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Residential Exposures Associated With Asthma in US Children

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ABSTRACT. *Objective.* Residential exposures are recognized risk factors for childhood asthma, but the relative contribution of specific risk factors and the overall contribution of housing to asthma in US children is unknown. The objective of this study was to identify risk factors and estimate the population attributable risk of residential exposures for doctor-diagnosed asthma for US children.

Methods. A cross-sectional survey was conducted from 1988 to 1994. Survey participants were 8257 children who were <6 years old and who participated in the Third National Health and Nutrition Examination Survey, a survey of the health and nutritional status of children and adults in the United States. The main outcome measure was doctor-diagnosed asthma, as reported by the parent.

Results. Six percent of children had doctor-diagnosed asthma. The prevalence of asthma was higher among boys (6.7%) than girls (5.1%) and was higher among black children (8.9%) than white children (5.2%). Risk factors for doctor-diagnosed asthma included a family history of atopy (odds ratio [OR]: 2.2; 95% confidence interval [CI]: 1.5, 3.1), child's history of allergy to a pet (OR: 24.2; 95% CI: 8.4, 69.5), exposure to environmental tobacco smoke (OR: 1.8; 95% CI: 1.2–2.6), use of a gas stove or oven for heat (OR: 1.8; 95% CI: 1.02–3.2), and presence of a dog in the household (OR: 1.6; 95% CI: 1.1, 2.3). The population attributable risk of ≥ 1 residential exposure for doctor-diagnosed asthma in US children <6 years old was 39.2%, or an estimated 533 000 excess cases, whereas having a family history of atopy accounted for 300 000. The attributable cost of asthma as a result of residential exposures for children <6 years old was \$402 million (95% CI: \$296–\$507 million) annually.

Conclusions. The elimination of identified residential risk factors, if causally associated with asthma, would result in a 39% decline in doctor-diagnosed asthma among US children <6 years old. *Pediatrics* 2001; 107:505–511; *NHANES, children, pediatric, prevention, epidemiology, allergic rhinitis, medical costs, day care, housing, pets and environment.*

ABBREVIATIONS. ETS, environmental tobacco smoke; PAR, population attributable risk; NHANES, National Health and Nutrition Examination Survey; 95% CI, confidence interval; OR, odds ratio.

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Asthma, the most common chronic illness of childhood, is estimated to affect more than 4 million children in the United States.¹ Despite advances in therapy and in our understanding of the pathophysiology of this disease, there has been an increase in the prevalence, morbidity, and mortality of children with asthma during the past 2 decades.^{1–3} From 1980 to 1993, the prevalence of asthma increased by 75%, with the largest increase occurring in children <5 years old.¹ Each year, asthma leads to more than 3 million clinic visits, 550 000 emergency visits, 150 000 hospitalizations, and in excess of 150 deaths in children <15 years old.¹

Numerous risk factors for childhood asthma have been identified. Residential exposures, including environmental tobacco smoke (ETS) and indoor allergens, have been shown consistently to be potent risk factors for the development and exacerbation of asthma.^{4–19} Specific risk factors implicated in childhood asthma often vary by geography, urbanization, and poverty. Thus, with the exception of ETS,^{8,9} the relative contribution of specific residential exposures and the overall contribution of housing factors to asthma in US children remains unclear. Estimating the contribution of residential exposures and housing is critical to developing a strategy to prevent childhood asthma. The objective of this study was to identify risk factors and estimate the population attributable risk (PAR) of residential exposures for doctor-diagnosed asthma among a representative sample of children <6 years old in the United States.

METHODS

The Third National Health and Nutrition Examination Survey (NHANES III), conducted from 1988 to 1994, was the source of data for this study. NHANES III is a cross-sectional, random household survey of the civilian, noninstitutionalized population that used a complex, multistage probability sampling design. The definition of asthma used in this study was based on parent report, as determined by a positive response to the survey question, "Were you ever told by a doctor that your child had asthma?"

A review of the literature on the cause of asthma was conducted to identify environmental and residential risk factors for childhood asthma. We focused on modifiable factors that could be investigated by using NHANES III. These included features of housing,²⁰ type of heating,²¹ children's exposure to ETS,^{5,8,9,11–13} exposure or allergies to pets,^{6,7,10} use of a gas stove or oven for heat,²² and day care attendance. Unfortunately, skin tests were not performed for children <6 years old in NHANES III.

Although we were interested primarily in environmental risk factors, we included host-related variables to adjust for possible confounding. These variables included child's age,^{6,9} gender,^{9,11} race or poverty,^{20,23,24} index child's history of hay fever or allergic rhinitis,^{18,25} stay in the neonatal intensive care unit, and low birth weight.²⁰ Measures of socioeconomic status, such as educational

achievement of household head and family income below the federal poverty level, were also included in the analyses.^{23,24} Finally, we used parental history of atopy (defined as history of asthma or allergic rhinitis) as a measure of genetic predisposition to asthma.^{14,18,19}

Bivariate analyses were conducted to determine associations with doctor-diagnosed asthma. Then we developed a summary logistic regression model that included all variables that were associated significantly with doctor-diagnosed asthma in bivariate analyses ($P < .10$).

The independent associations of various factors with doctor-diagnosed asthma were estimated by using logistic regression analysis. The Hosmer-Lemeshow goodness-of-fit test statistic was calculated using SAS software (SAS Institute, Inc, Cary, NC) to measure how well the logistic regression model fit the data. The Hosmer-Lemeshow test statistic indicated that the model provided a good fit (H-L statistic = 5.23; $P = .73$). Ninety-five percent confidence intervals (CIs) were calculated for odds ratios (ORs); those that did not include one were considered statistically significant. Analyses using SUDAAN software (Research Triangle Park Institute, Research Triangle Park, NC) were used to account for the complex, multistaged sampling design of the survey. Sample weights were used to produce national estimates by adjusting for the oversampling of young children and minority groups. The PAR was calculated for independent associations.²⁶

We used published data on the economic cost of asthma to estimate the cost attributable to residential factors.²⁷ Direct costs included clinic and emergency department visits, hospital outpatient services, hospitalization, and medications. Indirect costs included loss of work as a result of school absence and illness days. We presented costs inflated to 1997 dollars using inflation factors based on the *Statistical Abstract of the United States*.²⁸ We compared health services use for children who had residential-associated asthma with those who had no such exposure. Because there was no difference in health service use by residential exposure status, the cost of residential asthma was the product of the fraction of cases attributable to residential exposures and the total cost.

RESULTS

Overall, 491 (5.9%) of the sample of 8257 children surveyed had doctor-diagnosed asthma (Table 1). This prevalence rate corresponds to 1.36 million children <6 years old in the US population. The prevalence of asthma was 5.2% among white children, 8.9% among black children, and 6.3% among children of other racial or ethnic backgrounds. The reported prevalence of doctor-diagnosed asthma was 2.5% in infants and increased to 8.4% in 5-year-old children.

Host factors that were independently associated with doctor-diagnosed asthma included a parental history of atopy (OR: 2.2; 95% CI: 1.5, 3.1) and black race (OR: 1.6; 95% CI: 1.1, 2.3; Table 2). Children with a history of allergic rhinitis, as reported by the parent, were 5 times more likely to have doctor-diagnosed asthma (OR: 5.6; 95% CI: 2.4, 13.0), and boys were at marginally increased risk for having asthma compared with girls (OR: 1.5; 95% CI: 0.97, 2.2).

We identified several residential exposures that were associated with childhood asthma after adjusting for potential confounders in a logistic regression analysis (Table 2). Children who had a history of allergies to a pet—defined as having ever given away or avoided pets due to allergies—were 24 times more likely to have doctor-diagnosed asthma. Presence of a dog in the house (OR: 1.6; 95% CI: 1.1, 2.3), exposure to ETS (OR: 1.7; 95% CI: 1.2, 2.5), and use of gas stove or oven for heat (OR: 1.8; 95% CI: 1.0, 3.1) also were independently associated with doctor-diagnosed asthma.

There was a trend toward age of residence as a risk factor for doctor-diagnosed asthma. Children who lived in housing that was built between 1946 and 1973 were 1.5 times more likely to have doctor-diagnosed asthma compared with children who lived in housing that was built before 1946, but this characteristic did not reach statistical significance (Table 2).

Analyzing for interactions revealed that the effect of tobacco was neither increased nor decreased by the presence of allergies. Similarly, there were no statistically significant interactions between tobacco exposure and family history of asthma or perinatal disease, including low birth weight or stay in a neonatal intensive care unit. There also was no interaction of family history of allergy and presence of a dog or cat in the household.

We calculated the PAR to estimate the contribution of various risk factors for asthma. The PAR represents the cases of asthma that would be prevented if the children were not exposed to specific agents or risk factors. More than 350 000 excess cases of childhood asthma were attributable to having a pet allergy—defined as having given up or avoided a pet because of allergies. Exposure to ETS accounted for 177 000 excess cases of doctor-diagnosed asthma, a dog in the household accounted for 140 000 excess cases, and using a gas stove or oven for heat accounted for 59 000 excess cases (Table 3).

Next, we estimated the PAR of having ≥ 1 residential exposure for doctor-diagnosed asthma. Of the 1.36 million cases of doctor-diagnosed asthma in US children <6 years old, we estimated that 533 000 (39.2%) were attributable to residential exposures. In contrast, having a parent with a history of atopy accounted for approximately 300 000 excess cases (Table 3).

We compared health care use among children who had residential-associated asthma (defined as children with asthma who had 1 or more residential exposures associated with asthma) with those who had nonresidential-associated asthma. Health services use and episodes of wheezing did not differ among children who had 1 or more residential exposures associated with asthma compared with those who did not have any such exposure (Table 4). There were no significant differences in the frequency of wheezing, wheezing apart from a cold, hospitalization, or emergency and clinic visits for children with or without identified residential exposures.

Because there were no significant differences in health services use for children with or without an identified residential exposure, we assumed that the costs associated with residential exposures were proportional to the PAR fraction. On the basis of a PAR of 39.2% (95% CI: 29.8%–49.5%), the total (direct and indirect) cost of asthma attributable to residential risk factors was \$402 million (95% CI: \$296–\$507 million) annually for children <6 years old.

DISCUSSION

The results of these analyses indicate that approximately 532 000, or 39%, of the 1.36 million cases of doctor-diagnosed asthma in US children <6 years

TABLE 1. Prevalence of Doctor-Diagnosed Asthma in NHANES III (1988 to 1994) According to Demographic, Host, and Residential Factors

Variable	Sample Size	Percent Prevalence (95% CI)
Total	8257	5.9 (5.1, 6.7)
Gender		
Male	4086	6.7 (5.7, 7.7)
Female	4171	5.1 (4.0, 6.2)
Race		
Black	2274	8.9 (7.5, 10.3)
White	5642	5.2 (4.2, 6.2)
Other	341	6.3 (2.4, 10.2)
Poverty status		
<100%	2858	8.8 (7.3, 10.3)
101%–150%	1124	7.0 (5.1, 8.9)
>150%	3492	4.7 (3.7, 5.7)
Education of head of family		
<High school graduate	2858	7.6 (6.1, 9.1)
High school graduate	2699	6.6 (4.9, 8.3)
>High school graduate	2512	4.5 (3.3, 5.7)
Received care in neonatal intensive care unit		
Yes	936	6.0 (3.7, 8.3)
No	7308	5.8 (5.1, 6.7)
Birthweight		
<2500 g	672	7.2 (4.3, 10.1)
≥2500	7377	5.8 (5.0, 6.6)
Dog in home		
Yes	1387	6.3 (4.4, 8.3)
No	6844	5.8 (5.0, 6.6)
Smoking		
Prenatal and postnatal	1360	9.5 (7.5, 11.5)
Prenatal only	273	7.6 (3.2, 11.9)
Postnatal only	1741	5.7 (3.9, 7.5)
None	4814	4.8 (3.9, 5.6)
Cat in home		
Yes	969	4.8 (3.0, 6.7)
No	7262	6.2 (5.3, 7.0)
Given up pet due to allergies		
Yes	111	59.3 (41.0, 77.6)
No	8119	5.1 (4.3, 5.9)
Use gas stove or oven for heat		
Yes	775	10.8 (6.6, 15.0)
No	7448	5.6 (4.8, 6.5)
Urban residence		
Yes	4394	5.2 (4.2, 6.2)
No	3863	6.7 (5.5, 7.9)
Year house built		
<1946	1422	5.4 (3.9, 6.9)
1946–1973	3229	7.4 (5.8, 9.0)
>1973	2393	4.6 (3.6, 5.6)
Attend day care		
≥10 h/wk	1981	7.7 (5.9, 9.5)
<10 h/wk	439	5.6 (2.8, 8.4)
None	5837	5.1 (4.2, 6.0)
Hay fever		
Yes	144	32.8 (22.2, 43.5)
No	8113	5.3 (4.5, 6.1)
Biological parent ever told has asthma or hay fever		
Yes	1566	11.0 (9.0, 12.9)
No	6582	4.4 (3.6, 5.6)
Ever breastfed		
Yes	3876	4.8 (3.7, 5.9)
No	4369	7.2 (6.1, 8.2)

old could be prevented by eliminating exposures to indoor pollutants and allergens in housing. The effect of eliminating residential risk factors, if they are indeed causal, would have a profound impact on hospitalization rates, emergency and clinic visits, direct and indirect medical costs, school absences, and health and functioning of children.²⁹

Children with a history of allergic rhinitis were more likely to have doctor-diagnosed asthma. Aller-

gic rhinitis, as measured by a history of hay fever or nasal eosinophilia, has been reported to be an especially potent risk factor for the subsequent development of asthma.^{18,25} It is difficult to disentangle the diagnosis of allergic rhinitis as an indicator of predisposition to asthma or the result of environmental exposure. Similarly, family history of atopy may represent shared genes or common environmental exposures that predispose to asthma.

TABLE 2. Adjusted ORs for Doctor-Diagnosed Asthma in Children Less Than Six Years Old, NHANES III (1988–1994)

Variable	Odds Ratio (95% CI)	P Value
Gender		
Male	1.5 (0.97, 2.2)	.07
Female*	Referent	—
Race		
Black	1.6 (1.1, 2.3)	.02
Other	1.2 (0.6, 2.6)	.64
White*	Referent	—
Poverty status		
<100%	1.5 (0.9, 2.5)	.16
101%–150%	Referent	—
>150%*	0.7 (0.4, 1.2)	.20
Education of head of family		
<High school graduate	0.9 (0.6, 1.4)	.63
High school graduate	0.8 (0.5, 1.2)	.35
>High school graduate*	Referent	—
Received care in neonatal intensive care unit		
Yes	0.7 (0.4, 1.2)	.18
No*	Referent	—
Birth weight		
<2500 g	1.6 (0.8, 3.1)	.21
≥2500*	Referent	—
Smoking		
Prenatal and postnatal	1.7 (1.2, 2.5)	.01
Prenatal only	1.3 (0.6, 3.0)	.47
Postnatal only	0.9 (0.6, 1.3)	.50
None*	Referent	—
Dog in home		
Yes	1.6 (1.1, 2.3)	.02
No*	Referent	—
Cat in home		
Yes	0.9 (0.5, 1.4)	.50
No*	Referent	—
Ever given up pet because of allergies		
Yes	24.7 (8.4, 72.9)	<.001
No*	Referent	—
Use gas stove or oven for heat		
Yes	1.8 (1.02, 3.1)	.04
No*	Referent	—
Urban residence		
Yes	0.7 (0.5, 1.1)	.11
No*	Referent	—
When house built		
>1973	1.0 (0.7, 1.5)	.82
1946–1973	1.5 (0.9, 2.5)	.10
<1946*	Referent	—
Attend day care		
≥10 h/wk	1.6 (1.1, 2.3)	.01
<10 h/wk	1.8 (0.9, 3.7)	.11
None*	Referent	—
Hay fever		
Yes	5.6 (2.4, 13.0)	<.001
No*	Referent	—
Biological parent ever told has asthma or hay fever		
Yes	2.2 (1.5, 3.1)	<.001
No*	Referent	—
Ever breastfed		
Yes	0.8 (0.6, 1.2)	.31
No*	Referent	—

* Reference category; adjusted for gender, race, poverty status, education of head of family, ever breastfed, received care in neonatal intensive care unit, birth weight, cat in home, urban residence, and age house built.

The presence of a dog in the household and having an allergy to a pet were major risk factors for doctor-diagnosed asthma in US children. Pet allergens have been associated with asthma in numerous studies.^{4,10,30,31} In contrast, another report suggested that exposure to pets during infancy was associated with a lower prevalence of asthma at 12 to 13 years of

age.³² Consistent with national surveys conducted in Europe,^{30,31} this present analysis indicates that pets were the predominant risk factor for asthma in US children. These variables may overestimate the risk of asthma from exposure to pets, however, because the temporal relationship of exposure and onset of asthma was unknown. A family may have given

TABLE 3. Population Attributable Fraction for Host Factors and Residential Exposures Associated With Doctor-Diagnosed Asthma in Children Less Than Six Years Old, NHANES III (1988–1994)*

Characteristic	Exposed (%)	OR	95% CI	Attributable Fraction (%)	Excess Cases
Host factors					
Allergic rhinitis in index child	2.2	5.6	2.4, 13.0	9.3	126 000
Parental history	23.8	2.2	1.5, 3.1	21.7	294 000
Environmental factors					
Given up pet because of allergies	1.5	24.7	8.4, 72.9	26.3	358 000
Exposure to ETS	19.0	1.7	1.2, 2.5	12.0	164 000
Dog in the house	20.5	1.6	1.1, 2.3	10.4	142 000
Gas stove or oven for heat	5.7	1.8	1.0, 3.1	4.3	58 000
Day care attendance	29.1	1.6	1.1, 2.3	14.5	198 000
Residential factors†					
≥1 Residential exposure	39.6	2.6	1.8, 3.9	39.2	533 000
Given up a pet because of allergies					
Environmental tobacco smoke					
Dog in household					
Use of gas stove					

* The risk factors are not mutually exclusive, and the estimates of attributable risk are not additive. All odds ratio and attributable risks are adjusted for gender, race, poverty status, education of head of family, ever breastfed, received care in neonatal intensive care unit, birth weight, cat in home, urban residence, and age house built.

† Residential factors did not include day care attendance.

TABLE 4. Asthma Symptoms and Health Services Use Among Children With Asthma by Residential Exposure, NHANES III (1988–1994)*

Variable	Prevalence in Children With Residential Asthma (n = 255)	Prevalence in Children With Nonresidential Asthma (n = 236)	P Value
Wheezing apart from a cold In the past 12 mo . . .	48.3%	40.7%	.26
Wheezing	77.8%	75.5%	.74
Mean number of episodes (SD)	30.3 (±5.9)	47.4 (±11.4)	.18
Hospitalized for wheezing	12.7%	14.5%	.70
Emergency room or clinic visit for wheezing	67.9%	54.5%	.13

* Residential asthma is defined as children with asthma who had 1 or more residential exposures that were associated with asthma.

away a pet, for example, because the child received a diagnosis of asthma unrelated to pet allergy.

The impact of passive smoking on the prevalence and severity of childhood asthma is substantial.^{8,9,11–14} Exposure to ETS has been associated with bronchial hyperreactivity; the mean number of exacerbations of asthma increase and pulmonary function tests worsen as cotinine levels increased.^{5,9,11,14} It has been estimated that between 1988 and 1994, approximately 38% of US children <6 years old were exposed to ETS.⁹

Childhood asthma was also found to be associated with use of a gas stove or oven for heating. Most combustion heating appliances are vented to the outside of housing to facilitate removal of the products of combustion, including nitrogen dioxide, carbon monoxide, and carbon dioxide.²² Cooking devices that are used improperly for heating often are not vented and may therefore be associated with high levels of nitrogen dioxide and other emissions.³³ Gas stoves or ovens are most often used for heat in the southeastern United States, especially in poor and rural housing.³³

Targeting asthma prevention efforts is difficult. Consistent with other studies, we found that a family history of hay fever or asthma was a major risk factor

for doctor-diagnosed asthma^{14,18,19}; yet, although 44% of children with asthma in this analysis had a parental history of atopy, >50% did not have such a history. Moreover, only 11% of children with a family history of atopy had doctor-diagnosed asthma. Thus, if interventions are targeted only to families with a history of atopy, then 60% of children who will receive a diagnosis of asthma in the first 6 years of life would be neglected and only 11% of children who are targeted potentially would benefit. Although exposures to ETS and sensitization to indoor allergens are considered to be causally associated with asthma development or exacerbations,³⁴ this has not been tested in experimental trials. Thus, additional research is necessary to prove that the elimination of exposures will result in lower rates of asthma. Finally, it is relatively easy to attribute a large fraction of doctor-diagnosed asthma to housing factors, but it will be considerably more difficult to eliminate such exposures on a national level.

Certain limitations of these analyses should be acknowledged. Asthma was based on parental report. Cotinine and pulmonary function tests were not available for this age group, and a maternal postnatal smoking variable was not present. Instead, the independent association of the combined vari-

able for prenatal and postnatal tobacco exposure but not postnatal exposure alone most likely served as a proxy for maternal smoking. Because we were not able to measure exposure or sensitization to various allergens (eg, dust mite, fungi, cockroach), we may have underestimated the contribution of residential exposures to doctor-diagnosed asthma. We also did not have a measure of viral infections that have been associated with wheezing, such as respiratory syncytial virus, with the exception of day care attendance as a risk factor for viral infections. Finally, if children who are genetically predisposed develop asthma only if they are exposed to allergens, then our estimates of the PAR of residential exposures are low.

Similar to other studies, this analysis was limited by the lack of a universally accepted definition for asthma.³⁵ Some studies combined doctor-diagnosed asthma and wheezing as a measure for asthma, whereas we included only children with doctor-diagnosed asthma to enhance diagnostic specificity. Although 60% to 90% of individuals who eventually develop asthma received that diagnosis by 6 years of age,^{36,37} some children who receive a diagnosis of asthma in the first 3 years of life will not develop persistent asthma.¹⁸ Thus, these findings may not be relevant for older children who spend less time in their primary residence and for whom allergen-induced asthma is increasingly important. Still, wheezing in the first 6 years of life is associated with considerable morbidity and mortality.

Taken together, these and other data demonstrate clearly that children's health is inextricably linked with housing. More than 60% of fatal injuries among children <15 years old occurred in the home.³⁸ Exposure to ETS competes with injuries as the leading cause of disease and disability in children,³⁹ and subclinical lead toxicity, affecting 1 in every 20 US children, is primarily attributable to residential lead hazards.^{40,41} Unfortunately, despite growing evidence that residential exposures have a dramatic impact on children's health, housing is largely ignored as a public health problem.

CONCLUSION

We identified a number of residential exposures that were associated with doctor-diagnosed asthma in US children who were surveyed from 1988 to 1994. Parents need to consider carefully the risks and benefits of owning a pet, particularly during early childhood and especially if there is a maternal history of an allergic condition. Exposure to ETS increases the risk of asthma for young children, regardless of the presence of a coexisting allergic disorder or pulmonary disease. The results of these analyses indicate that the elimination of residential exposures, if causally related with asthma, could have a profound effect on medical costs in the United States and, more importantly, on the health of children.

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REFERENCES

- Centers for Disease Control and Prevention. Asthma mortality and hospitalization among children and young adults, 1980–1993. *MMWR Morb Mortal Wkly Rep.* 1996;45:350–353
- Weiss KB, Gergen PJ, Wagener DK. Breathing better or wheezing worse? The changing epidemiology of asthma morbidity and mortality. *Annu Rev Public Health.* 1993;14:491–513
- Weitzman M, Gortmaker SL, Sobol AM, Perrin JM. Recent trends in the prevalence and severity of childhood asthma. *JAMA.* 1992;268:2673–2677
- Pope AMR, Patterson R, Burge H. *Indoor Allergens: Assessing and Controlling Adverse Health Effects.* Washington, DC: National Academy Press; 1993
- Chilmonczyk BA, Salmun LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med.* 1993;328:1665–1669
- Duff AL, Pomeranz ES, Gelber LE. Risk factors for acute wheezing in infants and children: viruses, passive smoke, and IgE antibodies to inhalant allergens. *Pediatrics.* 1993;92:535–540
- Gelber L, Seltzer LH, Bouzoukis JK, Pollart SM, Chapman MD, Platts-Mills TA. Sensitization and exposure to indoor allergens as risk factors for asthma among patients presenting to hospital. *Am Rev Respir Dis.* 1993;147:573–578
- Stoddard JJ, Miller T. Impact of parental smoking on the prevalence of wheezing respiratory illness in children. *Am J Epidemiol.* 1995;141:96–102
- Gergen PJ, Fowler JA, Maurer KR, Davis WW, Overpeck MD. The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988 to 1994. *Pediatrics.* 1998;101(2). URL: <http://www.pediatrics.org/cgi/content/full/101/2/e8>
- Ingram J, Sporik R, Rose G, Honsinger R, Chapman M, Platts-Mills TAE. Quantitative assessment of exposure to dog (*Can f 1*) and cat (*Fel d 1*) allergens: relationship to sensitization and asthma among children living in Los Alamos, New Mexico. *J Allergy Clin Immunol.* 1995;96:449–456
- Martinez FD, Antognoni G, Macri F, et al. Parental smoking enhances bronchial responsiveness in nine-year-old children. *Am Rev Respir Dis.* 1988;138:518–523
- Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. *Pediatrics.* 1992;89:21–26
- Weitzman M, Gortmaker S, Walker DK, Sobol A. Maternal smoking and childhood asthma. *Pediatrics.* 1990;85:505–511
- Young S, LeSouef PN, Geelhoed GC, Stick SM, Turner KJ, Landau LI. The influence of a family history of asthma and parental smoking on airway responsiveness in early infancy. *N Engl J Med.* 1991;324:168–173
- Sporik R, Holgate ST, Platt-Mills TAE. Exposure to house-dust mite allergen (*Der p 1*) and the development of asthma in childhood. *N Engl J Med.* 1990;323:502–507
- Platts-Mills TAE, Sporik RB, Wheatley LM, Heymann PW. Is there a dose-response relationship between exposure to indoor allergens and symptoms of asthma? *J Allergy Clin Immunol.* 1995;96:435–440
- Rosenstreich DL, Eggleston P, Kattan M, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med.* 1997;336:1356–1363
- Martinez FD, Wright AL, Taussig LM, et al. Asthma and wheezing in the first six years of life. *N Engl J Med.* 1995;332:133–138
- Litonjua AA, Carey VJ, Burge H, Weiss ST, Gold DR. Parental history and the risk for childhood asthma. *Am J Respir Crit Care Med.* 1998;158:176–181
- Weitzman M, Gortmaker S, Sobol A. Racial, social, and environmental risks for childhood asthma. *AJDC.* 1990;144:1189–1194
- Infante-Rivard C. Childhood asthma and indoor environmental risk factors. *Am J Epidemiol.* 1993;137:834–844
- Garrett MH, Hooper MA, Hooper BM, Abramson MJ. Respiratory symptoms in children and indoor exposures to nitrogen dioxide and gas stoves. *Am J Respir Crit Care Med.* 1998;158:891–895
- Weiss KB, Gergen PJ, Crain EF. Inner-city asthma: the epidemiology of

- an emerging US public health concern. *Chest*. 1992;101:362S–367S
24. Halfon N, Newacheck PW. Childhood asthma and poverty: differential impacts and utilization of health services. *Pediatrics*. 1993;91:56–61
 25. Zeiger RS, Heller S. The development and prediction of atopy in high-risk children: follow-up at seven years in a prospective randomized study of combined maternal and infant food allergen avoidance. *J Allergy Clin Immunol*. 1995;95:1179–1190
 26. Coughlin S, Benichou J, Weed D. Attributable risk estimation in case-control studies. *Epidemiol Rev*. 1994;16:51–64
 27. Smith DH, Malone DC, Lawson KA, Okamoto LJ, Battista C, Saunders WB. A national estimate of the economic costs of asthma. *Am J Respir Crit Care Med*. 1997;156:787–793
 28. *Statistical Abstract of the United States*. 199th ed. Washington, DC: US Department of Commerce, Bureau of the Census; 1999
 29. Taylor WR, Newacheck PW. Impact of childhood asthma on health. *Pediatrics*. 1992;90:657–662
 30. Burr ML, Anderson HR, Austin JB, et al. Respiratory symptoms and home environment in children: a national survey. *Thorax*. 1999;54:27–32
 31. Plaschke P, Janson C, Norman E, Bjornsson E, Ellbjar S, Jarvholm B. Association between atopic sensitization and asthma and bronchial hyperresponsiveness in Swedish adults: pets, and not mites, are the most important allergens. *J Allergy Clin Immunol*. 1999;104:58–65
 32. Hesselmar B, Aberg N, Aberg B, Eriksson B, Bjorksten B. Does early exposure to cat or dog protect against later allergy development? *Clin Exp Allergy*. 1999;29:611–617
 33. Centers for Disease Control and Prevention. Use of unvented residential heating appliances—United States, 1988–1994. *MMWR Morb Mortal Wkly Rep*. 1997;46:1221–1224
 34. *Clearing the Air: Asthma and Indoor Air Exposures*. Washington, DC: Committee on the Assessment of Asthma and Indoor Air, Institute of Medicine, National Academy Press; 2000
 35. Samet JM. Epidemiologic approaches for the identification of asthma. *Chest*. 1987;91:74S–78S
 36. Croner S, Kjellman NIM. Natural history of bronchial asthma in childhood. *Allergy*. 1992;47:150–157
 37. Yuninger JW, Reed CE, O'Connell EJ, Melton J, O'Fallon WM, Silverstein MD. A community based study of the epidemiology of asthma. *Am Rev Respir Dis*. 1992;146:888–894
 38. Pollock DA, McGee DL. Deaths due to injury in the home among persons under 15 years of age, 1970–1984. *MMWR Morb Mortal Wkly Rep*. 1988;37:13–20
 39. Aligne CA, Stoddard J. Tobacco and children. An economic evaluation of the medical effects of parental smoking. *Arch Pediatr Adolesc Med*. 1997;151:648–653
 40. Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the US population to lead, 1991–1994. *Environ Health Perspect*. 1998;11:745–750
 41. Lanphear BP. The paradox of lead poisoning prevention. *Science*. 1998; 281:1617–1618

KIDS ARE NOT PROTECTED

The Food and Drug Administration (FDA) has the budget and the authority to test products before they are put on the market, and the National Highway Traffic Safety Administration has extensive authority to set mandatory safety standards for motor vehicles, but the Consumer Product Safety Commission, which oversees children's products, can do little to prevent inadequately tested products from being sold . . . millions of products that are unsafe can be sold, and scores of kids killed before the product is withdrawn for sale.

Felcher EM. Children's products and risk. *Atlantic Monthly*. November 2000

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