma and bronchitis and maternal smoking. Maternal smoking and the presence of asthma and bronchitis in the Genesee County sample were all associated with the likelihood of a disability day in the past two weeks. When the presence of asthma and bronchitis was controlled, maternal smoking was still significantly ($p = .04$) related to the odds of a disability day, although the magnitude of the estimated relationships was reduced; e.g., the estimated odds-ratio changed from 1.46 to 1.29 when these controls were introduced. Thus, it appears that the relationships estimated above between maternal smoking and the prevalence of chronic respiratory conditions do not account for all of the often reported relationships between parental smoking and acute respiratory illnesses in children.

Discussion

It is useful to conceptually distinguish three possible roles by which an exogenous factor like tobacco smoke might play a role in the pathogenesis of asthma. Such a factor

> ". . . might be responsible for the inception of asthma by inducing a state of hyperreactivity in the bronchi. Second, it might maintain and reinforce hyperreactivity. Third, it might provoke the expression of hyperreactivity, giving rise to clinically recognizable attacks of asthma.[35]

While the present data do not shed light on the precise role of maternal smoking in the development of childhood asthma, the analyses clearly indicate an influence controlling for other known factors such as hayfever and allergies.[11] Although estimates of attributable risk should be interpreted with these comments in mind, the present data suggest that maternal smoking can be considered an important factor in the occurrence of childhood asthma.

These data can also be considered as another illustration of the extent to which indoor air pollution is a significant determinant of individual health status. As current energy conservation measures in the United States reduce the flow of air through households, the consequences of indoor pollution due to cigarette smoking could certainly increase in magnitude.

The findings from this study suggest that an opportunity exists for health care providers to help prevent asthma in children, and to reduce the level of functional impairment of asthmatic children. One strategy would be to encourage smoking mothers of children with clinically diagnosed asthma to quit smoking. Some effective therapies have been devised to help smokers end their habit,[39] and referrals to such programs could be arranged.

A number of current pediatric texts do not mention such an approach to treating the asthmatic child.[11,36] Evaluations of the possible efficacy of these interventions in reducing the prevalence of asthma and functionally impairing asthma could add significantly to current knowledge.

A broader preventive strategy could involve encouraging the reduction or elimination of maternal smoking among families with a history of allergies. Such families have a greater risk of producing allergic children. This approach parallels the commonly accepted practice of avoidance therapy with household pets for allergic families.[40] There are also substantial benefits to be gained by the mothers themselves if these interventions are successful.

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APPENDIX A

Checklist of Chronic Conditions and Impairments:

(a) Asthma
(b) Hay Fever
(c) Any other kind of allergy
(d) Any trouble with his/her kidneys
(e) Anything wrong with his/her heart
(f) Any difficulty hearing
(g) Any difficulty seeing (even with glasses)
(h) Trouble speaking (stammering, lisping, hard to understand)
(i) Missing fingers, hand, arm, toes, foot or leg
(j) Any permanent stiffness or deformity of foot, leg, fingers, arm or back
(k) Condition present since birth, such as club foot or cleft palate
(l) Paralysis of any kind
(m) Mental impairment or retardation
(n) Arthritis
(o) Bronchitis
(p) Epilepsy, convulsions
(q) Cerebral palsy
(r) Diabetes
(s) Anything else