# Interior Surface Materials in the Home and the Development of Bronchial Obstruction in Young Children in Oslo, Norway

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## ABSTRACT

*Objectives.* This study assessed the role of polyvinyl chloride (PVC) plastics and textile materials in the home in the development of bronchial obstruction during the first 2 years of life.

*Methods.* The study was a matched pair case–control study based on a cohort of 3754 newborns in Oslo in 1992 and 1993 who were followed up for 2 years. The case group consisted of 251 children with bronchial obstruction; the control group was matched one-to-one for date of birth.

*Results.* In conditional logistic regression analysis, the risk of bronchial obstruction was related to the presence of PVC flooring (adjusted odds ratio [OR] = 1.89; 95% confidence interval [CI] = 1.14, 3.14) and textile wall materials (adjusted OR = 1.58; 95% CI = 0.98, 2.54). The reference category was wood or parquet flooring and painted walls and ceiling. Further analysis revealed an exposure–response relationship between the assessed amount of PVC and other plasticizer-containing surface materials and the risk of bronchial obstruction.

*Conclusions.* This study provides new evidence of the role of PVC and textile wall materials in the development of bronchial obstruction in young children. (*Am J Public Health.* 1999; 89:188–192)

The home environment has changed considerably during the past 3 decades because of rapid change in building technology, as well as in the type of materials used in interior decoration, furniture, and textiles. Some of the new surface materials are potential emitters of chemical compounds and particulates with allergenic properties. Little is known about whether exposure to these indoor air pollutants in the home environment contributes to bronchial obstruction and asthma.

Inflammation of the airways is an important part of the mechanism in asthma and bronchial reactivity.[1] Most allergens stimulate production of IgE antibodies that bind to mast cells, and with linkage of antigens the mast cells release inflammatory mediators causing bronchospasm and mucus production. There also appear to be chemical compounds with a capacity to trigger inflammation without involving IgE.[2] Long-term occupational exposure to relatively high levels of chemicals such as formaldehyde,[3] toluene di-isocyanate,[4] and acid anhydrides[5] is known to increase the risk of asthma. Exposure to some of these chemical compounds, such as formaldehyde, may also lead to development of specific airway hypersensitivity.[3]

Plasticized polyvinyl chloride (PVC) materials are presently among the most frequently used wall and floor covering materials in homes because they provide inexpensive, easy-to-clean surfaces that are especially practical in kitchens, bathrooms, and children's playrooms and bedrooms.[6] PVC materials are potential emission sources of chemicals used as plasticizers, viscosity modifiers, and stabilizers, and these emissions are usually long-lasting.

Textile surface materials may emit chemical compounds and may serve as sources of particulate pollution. Studies of office workers have shown that mucosal and allergic symptoms are more common in offices with large amounts of textile surface materials.[7,8]

We assessed the role of interior surface materials in the home in the development of bronchial obstruction during the first 2 years of life. In particular, we hypothesized a priori that PVC plastics and textile materials were determinants of bronchial obstruction.

## Methods

### *Study Design*

The study was a matched pair case–control study based on the Oslo Birth Cohort, a cohort of 3754 children born in Oslo during a period of 15 months in 1992 and 1993. Details of the recruitment and follow-up of the cohort have been presented elsewhere.[9] Information on the child's health and environmental exposures was collected at birth and when the child was aged 6 months (follow-up rate 95%), 12 months (92%), 18 months (92%), and 24 months (81%).

This study was approved by the Regional Ethical Committee and permission for data registration was given by the Norwegian Data Inspectorate.

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*Selection of Subjects*

The outcome of interest was bronchial obstruction, defined as 2 or more episodes with symptoms and signs of bronchial obstruction or 1 episode lasting more than 1 month.[10,11] All of the children from the cohort who had had bronchial obstruction were recruited for the case group. We obtained information on episodes of bronchial obstruction from the project pediatrician, family physician records, follow-up questionnaires, and medical records. The final decision on which children were to be included in the case group was made by a committee of 3 senior pediatricians, as described in more detail elsewhere.[10,11]

As soon as a case subject was identified, the child born closest in time to that child was selected as the age-matched control, providing he or she had no medical history suggestive of bronchial obstruction at the time of selection. Only children still living in Oslo and with the same home address during the last 3 months before becoming candidates for the study were included.

*Assessment of Surface Materials*

For the assessment of interior surface materials, the residence was divided into 2 areas: the child's bedroom and the remainder of the residence. The interior surfaces were categorized as floors, walls, ceiling, furniture, and the child's bed. The materials commonly used in the interior surfaces were identified and classified into 13 categories. These categories were given material-specific values from 1 to 8 based on their expected emission of plasticizers: metal, tile, wood or parquet, wallpaper, and wall fiberglass were given a value of 1; cork, paint, lacquer, and textile wallpaper, 2; rubber, 3; carpet and linoleum, 4; plastic laminate, 6; and PVC, 8.[12,13] Each surface category was given a weight reflecting the child's potential exposure. The weights for the child's bedroom and the remaining rooms, respectively, were as follows: floors, 0.20 and 0.40; walls, 0.10 and 0.20; ceilings, 0.05 and 0.10; furniture, 0.15 and 0.30; child's bed, 0.50 and 0.0. These weights and material-specific values were used to calculate a plasticizer exposure index.

*Data Collection*

Two trained persons assessed exposures and interviewed parents during site inspections. The home visits were performed in the same 2-week period for the matched case–control pairs. The families were urged not to perform any interventions before the visit, which usually was conducted within 1 week after the first contact.

Building characteristics were evaluated in detail: interior surface materials, type of building, type of ventilation system, presence or absence of a kitchen range hood, crowdedness (square meters per occupant), bedroom arrangements, and home dampness problems. The air change rates ($h^{-1}$) were measured in a subgroup of 172 pairs with a passive (perfluorocarbon) tracer gas method,[14] over a 14-day integrated sampling period.

*Statistical Methods*

We used the odds ratio (OR) as a measure of association between outcome and exposure. We calculated crude odds ratios as ratios between the numbers of discordant pairs, and we calculated 95% confidence intervals (CIs) on the basis of the Mantel-Haenszel test statistics. We estimated the adjusted odds ratios in conditional logistic regression analysis.[15]

First, in bivariate analyses using dichotomous variables, we assessed the role of type of floor, wall, and ceiling materials. We fitted all these exposure variables in the model with other covariates. Information on covariates was taken from the follow-up questionnaires and home inspections. Sex, parental history of atopy, breast-feeding, having siblings, day-care attendance at the age of 1 year, exposure to environmental tobacco smoke, and the presence of dampness in the home were considered core covariates. Information on one (usually) or more covariates was missing for one subject in 42 case–control pairs. In this situation the information was assumed to be the same for both subjects and thus all pairs were retained in the analyses to maximize precision. Models that excluded the pairs with missing information produced almost identical point estimates.

Second, we assessed the role of plastic materials, fitting the plasticizer exposure index both as a categorical and as a numerical variable. The exposure categories were the 2 highest quartiles of the exposure indices (50th–75th and 75th–100th percentiles); the 0 through 50th percentile was the reference category. The core covariates were included in all the models. Other variables, such as birthweight, maternal age, maternal education, annual family income, pets, type of building, type of ventilation system, presence of kitchen range hood, crowdedness, and bedroom arrangement, were included in the model only if they changed the point estimate or improved the precision of the studied relationship.[16] The categorization of building variables is presented in Table 1; that of other variables is presented elsewhere.[11] Children living in homes in which one or more inhabitants smoked (as reported in the birth questionnaire) were considered to be exposed to environmental tobacco smoke. Children with at least one parent reporting a history of asthma or hay fever were considered as having atopic parents.

## Results

A total of 304 children from the Oslo Birth Cohort fulfilled the case definition. Of these, 256 were still living in Oslo at the time of diagnosis and were therefore candidates for the case–control study. A total of 5 pairs were lost to follow-up: the parents of 3 case subjects were unwilling to have a home visit, and the homes of 2 pairs were not visited by mistake.

The case group had a higher proportion of boys, atopic parents, children with siblings, and exposure to environmental tobacco smoke and a lower proportion of breast-feeding after the age of 6 months than did the control group (see Table 2 in Nafstad et al.[11]). There were few differences between the case and control groups in type of building and ventilation, presence or absence of a kitchen range hood, crowdedness, and child's bedroom arrangement (Table 1).

*Surface Materials and Bronchial Obstruction*

Both in the crude analyses and in the conditional logistic regression adjusting for other materials and potential confounders, the risk of bronchial obstruction was greater in the presence of PVC in the floors (adjusted OR = 1.89; 95% CI = 1.14, 3.14) and of textile wallpaper in one or more rooms (adjusted OR = 1.58; 95% CI = 0.98, 2.54), compared with the reference category of wood or parquet flooring and painted walls and ceiling (Table 2).

*Plasticizers and Bronchial Obstruction*

Figure 1 shows the cumulative frequency distribution of the plasticizer exposure index for case and control subjects. The curve deviates for index values above 2.5. The mean exposure index was significantly higher in case subjects (3.09; SD = 0.88) than in control subjects (2.87; SD = 0.73) (paired $t$ test $P = .001$).

In the conditional logistic regression analysis controlling for potential confounders (Table 3), bronchial obstruction was significantly associated with assessed exposure to plasticizers. The estimated odds ratio in the

contrast between the highest quartile of exposure and the reference category was 2.71 (95% CI = 1.50, 4.91) when adjustments were made for the core covariates, maternal education, and family annual income. The inclusion of other covariates did not influence the point estimate or improve precision; however, in a subgroup of 172 pairs, air change rate was also included as a covariate. The resulting estimated odds ratios were 1.08 (95% CI = 0.55, 2.12) for the 50th–75th percentile and 3.29 (95% CI = 1.59, 6.81) for the 75th–100th percentile in comparison to the reference category (0–50th percentile).

Fitting of the continuous exposure variable indicated a significant exposure–response pattern, with an increase of 1.65 (95% CI = 1.20, 2.27) in the odds ratio per unit increase in the plasticizer exposure index (range, 1 to 8). This estimate was from a model that included the core covariates, maternal education, and family annual income, and it remained relatively stable when other covariates were included. In the subgroup of 172 pairs, air change rate was also included, resulting in a slightly higher odds ratio of 1.84 (95% CI = 1.24, 2.74).

**TABLE 1—Distribution of Building Characteristics Among Case and Control Subjects in a Study of Bronchial Obstruction in Young Children: Oslo, Norway, 1992–1995**

| | Case Subjects (n = 251) | Control Subjects (n = 251) |
|---|---|---|
| | % (95% CI) | % (95% CI) |
| Type of building | | |
| Single-family house | 19 (14, 24) | 25 (20, 30) |
| Detached or semidetached house | 28 (23, 34) | 23 (18, 28) |
| Apartment building | 52 (46, 58) | 52 (46, 58) |
| Type of ventilation | | |
| Natural | 68 (62, 74) | 66 (60, 72) |
| Mechanical exhaust | 32 (26, 38) | 32 (26, 38) |
| Balanced | 0 (0, 1) | 2 (1, 4) |
| Kitchen range hood (exhaust) | 53 (47, 59) | 55 (49, 61) |
| Interior surface material in child's bedroom, living room, and bathroom | | |
| Floor (in 1 or more of 4 rooms) | | |
| Wood/parquet | 69 (63, 75) | 76 (70, 81) |
| PVC* | 72 (66, 77) | 59 (53, 65) |
| Tile | 61 (55, 76) | 69 (63, 75) |
| Carpet | 39 (33, 45) | 46 (40, 52) |
| Linoleum | 7 (4, 10) | 8 (5, 12) |
| Wall (in 1 or more of 4 rooms) | | |
| Paint | 92 (89, 95) | 95 (92, 98) |
| Tile | 48 (42, 54) | 45 (38, 51) |
| PVC-coated wallpaper | 32 (26, 38) | 34 (28, 40) |
| Textile wallpaper* | 31 (25, 36) | 22 (17, 27) |
| Wallpaper | 23 (18, 28) | 24 (19, 30) |
| Wood panel | 13 (9, 17) | 11 (7, 15) |
| Ceiling | | |
| Paint | 90 (87, 94) | 93 (90, 96) |
| Wood panel | 17 (12, 21) | 22 (16, 27) |
| Crowdedness (<20 $m^2$ per occupant) | 22 (17, 27) | 22 (17, 27) |
| Separate bedroom for child | 55 (48, 61) | 58 (52, 64) |
| Home dampness problems (verified)* | 27 (22, 33) | 14 (9, 18) |

*Note.* CI = confidence interval; PVC = polyvinyl chloride.
*$P < .05$ ($\chi^2$ test).

**TABLE 2—Crude and Adjusted Odds Ratios (ORs) for Bronchial Obstruction During the First 2 Years of Life According to Interior Surface Materials in the Home (Conditional Logistic Regression of 251 Pairs): Oslo, Norway, 1992–1995**

| | Crude OR (95% CI) | Adjusted OR[a] (95% CI) |
|---|---|---|
| Flooring material | | |
| PVC in 1 or more rooms | 1.73 (1.19, 2.50) | 1.89 (1.14, 3.14) |
| Carpet in 1 or more rooms | 0.76 (0.54, 1.07) | 0.74 (0.48, 1.13) |
| Wall coating material | | |
| PVC-coated wallpaper in 1 or more rooms | 0.92 (0.63, 1.36) | 0.72 (0.42, 1.22) |
| Textile wallpaper in 1 or more rooms | 1.53 (1.02, 2.27) | 1.58 (0.98, 2.54) |

*Note.* CI = confidence interval; PVC = polyvinyl chloride.
[a]Adjusted for other interior surface material variables (tile and linoleum on floor; tile, wallpaper, and wood panel on wall; wood panel on ceiling), the core covariates (sex, parental atopy, having siblings, day-care attendance, breast-feeding, exposure to environmental tobacco smoke, and dampness problems), maternal education, and family annual income. The reference category is wood or parquet floors and painted walls and ceilings.

## *Discussion*

In our case–control study, children with bronchial obstruction were more likely to have PVC flooring and textile wall materials in their homes than were children without bronchial obstruction. With adjustment for confounding, the estimated odds ratio for bronchial obstruction was 1.89 (95% CI = 1.14, 3.14) among children with PVC flooring and 1.58 (95% CI = 0.98, 2.54) among children with textile wall materials, in contrast to the reference category of wood or parquet floor and painted walls and ceilings. We further evaluated the plasticizers used in PVC materials as a potential cause of bronchial obstruction and found that the risk of bronchial obstruction increased in relation to the amount of plasticizer-emitting materials in the home.

### *Validity of Results*

Given the vast number of different chemicals commonly present in building materials, it would be infeasible to measure the concentrations of all the separate components. Therefore, we used the type of materials in different interior surfaces as a proxy for exposure to chemicals and particles.

Many previous studies have used wheezing as an outcome when studying causes of lower airway diseases. However, reported wheezing is probably not an optimal measure of bronchial obstruction. We attempted to use a more accurate measure by restricting the case definition to recurrent or prolonged episodes of bronchial obstruction, using a specialist committee's judgment based on data from clinical examinations and available health records. We also made an effort to detect all the cases by reminding the

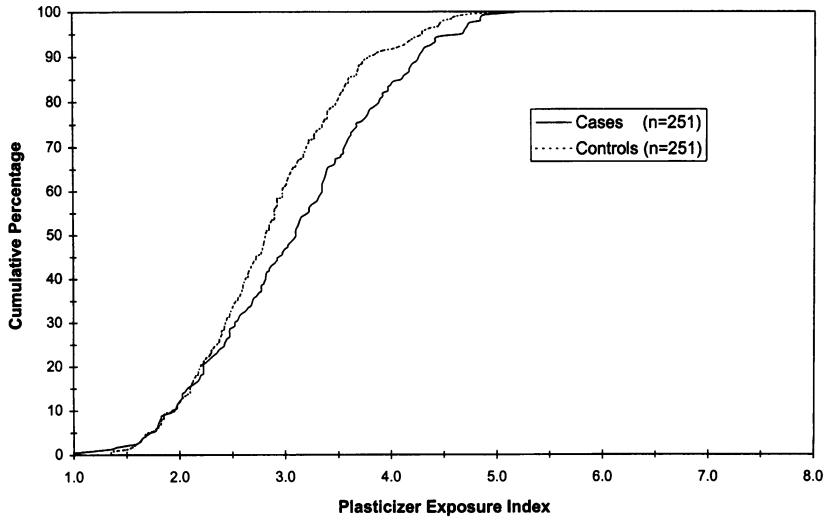


**FIGURE 1—Cumulative frequency distribution of the plasticizer exposure index for case and control subjects in a study of bronchial obstruction in young children: Oslo, Norway, 1992–1995.**

**TABLE 3—Crude and Adjusted Odds Ratios (ORs) for Bronchial Obstruction During the First 2 Years of Life According to Plasticizer Exposure Index (Conditional Logistic Regression of 251 Pairs): Oslo, Norway, 1992–1995**

| | Crude OR (95% CI) | Adjusted OR[a] (95% CI) |
|---|---|---|
| Model 1 | | |
| 0–50th percentile (reference category) | 1.00 . . . | 1.00 . . . |
| 50th–75th percentile | 1.28 (0.81, 2.04) | 1.34 (0.78, 2.30) |
| 75th–100th percentile | 2.46 (1.50, 4.02) | 2.71 (1.50, 4.91) |
| Model 2 (continuous) | 1.56 (1.20, 2.04) | 1.65 (1.20, 2.27) |

*Note.* Information on plasticizer exposure index was missing for 12 of the 251 pairs. CI = confidence interval.
[a]Adjusted for the core covariates (sex, parental atopy, having siblings, day-care attendance, breast-feeding, exposure to environmental tobacco smoke, and dampness problems), maternal education, and annual family income.

parents, in each questionnaire, to contact the project pediatrician if the child experienced respiratory problems; by asking the outpatient clinics to refer all possible cases to the project pediatrician; and by contacting families who reported children's respiratory symptoms in the questionnaires. To evaluate the detection of bronchial obstruction, a physician conducted telephone interviews with 100 parents who had reported no symptoms or signs of bronchial obstruction in their children. No overlooked episodes were revealed.

Participation rates among both case subjects (98.4%) and control subjects (100%) were very high and could not introduce bias. Parents' conscious choice of interior materials is a potential source of bias if related to risk of bronchial obstruction. The adjustment for parental atopy reduced any selection bias related to the child's predisposition to bronchial obstruction. Avoidance of plastic or textile materials among parents of children experiencing symptoms would have led to underestimation of the relationship between exposure and outcome. The assessment of exposure shortly after the case definition reduced this potential source of bias. Thus, selection bias is not a likely explanation for the observed relationship between bronchial obstruction and surface materials.

The information for case and control subjects was comparable for several reasons. The assessment of interior materials and other factors was a standard procedure that was carried out concurrently in the homes of each case–control pair. The investigators were advised not to ask about the child's case or control status, but some parents may have provided this information during the home visit. Furthermore, information on a number of other potential determinants of bronchial obstruction was collected in the baseline and follow-up questionnaires given to parents of the cohort before the development of bronchial obstruction.

Potential confounding by age and season was controlled by one-to-one matching of case and control subjects. Other potential confounders were adjusted for in the conditional logistic regression analysis.

### *Synthesis With Previous Knowledge*

To our knowledge there is little previous empirical evidence of an increased risk of bronchial obstruction or asthma related to interior materials in the indoor environment.

Plastic interior materials, especially plasticizers used in the synthesis of PVC plastics, are potential sources of chemicals that may cause inflammation of the airways and increase the risk of bronchial obstruction and asthma. Plasticizer di-(2-ethylhexyl) phthalate (DEHP) is widely used in PVC plastics, and it may constitute 40% of PVC. There is some suggestive empirical evidence of plasticizers' effect on airways in rats and human infants. Mono(2-ethylhexyl) phthalate (MEHP), the primary hydrolysis product of DEHP, was found to induce bronchial hypersensitivity in rats,[17] and preterm infants exposed to DEHP from respiratory tubing systems were reported to have a higher risk of bronchial asthma.[18] Furthermore, it is hypothesized that MEHP mimics the inducing prostaglandins (PGs) $PGD_2$, $9\alpha,11\beta PGF_2$, $PGF_{2\alpha}$, and thromboxanes in the lungs and thereby increases the risk of inducing inflammation in the airways, which is a characteristic feature of asthma.[19]

Plasticizers have a high affinity[20] for particles, and they have been shown to migrate from PVC floor to sedimented house dust.[19] Thus exposure from PVC floor materials could be stronger than exposure from wall coverings, especially in young children, whose breathing zone is close to the floor. This could explain why the risk of bronchial obstruction was related to PVC flooring but not to PVC wall materials.

## *Conclusion*

Our results provide new evidence supporting the hypothesis that PVC materials and textile wall materials used in the home environment are possible determinants of bronchial obstruction in early childhood. Possible direct causal agents could be plasticizers emitted from PVC plastic materials. □

## Contributors

Jouni Jaakkola planned and supervised the data analysis and wrote the paper. Leif Øie was responsible for the environmental data collection and analyzed the data. Per Nafstad participated in the planning of the study and was responsible for the health data collection. Grete Botten and Sven Samuelsen participated in the planning of the study, and Grete Botten supervised the data collection. Per Magnus participated in data analysis and contributed to the text. All authors contributed to the text and are guarantors of the integrity of the research.

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## References

1. Laitinen LA, Heino M, Laitinen A, Kava T, Haahtela T. Damage of the airway epithelium and bronchial reactivity in patients with asthma. *Am Rev Respir Dis*. 1985;131:399–406.
2. Frew AJ. The immunology of respiratory allergies. *Toxicol Lett*. 1996;86:65–72.
3. Nordman H, Keskinen H, Tuppurainen M. Formaldehyde asthma—rare or overlooked? *J Allergy Clin Immunol*. 1985;75:91–99.
4. Patterson R, Hargreave F, Grammer L, Harris K, Dolovich J. Toluene diisocyanate respiratory reactions, I: reassessment of the problem. *Int Arch Allergy Appl Immunol*. 1987;84:93–100.
5. Newman Taylor AJ, Venables KM, Durham SR, Graneek BJ, Topping MD. Acid anhydrides and asthma. *Int Arch Allergy Appl Immunol*. 1987;82:435–439.
6. 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: Elsevier Science Publishers BV (Biomedical Division); 1989:329–336.
7. Skov P, Valbjørn O, Pedersen BV, and DISG. Influence of indoor climate on the sick building syndrome in an office environment. *Scand J Work Environ Health*. 1990;16:363–371.
8. Jaakkola JJK, Tuomaala P, Seppänen O. Textile wall materials and sick building syndrome. *Arch Environ Health*. 1994;49:175–181.
9. Nafstad P, Jaakkola JJK, Hagen JA, Botten G, Kongerud J. Breastfeeding, maternal smoking and lower respiratory infections. *Eur Respir J*. 1996;9:2623–2629.
10. Nafstad P, Kongerud J, Botten G, Hagen JA, Jaakkola JJK. The role of passive smoking in the development of bronchial obstruction during the first two years of life. *Epidemiology*. 1997;8:293–297.
11. Nafstad P, Øie L, Mehl R, et al. Residential dampness problems and symptoms and signs of bronchial obstruction in young Norwegian children. *Am J Respir Crit Care Med*. 1998;157:410–414.
12. Sundmark H. *Environmental Risk Assessment of Phthalates Used as Plasticizers for PVC*. Oslo, Norway: Norsk Hydro; 1995.
13. *The Use of Phthalates in Denmark*. Copenhagen, Denmark: Danish Environmental Protection Agency; 1984.
14. Stymne H, Boman CA, Kronvall J. Measuring ventilation rates in the Swedish housing stock. *Bld Environ*. 1994;29:373–379.
15. Breslow NE, Day NE. *Statistical Methods in Cancer Research. Vol. 1. The Analysis of Case-Control Studies*. Lyon, France: International Agency for Research on Cancer; 1980.
16. Kleinbaum DG. *Logistic Regression: A Self-Learning Text*. New York, NY: Springer Verlag; 1994.
17. Doelman CJA, Borm PJA, Bast A. Plasticiser and bronchial hyperreactivity. *Lancet*. 1990; 335:725.
18. Roth B, Herkenrath P, Lehman H-J, Ohlens H-D, Homig HJ, Benz-Bohm G. D-(2-ethyl)-phthalate as plasticizer in PVC respiratory tubing systems: indication of hazardous effects on pulmonary function in mechanically ventilated, preterm infants. *Eur J Pediatr*. 1988;147:41–46.
19. 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.
20. *Toxicological Profile for Di(2-ethylhexyl)phthalate (DEHP)*. Washington, DC: US Dept of Health and Human Services; April 1993. TP-92/05.