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Increased risk of allergy in children due to formaldehyde exposure in homes

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Key words: allergic sensitization; asthma; atopy; child health; formaldehyde; indoor air; respiratory symptoms.

Background: Formaldehyde levels were measured in 80 houses in the Latrobe Valley, Victoria, Australia. An association between exposure to formaldehyde and sensitization to common aero-allergens has been suggested from animal trials, but no epidemiologic studies have tested this hypothesis.

Methods: A total of 148 children 7–14 years of age were included in the study, 53 of whom were asthmatic. Formaldehyde measurements were performed on four occasions between March 1994 and February 1995 with passive samplers. A respiratory questionnaire was completed, and skin prick tests were performed.

Results: The median indoor formaldehyde level was 15.8 $\mu\text{g}/\text{m}^3$ (12.6 ppb), with a maximum of 139 $\mu\text{g}/\text{m}^3$ (111 ppb). There was an association between formaldehyde exposure and atopy, and the adjusted odds ratio was 1.40 (0.98–2.00, 95% CI) with an increase in bedroom formaldehyde levels of 10 $\mu\text{g}/\text{m}^3$. Furthermore, more severe allergic sensitization was demonstrated with increasing formaldehyde exposure. On the other hand, there was no significant increase in the adjusted risk of asthma or respiratory symptoms with formaldehyde exposure. However, among children suffering from respiratory symptoms, more frequent symptoms were noted in those exposed to higher formaldehyde levels.

Conclusions: Low-level exposure to indoor formaldehyde may increase the risk of allergic sensitization to common aeroallergens in children.

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Formaldehyde is a well-known irritant of the upper respiratory tract with symptoms such as eye, nose, and throat irritation commonly associated with indoor exposure to the gas (1–3). Ambient formaldehyde levels are usually

very low (below $6 \mu\text{g}/\text{m}^3$), but may be higher in urban areas (4). Indoor sources of formaldehyde include particle board, plywood, fibreboard, panelling, urea formaldehyde foam insulation, and some carpets and furniture, as well as some household chemicals. Due to the common use of formaldehyde-emitting materials in residential houses, indoor concentrations generally exceed those outdoors. In the USA, a mean formaldehyde concentration of $38 \mu\text{g}/\text{m}^3$ has been reported in residential houses, with a typical range of $13\text{--}350 \mu\text{g}/\text{m}^3$ (4). An indoor guideline of 100 ppb ($125 \mu\text{g}/\text{m}^3$) has been set in Australia, and this concentration is seldom exceeded in conventional houses (5). However, substantially higher formaldehyde levels can be found in caravans, mobile homes, and houses insulated with urea formaldehyde insulation.

While irritant effects of the upper respiratory tract are a well-established consequence of formaldehyde exposure above $150 \mu\text{g}/\text{m}^3$ (4), there have also been suggestions of a long-term effect on the lower respiratory system of formaldehyde exposure (6, 7), but the results have not been consistent (4). Furthermore, the mechanism by which formaldehyde exposure could affect the lower respiratory tract is unclear. At low exposure levels, most formaldehyde is deposited in the upper respiratory tract (4), and irritant effects on the lower respiratory system are therefore not likely. Another possible mechanism would be sensitization to tissue proteins that have reacted to formaldehyde, an effect which would in turn lead to respiratory symptoms. Indeed, such sensitization has been demonstrated at high exposure levels which would only be encountered in occupations such as pathology or embalming (8). More recently, it has also been demonstrated that low-level exposure to formaldehyde can trigger production of specific IgE in children, but this sensitization was not associated with symptoms, making its relevance unclear (9). A third possible mechanism for an effect of formaldehyde on the lower respiratory system would be an increased risk of allergic sensitization to common aeroallergens with formaldehyde exposure. The biologic plausibility for such an effect has been demonstrated in animal trials at high exposure levels (10). On the other hand, no evidence for such an association in man has yet been published, making it uncertain whether the lower exposure levels in conventional homes have the potential to interact with allergen exposure.

This paper presents the association of formaldehyde with atopy, asthma, and respiratory symptoms in children. The data were collected as part of an indoor environmental study, which also included measurements of indoor nitro-

gen dioxide, house-dust-mite allergen (Der p 1), and airborne fungal spores, results for which are being published separately (11–14).

Material and methods

A total of 80 households from the rural towns Moe and Morwell in the Latrobe Valley, Victoria, Australia, were recruited for the study. Both towns have a population of around 18 000 people, and they are surrounded by open-cut brown coal mines and power stations, which provide considerable employment. Brief information sheets with a reply-paid slip were distributed via schools and medical centres seeking households with at least one child in the age-group 7–14 years to participate in this multicontaminant study. Equal numbers of households with asthmatic and nonasthmatic children were sought. In the later stages of the recruiting process, advertisements in the local press were utilized to encourage households with nonasthmatic children to participate, as they were proving more difficult to recruit. Forty-three of the participating households had at least one asthmatic child in the age-group, diagnosed by a doctor, but any nonasthmatic children were also included as study participants, making 53 asthmatic and 30 nonasthmatic children. The remaining 37 households had only nonasthmatic children with a total of 65 in the age-group. Across the 80 households, a total of 148 study children was thus included. Their mean age was 10.2 years at the start of the study with equal numbers of girls and boys. Approval for the study was obtained from the Standing Committee for Ethics in Research on Humans at Monash University (project no. 73/93).

Formaldehyde measurements

Formaldehyde samples were collected from participating households on four occasions over 1 year, in March–April, May, and September 1994, and January–February 1995. Passive samplers were exposed for 4 days in bedrooms of participating children, living rooms, kitchens, and outdoors. Thus, the total number of samples in each house varied depending on the number of children participating in the study. The method was based on the work by Levin et al. (15), with slight modifications (16).

Table 1. Formaldehyde levels in association with atopic and nonatopic children, geometric means (and 95% confidence intervals)

	Atopic children n=88	Nonatopic children n=57	P value
Bedroom levels	19.0 (16.7–21.7)	16.4 (14.3–18.8)	0.06
Highest recorded level	38.3 (33.8–43.3)	28.6 (24.6–33.3)	0.002

Health outcomes

A validated respiratory questionnaire was modified for use in the study (17). One questionnaire was completed for each child during an interview with a parent at the last visit to the household. Information on parental allergy, parental asthma, and presence of pets was also collected. The frequency of respiratory symptoms experienced during the past year was recorded in four categories (none, 1–3 times, 4–12 times, or >12 times). Eight respiratory symptoms were included: cough, cough in the morning, shortness of breath, waking due to shortness of breath, wheeze/whistling, asthma attacks, chest tightness, and chest tightness in the morning. During data analysis, a respiratory symptom score was calculated for each child. For this purpose, the four symptom frequency categories were coded as 0, 1, 2, and 3, and the scores for each individual symptom were then added to give a total respiratory symptom score.

Skin prick tests were performed on 145 of 148 study children with 12 environmental allergens (Hollister Stier, Spokane, WA, USA): cat, dog, grass mix no. 7, Bermuda grass, house dust, house-dust mite (*Dermatophagoides pteronyssinus* and *D. farinae*), and fungi (*Alternaria tenuis*, *Hormodendrum cladosporioides*, *Penicillium* mix, *Aspergillus* mix, and mould mix A). A saline solution obtained

with the extracts was used as a negative control, and a histamine solution (10 mg/ml) was used as a positive control. The tests were performed by a trained technician between August and October 1994. Largest wheal diameters were measured 15 min after pricking. The ratio of allergen wheal size to histamine wheal size was calculated, and tests were considered positive if this ratio was greater than or equal to 0.5 (18).

Statistical considerations

Statistical analyses were performed with SPSS for Windows, version 6.0 (SPSS, Inc., Chicago, IL, USA, 1993), with Genstat 5, release 3.1 (New York: Oxford University Press, 1994) being used for logistic regression modelling. The distribution of formaldehyde measurements was positively skewed, and nonparametric tests such as the Mann-Whitney U were therefore used for initial comparisons. The distributions of children in three categories of formaldehyde exposure were compared by chi-square tests with follow-up assessment of linear trends. Logistic-regression models (19) were applied to calculate adjusted odds ratios for atopy, asthma, and respiratory symptoms with formaldehyde exposure. It is acknowledged that one of the assumptions of logistic regression is independence between observations. In this study, such an assumption may not be totally justified for observations on individual children, since many of the households included more than one study child, and children in the household are likely to be less genetically diverse and/or subject to relatively similar unmeasured environmental conditions. However, under the assumptions of the usual overdispersion model, the estimates of the regression parameters are the same as those obtained for the independence model (20). Health outcomes with several

Table 2. Distribution of atopic and nonatopic children by formaldehyde-exposure group (bedroom and highest recorded levels)

Formaldehyde-exposure group	n	Asthmatics n=57	Nonasthmatics n=88	Proportion asthmatic
Bedroom levels				
<10 µg/m ³	26	13	13	0.50
10–30 µg/m ³	88	36	52	0.59
>30 µg/m ³	31	8	23	0.74
Linear trend P=0.06				
Highest level				
<20 µg/m ³	30	20	10	0.33
20–50 µg/m ³	75	27	48	0.64
>50 µg/m ³	40	10	30	0.75
Linear trend P<0.001				

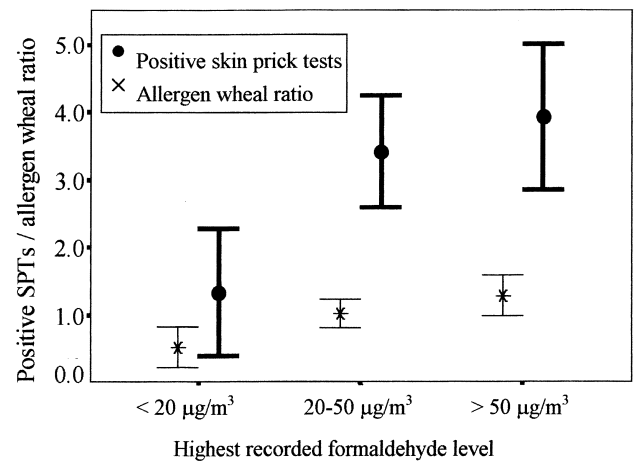
Table 3. Highest recorded formaldehyde levels ($\mu\text{g}/\text{m}^3$) for children with and without atopy by parental allergy/asthma. Medians are given, with *P* value for difference between groups (Mann-Whitney)

		<i>n</i>	Atopics	Nonatopics	<i>P</i> value
Parental allergy	Yes	100	41.2	30.8	0.02
	No	48	30.6	25.9	0.17
Parental asthma	Yes	39	45.7	44.1	0.65
	No	109	34.7	27.2	0.02

response categories were analysed for an association with formaldehyde exposure by comparing the mean in three formaldehyde exposure groups by analysis of variance with Bonferroni's test for multiple comparisons. In addition, multiple linear regression models were used to make adjustments for possible confounding factors. The three formaldehyde-exposure groups were entered to test for a linear and a quadratic association. All possible interaction terms between parental asthma, parental allergy, and formaldehyde exposure groups were tested in the models, but only those which were significant were included in the final models.

Results

The median indoor formaldehyde level calculated from individual measurements was $15.8 \mu\text{g}/\text{m}^3$ (12.6 ppb), with a maximum of $139 \mu\text{g}/\text{m}^3$ (111 ppb) (16). The median outdoor level was $0.7 \mu\text{g}/\text{m}^3$ with a range of <0.3 – $15.3 \mu\text{g}/\text{m}^3$. A total of three indoor samples exceeded the current Australian indoor guideline of 100 ppb. Glued wood products, such as particle board and fibreboard, were the main sources of indoor formaldehyde (16). No significant associations between formaldehyde levels and other contaminants (nitrogen dioxide levels, house-dust-mite allergen levels, or fungal spore concentrations) or housing factors were found. Some 46% of children were exposed to indoor pets, while 33% were exposed to passive smoking. Of the asthmatic children, 83% were atopic (recorded at least one

**Figure 1.** Severity of allergic sensitization in children by formaldehyde-exposure group (highest recorded formaldehyde level). Mean number of positive skin prick tests (SPTs) and largest allergen wheal ratios are shown with 95% confidence interval.

positive skin prick test), while 48% of nonasthmatic children were atopic. The most common allergy for both asthmatics and nonasthmatics was that to the house-dust mite (*D. pteronyssinus*), 81% of asthmatic children showing a positive skin prick test, compared to 39% of nonasthmatics. Some 68% of children had at least one parent with allergic problems (parental allergy), while 26% of children had at least one asthmatic parent (parental asthma).

Some 94% of asthmatic children suffered from at least one respiratory symptom, compared to 48% of nonasthmatic children. A respiratory symptom score was calculated for each child based on questionnaire data. Asthmatic children recorded a mean score of 4.6 (range 0–8.0), while nonasthmatics recorded a mean score of 0.7 (range 0–4.0). The difference between the two groups was highly significant ($P<0.001$). Parental asthma and parental allergy were found to be significantly associated with health outcomes in the children. Parental asthma was associated with asthma ($P<0.001$), allergy ($P=0.02$), and respiratory symptoms ($P=0.03$). Parental allergy was associated with asthma ($P=0.02$), but not with atopy or respiratory symptoms in the children. Boys were more likely than girls to be atopic

Table 4. Distribution of asthmatic and nonasthmatic children by formaldehyde-exposure category (highest recorded levels)

Formaldehyde-exposure group	<i>n</i>	Nonatopic children, <i>n</i> =53	Atopic children, <i>n</i> =95	Proportion atopic
<20 $\mu\text{g}/\text{m}^3$	31	5	26	0.16
20–50 $\mu\text{g}/\text{m}^3$	76	30	46	0.39
>50 $\mu\text{g}/\text{m}^3$	41	18	23	0.44
Linear trend $P=0.02$				

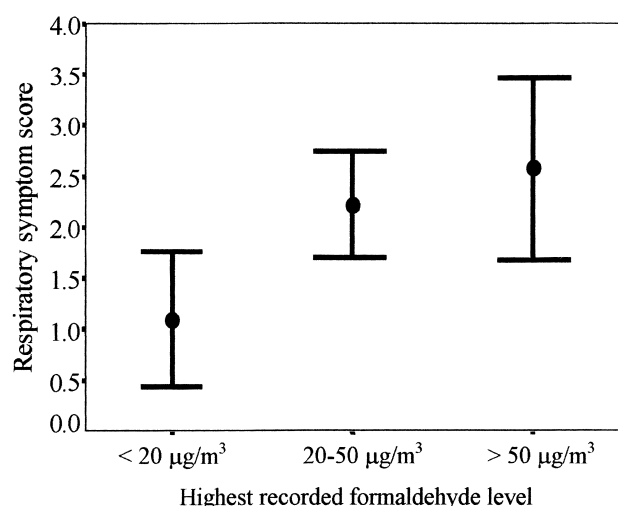


Figure 2. Respiratory symptom score by formaldehyde-exposure group. Means with 95% confidence intervals.

($P=0.04$). In addition, an association between parental asthma and formaldehyde levels was seen, with higher levels recorded for children whose parents were asthmatic ($P<0.01$).

Allergy

Marginally higher mean formaldehyde levels were recorded in the bedrooms of atopic than nonatopic children ($P=0.06$) (Table 1). A significant difference in formaldehyde levels was seen when comparing highest recorded levels in the home of the child ($P=0.002$). The children's formaldehyde exposure was categorized into three categories based on bedroom levels, and another three categories based on highest recorded levels were created. Table 2 shows the distribution of atopic and nonatopic children between these exposure groups. For bedroom levels, the groups were not significantly different. However, the difference between the groups based on highest recorded formaldehyde levels was significant ($\chi^2=13.2$, $df=2$, $P=0.001$), and a linear trend was present, suggesting a greater risk of atopy with higher exposure levels ($P<0.001$). As mentioned above, parental asthma and parental allergy were also associated with formaldehyde levels. For this reason, it is possible that any association between formaldehyde levels and health outcomes could be confounded by the concurrent association between formaldehyde levels and family history. In order to clarify whether this was the case, children with and without parental asthma/allergy were analysed separately for an association with highest recorded formaldehyde levels (Table 3). Formaldehyde levels were consistently higher for atopic children, and the difference was significant for

children without parental asthma, suggesting that the association between formaldehyde and atopy was not confounded by this factor. Logistic regression was used to calculate odds ratios for the association between atopy and formaldehyde exposure. The crude odds ratio for atopy with an increase in bedroom formaldehyde levels by $10 \mu\text{g}/\text{m}^3$ was 1.34, and adjusted for parental asthma and sex, it was 1.40 (95% confidence interval, 0.98–2.00). Similarly, an adjusted odds ratio of 1.42 (95% CI, 0.99–2.04) for atopy with an increase in highest recorded formaldehyde level by $20 \mu\text{g}/\text{m}^3$ was found (crude odds ratio 1.43). Data on passive smoking, presence of pets, indoor nitrogen dioxide levels, airborne fungal spores, or house-dust-mite allergen levels did not significantly influence the estimates and were therefore not included in the model.

In addition to the evidence that formaldehyde exposure is a risk factor for allergic sensitization, there was evidence of a more severe sensitization with greater formaldehyde exposure. There were significant differences in the average number of positive skin prick tests for children in three formaldehyde-exposure categories based on highest recorded levels ($P=0.004$) (Fig. 1). Similarly, the average relative allergen wheal size for the largest allergen wheal was different between formaldehyde-exposure groups ($P=0.002$) (Fig. 1). Both these measures of the severity of allergic sensitization tended to increase with increasing formaldehyde exposure, suggesting that greater severity of the allergic sensitization was associated with higher formaldehyde exposure. In both cases, the two higher-exposure group means were significantly higher than the low-exposure group mean in multiple comparison tests ($P<0.05$). Multiple linear-regression models were used to adjust the associations for potential confounding variables. A significant linear influence of formaldehyde exposure remained after controlling for parental asthma/allergy and sex on both the number of positive skin prick tests and maximum relative wheal size.

Asthma

An association between formaldehyde exposure and asthma in children was tested by looking at the distribution of asthmatic children in three exposure groups of formaldehyde (Table 4). Bedroom formaldehyde-exposure groups showed no significant difference between groups, but there were significant differences between highest recorded formaldehyde level groups ($\chi^2=6.84$, $df=2$, $P=0.03$). A higher proportion of asthmatics was seen with higher formaldehyde exposure, with a significant linear trend present ($P=0.02$).

However, after adjusting for parental allergy and parental asthma by logistic regression, the odds ratio for asthma was not significantly different from 1.0.

Respiratory symptoms

An association between the presence of any respiratory symptoms and formaldehyde levels was tested in a similar manner as for atopy and asthma, comparing the distribution of children between formaldehyde-exposure groups. No significant differences between exposure groups were seen, but there was a weak trend to more children with respiratory symptoms in higher formaldehyde-exposure groups with the highest recorded formaldehyde-level groups. The linear trend was not significant. In addition, a mean respiratory symptom score was calculated for children in the three formaldehyde-exposure groups. Significant differences between groups were seen with the exposure groups based on highest recorded formaldehyde levels ($P=0.03$) (Fig. 2). The highest exposure group recorded a significantly higher average score than the low-exposure group in multiple comparisons. A multiple linear-regression model was used to adjust for the effect of parental asthma/allergy and for any interaction between parental asthma, parental allergy, and formaldehyde-exposure group. A significant influence of high formaldehyde exposure on the respiratory symptom score remained after these adjustments.

Discussion

The findings presented in this paper suggest that formaldehyde exposure at levels below current guidelines may lead to an increased risk of allergic sensitization to common aeroallergens in children. For each increase in bedroom formaldehyde levels of $10 \mu\text{g}/\text{m}^3$ (8 ppb), there was an approximate increase in the risk of atopy of 40%, as estimated from the odds ratio. This effect of formaldehyde exposure on allergic sensitization to common aeroallergens has not previously been reported in epidemiologic studies. Such an effect has, however, been suggested from animal studies at high formaldehyde-exposure levels ($2000 \mu\text{g}/\text{m}^3$ or 1600 ppb) (10). The authors of that study propose that formaldehyde may facilitate sensitization to allergens due to changes in the upper respiratory tract. Increased permeability of the respiratory epithelial layer, or suppression of mucosal immune defences are possible mechanisms.

A potential problem with selection bias in the present study should be recognized, as participating households

were recruited as volunteers. It is likely that families with allergic problems were more likely to take part in the study. Some 48% of nonasthmatic children were atopic, and this is somewhat higher than in comparable community studies. However, this is not believed to have influenced the results in any significant way. Another potential problem is that the formaldehyde exposure was measured for children 7–14 years of age, while sensitization to indoor allergens occurs mainly in early childhood. Ideally, only children still residing in the dwelling where they were born should have been studied, but this was not possible. Some 74% of the study children had lived in the same house for 5 years or more, while 36% had never moved since birth. It is possible that families with children who developed allergies in early childhood subsequently chose to live in newer dwellings, which also tend to have higher formaldehyde levels, thereby causing the observed association between formaldehyde and atopy. However, there was no significant association between formaldehyde levels and house age; therefore, the latter factor is unlikely to have confounded the results. There is also a potential problem of recall bias, as data on respiratory symptoms were collected retrospectively. However, in this study, major recall bias was unlikely, as participants were asked to remember only events which actually occurred during the period of study. There were no significant associations between formaldehyde levels and other housing factors associated with health outcomes, making confounding by such factors unlikely.

Evidence for an effect of formaldehyde exposure on atopy has been provided by the present study. An approximate increase in risk of atopy of 40% with either $10 \mu\text{g}/\text{m}^3$ higher bedroom levels of formaldehyde, or with $20 \mu\text{g}/\text{m}^3$ higher exposure levels, was seen after adjusting for potential confounding factors. The odds ratio for bedroom exposure levels was significant, but the association with highest recorded formaldehyde levels was stronger throughout the analyses. The main sources of formaldehyde in the study houses were glued wood products (16). It is possible that the stronger association with highest recorded levels is due to better characterization of houses, since ventilation may have occurred on most sampling occasions, thus diluting the average concentrations significantly. It is also possible that peak levels are most important in producing sensitization. In addition to an increased risk of atopy, there was evidence of more severe allergic sensitization with higher formaldehyde exposure (Fig. 1). This suggests a dose-response association between formaldehyde exposure and allergy in children. The associations with severity of allergic sensi-

zation remained significant after adjusting for confounding factors.

A significantly higher respiratory symptom score was seen for children with high formaldehyde exposure (Fig. 2), and the association remained significant after adjusting for confounding factors. This respiratory symptom score can be considered as a measure of the severity of respiratory symptoms. The odds ratio for the presence of any respiratory symptoms was, on the other hand, not significant. An apparent association between asthma in children and formaldehyde exposure is suggested from bivariate analysis (Table 4), but the association did not remain after adjusting for family-history-related factors. Previously, asthma-like symptoms have been associated with formaldehyde levels (7), and another study showed a direct association between formaldehyde levels and diagnosed asthma in children (4). Therefore, it is quite possible that an association does exist, but further evidence is required.

Implications

If low-level formaldehyde exposure, well below current guidelines, does cause children to become more prone to developing allergies, it would have widespread implications in the developed world. The use of materials emitting formaldehyde in buildings is very common, and some exposure occurs in virtually every modern home. Thus,

even a small increase in the risk of allergy would lead to large numbers of children being affected. In recent decades, it is clear that the prevalence of allergic diseases has increased substantially in many Western countries (21). At the same time, materials emitting formaldehyde have become increasingly popular in residential homes (4). Thus, it is possible that at least some part of the increase in allergic disease could have been brought about by an increase in indoor formaldehyde exposure. Coupled with an increase in the exposure to some indoor allergens (22), the effect may have been synergistic, thus offering a possible explanation of the rapid increase in allergic diseases. Indoor guidelines for formaldehyde exposure may need to be reviewed in light of recent findings, and the use of formaldehyde-emitting materials in homes should be limited until the completion of larger-scale longitudinal epidemiologic studies to clarify the role of formaldehyde in allergic disease and respiratory health.

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References

- Godish T, Zollinger TW, Konopinski V. Residential formaldehyde. Increased exposure levels aggravate adverse health effects. *J Environ Health* 1990;53:34–37.
- Hanrahan LP, Dally KA, Anderson HA, Kanarek MS, Rankin J. Formaldehyde vapour in mobile homes: a cross sectional survey of concentrations and irritant effects. *Am J Public Health* 1984;74:1026–1027.
- Ritchie IM, Lehnen RG. Formaldehyde-related complaints of residents living in mobile and conventional homes. *Am J Public Health* 1987;77:323–328.
- Stenton SC, Hendrick DJ. Formaldehyde. *Immunol Allergy Clin North Am* 1994;14:635–657.
- McPhail S. Formaldehyde in homes. *J Occup Health Safety Aust N Z* 1991;7:139–144.
- Krzyzanowski M, Quackenboss JJ, Lebowitz MD. Chronic respiratory effects of indoor formaldehyde exposure. *Environ Res* 1990;52:117–125.
- Norbäck D, Björnsson E, Janson C, Widström J, Boman G. Asthmatic symptoms and volatile organic compounds, formaldehyde, and carbon dioxide in dwellings. *Occup Environ Med* 1995;52:388–395.
- Nordman H, Keskinen H, Tuppurainen M. Formaldehyde asthma – rare or overlooked? *J Allergy Clin Immunol* 1985;75:91–99.
- Wantke F, Demmer CM, Tappler P, Götz M, Jarisch R. Exposure to gaseous formaldehyde induces IgE-mediated sensitisation to formaldehyde in schoolchildren. *Clin Exp Allergy* 1996;26:276–280.
- Tarkowski M, Gorski P. Increased IgE antiovalbumin level in mice exposed to formaldehyde. *Int Arch Allergy Immunol* 1995;106:422–424.
- Garrett MH, Hooper BM, Hooper MA, Abramson MJ, Rayment PR. Indoor airborne fungal spores, dampness and associations with environmental factors and respiratory health in children. *Clin Exp Allergy* 1998;28:459–467.
- Garrett MH, Hooper MA, Hooper BM, Abramson MJ. Respiratory symptoms in children and indoor exposure to nitrogen dioxide and gas stoves. *Am J Respir Crit Care Med* 1998;158:891–895.
- Garrett MH, Hooper BM, Hooper MA. Indoor environmental factors associated with house-dust-mite allergen (Der p 1) levels in south-eastern Australian houses. *Allergy* 1998;53:1060–1066.
- Garrett MH, Abramson MJ, Hooper BM, Rayment PR, Strasser RP, Hooper MA. Indoor environmental risk factors for respiratory health in children. *Indoor air* 1998 (in press).
- Levin J-O, Lindahl R, Andersson K. Monitoring parts-per-billion levels of formaldehyde using a diffusive sampler. *J Air Pollut Control Assoc* 1989;39:44–47.
- Garrett MH, Hooper MA, Hooper BM. Formaldehyde in Australian homes: levels and sources. *Clean Air Aust* 1997;31:28–32.

17. Abramson M, Kutin J, Bowes G. The prevalence of asthma in Victorian adults. *Aust N Z J Med* 1992;**22**:358–363.
18. Meinert R, Frischer T, Karamaus W, Kuehr J. Influence of skin prick test criteria on estimation of prevalence and incidence of allergic sensitisation in children. *Allergy* 1994;**49**:526–532.
19. Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley, 1989.
20. McCullagh P, Nelder J. Generalised linear models. London: Chapman and Hall, 1983;sect. 2.1.2–2.1.3.
21. Strachan DP. Time trends in asthma and allergy: ten questions, fewer answers. *Clin Exp Allergy* 1995;**25**:791–794.
22. Peat JK, van den Berg RH, Green WF, Mellis CM, Leeder SR, Woolcock AJ. Changing prevalence of asthma in Australian children. *BMJ* 1994;**308**:1591–1596.