Plastic Wall Materials in the Home and Respiratory Health in Young Children

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A B S T R A C T

Objectives. The relation between the presence of plastic wall materials in the home and respiratory health in children was assessed.

Methods. This population-based cross-sectional study involved 2568 Finnish children aged 1 to 7 years.

Results. In logistic regression models, lower respiratory tract symptoms—persistent wheezing (adjusted odds ratio [OR]=3.42, 95% confidence interval [CI]=1.13, 10.36), cough (OR=2.41, 95% CI=1. 04, 5.63), and phlegm (OR=2.76, 95% CI=1.03, 7.41)—were strongly related to the presence of plastic wall materials, whereas upper respiratory symptoms were not. The risk of asthma (OR=1.52, 95% CI=0.35, 6.71) and pneumonia (OR=1.81, 95% CI=0.62, 5.29) was also increased in children exposed to such materials.

Conclusions. Emissions from plastic materials indoors may have adverse effects on the lower respiratory tracts of small children. (*Am J Public Health*. 2000;90:797–799)

Plastic materials provide inexpensive, easy-to-clean surfaces, and they are increasingly used to cover walls and floors of kitchens, bathrooms, and children's playrooms and bedrooms. However, these materials emit into the indoor air many chemicals, such as plasticizers used in the synthesis of polyvinyl chloride.[1] These chemicals may cause airway inflammation and thus increase the risk of bronchial obstruction, asthma, and perhaps susceptibility to respiratory infections.[2]

A recent case–control study of 251 case patients and one-to-one matched controls in Oslo, Norway, indicated that the presence of polyvinyl chloride and other plasticizer-containing surface materials in the home increases the risk of bronchial obstruction during the first 2 years of life.[3] In another population of children, we elaborated further on the relation between the presence of plastic materials in the home and the risk of asthma and asthmalike symptoms, using information from the Children's Environment and Health Study carried out in 1991 in Espoo, Finland.[4,5] We also extended the hypothesis to include other plausible effects in the airways such as symptoms of the upper and lower respiratory tracts, allergic rhinitis, asthma, and respiratory infections.

Methods

Study Population and Data Collection

The source population for the Children's Environment and Health Study comprised all children of the city of Espoo born between January 1, 1984, and December 31, 1989. Espoo is an urban–suburban municipality located near Helsinki. In March 1991, we distributed a questionnaire to parents of a random sample of the source population.[4,5] The study population included 2568 children whose parents or other guardians completed a questionnaire (response rate: 80.3%).

The questionnaire inquired about the child's personal characteristics; the child's respiratory health and atopic diseases, as well as the number of infections during the previous year; parents' education and job category, as indicators of socioeconomic status, along with their smoking habits and history of respiratory and allergic diseases; duration of breast-feeding; type of day care; and details on home environment and building characteristics. The questions on respiratory health were derived partly from the 1978 American Thoracic Society questionnaire for children translated to Finnish and Swedish, the 2 official languages of Finland. The questions were modified to correspond to everyday use of these languages.

Methods

The exposure variable of interest was the presence of any plastic wall materials in the child's home. Such exposure was based on responses to a question assessing the proportion of plastic wall surfaces in the home; the question had 3 response alternatives: (1) no; (2) yes, less than 50%; and (3) yes, at least 50%. This information was missing for 15 children, so analyses were carried out with 2553 children. In the final analyses, we used a dichotomous exposure variable (yes/no) because the number of children in the highest exposure category was small (n=19).

We used the following health outcomes (and definitions)[4,5]: current asthma (doctor-diagnosed asthma with symptoms and/or medication during the previous 12 months), allergic rhinitis (doctor-diagnosed asthma with nasal symptoms during the previous 12 months), persistent wheezing (wheezing apart from colds or wheezing most days or nights during the previous year), persistent cough (a cough apart from colds for 3 months of the previous year or more), persistent phlegm (phlegm production or dyspnea due to stuffiness in breathing apart from colds for 3 months of the previous year or more), weekly nasal congestion/excretion (nasal congestion/excretion apart from colds for 1 to 3 days per week or more), and respiratory infections (at least 1 episode of corresponding infection during the previous 12 months).

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The odds ratio (OR) was used as a measure of effect. Logistic regression models with exposure and potential confounders as covariates provided adjusted odds ratios and their corresponding 95% confidence intervals (CIs). The following potential confounders were included in the analyses as covariates: sex, age, parental education, single guardian, day-care center attendance, presence of furry or feathery pets, and exposures to environmental tobacco smoke and any mold and dampness problems (defined as occurrence of mold odor, visible mold, moisture, or water damage during the previous year or earlier).

Results

A total of 49 children (1.9%) had asthma, and 92 (3.6%) had allergic rhinitis (Table 1). The prevalence of respiratory symptoms varied from 1.7% to 5.4%. The prevalence of asthma and wheezing was relatively low in comparison with figures reported recently in Europe. However, another Finnish study[6] of children aged 7 to 12 years reported prevalences of 4.4% for doctor-diagnosed asthma and 5.4% for wheezing during the previous 12 months, figures that correspond to those of the present study when the slightly higher age range is taken into account.

Plastic wall materials were reported in the homes of 72 children (2.8%). Table 1 shows crude and adjusted estimates of odds ratios due to exposure. Risk of asthma was associated with exposure, although the 95% confidence interval for the adjusted odds ratio included unity (OR = 1.52, 95% CI = 0.35, 6.71). Lower respiratory tract symptoms—persistent wheezing (OR = 3.42, 95% CI = 1.13, 10.36), cough (OR = 2.41, 95% CI = 1.04, 5.63), and phlegm (OR = 2.76, 95% CI = 1.03, 7.41)—were associated with the presence of plastic wall materials, whereas upper respiratory symptoms were not (Table 1). The relative risk estimates for pneumonia, bronchitis, and otitis media were slightly increased.

Discussion

In agreement with our hypothesis, risks of chronic respiratory symptoms typical of asthma were associated with the presence of plastic wall materials. The risks of asthma and pneumonia were also higher in children exposed to plastic materials than in unexposed children, although the 95% confidence intervals for the risk estimates were wide and included unity.

The present study was designed to evaluate the role of home and day-care environments as determinants of children's respiratory health. We collected information on a number of potential sources of chemical and microbiological air pollutants in order to assess indirectly exposures to indoor air pollution. Emissions from plastic materials were one of the central issues. Given the vast number of chemicals present in plastics and other building materials, it is not feasible to measure all of the relevant compounds in indoor air.

We asked respondents about the presence of plastic wall materials in their current residence and used this source information as a measure of exposure. The information on exposure probably included some misclassification; because there was no public awareness of hazards of plastic materials, however, it is likely that this misclassification was nondifferential.

Renovations in the current residence or occupancy in other residences could also have introduced error into assessments of previous exposure; this issue was most relevant in assessments of the risk of asthma and allergic rhinitis. Avoidance of plastic materials among parents of asthmatic or allergic children would have led to underestimation of the relation between exposure and outcomes.

Information on respiratory symptoms and other outcomes was also based on parental reports. Any error in outcome assessments was probably nondifferential, again because of the lack of public awareness of the studied relation. Given the observed association between exposure and lower respiratory tract symptoms, the lack of association between exposure and upper respiratory symptoms speaks further against systematic misclassification of both exposure and outcomes, because similar mechanisms should have produced increased risk estimates for upper respiratory symptoms as well.

We were able to adjust for confounding by most of the known determinants of the outcomes. The estimated odds ratios for wheezing, cough, and phlegm were not sensitive to different combinations of confounders. Therefore, we would not expect residual confounding by the covariates to be large.

Our results are in line with the findings of the Norwegian case–control study mentioned earlier.[3] In that study, the risk of bronchial obstruction during the first 2 years of life increased in relation to the amount of plasticizer-emitting materials in the home.[3] This relation was substantially stronger (OR = 12.3, 95% CI = 1.00, 1.59) when the ventilation rate in the home was low than when it was high (OR = 2.6, 95% CI = 1.02, 6.58).[7] This finding was consistent with the idea that indoor concentrations of chemical emissions from surface materials would be reduced with a higher ventilation rate.

Our results provide additional evidence that indoor plastic materials may emit chemicals that have adverse effects on the lower respiratory tracts of small children. The qual-

TABLE 1—Occurrence of Asthma, Allergic Rhinitis, Respiratory Symptoms, and Respiratory Infections in Relation to the Presence of Plastic Wall Materials: Preschool Children (n = 2553), Espoo, Finland, 1991

Outcome[a]	Exposed (n = 72) %	Reference (n = 2481) %	Crude Odds Ratio	Adjusted Odds Ratio[b]	95% Confidence Interval
Asthma	2.8	1.9	1.49	1.52	0.35, 6.71
Allergic rhinitis	4.2	3.6	1.17	1.20	0.36, 3.97
Symptoms					
Persistent wheezing	5.9	1.6	3.86	3.42	1.13, 10.36
Persistent cough	9.7	3.7	2.80	2.41	1.04, 5.63
Persistent phlegm	6.9	2.3	3.15	2.76	1.03, 7.41
Weekly nasal congestion	5.6	5.4	1.04	0.95	0.33, 2.71
Weekly nasal excretion	5.6	5.2	1.08	0.90	0.32, 2.57
Infections[c]					
Pneumonia	5.6	2.9	1.99	1.81	0.62, 5.29
Bronchitis	20.8	16.2	1.36	1.34	0.74, 2.43
Otitis media	51.4	42.6	1.43	1.38	0.85, 2.24
Tonsillitis	8.3	8.0	1.04	1.04	0.44, 2.47

[a]The time period of interest is the previous 12 months.
[b]Odds ratios from logistic regression analysis adjusting for sex, age, highest parental education, single guardian, day-care center attendance, presence of furry or feathery pets, and exposures to environmental tobacco smoke and dampness problems.
[c]At least 1 infection during the previous 12 months.

ity of plastic materials used in homes varies, and therefore it is reasonable to expect as well that emissions of chemical compounds vary qualitatively and quantitatively. Our study was population based and therefore comprised a representative cross section of the types of materials on the market. In spite of the limitations of the study, the results warrant further attention to the types of plastic materials used in interior decoration. □

Contributors

J. Jaakkola initiated and designed the study, supervised data collection and analysis, and wrote the paper. P. Verkasalo performed the data analyses and contributed to interpreting the results and writing the paper. N. Jaakkola participated in planning the study, collecting the data, interpreting the results, and writing the paper.

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References

1. Saarela K, Kaustia K, Kiviranta A. Emissions from materials: the role of additives in PVC. In: Bieva CJ, Courtois Y, Covaerts M, eds. *Present and Future of Indoor Air Quality.* Amsterdam, the Netherlands: Elsevier Science Publishers; 1989:329–336.
2. Øie L, Hersoug L-G, Madsen JØ. Residential exposure to plasticizers and its possible role in the pathogenesis of asthma. *Environ Health Perspect.* 1997;105:972–978.
3. Jaakkola JJK, Øie L, Nafstad P, Botten G, Samuelsen SO, Magnus P. Interior surface materials in the home and the development of bronchial obstruction in young children in Oslo, Norway. *Am J Public Health.* 1999;89:188–192.
4. Jaakkola JJK, Jaakkola N, Ruotsalainen R. Home dampness and molds as determinants of respiratory symptoms and asthma in pre-school children. *J Exp Anal Environ Epidemiol.* 1993;3(suppl 1):129–142.
5. Louhiala PJ, Jaakkola N, Ruotsalainen R, Jaakkola JJK. Form of day care and respiratory infections among Finnish children. *Am J Public Health.* 1995;85:1109–1112.
6. Timonen KL, Pekkanen J, Korppi M, Vaheristo M, Salonen RO. Prevalence and characteristics of children with chronic symptoms in eastern Finland. *Eur Respir J.* 1995;8:1155–1160.
7. Øie L, Nafstad P, Botten G, Magnus P, Jaakkola JJK. Ventilation in homes and bronchial obstruction in young children. *Epidemiology.* 1999;10:294–299.