Journal of Epidemiology and Community Health 1993; **47**: 282–286

Increased wheeze but not bronchial hyper-reactivity near power stations

J A Halliday, R L Henry, R G Hankin, M J Hensley

Department of Paediatrics and Epidemiology, University of Newcastle, 2308 NSW Australia
J A Halliday
R L Henry
M J Hensley
Department of Paediatrics John Hunter Hospital, New Lambton Heights, 2305 NSW Australia
R G Hankin

Correspondence to:
Professor R Henry
Department of Paediatrics,
University of Newcastle,
John Hunter Hospital,
Locked Bag 1, Newcastle
Mail Centre, 2310 NSW
Australia

Accepted for publication October 1992

Abstract

***Study Objective*—In a previous study a higher than expected prevalence of asthma was found in Lake Munmorah, a coastal town near two power stations, compared with another coastal control town. This study aimed to compare atopy, bronchial hyper-reactivity, and reported symptoms of asthma in the power station town and a second control area with greater socioeconomic similarity.**

***Design*—A cross sectional survey was undertaken.**

***Settings*—Lake Munmorah, a coastal town near two power stations, and Dungog, a country town in the Hunter Valley, NSW, Australia**

***Participants*—All children attending kindergarten to year 6 at all schools in the two towns were invited to participate in 1990. The response rates for the questionnaire for reported symptoms and associated demographic data were 92% in Lake Munmorah and 93% in Dungog, with 84% and 90% of children respectively being measured for lung function, atopy, and bronchial reactivity. There were 419 boys and 432 girls aged 5 to 12 years.**

***Measurements and main results*—Main outcome measures were current wheeze and bronchial hyper-reactivity, defined as a fall in forced expiratory volume in 1 second (FEV_1) or peak expiratory flow (PEF) of 20% or more. Current wheeze was reported in 24·8% of the Lake Munmorah children compared with 14·6% of the Dungog children. Bronchial hyper-reactivity was similar for both groups—25·2% in Lake Munmorah and 22·3% in Dungog. The mean baseline FEV_1 was lower in Lake Munmorah than in Dungog ($p<0·001$). Dungog children were more likely to have positive skin test to house dust mite (Dungog 27·0%, Lake Munmorah 20·2%, $p=0·028$) but there were no other differences in skin test atopy in the two towns. After adjusting for age, gender, any smoker in the house, and positive dust mite skin test, the odds of current wheeze in Lake Munmorah compared with Dungog was 2·16 (95% confidence interval 1·45, 3·15).**

***Conclusions*—Baseline lung function was lower and reported symptoms of asthma were higher in the power station town, but bronchial hyper-reactivity and skin test defined atopy were similar in the two communities. These results are consistent with the previous study and confirm the increased presence of reported symptomatic illness in the town near power stations.**

J Epidemiol Community Health 1993; **47**: 282–286

A number of environmental risk factors have been reported for childhood asthma. Both outdoor and indoor air pollution, including parental smoking and exposure to house dust mite allergen, have been associated with childhood asthma.[1–15] One potential risk factor for asthma is emission from power stations. There is controversy in the published reports about the occurrence of asthma in the vicinity of power stations. In 1986 and 1987 we carried out cross sectional and longitudinal studies to investigate whether there was more asthma in the town of Lake Munmorah which is situated within 4 km of two coal fired power stations. We found that the prevalence of childhood asthma in Lake Munmorah was approximately twice that of a control area, Nelson Bay.[16] In the longitudinal study of daily symptoms and daily air pollutant levels at Lake Munmorah, however, no relationship between pollution levels and symptoms was shown.[17] Unfortunately, there were some aspects of these studies which indicated the need for further examination of the problem: a low response rate (72% of eligible children), substantial socioeconomic differences between the two communities, and the possibility of parental over-reporting of symptoms at Lake Munmorah might have explained some of the observed difference in prevalence of asthma.

Given the unexplained differences in asthma symptoms between Lake Munmorah and the control area, a further study was planned to compare Lake Munmorah with a second control area. The research question was whether there really was a higher prevalence of asthma in Lake Munmorah, and if so, why.

Methods

Cross sectional surveys of asthma were performed in primary school children from Lake Munmorah and the control area, Dungog, an inland Hunter Valley community free of industrial sources of pollution and about the same distance (40 km) from Newcastle as Lake Munmorah. Dungog was chosen on the basis of data from the Australian Bureau of Statistics which suggested that Lake Munmorah and Dungog were communities with similar socioeconomic status.

All children attending kindergarten to year 6 at both state and Catholic schools in the two towns were invited to participate in this study. In October 1990 there were 487 primary and infant school children in Lake Munmorah and 434 in Dungog.

A questionnaire was distributed to parents to obtain demographic data and information about symptoms of asthma, atopy, medication use, family history of asthma and atopy, smoking in the home, and birth weight. Additional questions about abdominal pain and headache were asked to assess levels of general symptoms and to find out if headaches and asthma were associated. The father's occupation, was coded between 1 and 7 from a schedule in *Status and Prestige in Australia* by A A Congalton,[18] and grouped into four categories: professional/technical (Congalton scale 1 to 4), trades (Congalton scale 5), manual (Congalton scale 6 and 7), and pensioner/home duties/ unemployed.

Measurement of lung function, bronchial reactivity, and skin test atopy was performed at the schools using standard techniques.[19 20] Bronchial reactivity was measured by histamine challenge—a cumulative dose of histamine from 0·03 μmol (dose 1) to 7·8 μmol (dose 9) was given, stopping when the forced expiratory volume in 1 second (FEV_1) fell by 20% or more. Bronchial hyper-reactivity was considered to be present if there was a fall of 20% or more in FEV_1 from the baseline post-saline level during the histamine challenge. Peak expiratory flow (PEF) was used instead of FEV_1 to define bronchial hyper-reactivity in the younger children who were unable to perform spirometry. Baseline measures of PEF and FEV_1 were standardised for height and gender by expressing the measured value as a percentage of the predicted value calculated by the method of Cotes.[21] A continuous measure of bronchial reactivity, the dose-response slope, was also calculated for those children with FEV_1 measurements for the histamine challenge.[22] The dose-response slope is the percentage change in FEV_1 from baseline to the last dose divided by the total dose of histamine. It was transformed logarithmically after adding 3% per μmol to eliminate negative and zero values. Skin test reactions to cat fur, house dust mite, rye grass, mixed grass, aspergillus, and alternaria were classified as positive if the weal was 3 mm or more in diameter. Skin test atopy was considered to be present if at least one of these allergens produced a positive result.

Table I Frequency of asthma related variables in Lake Munmorah and Dungog

Outcome	*Lake Munmorah (n=447)* (%)*	*Dungog (n=404)* (%)*	*Odds ratio (95% CI)*
Ever wheezed	39·6	23·1	2·18 (1·62, 2·94)
Attacks of breathlessness	27·5	19·6	1·56 (1·13, 2·15)
Diagnosed asthma	33·2	20·1	1·97 (1·45, 2·70)
Current wheeze	24·8	14·6	1·93 (1·36, 2·73)
Bronchial hyper-reactivity	25·2	22·3	1·18 (0·85, 1·63)
Current wheeze plus bronchial hyper-reactivity	10·2	6·5	1·62 (0·97, 2·71)

*Total numbers for bronchial hyper-reactivity measurement were 409 Lake Munmorah and 382 in Dungog

Table II Baseline lung function and bronchial reactivity in Lake Munmorah and Dungog

	Lake Munmorah	*Dungog*	*p value*
PEF (% predicted)	n=415	n=386	
Mean (SD)	94·6 (14·7)	97·3 (14·7)	0·01*
FEV_1 (% predicted)	n=291	n=301	
Mean (SD)	95·9 (13·0)	99·8 (12·7)	<0·001*
Bronchial reactivity† (log dose-response slope)	n=288	n=297	
Median	1·33	1·31	0·928‡

**t* test; †Calculated only for children with completed FEV_1 histamine challenge; ‡Rank sum test, PEF=peak expiratory flow; FEV_1=forced expiratory volume in 1 second

Approval was granted for all components of the study by the relevant local committees and by the Ethics Committee of the University of Newcastle. Written informed consent was obtained from the parents of all participating children.

The data were checked manually against the questionnaires and measurement result sheets after entry. Range and logic checks of the data and all other analyses were performed using SAS procedures. Dichotomous demographic, medical history, and family history data were compared with estimated odds ratios and 95% confidence intervals (CI) (using the method of Woolf[23]). Comparison of the two towns on variables with more than two categories was performed by cross-tabulation and the χ^2 statistic.

The means of PEF and FEV_1 in each town, expressed as the percentage predicted, were compared by the *t* test. The rank sum test was used to compare the dose-response slope measure of bronchial reactivity, since the distributions were skewed, even after log transformation.

Three dichotomous outcome measures were used:

(a) Current wheeze (wheeze in the last 12 months, as reported by the parent)
(b) Bronchial hyper-reactivity
(c) Current wheeze plus bronchial hyper-reactivity.

The comparison between Lake Munmorah and Dungog on each of these outcome variables was performed via crude odds ratios and 95% CI. Adjusted odds ratios were estimated from logistic regression models for each of the three outcome definitions. Each model started with the same explanatory variables—location (Lake Munmorah or Dungog), age, gender, atopy, father's occupation, and smoking in the home; two way interactions of location with all other explanatory variables were also checked. Separate models were run with smoking defined as 'maternal smoking' and with atopy defined as 'positive reaction to dust mite'. The best model was chosen using the log likelihood statistic to compare sub-models. Goodness of fit of the models was examined by calculating the C* statistic as described by Hosmer and Lemshow.[24]

Diagnostic statistics produced by SAS PROC LOGISTIC were used to check for influential observations.

Results

Completed questionnaires were returned by 447 (92%) primary school children from Lake Munmorah, and 404 (93%) from Dungog. There were 419 boys and 432 girls, whose ages were between 5 and 12 years. A short questionnaire which gave information on age, gender, current wheeze, cough and asthma, was returned by 18 of the 40 non-responders from Lake Munmorah and 10 of the 27 from Dungog. When these children were included as responders, the overall questionnaire response rate was 95% of all primary school children in each town. Eighty four per cent of the children completed all components of the study (questionnaire, skin tests, and lung function) in Lake Munmorah and 90% in Dungog.

Table III Prevalence of positive allergen skin test responses in Lake Munmorah and Dungog

	Lake Munmorah (n=447) (% with positive skin test)	Dungog (n=404) (% with positive skin test)	Odds ratio (95% CI)
Cat fur	5·8	7·0	0·81 (0·46, 1·43)
Dust mite	20·2	27·0	0·68 (0·49, 0·95)
Rye grass	16·6	19·5	0·82 (0·57, 1·18)
Mixed grass	16·1	17·9	0·88 (0·61, 1·27)
Aspergillus	6·7	5·5	1·25 (0·70, 2·24)
Alternaria	8·4	10·4	0·80 (0·49, 1·28)
One or more allergen tested	29·1	33·0	0·83 (0·62, 1·13)

Total numbers for some cells differ because of small numbers of missing data

Table IV Father's occupation and smoking in the home at Lake Munmorah and Dungog

	Lake Munmorah (n=444) (%)	Dungog (n=404) (%)	p value*
Father's occupation:			
Professional/technical	17·3	38·6	<0·001
Trade	22·0	18·6	
Manual	46·6	27·0	
Pensioner/home duties	14·1	15·8	
Smokers living in the home:			
No-one smokes	43·2	56·2	
One smoker	32·9	30·9	<0·001
Two or more smokers	23·9	12·9	
Cigarettes smoked in the home per day:†			
None	48·1	58·9	<0·001
1–19‡	25·5	26·8	
20–30	19·6	12·0	
>30	6·8	2·3	

*χ^2 statistic; †some smokers did not smoke at home; ‡includes three pipe smokers in Dungog

Table V Percentages with reported symptoms in Lake Munmorah and Dungog

Symptoms	Lake Munmorah (n=447) (%)	Dungog (n=404) (%)	Odds ratio (95% CI)
Bronchitis before 2 years of age	24·7	9·9	2·98 (2·04, 4·36)
Bronchiolitis before 2 years of age	24·9	9·6	3·13 (2·13, 4·60)
Episodes of pain in the abdomen	19·6	12·4	1·72 (1·18, 2·51)
Frequent headaches	23·2	21·2	1·12 (0·81, 1·56)
Other illness, eg epilepsy, appendicitis	14·6	14·7	0·99 (0·67, 1·46)
Dry cough at night	75·3	57·8	2·23 (1·67, 2·98)
Dry cough at night (apart from cold/chest infection)	39·7	23·6	2·13 (1·59, 2·87)
Chest colds	73·3	53·0	2·43 (1·83, 3·23)
Eczema	29·2	19·7	1·68 (1·22, 2·32)
Hay fever	35·0	23·2	1·79 (1·32, 2·42)
Allergies	24·1	17·3	1·51 (1·07, 2·14)

Table I shows the percentage of children with reported respiratory symptoms, as well as the crude odds ratio and 95% CIs for the odds of each outcome in Lake Munmorah compared with the odds of that outcome in Dungog. The odds of 'ever having wheezed', 'ever having attacks of breathlessness', 'asthma diagnosed by doctor', and 'current wheeze' were all statistically significantly higher in Lake Munmorah. Baseline PEF and FEV_1 were lower at Lake Munmorah than at Dungog (table II). Bronchial hyper-reactivity was similar in the two towns, as was the median log dose-response slope measure of bronchial reactivity.

The percentages of children in each area with positive reactions to allergen skin tests are listed in table III. About 30% of the children in each area had at least one positive reaction. Dungog children were more likely to have a positive reaction to house dust mite.

Dungog had been chosen as the control area on the basis of 1986 census data, with the aim of having two areas with similar socioeconomic status. In particular, Lake Munmorah and Dungog were similar for family income and occupational status of workers. Our questionnaire data, however, indicated some unexpected differences in socioeceonomic status for our samples (table IV). Smoking habits also differed in the two areas—57% of the Lake Munmorah children came from homes with at least one smoker compared with 43% in Dungog (OR 1·72, 95% CI 1·31, 2·25); 40% of mothers in Lake Munmorah were smokers compared with 24% in Dungog (OR 2·05, 95% CI 1·53, 2·75). These factors were therefore considered when calculating adjusted odds ratios.

Data on the medical history of the children are listed in table V. The Lake Munmorah group was more likely to report having had bronchitis and bronchiolitis before the age of 2 years, dry cough at night, chest colds, eczema, hay fever, allergies, and frequent episodes of abdominal pain. Twenty nine per cent of Lake Munmorah children were currently using at least one medication compared with 20% of those from Dungog (OR 1·62, 95% CI 1·18, 2·23). There were, however, no significant differences between the two groups for frequent headache or other illnesses (for example, epilepsy, appendicitis). Extra information was obtained from the children who had ever been diagnosed as having asthma (table VI). A total of 147 children (32%) in Lake Munmorah had been diagnosed as having asthma compared with 81 (20·1%) Dungog children (OR 1·97, 95% CI 1·45, 2·70). There were no significant differences between the two areas with respect to the frequency of attacks, their severity, or treatment. In addition, a similar proportion of 'children with asthma' in each group had had two or more attacks in the previous year—44% in Lake Munmorah, 47% in Dungog (OR 0·87, 95% CI 0·51, 1·151). A similar analysis of other questions about children reported as ever having wheezed showed that there was no significant difference between the two groups for 'age at which wheezing began', 'time since last wheezing', 'number of wheezing attacks in the last 12 months', or 'attacks of wheezing after exercise'. Of the children who had ever wheezed, 14% in both towns were currently using preventive asthma medication: similarly, 32% of the Lake Munmorah children and 34% of those from Dungog were currently using bronchodilators.

Similar percentages of parents reported a history of asthma, hay fever, eczema, and allergies, but more Lake Munmorah parents reported that their children had had chest disease (apart from asthma) (table VII). For siblings, the odds of asthma, eczema, and chest disease were higher at Lake Munmorah. The proportion of families where a sibling had hay fever or allergy was similar for both towns.

The results of the logistic regression analysis are shown in table VIII for each of the outcomes current wheeze, bronchial hyper-reactivity, and current wheeze plus bronchial hyper-reactivity. Location (Lake Munmorah or Dungog), atopy, any smoking in the home, age, and gender were the independent variables in the final model. Atopy was an independent risk factor for each of the three outcomes. Retention of 'any smoker in the home' was justified by a statistically significant change in the log likelihood statistic when this variable was included. Gender and age were included for standard biological reasons. Father's occupation was neither an independent risk factor, nor was its inclusion supported by improvement in

Table VI Comparison of disease in children reported to have asthma diagnosed by a doctor in Lake Munmorah and Dungog

Children with diagnosed asthma	Lake Munmorah (n=147) (33·2%) (%)	Dungog (n=81) (20.1%) (%)	p value*
Age of asthma diagnosis (y):			
≤1	20·4	20·3	0·24
1–2	19·7	13·9	
2–3	19·7	12·7	
3–4	13·4	12·7	
≥4	26·8	40·5	
Time since last attack (mth):			
<1	27·7	24·1	0·42
1–6	15·6	22·8	
6–12	12·8	16·5	
>12	44·0	36·7	
No of attacks in last year:			
0	40·3	39·5	0·75
1	14·6	13·6	
2–3	22·2	25·9	
4–12	17·4	12·4	
>12	5·6	8·6	
Now using preventive asthma medication	19·1	17·3	0·84
Now using bronchodilators (without preventive medication)	39·5	43·2	0·58
Now using any medication	67·8	66·7	0·86
Ever used any asthma medication	98·0	97·5	0·84
Started medication:			
In the last year	7·6	13·9	0·31
More than a year ago	89·7	83·5	
Never	2·8	2·5	
Frequency of medication in last 12 months			
Daily	12·5	22·2	0·30
More than once per mth	17·4	14·8	
Less than once per mth	36·8	33·3	
Never	33·3	29·6	

*χ^2 statistic

the log likelihood statistic. There were no statistically significant two way interactions for location with the other explanatory variables. The fit of all the models was improved by defining atopy as 'positive reaction to house dust mite' rather than 'positive reaction to one or more (of the six tested)', and by defining 'smoking in the home' as 'any smoker' rather than 'maternal smoking'. On the basis of the C* statistic, there was no evidence of poor fit for the models used to estimate ajusted odds ratios in table VIII.

After adjusting for age, gender, house dust mite atopy, and any smoking in the home, the odds of current wheeze in Lake Munmorah compared with the odds of current wheeze in Dungog was 2·16 (95% CI 1·45, 3·15), the odds of bronchial hyper-reactivity in Lake Munmorah compared with Dungog was 1·20 (95% CI 0·85, 1·71) and the odds of current wheeze plus bronchial hyper-reactivity was 1·96 (95% CI 1·12, 3·45). The similarity between crude and adjusted odds ratios indicates little if any confounding by the other factors in the model.

Table VII Comparison of family history of respiratory and allergic diseases in Lake Munmorah and Dungog

History	Lake Munmorah (n=447) (%)	Dungog (n=404) (%)	Odds ratio (95% CI)
Sibling ever had asthma	39·2	27·6	1·68 (1·26, 2·24)
Sibling ever had eczema	25·1	18·2	1·51 (1·08, 2·11)
Sibling ever had hay fever	17·1	17·4	0·98 (0·68, 1·40)
Sibling ever had allergies	19·6	21·4	0·90 (0·64, 1·25)
Sibling ever had chest disease	16·6	10·5	1·71 (1·14, 2·56)
Father ever had asthma	13·7	10·7	1·34 (0·87, 2·05)
Father ever had eczema	8·6	6·6	1·34 (0·79, 2·29)
Father ever had hay fever	25·2	23·7	1·09 (0·79, 1·50)
Father ever had allergies	19·2	20·8	0·91 (0·64, 1·29)
Father ever had chest disease	18·5	10·0	2·05 (1·36, 3·10)
Mother ever had asthma	14·5	18·1	0·77 (0·53, 1·11)
Mother ever had eczema	17·9	13·6	1·39 (0·94, 2·03)
Mother ever had hay fever	35·6	33·8	1·08 (0·81, 1·45)
Mother ever had allergies	27·4	30·0	0·88 (0·65, 1·20)
Mother ever had chest disease	17·8	9·0	2·18 (1·43, 3·32)

Discussion

In this cross sectional survey of the schoolchildren in two towns we found that the odds of having current asthma (defined as current wheeze or current wheeze plus bronchial hyper-reactivity) was higher in a town near two power stations than in a similar sized country town with no nearby source of industrial emissions. The interpretation of this result requires consideration of the representativeness of the study sites and participants, the validity of the measurements, and the degree to which adjustment can be made for confounding.

The presence of asthma and asthma related symptoms was very high in Lake Munmorah and, on a national scale, low in Dungog. The 1990 Australian National Asthma Campaign survey of 8753 children from four centres (Sydney, Melbourne, Brisbane, Hunter Valley), found 17% with diagnosed asthma, 20% with wheeze in the last 12 months, and 30% with history of wheezing.[25] The corresponding Lake Munmorah figures of 33%, 24%, and 40% were all higher: those for Dungog were 20%, 15%, and 23% respectively. A previous survey had also found a higher prevalence of reported symptoms in the power station town.[16] The response rates in the current study for questionnaire data were very good, 92% and 93% in the two towns, and this overcomes some of the criticism of the previous study in which rates were 76% and 70%. The response rates for lung function and skin tests were also good and it is reasonable to assume that the data are representative of the two communities.

The measurement of asthma in epidemiological studies is not standardised and hence a range of measures is necessary. Disparity between symptoms of asthma and bronchial hyper-reactivity has been reported.[26] Some children may have a history compatible with asthma but their measured airway responsiveness does not lie within the usually accepted 'asthmatic' range, while other children with no respiratory symptoms have increased responsiveness. Since asthma is a clinical syndrome, both the severity of symptoms and diagnosis by a doctor are essential but open to reporting and diagnostic bias. There would need to be substantial bias if the difference found in this study were incorrect. With respect to the factors related to asthma which could be objectively measured, mean baseline lung function was worse in Lake Munmorah, but bronchial hyper-reactivity was similar in both towns.

The possibility of over-reporting of symptoms in Lake Munmorah must be considered a possible bias. There was no difference, however, between the two groups' reporting of frequent headache and other illnesses (for example, epilepsy, appendicitis) for the children. There was also no

Table VIII Adjusted odds ratios (OR) and 95% confidence intervals (CI) estimated from logistic regression models for three outcomes—current wheeze, bronchial hyperreactivity (BHR), and current wheeze plus BHR

Factor	Current wheeze (159 cases/ 628 controls) OR (95% CI)	BHR (184 cases/ 599 controls) OR (95% CI)	Current wheeze plus BHR (65 cases/ 711 controls) OR (95% CI)
Age (y) (odds ratio for a 1 year increase in age)	0·97 (0·89, 1·06)	0·86 (0·79, 0·94)	0·87 (0·76, 1·00)
Male-female	1·38 (0·96, 1·98)	1·09 (0·77, 1·54)	1·22 (0·71, 2·12)
Place of residence Lake Munmorah/Dungog	2·16 (1·45, 3·15)	1·20 (0·85, 1·71)	1·96 (1·12, 3·45)
House dust mite atopy	2·53 (1·71, 3·74)	3·38 (2·33, 4·91)	7·79 (4·47, 13·58)
Any smoker in the home	1·02 (0·71, 1·47)	1·30 (0·92, 1·84)	1·11 (0·64, 1·91)

difference in reported asthma, hay fever, eczema, and allergies for the two groups of parents. On the other hand both parents and siblings in Lake Munmorah reported more chest disease, apart from asthma. There does not seem to be a consistent trend of over-reporting in Lake Munmorah. The possibility of bias due to different criteria for diagnosing asthma and over-representation of mild asthma in Lake Munmorah was also considered, using the data available for children with diagnosed asthma and current wheeze. There was no evidence to suggest that the severity of the reported asthma in Lake Munmorah was less that that in Dungog with respect to frequency of attacks and use of asthma medications. Therefore, at this stage, we have no evidence that diagnostic bias by medical practitioners could be responsible for the difference.

It is difficult not to accept the measured excess of wheeze and decreased lung function at Lake Munmorah is real. Possible explanations include the indoor and outdoor environment, social class, genetics, and atopy. Wheezing, asthma, chronic infections of the lower respiratory tract, and decreased FEV_1 have been found to be more frequent in children exposed to tobacco smoke at home,[13-15] and conflicting views are held about the effects of socioeconomic status on the prevalence of asthma.[12] The multivariable analysis, however, indicated that the effect of location on current wheeze persisted after adjustment for atopy, parental smoking, and father's occupation. Other factors must have been present to explain the differences in reported wheeze and baseline lung function. These could include differences in air quality and genetic factors. A previous study showed very low levels of sulphur dioxide and nitrogen oxide gas compounds in the Lake Munmorah area,[17] but more comprehensive measurements of air pollutants are now being made.

Data are currently being collected which will enable comparison of daily measurements of temperature, humidity, wind, pollens, and other airborne particulates with daily asthma symptoms and PEF of children with asthma in the two towns. Analysis of the new data may help to clarify the relationship of childhood asthma with natural and industrial environmental hazards.

The authors acknowledge the financial support of the Electricity Commission of New South Wales. We wish to thank Pauline Brown, Patricia Kiehne, Majella Maher, Patricia Marks, Jane Sedgewick and Alexandra Stone for their assistance with data collection and Joan Welsh for typing the manuscript.

1 Pope CA. Respiratory disease associated with community air pollution and a steel mill, Utah valley. *Am J Public Health* 1989; **79(5)**: 623–8.
2 Pope CA. Respiratory hospital admissions associated with PM10 pollution in Utah, Salt Lake, and Cache Valleys. *Arch Environ Health* 1991; **46(2)**: 90–7.
3 Ware JH, Ferris BG, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. *Am Rev Respir Dis* 1986; **133**: 834–42.
4 Whittemore AS, Korn EL. Asthma and air pollution in the Los Angeles area. *Am J Public Health* 1980; **70**: 687–96.
5 Zwi S, Davies JC, Becklake MR, Goldman HI, Reinach SG, Kallenbach JM. Respiratory health status of children in the eastern Transvaal highveld. *S Afr Med J* 1990; **78(11)**: 647–53.
6 Andrae S, Axelson O, Bjorksten B, Fredriksson M, Kjellman N-I M. Symptoms of bronchial hyper-reactivity and asthma in relation to environmental factors. *Arch Dis Child* 1988; **63**: 473–8.
7 Hosein HR, Corey P, Robertson J McD. The effect of domestic factors on respiratory symptoms and FEV_1. *Int J Epidemiology* 1989; **18(2)**: 390–6.
8 Daigler GE, Markello SJ, Cummings KM. The effect of indoor air pollutants on otitis media and asthma in children. *Laryngoscope* 1991; **101(3)**: 293–6.
9 Neas LM, Dockery DW, Ware JH, Spengler JD, Speizer FE, Ferris BG. Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *Am J Epidemiol* 1991; **134(2)**: 204–19.
10 Dales RE, Zwanenburg H, Burnett R, Franklin C. Respiratory health effects on home dampness and molds among Canadian children. *Am J Epidemiol* 1991; **134(2)**: 196–203.
11 Sporik R, Holgate ST, Platt-Mills TAE, Cogswell JJ. Exposure to house-dust mite allergen (Der p I) and the development of asthma in childhood. *N Eng J Med* 1990; **323(8)**: 502–7.
12 Mitchell EA, Stewart AW, Pattemore PK, Asher MI, Harrison AC, Rea HH. Socioeconomic status in childhood asthma. *Int J Epidemiology* 1989; **18(4)**: 888–90.
13 Weitzman M, Gortmaker S, Walker D, Sobol A. Maternal smoking and childhood asthma. *Paediatrics* 1990; **85(4)**: 505–11.
14 Martinez FD, Antognoni G, Macri F, Bonci E, Midulla F, De Castro G, Ronchetti R. Parental smoking enhances bronchial responsiveness in nine-year-old children. *Am Rev Respir Dis* 1988; **138**: 518–23.
15 Zmirou D, Blatier JF, Andre E, Ferley JP, Balducci F, Rossum F, Delormas P. Passive smoking respiratory risk. A quantitative synthesis of the literature. *Rev Mal Respir* 1990; **7(4)**: 361–71.
16 Henry RL, Abramson R, Adler JA, Wlodarczyk J, Hensley MJ. Asthma in the vicinity of power stations: I. A prevalence study. *Pediatr Pulmonol* 1991; **11**: 127–133.
17 Henry RL, Bridgman HA, Wlodarczyk J, Abramson R, Adler JA, Hensley MJ. Asthma in the vicinity of power stations: II. Outdoor air quality and symptoms. *Pediatr Pulmonol* 1991; **11**: 134–40.
18 Congalton AA. *Status and prestige in Australia*. Melbourne. FW Cheshire Publishing, 1969: appendix B 142–148.
19 Yan K, Salome C, Woolcock AJ. Rapid method for measurement of bronchial responsiveness. *Thorax* 1983; **38**: 760–6.
20 Van Asperen PP, Mellis CM, South RT, Simpson SJ. Allergen skin-prick testing in asthmatic children. *Med J Aust* 1980; 2: 266–8.
21 Cotes JE. *Lung function; assessment and application in medicine*. 4th ed. Oxford: Blackwell, 1979.
22 O'Connor G, Sparrow D, Taylor D, Segal M, Weiss S. Analysis of dose-response curves to methacoline. *Am Rev Respir Dis* 1987; **136**: 1412–7.
23 Kleinbaum D, Kupper L, Morgenstern H. *Epidemiological research; principles and quantitative methods*. Belmont, California: Wadsworth Inc, 1982: 299.
24 Lemeshow S, Hosmer DW. A review of goodness of fit statistics for use in the development of logistic regression models. *Am J Epidemiol* 1982; **115(1)**: 92–106.
25 Bauman A, Mitchell CA, Henry RL, Robertson CF, Abramson MJ, Comino E, Hensley MJ, Leeder SR. Asthma morbidity in Australia: an epidemiological study. *Med J Aust* 1992; **156**: 827–31.
26 Sears MR, Jones DT, Holdaway MD, Hewitt CL, Flannery EM, Herbison GP, Silva PA. Prevalence of bronchial reactivity to inhaled methacholine in New Zealand children. *Thorax* 1986; **41**: 283–9.