Respiratory Symptoms in Children and Indoor Exposure to Nitrogen Dioxide and Gas Stoves

MARIA H. GARRETT, MARTIN A. HOOPER, BEVERLEY M. HOOPER, and MICHAEL J. ABRAMSON

School of Applied Sciences, Monash University, Churchill, Victoria, Australia, and Department of Epidemiology and Preventive Medicine, Monash Medical School, Alfred Hospital, Prahran, Victoria, Australia

Nitrogen dioxide levels were measured in 80 homes in the Latrobe Valley, Victoria, Australia, using passive samplers. Some 148 children between 7 and 14 yr of age were recruited as study participants, 53 of whom had asthma. Health outcomes for the children were studied using a respiratory questionnaire, skin prick tests, and peak flow measurements. Nitrogen dioxide concentrations were low, with an indoor median of 11.6 $\mu g/m^3$ (6.0 ppb), and a maximum of 246 $\mu g/m^3$ (128 ppb). Respiratory symptoms were more common in children exposed to a gas stove (odds ratio 2.3 [95% CI 1.0–5.2], adjusted for parental allergy, parental asthma, and sex). Nitrogen dioxide exposure was a marginal risk factor for respiratory symptoms, with a dose–response association present ($p = 0.09$). Gas stove exposure was a significant risk factor for respiratory symptoms even after adjusting for nitrogen dioxide levels (odds ratio 2.2 [1.0–4.8]), suggesting an additional risk apart from the average nitrogen dioxide exposure associated with gas stove use. Atopic children tended to have a greater risk of respiratory symptoms compared with nonatopic children with exposure to gas stoves or nitrogen dioxide, but the difference was not significant. 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.

Nitrogen dioxide is produced during combustion processes. Indoor sources include gas stoves, gas heaters, and smoking. Because these sources are not always well ventilated during use, any nitrogen dioxide generated is trapped indoors, causing higher levels than outdoors. Concerns about adverse health effects of such exposure have therefore been raised. Controlled clinical trials have demonstrated that high nitrogen dioxide exposure can cause lung injury and a decrease in pulmonary defense mechanisms due to the oxidant effects of the gas (1). On the other hand, acute effects are less likely at low exposure levels such as those experienced in residential homes. Sensitive subgroups of the population may still be affected (2). One such subgroup may be the atopic population, as it has been demonstrated that nitrogen dioxide exposure can potentiate the effect of allergen exposure (3).

Results from epidemiological studies have been inconclusive with some studies reporting an association between exposure to nitrogen dioxide and respiratory symptoms in children (4–6), while others have found no such association (7–10). Possible reasons for these conflicting results include: misclassification of nitrogen dioxide exposure owing to few measurements, or only using the presence of a gas stove to classify nitrogen dioxide exposure; misclassification of health outcomes when using questionnaires to collect retrospective data; or a small sample size. Each of these problems in study design lead to a lower likelihood of revealing a positive association between exposure and health outcomes. Thus, Samet and Utell (1) concluded that the problems with conflicting results can largely be attributed to problems in methodology.

This report presents nitrogen dioxide exposure data and associations with respiratory health outcomes from an indoor environmental study conducted in the Latrobe Valley, Victoria, Australia. Comprehensive direct measurements of indoor nitrogen dioxide exposure were made on five occasions over 1 yr, thus reducing the risk of misclassification of exposure. Health data were collected during an interview with a parent, and only respiratory symptoms experienced during the sample collection period were required, leading to a reduced risk of misclassifying health outcomes. The sample size was sufficient and the children were selected on the basis of having a high proportion of asthmatics/atopics, thus including many potentially sensitive individuals. The aim of the analysis was to determine the association between nitrogen dioxide exposure and respiratory symptoms/asthma.

METHODS

Eighty households in the Latrobe Valley, Victoria, Australia with children between 7 and 14 yr of age were recruited for an indoor environmental study. An overview of the methodology for the full study protocol has been published (11). Approval was obtained from the Standing Committee on Ethics in Research on Humans at Monash University (project 73/93). Forty-three households had at least one asthmatic child, while the remaining 37 households had only nonasthmatic children. All children in the age group residing in the households were included in the study, making a total of 148 children, 53 of

(*Received in original form January 24, 1997 and in revised form May 20, 1998*)

M. H. Garrett was the recipient of a Monash University graduate scholarship and a Monash University postgraduate publications award. Financial support was also provided by the Australian Research Council, and Monash University Gippsland Campus Research Fund.

Correspondence and requests for reprints should be addressed to Associate Professor Martin Hooper, School of Applied Sciences, Monash University, Churchill, Victoria 3842, Australia. E-mail: Martin.Hooper@sci.monash.edu.au

Am J Respir Crit Care Med Vol 158. pp 891–895, 1998
Internet address: www.atsjournals.org

whom were asthmatic. The remaining 95 children were nonasthmatic and 30 of these resided in households with asthmatic children. The mean age of children was 10.2 yr at the start of the study, ranging from 7 to 14 yr. There were equal numbers of boys and girls, making 74 of each sex. Sampling visits were made on six occasions over 1 yr, with the first visit in March–April 1994, and the following on a two monthly cycle finishing in January–February 1995. Nitrogen dioxide samples were collected for all sampling visits except for November 1994, making a total of five sampling occasions per household. For each visit, nitrogen dioxide samples were collected from bedrooms of children between 7 and 14 yr of age, living rooms, kitchens, and outside each house. Passive nitrogen dioxide monitors were used (12), with some modifications (13), and monitors were exposed for 4 d.

A health questionnaire adapted from the validated Monash Respiratory questionnaire (14) was completed during an interview with a parent. The frequency of respiratory symptoms over the year of sample collection was recorded in four categories: none, 1–3 times, 4–12 times, and > 12 times. Eight respiratory symptoms were included: cough, shortness of breath, waking due to shortness of breath, wheeze, asthma attacks, chest tightness, cough in the morning, and chest tightness in the morning. Presence of respiratory symptoms in a child was defined as presence of at least one of these symptoms during the year of sample collection. In addition, a respiratory symptom score was calculated by coding the four categories as 0, 1, 2, and 3, and then adding the scores for the eight respiratory symptoms for each child. In addition, the questionnaire included items about parental asthma and parental allergy.

Skin prick tests were performed on all study children using 12 common aeroallergens (Hollister Stier, Spokane, WA): 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 mold mix A). A saline solution 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 divided by the histamine wheal size was calculated, and tests were considered positive if this ratio was equal to or exceeded 0.5 (15). Children with at least one positive skin test were defined as atopic.

Peak expiratory flow rates (PEFR) were monitored by asthmatic children for 2 wk in winter (July) and for another 2 wk in late spring (November). A peak flow meter (Allersearch, Melbourne) was supplied to the households with instructions for use. The highest of three measurements of PEFR in the morning was recorded. Three further measurements were taken following bronchodilator and again the highest recorded. The procedure was repeated at night. The first 3 d of readings in each period were excluded from analysis to avoid the influence of a learning effect. The daily variability in peak flow (%) was calculated as [(highest − lowest)/highest] × 100 (16). The average variability for each period was used in further analysis.

Statistical analysis was performed using SPSS for Windows, version 6.0 (SPSS Inc., Chicago, IL, 1993), with Genstat 5, release 3.1 (Oxford University Press Inc., New York, 1994) used for multiple logistic regression modeling. Nitrogen dioxide data were positively skewed, and nonparametric tests such as Mann-Whitney were therefore used for initial comparisons. In order to clarify which measure of nitrogen dioxide exposure was most appropriate for testing the association with respiratory symptoms, we compared a continuous nitrogen dioxide variable, nitrogen dioxide exposure categories, and a dichotomous classification based on the presence of a gas stove. Crude and adjusted odds ratios for respiratory symptoms with exposure to nitrogen dioxide and gas stoves were calculated (17). For the continuous nitrogen dioxide variable, the risk associated with an increase in concentration by 10 $\mu g/m^3$ was calculated. This value was chosen as such an increase could be attributed to the use of a gas stove in the studied households (13). Because more than one child was included from each household, the proportion of children with respiratory symptoms in each household was used as the dependent variable in the logistic regression models to allow for clustering in the data. Linear regression was used to test an association between nitrogen dioxide exposure measures and frequency of respiratory symptoms, and peak flow variability.

RESULTS

Nitrogen dioxide concentrations in Latrobe Valley houses were found to be relatively low with an indoor median of 11.6 $\mu g/m^3$ (6.0 ppb). The 10th and 90th percentiles were 5.01 $\mu g/m^3$ and 27.9 $\mu g/m^3$, respectively, and the maximal concentration was 246 $\mu g/m^3$ (128 ppb). The main indoor nitrogen dioxide sources were identified as gas stoves, vented gas heaters, and smoking. One unvented gas heater was used among the study houses, and it was associated with the highest recorded levels. Boys were more likely to suffer from allergies than girls ($p = 0.04$), whereas there was no significant difference in the prevalence of asthma or respiratory symptoms by sex. Some 26% of children had one or two asthmatic parents (parental asthma), while 68% of children had one or two allergic parents (parental allergy). There was a significantly increased risk for children with parental asthma to suffer from asthma, atopy, and respiratory symptoms, whereas parental allergy was a significant risk factor for asthma only.

Some 61% of all children had at least one positive skin prick test, and were classified as atopic. Among asthmatics, 83% were atopic, compared with 48% of nonasthmatics. The most common allergy was to the house dust mite (*D. pteronyssinus*) with 81% of asthmatic children showing a positive reaction on skin testing, compared with 39% of nonasthmatics.

Respiratory Symptoms

In total, 65% of children suffered from at least one respiratory symptom during the study. Among asthmatic children, 94% reported having at least one respiratory symptom, compared with 48% of nonasthmatic children. The proportion of children suffering from each individual respiratory symptom is included in Table 1. The median respiratory symptom score was 5.0 among asthmatic children compared with 0.0 for nonasthmatics.

Nitrogen dioxide exposure was tested for an association with respiratory symptoms using multiple logistic regression models. The continuous nitrogen dioxide variables "mean indoor exposure," "mean bedroom exposure," "summer levels," and "winter levels" were treated as having a linear relationship with the probability of respiratory symptoms, and the risk associated with an increase of 10 $\mu g/m^3$ was calculated. Odds ratios for these various measures of exposure are presented in Table 2. Whereas the presence of a gas stove significantly increased the likelihood of respiratory symptoms, neither of the other major nitrogen dioxide sources, gas heaters or smoking, were found to be significantly associated with respiratory symptoms. Results are therefore not presented. The adjusted

TABLE 1

ADJUSTED ODDS RATIOS (OR) WITH 95% CONFIDENCE INTERVALS (CI) FOR EACH INDIVIDUAL RESPIRATORY SYMPTOM WITH GAS STOVE EXPOSURE AND BEDROOM NITROGEN DIOXIDE EXPOSURE (+10 $\mu g/m^3$)

Respiratory Symptom	% of Children	Gas Stove Exposure		Bedroom NO_2	
		OR*	95% CI	OR*	95% CI
Cough	59	2.25	1.13–4.49	1.47	0.99–2.18
Shortness of breath	31	1.49	0.72–3.08	1.23	0.92–1.64
Waking short of breath	17	1.01	0.42–2.45	1.04	0.71–1.53
Wheeze	24	1.79	0.80–3.99	1.15	0.85–1.54
Asthma attacks	23	1.73	0.77–3.90	1.06	0.77–1.46
Chest tightness	13	3.11	1.07–9.05	1.12	0.81–1.56
Cough in the morning	24	1.42	0.63–3.19	1.25	0.92–1.69
Chest tightness in morning	14	1.10	0.42–2.88	1.32	0.95–1.84

* Adjusted for parental asthma, parental allergy, and sex.

TABLE 2

OR WITH 95% CI FOR RESPIRATORY SYMPTOMS WITH VARIOUS MEASURES OF NITROGEN DIOXIDE EXPOSURE (n = 148 CHILDREN)

NO_2 Exposure Measure	Crude OR	Adjusted OR*	95% CI
NO_2 indoor mean†	1.26	1.24	0.91–1.68
NO_2 winter†	1.13	1.12	0.93–1.35
NO_2 summer†	2.81	2.71	1.11–6.59
NO_2 bedroom†	1.47	1.41	0.95–2.10
$< 10\ \mu g/m^3$‡	1.00	1.00	
$10–20\ \mu g/m^3$‡	1.70	1.47	0.69–3.13
$> 20\ \mu g/m^3$‡	4.03	3.62	1.08–12.08
Gas stove	2.77	2.32	1.04–5.18
Gas stove§	2.77	2.24§	1.04–4.82

* Adjusted for parental asthma, parental allergy, and sex.
† OR for an increase by 10 $\mu g/m^3$.
‡ Bedroom nitrogen dioxide levels, coded as two dummy variables.
§ Adjusted for bedroom nitrogen dioxide levels as well as parental allergy, parental asthma, and sex.

odds ratios have been adjusted for the genetic factors, parental asthma, parental allergy, and sex. Other potential confounding factors, such as child age, passive smoking (30%), and socioeconomic status measured by house ownership (15% rented), were considered in the analysis, but neither had a significant influence on the risk estimates.

A significant increase in the risk of respiratory symptoms was seen with the presence of a gas stove, although high bedroom concentrations of nitrogen dioxide were a marginal risk factor (Table 2). On the other hand, average indoor nitrogen dioxide levels were not significantly associated with respiratory symptoms. It can also be noted that summer levels of indoor nitrogen dioxide were significantly associated with respiratory symptoms, while winter levels were not. Three nitrogen dioxide exposure categories were created from bedroom levels, and a dose–response association with respiratory symptoms is demonstrated by a higher odds ratio with higher exposure (Table 2). Further, it was found that the risk of respiratory symptoms with gas stove exposure was still significant even after adjusting for bedroom nitrogen dioxide concentrations (Table 2). This suggests that there is an additional effect of gas stove exposure on respiratory symptoms beyond average nitrogen dioxide exposure.

Gas stove exposure and bedroom nitrogen dioxide levels (continuous) were used to calculate odds ratios for each individual respiratory symptom. Adjusted odds ratios are presented in Table 1. Cough has an odds ratio significantly greater than 1.0 for gas stove exposure, and a marginally significant odds ratio for bedroom nitrogen dioxide levels. In addition, chest tightness was significantly more likely with gas stove exposure.

Respiratory symptom scores for children were compared by nitrogen dioxide exposure groups based on bedroom levels and gas stove exposure. Significantly higher respiratory symptom scores were seen for children exposed to a gas stove ($p = 0.01$, Mann-Whitney). However, the median score was 1.0 for both groups owing to the large proportion of the scores being 0 or 1.0 in both groups. The difference between groups is reflected by a mean score for children exposed to gas stoves of 2.49 compared with 1.72 for children not exposed to a gas stove. There was a marginal difference between the respiratory symptom scores for children in three nitrogen dioxide exposure groups ($p = 0.09$, Kruskal-Wallis). Again, the medians do not reflect the difference due to the skewed distribution of data, but a comparison of means for the three exposure groups shows a tendency for higher scores with higher exposure: (*1*) $< 10\ \mu g/m^3$, 1.78; (*2*) $10–20\ \mu g/m^3$, 2.15; and (*3*) $> 20\ \mu g/m^3$, 2.71.

TABLE 3

OR WITH 95% CI FOR ASTHMA WITH VARIOUS MEASURES OF NITROGEN DIOXIDE EXPOSURE (n = 148 CHILDREN)

NO_2 Exposure Measure	Crude OR	Adjusted OR*	95% CI
NO_2 bedroom†	1.03	1.01	0.75–1.37
NO_2 indoor mean†	0.99	1.00	0.75–1.31
NO_2 winter†	0.99	0.99	0.84–1.16
NO_2 summer†	1.82	2.52	0.99–6.42
Gas stove	2.11	2.23	1.06–4.72

* Adjusted for parental asthma, parental allergy, sex, and presence of pets.
† OR for an increase by 10 $\mu g/m^3$.

Asthma

Some 36% of children were diagnosed as asthmatics by a doctor. An association between asthma diagnosis and exposure to gas stoves and nitrogen dioxide was tested in a similar manner as for respiratory symptoms. Odds ratios for the associations are presented in Table 3. There is a significant association between the presence of a gas stove and asthma in children, and a marginally significant association with nitrogen dioxide summer measurements, whereas bedroom levels, indoor mean levels, and winter levels were not significantly associated with asthma.

Allergy

No direct association between atopy and nitrogen dioxide concentrations or presence of gas stoves was seen. Separate adjusted odds ratios for respiratory symptoms were calculated for atopic and nonatopic children. The results presented in Table 4 show that the odds ratios are consistently higher for atopic children, but the difference between the groups was not statistically significant ($p > 0.05$).

Pulmonary Function

Lung function, measured as PEFR, was recorded by asthmatic children (n = 53) in two seasons; winter (July) and late spring (November). Winter results showed a mean variability of 14.5%; late spring results had a mean variability of 13.3%. Multiple regression was used to test an association between nitrogen dioxide concentrations and peak flow variability in winter. Similarly, the association between nitrogen dioxide exposure in early spring (September) and peak flow variability in late spring (November) was tested (no nitrogen dioxide data were available for late spring). For winter data, a significant but weak association was present ($r = 0.31$, $p = 0.03$), whereas there was a trend to an association in spring ($r = 0.25$, $p = 0.08$). After adjusting for passive smoking, peak flow vari-

TABLE 4

OR WITH 95% CI FOR RESPIRATORY SYMPTOMS IN ATOPIC AND NONATOPIC CHILDREN WITH EXPOSURE TO GAS STOVES AND NITROGEN DIOXIDE

	Nonatopic Children (*n = 57*)		Atopic Children (*n = 88*)	
Exposure Measure	OR*	95% CI	OR*	95% CI
Gas stove	2.08	0.67–6.48	2.58	0.91–7.34
Bedroom NO_2, $+10\ \mu g/m^3$	1.09	0.60–1.97	2.08	0.88–4.90
Summer NO_2, $+10\ \mu g/m^3$	1.37	0.74–2.53	3.48	0.89–13.58

* Adjusted for parental allergy, parental asthma, and sex.

ability was no longer significantly associated with nitrogen dioxide exposure in multiple linear regression models. Gas stove presence was not associated with peak flow variability in either month ($p > 0.35$).

DISCUSSION

In the current analysis, the risk of gas stove exposure for respiratory symptoms remains significant even after adjusting for nitrogen dioxide levels (Table 2). This implies that the risk associated with gas stove exposure can not be fully accounted for by average nitrogen dioxide measurements. A possible reason for the additional increase in the risk of respiratory symptoms due to gas stove exposure is the high peak concentration of nitrogen dioxide known to occur during gas stove use. Exposure to such peak levels may be more detrimental to respiratory health than cumulative exposure levels (1), and they are not always well characterized by average nitrogen dioxide measurements (18). If these short-term exposures are important factors for respiratory health, it is possible that a failure to measure such exposures can explain much of the inconsistency in the data published to date. Therefore, it is strongly recommended that short-term measurements of nitrogen dioxide concentrations are included in future epidemiological studies studying the relationship between exposure to nitrogen dioxide and respiratory symptoms. Passive monitors with a detection limit of 35 ppb ($\sim$ 70 $\mu g/m^3$) for a sampling period of 2 h can be achieved by slightly modifying the traditional passive monitors to increase sensitivity (19).

Nitrogen dioxide concentrations in the study houses were low, with a total of 10 samples exceeding 60 ppb (115 $\mu g/m^3$), which is an ambient 24-h guideline in Victoria, Australia (State Environment Protection Policy, 1981) (20). A similar ambient standard is set by the U.S. Environmental Protection Agency at 100 $\mu g/m^3$. Despite the low exposure levels, indoor nitrogen dioxide was found to be a significant risk factor for respiratory symptoms in children. An even greater risk of symptoms was associated with gas stove exposure.

A 41% increase in the risk of respiratory symptoms was seen with an increase in bedroom nitrogen dioxide levels by 10 $\mu g/m^3$ (Table 2). Although this risk was only marginally significant, the apparent dose–response association between bedroom levels and respiratory symptoms supports an important association. Similarly, the odds ratio for respiratory symptoms with exposure to a gas stove was 2.32. Previous studies reporting results related to the association between nitrogen dioxide exposure or gas stove exposure and respiratory symptoms in children have been reviewed (21). From a meta-analysis of 11 studies the authors conclude that the risk of respiratory symptoms increases by at least 20% with exposure to a gas stove. If only data from studies measuring nitrogen dioxide levels were used, a higher estimate of 27% was gained. The authors of this study point out that the calculated estimate is likely to be an underestimate because of problems with misclassification of exposure and health outcomes in the original studies. More recent studies have indeed reported higher odds ratios, similar to those in the present study for gas stove exposure. Jarvis and coworkers (22) reported odds ratios ranging from 1.62 to 2.60 for various respiratory symptoms in young adult women using gas for cooking. Infante-Rivard (6) reported an odds ratio of 2.51 for asthma in children with a personal exposure to about 20–30 $\mu g/m^3$ nitrogen dioxide. Thus, it is likely that the true increase in the risk of respiratory symptoms with gas stove exposure is greater than 27%.

Respiratory symptoms scores were calculated as a measure of the severity of the respiratory symptoms in children. Significantly higher scores were evident with gas stove exposure. On the other hand, no significant difference in scores between nitrogen dioxide groups was seen, but a suggestion of a dose–response association was present. It is speculated that more severe respiratory symptoms may result from higher nitrogen dioxide exposure in children, in addition to the increased risk of any respiratory symptom.

Great differences in the results obtained from monitoring nitrogen dioxide concentrations in different seasons can occur as seen in Tables 2 and 3. It is likely that seasonal factors have been responsible for some of the variation in results from previous epidemiological studies where nitrogen dioxide has usually only been measured in one season.

Recent experimental studies (3, 23) suggest an interaction between nitrogen dioxide exposure and indoor allergens. Accordingly, allergic subjects would be expected to react to lower nitrogen dioxide exposure levels than nonallergic subjects, which was also suggested to be the case in a recent study of young women (20). Although the difference was not significant, it does suggest that the possible interaction implied from controlled clinical trials is present in real-life situations. The present study of children shows similar results, but again the difference was not statistically significant (Table 4). The importance of an interaction between nitrogen dioxide exposure and atopy warrants further investigation with a larger sample size to clarify the association.

There are previous reports of a reduced pulmonary function in children exposed to nitrogen dioxide in their home. Hosein and coworkers (24) reported a significantly lower forced expiratory volume (FEV_1) in children exposed to a gas stove, while Jarvis and coworkers (22) reported a small reduction in FEV_1 in women using gas for cooking. In another study, young adult asthmatics had reduced PEFR with gas stove use (25) suggesting an effect in sensitive subjects. In the present study, a weak association between nitrogen dioxide levels and PEFR variability in children was seen, but when adjusted for passive smoking, the association was no longer significant. No association with gas stove exposure was evident. The likely reason for the lack of a significant association in the present study is a small sample size ($n = 53$). In addition, nitrogen dioxide measurements were only available for one of two PEFR measurement periods, making misclassification probable.

In conclusion, the present study shows a significant adverse effect of gas stove exposure on respiratory health in children. It is suggested that peak nitrogen dioxide exposure is the critical factor in this association, and measurements of such exposure in epidemiology will be necessary to clarify the association. Meanwhile, these results suggest that alternative methods of cooking should be used by families with young children, particularly children with asthma. Appropriate ventilation of all indoor combustion appliances, including gas stoves, is strongly recommended.

***Acknowledgment*:** The participating households are thanked for their valuable contribution to the study. Gippsland Pathology Service generously performed the skin prick tests, and Associate Professor Philip Rayment provided statistical advice.

References

1. Samet, J. M., and M. J. Utell. 1990. The risk of nitrogen dioxide: what have we learned from epidemiological studies? *Toxicol. Ind. Health* 6: 247–262.
2. McKee, D. J., and R. M. Rodriguez. 1991. Health effects associated with ozone and nitrogen dioxide exposure. *Water Air Soil Pollut.* 67:11–35.
3. Devalia, J. L., C. Rusznak, M. J. Herdman, C. J. Trigg, H. Tarraf, and R. J. Davies. 1994. Effect of nitrogen dioxide and sulphur dioxide on airway response of mild asthmatic patients to allergen inhalation. *Lancet*

344:1668–1671.
4. Melia, R. J. W., C. du V. Florey, and S. Chinn. 1979. The relation between respiratory illness in primary school children and the use of gas for cooking: I. Results from a national survey. *Int. J. Epidemiol.* 8:333–338.
5. Neas, L. M., D. W. Dockery, J. H. Ware, J. D. Spengler, F. E. Speizer, and B. G. Ferris. 1991. Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *Am. J. Epidemiol.* 134:204–219.
6. Infante-Rivard, C. 1993. Childhood asthma and indoor environmental risk factors. *Am. J. Epidemiol.* 137:834–844.
7. Keller, M. D., R. R. Lanese, R. I. Mitchell, and R. W. Cote. 1979. Respiratory illness in households using gas and electricity for cooking: I. Survey of incidence. *Environ. Res.* 19:495–503.
8. Koo, L. C., J. H.-C. Ho, C.-Y. Ho, H. Matsuki, H. Shimizu, T. Mori, and S. Tomigana. 1990. Personal exposure to nitrogen dioxide and its association with respiratory illness in Hong Kong. *Am. Rev. Respir. Dis.* 141:1119–1126.
9. Samet, J. M., W. E. Lambert, B. J. Skipper, A. H. Cushing, W. C. Hunt, S. A. Young, L. C. McLaren, M. Schwab, and J. D. Spengler. 1993. Nitrogen dioxide and respiratory illness in infants. *Am. Rev. Respir. Dis.* 148:1258–1265.
10. Spengler, J., L. Neas, S. Nakai, D. Dockery, F. Speizer, J. Ware, and M. Raizenne. 1994. Respiratory symptoms and housing characteristics. *Indoor Air* 4:72–82.
11. Garrett, M. H., B. M. Hooper, R. P. Strasser, M. J. Abramson, and M. A. Hooper. 1995. Home environment and asthma—research methodology. *In* L. Morawska, N. D. Bofinger, and M. Maroni, editors. Indoor Air—An Integrated Approach. Elsevier Science Ltd., Exeter, UK. 219–222.
12. Palmes, E. D., A. F. Gunnison, J. DiMattio, and C. Tomczyk. 1976. Personal sampler for nitrogen dioxide. *Am. Ind. Hyg. Assoc. J.* 37:570–577.
13. Garrett, M. H., M. A. Hooper, and B. M. Hooper. 1998. Nitrogen dioxide in Australian homes: levels and sources. *J. Air Waste Manage. Assoc.* (In press)
14. Abramson, M., J. Kutin, and G. Bowes. 1992. The prevalence of asthma in Victorian adults. *Aust. N.Z. J. Med.* 22:358–363.
15. Meinert, R., T. Frischer, W. Karamaus, and J. Kuehr. 1994. Influence of skin prick test criteria on estimation of prevalence and incidence of allergic sensitization in children. *Allergy* 49:526–532.
16. Woolcock, A. J., A. R. Rubinfield, J. P. Seale, L. L. Landau, R. Antic, C. Mitchell, H. H. Rea, and P. Zimmerman. 1989. Asthma management plan, 1989. *Med. J. Aust.* 151:650–653.
17. Hosmer, D. W., and S. Lemeshow. 1989. Applied Logistic Regression. John Wiley & Sons, New York.
18. Brunekreef, B., D. Houthuijs, L. Dijkstra, and S. M. Boleij. 1990. Indoor nitrogen dioxide exposure and children's pulmonary function. *J. Air Waste Manage. Assoc.* 40:1252–1256.
19. Standards Australia. 1990. Methods for sampling and analysis of indoor air. Method 1.2: Determination of nitrogen dioxide—spectrophotometric method—treated filter/passive badge sampling procedure. Australian Standard 23656.1.2-1990.
20. State Environment Protection Policy (Air Environment). 1981. Victorian Government Gazette, Melbourne, No. 63, July 13, 1981, Clause 5.
21. Hasselblad, V., D. M. Eddy, and D. J. Kotchmar. 1992. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J. Air Waste Manage. Assoc.* 42:662–671.
22. Jarvis, D., S. Chinn, C. Luczynska, and P. Burney. 1996. Association of respiratory symptoms and lung function in young adults with use of domestic gas appliances. *Lancet* 347:426–431.
23. Tunnicliffe, W. S., P. S. Burge, and J. G. Ayres. 1994. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 344:1733–1736.
24. Hosein, H. R., P. Corey, and J. McD. Robertson. 1989. The effect of domestic factors on respiratory symptoms and FEV_1. *Int. J. Epidemiol.* 18:390–396.
25. Lebowitz, M. D., C. J. Holberg, B. Boyer, and C. Hayes. 1985. Respiratory symptoms and peak flow associated with indoor and outdoor air pollution in the Southwest. *J.A.P.C.A.* 35:1154–1158.