Changing prevalence of asthma in Australian children

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Abstract

Objective—To investigate whether prevalence of asthma in children increased in 10 years.

Design—Serial cross sectional studies of two populations of children by means of standard protocol.

Setting—Two towns in New South Wales: Belmont (coastal and humid) and Wagga Wagga (inland and dry).

Subjects—Children aged 8–10 years: 718 in Belmont and 769 in Wagga Wagga in 1982; 873 in Belmont and 795 in Wagga Wagga in 1992.

Main outcome measures—History of respiratory illness recorded by parents in self administered questionnaire; airway hyperresponsiveness by histamine inhalation test; atopy by skin prick tests; counts of house dust mites in domestic dust.

Results—Prevalence of wheeze in previous 12 months increased in Belmont, from 10.4% (75/718) in 1982 to 27.6% (240/873) in 1992 ($P < 0.001$), and in Wagga Wagga, from 15.5% (119/769) to 23.1% (183/795) ($P < 0.001$). The prevalence of airway hyperresponsiveness increased twofold in Belmont to 19.8% (173/873) ($P < 0.001$) and 1.4-fold in Wagga Wagga to 18.1% ($P < 0.05$). The prevalence of airway hyperresponsiveness increased mainly in atopic children only, but the prevalence of atopy was unchanged (about 28.5% in Belmont and about 32.5% in Wagga Wagga). Numbers of house dust mites increased 5.5-fold in Belmont and 4.5-fold in Wagga Wagga.

Conclusions—We suggest that exposure to higher allergen levels has increased airway abnormalities in atopic children or that mechanisms that protected airways of earlier generations of children have been altered by new environmental factors.

Introduction

Mortality is almost the only guide to the prevalence of severe asthma in a community. In Australia mortality from asthma in people aged under 35 years increased steadily between 1978 and 1988.¹ Although mortality from asthma has decreased since then, Australia appears to have the highest rate for young people among the countries that report causes of deaths. At the same time as the increase in asthma mortality, morbidity from asthma (as measured by hospital admissions) and symptom prevalence in children also increased.² Serial epidemiological studies show that the cumulative prevalence of wheeze increased in children between 1968 and 1990³ and in young adults between 1981 and 1990.⁴ Several studies conducted between 1982 and 1992 that used a standardised question of "wheeze in the previous year" indicate an average 1.5-fold increase in the prevalence of asthma symptoms in children over the decade.⁵ However, these studies were all based on questionnaire measurements, and

few studies have included an objective test of airway hyperresponsiveness.

The alarming increase in the reporting of symptoms of wheeze in children makes it important to ascertain whether the changes indicate a true increase in the incidence of asthma, an increasing frequency or severity of symptoms, or a decrease in the under-recognition of asthma. To examine this, we repeated studies in 1982 and 1992 in populations of children living in two different climatic regions of Australia. In these studies we made objective measurements of airway responsiveness and of sensitisation to allergens in addition to questionnaire measurements of symptom history.

Subjects and methods

POPULATION SAMPLES

In June (winter) of 1982 and 1992 we studied a random sample of children in the two study towns of Wagga Wagga and Belmont, both in New South Wales.^{6,7} Wagga Wagga is situated in a dry inland pastoral region with an average yearly rainfall of 553 mm and humidity range of 29–67%. Belmont is a coastal town with an average yearly rainfall of 1211 mm and a humidity range of 62–75%. The sampling frame was all primary schools, and schools were selected randomly to give the required numbers of children. We estimated that each sample should comprise about 800 children to detect a difference of 4% in the prevalence of airway hyperresponsiveness between regions (power 80%, significance 5%). All children aged 8–10 years at each selected school were invited to participate and were studied if the parents' informed consent was obtained.

RESPIRATORY SYMPTOMS AND INTERVIEWS

We gave a self administered questionnaire to the children's parents to measure symptom history. Standard questions for all studies were whether the child had ever had wheeze, had ever used any drug for asthma, or had ever had asthma diagnosed by a doctor. Further questions were whether the symptoms had occurred in the previous 12 months (symptoms of wheeze in the 12 months before the study were defined as recent); if the child had ever had hay fever or nasal allergy; if the child had ever had eczema; and if either parent had ever had asthma (such children were considered to have a history of parental asthma). Children whose parents reported that they had required treatment by a doctor or at a hospital for a respiratory infection before the age of two years were classified as having had an early respiratory infection.

We tested the questionnaire to show that items used in outcome definitions had a high degree of repeatability.⁷ To assess possible sampling bias, in 1992 we asked all attenders and children for whom consent for study was not obtained (non-respondents)

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if they had used any drug for asthma in the previous month.

LUNG FUNCTION AND AIRWAY RESPONSIVENESS

We administered a histamine inhalation challenge test by the rapid method.⁸ In 1982 we recorded lung function with spirometers from Vitalograph (Buckingham, United Kingdom) and read the tracings manually, while in 1992 we used rolling seal spirometers (Mijnhardt BV, Bunnik, Holland) connected to a computer (IBM-PC running Scientific and Medical software) for immediate data acquisition.

At all studies forced expiratory manoeuvres were repeated until two readings of forced expiratory volume in one second and forced vital capacity within 100 ml were obtained, of which the largest value was used in analyses. Subjects who had taken a β agonist within six hours of presenting were asked to withhold medication before returning for later testing. Histamine diphosphate was administered in doses ranging from 0.03 μ mol to 3.9 μ mol with hand held nebulisers (De Vilbiss 40 nebulisers in 1982 and De Vilbiss 45 in 1992). The test was stopped when forced expiratory volume in one second fell by 20% or more or when all histamine dose steps to 3.9 μ mol had been administered. If forced expiratory volume in one second fell by 10% or more, 200 mg of salbutamol aerosol was given to aid recovery. The fall in forced expiratory volume in one second was plotted against the logarithm of the dose administered, and, for subjects whose forced expiratory volume in one second fell by 20% or more, the dose of histamine that caused a 20% fall was estimated by interpolation. Subjects whose forced expiratory volume in one second fell by 20% or more were classified as having airway hyperresponsiveness, and subjects with both recent wheeze and airway hyperresponsiveness were classified as having current asthma because this defined the group with the most severe impairment in terms of symptoms present, drug requirements, atopy, and variability in peak flow.⁹

In order to report a response to histamine for each child, we calculated a dose-response ratio for all subjects as the percentage fall in forced expiratory volume in one second (FEV_1) at the last dose divided by total dose administered.¹⁰ We added a constant of 3 to all dose-response ratios in order to obtain a positive, normally distributed value for logarithmic conversion¹¹ so that values are indicated by the unit percentage fall $FEV_1/\mu\text{mol}+3$. Because there was a parallel shift in the cumulative frequency curve for dose-response ratios in normal subjects in 1982 compared with 1992 we added a further constant of 0.65 to the dose-response ratios for 1982. This shift was consistent with the slightly higher output of histamine with De Vilbiss 45 nebulisers.

ATOPY

Skin prick test reactions to common allergens were measured on the forearm.¹² The allergens tested were *Dermatophagoides farinae*, cat dander, ryegrass,

plantain, and *Alternaria tenuis* (Hollister-Stier). Histamine and glycerol were used as positive and negative controls. Weal size was recorded as the long axis and its perpendicular, and mean weal size was used in analyses. Weal size was recorded at 10 minutes after skin prick in 1982 and at 15 minutes in 1992. Weal size was recorded at both 10 and 15 minutes in Wagga Wagga in 1992, and it was estimated that a skin weal of 2.5 mm at 10 minutes was equal to a skin weal of 3 mm at 15 minutes. Thus, weal sizes of 2.5 mm or greater in 1982 and 3 mm or greater in 1992¹³ were regarded as positive. Subjects were considered atopic if they had a positive reaction to any of the five allergens.

DENSITIES OF HOUSE DUST MITES

Identical methods were used to collect household dust samples in 1982 and 1992. Parents of a random selection of children from each town were issued with a sealable polythene bag and asked to provide a sample of dust taken directly from their vacuum cleaner storage bag. When received, dust samples were stored in a freezer to halt further mite proliferation. Coarse material was removed before mites were separated by the flotation method.¹⁴ The mites were counted microscopically (all by the same examiner) and expressed as number per gram of fine dust.

STATISTICAL ANALYSIS

Analyses were undertaken with the statistical package SAS.¹⁵ Geometric means were calculated for dose-response ratios and numbers of house dust mites. Means and 95% confidence intervals were calculated for prevalences and for their differences between years. χ^2 Tests were used to determine the significance of differences in categorical variables between groups, and the Breslow-Day test was used to assess effect modification. Unpaired *t* tests were used to determine the significance of differences between mean values of continuous variables. When more than one comparison was being made analysis of variance, with Duncan's multiple range post-hoc test, was used to determine the significance of differences in dose-response ratios between atopy groups and age groups.

Results

In 1982 we studied 718 children (369 (51%) boys) in Belmont (consent rate 83%) and 769 children (417 (54%) boys) in Wagga Wagga (consent rate 88%), and in 1992 we studied 873 children (460 (53%) boys) in Belmont and 795 (354 (45%) boys) in Wagga Wagga (consent rate 83% in both towns). All the children were aged 8-10. In Belmont in 1992, 115 (13%) of the enrolled children and 27 (14%) of 187 non-respondents had used an asthma drug in the previous month ($P=0.5$). In Wagga Wagga in 1992, 95 (12%) of enrolled children and 21 (11%) of 189 non-respondents had used an asthma drug in the previous month ($P=0.75$).

Table I shows that the prevalence of doctor diagnosed

TABLE I—Changes in prevalence of respiratory symptoms, early respiratory infection, allergy, and parental history of asthma in children aged 8-10 in two towns between 1982 and 1992. Values are numbers (percentages) unless stated otherwise

	Belmont				Wagga Wagga			
	1982 (n=718)	1992 (n=873)	Difference (95% confidence interval)	P value	1982 (n=769)	1992 (n=795)	Difference (95% confidence interval)	P value
Asthma diagnosed	65 (9.1)	294 (37.7)	28.6 (24.8 to 32.4)	<0.001	99 (12.9)	236 (29.7)	16.8 (12.8 to 20.8)	<0.001
Use of asthma drug	69 (9.6)	247 (28.3)	18.7 (15.0 to 22.4)	<0.001	65 (8.5)	258 (32.5)	24.0 (20.2 to 27.8)	<0.001
Recent wheeze	75 (10.4)	240 (27.6)	17.2 (13.5 to 20.9)	<0.001	119 (15.5)	183 (23.1)	7.6 (3.7 to 11.5)	<0.001
No of attacks of wheeze a year:								
<4	57 (7.9)	102 (11.7)	3.8 (0.9 to 6.7)	<0.05	79 (10.3)	68 (8.6)	-1.7 (-4.6 to 1.2)	0.5
≥4	18 (2.5)	138 (15.8)	13.3 (10.6 to 16.0)	<0.001	40 (5.2)	115 (14.5)	9.3 (6.4 to 12.2)	<0.001
Early respiratory infection	153 (21.3)	141 (16.2)	-5.0 (-8.9 to -1.1)	<0.05	186 (24.2)	169 (21.3)	-1.9 (-7.1 to 1.3)	0.25
Hay fever	147 (20.5)	297 (34.0)	13.5 (9.2 to 17.8)	<0.001	223 (29.0)	343 (43.2)	14.2 (9.5 to 18.9)	<0.001
Eczema	146 (20.3)	213 (24.4)	4.1 (0 to 8.2)	0.1	173 (22.5)	254 (31.9)	7.4 (3.1 to 11.7)	<0.001
Parental asthma	129 (18.0)	236 (27.0)	9.0 (4.9 to 13.1)	<0.001	192 (25.0)	231 (29.1)	4.0 (-0.4 to 8.4)	0.1

TABLE II—Changes in prevalence of atopy and airway hyperresponsiveness in children aged 8-10 in two towns between 1982 and 1992. Values are numbers (percentages) unless stated otherwise

	Belmont				Wagga Wagga			
	1982 (n=718)	1992 (n=873)	Difference (95% confidence interval)	P value	1982 (n=769)	1992 (n=795)	Difference (95% confidence interval)	P value
Atopy	199 (27.7)	256 (29.3)	1.6 (-2.9 to 6.1)	0.1	233 (30.3)	277 (34.8)	4.5 (-0.1 to 9.1)	0.1
Airway hyperresponsiveness:	65 (9.1)	173 (19.8)	10.7 (7.3 to 14.1)	<0.001	90 (11.7)	144 (18.1)	6.4 (2.9 to 9.9)	<0.05
In non-atopic subjects	28/519 (5.4)	52/617 (8.4)	3.0 (0 to 5.9)	0.1	31/536 (5.8)	42/518 (8.1)	2.3 (-0.8 to 5.4)	0.25
In atopic subjects	37/199 (18.6)	121/256 (47.2)	28.6 (20.4 to 36.8)	<0.001	59/233 (25.3)	102/277 (36.8)	11.5 (3.5 to 19.5)	<0.01
Current asthma	32 (4.5)	105 (12.0)	7.5 (4.9 to 10.1)	<0.001	51 (6.6)	70 (9.4)	2.8 (0.1 to 5.5)	<0.05

TABLE III—Prevalence of house dust mites in samples of household dust in two towns in 1982 and 1992

	Belmont		Wagga Wagga	
	1982	1992	1982	1992
No (%) of samples with mites present	30/32 (94)	32/32 (100)	10/67 (15)	18/20 (90)
No of mites present in 1 g of sample:				
Range	0-2666	154-5144	0-120	0-1997
Geometric mean (95% confidence interval)*	224.9 (121.5 to 416.4)	1240.4 (888.1 to 1735.9)	66.7 (51.1 to 85.4)	301.2 (193.2 to 472.1)

*For samples with mites present only.

asthma, recent use of an asthma drug, and episodes of wheeze increased significantly in both regions, with the largest increases being for doctor diagnosed asthma in Belmont and recent use of an asthma drug in Wagga Wagga. The increases were larger in Belmont for all measures except recent use of an asthma drug. The increase in the rate of doctor diagnosed asthma was more than fourfold in Belmont and twofold in Wagga Wagga. The prevalence of children with fewer than four attacks of wheeze each year increased only slightly in Belmont and did not increase significantly in Wagga Wagga. However, the prevalence of children who had four or more attacks of wheeze in the previous year increased by 13% in Belmont (6.3-fold) and by 9% in Wagga Wagga (2.8-fold). The percentage of children with an early respiratory infection fell slightly in Belmont and did not change significantly in Wagga Wagga. There was a greater increase in the prevalence of hay fever (1.7-fold in Belmont and 1.5-fold in Wagga Wagga) than in the prevalence of eczema. The number of children with a parent who had had asthma increased significantly in Belmont but not in Wagga Wagga.

Table II shows the prevalence of the objective measurements of atopy and of airway hyperresponsiveness in each year together with the prevalence of current asthma, defined as the presence of both airway hyperresponsiveness and recent wheeze. The prevalence of atopy did not increase significantly, but the prevalence of airway hyperresponsiveness increased by 11% (twofold) in Belmont and by 6% (1.5-fold) in Wagga Wagga. The prevalence of current asthma increased by 8% (2.8-fold) in Belmont and by 3% (1.8-fold) in Wagga Wagga. The prevalence of airway hyperresponsiveness in non-atopic children was relatively low in both towns in both years and increased only slightly and not significantly during the study. In 1992, however, children who were atopic had significantly more airway hyperresponsiveness than in 1982. The prevalence of airway hyperresponsiveness increased by 29% (2.5-fold) in atopic children in Belmont and by 12% (1.5-fold) in atopic children in Wagga Wagga. Atopy was a significant effect modifier in the increase of airway hyperresponsiveness in Belmont ($\chi^2=7.45$, $df=1$, $P=0.006$) but not in Wagga Wagga ($\chi^2=0.33$, $df=1$, $P=0.57$).

Although the prevalence of atopy was similar in both towns, the profile of sensitisation to different allergens was different between towns and remained constant between years. More of the atopic children were sensitised to house dust mites in Belmont than in Wagga Wagga, and more were sensitised to alternaria or ryegrass in Wagga Wagga than in Belmont: in 1982,

77% (153/199) of atopic children in Belmont were sensitised to house dust mites compared with 52% (121/233) of atopic children in Wagga Wagga, while 72% (168/233) of atopic children in Wagga Wagga were sensitised to alternaria or ryegrass compared with 43% (86/199) of atopic children in Belmont. The rates of sensitisation in 1992 were similar.

The figure shows how the change in the prevalence of airway hyperresponsiveness is indicative of an increase in the severity of airway responsiveness, as measured by dose-response ratio. The curves for the non-atopic groups were symmetrical and S-shaped, indicating an approximately normal distribution of dose-response ratio in these groups. However, the curves for atopic children in 1982 were shifted towards the right, indicating skewness towards the region of severity, and the curves for atopic children in 1992

Relative cumulative frequency of dose-response ratio ((% fall in forced expiratory volume in one second)/(μ mol histamine administered) + 3) in non-atopic and atopic children living in Belmont and in Wagga Wagga. Dotted line indicates cut off point for abnormal responsiveness (calculated as 1.96 standard deviations from mean in lifelong asymptomatic children')

were skewed even further. The point at which the curves for atopic children in 1982 and 1992 depart indicates that about 70% of atopic children had more severe airway hyperresponsiveness in 1992 than did atopic children studied in 1982.

Table III shows that the numbers of house dust mites in domestic dust increased between 1982 and 1992 in both towns. In 1992 a greater percentage of the samples contained house dust mites, and in the samples with mites present there was a 5.5-fold increase in the numbers of mites in Belmont and a 4.5-fold increase in Wagga Wagga.

Discussion

The prevalence of asthma increased dramatically in two populations of Australian children living in different climatic regions. There was a large increase in the prevalence of diagnosed asthma, recent wheeze, and use of asthma drugs and a smaller but significant increase in the number of children with abnormal airway function, as measured by airway hyperresponsiveness. The finding in 1992 that the prevalence of diagnosed asthma and of use of asthma drugs was higher than the prevalence of recent wheeze suggests that there had been a substantial increase in awareness, but the increased prevalence of airway hyperresponsiveness in sensitised children indicates a real increase in the prevalence of airway abnormality in this group.

Because standard methods were used in all studies our estimates of changes in prevalence are likely to be valid. In both regions and in both years a large random sample of schools was sampled and high consent rates were achieved. In 1992 we found no bias to suggest that children with asthma were more likely to attend or refuse study. In Wagga Wagga in 1992 fewer boys attended but, because asthma is considered to be slightly more common in boys, this would have tended to reduce any increase in the prevalence of wheeze and airway hyperresponsiveness. In both years we used a self administered questionnaire that had good repeatability for measuring symptom history,^{7,16} and we maintained a core of standard and reliable questions. We used a widely accepted and repeatable measure of airway hyperresponsiveness¹¹ and a definition of current asthma that discriminated children with the greatest physiological impairment.⁹ We standardised the normal curves for dose-response ratios in 1982 to match 1992 to adjust for the small differences in nebuliser output. This adjustment decreased the differences in dose-response ratio between years.

INCREASING PREVALENCE OF SYMPTOMS AND AIRWAY HYPERRESPONSIVENESS

Many studies have documented an increased prevalence of asthma symptoms in recent years. In the United States the reported prevalence of ever having had asthma in 6-11 year old children increased from 4.8% in 1974-6 to 7.6% in 1980-4.¹⁷ Studies conducted in England and Australia in the 1960s and '70s showed a low prevalence of asthma plus wheezy bronchitis of 11-13%,^{3,18,19} but most estimates of the prevalence of wheeze measured since then have been much higher. In Australia the prevalence of recent wheeze measured in recent years has ranged from 20% to 25%.²⁰⁻²³ A similarly high prevalence of symptoms in recent years has been found in the United Kingdom^{24,25} and in New Zealand.²⁶ A serial study of Scottish children estimated that the prevalence of wheeze increased from 10.0% in 1964 to 19.8% in 1989,²⁷ although the context of the questionnaire and the disease definition was not standardised between studies so that this estimate of increase may be unreliable.

The prevalence of recent wheeze we measured in

1992 (28% in Belmont and 23% in Wagga Wagga) was slightly higher than the prevalence we found in other recent studies in Australia,²³ but it is consistent with a steadily increasing prevalence of wheeze in Australia and confirms that rates have doubled since 1982.²⁸ Despite consistent reports of an increase in symptoms, the only previous study of children that used an objective test of airway hyperresponsiveness at two time points was conducted in Wales in 1973 and 1988: this found that the prevalence of airway hyperresponsiveness induced by exercise increased only slightly and not significantly from 6.7% to 7.7%,²⁵ although the challenge protocol was not standardised.²⁹ Also the population was not classified according to atopy, so any increase of airway hyperresponsiveness in this group could not have been shown. In contrast we found about a twofold increase in airway hyperresponsiveness and current asthma, with the significant increase in airway hyperresponsiveness occurring mostly in atopic children.

An increase in the prevalence of airway hyperresponsiveness and current asthma was not found in a similar study of adults in Busselton, Western Australia, conducted at a nine year interval in the same decade.⁴ In that study the reporting of symptoms increased among young adults aged 20-30 but not among older people, and there was no increase in the prevalence of airway hyperresponsiveness at any age. Thus, the prevalence of asthma symptoms seems to have increased to a large extent in the current generation of children and to a lesser extent in young adults but not in older age groups, and the prevalence of airway hyperresponsiveness has increased in atopic children only. Increases in the prevalence of hay fever and of other symptoms closely associated with allergy, which have been reported from some studies, serve to validate the finding that allergic disease is increasing. It is interesting that hay fever is reported to be increasing in children, adolescents, and adults,^{4,30,31} while airway hyperresponsiveness does not seem to have increased in adults.⁴ We still do not have any indications of whether an increased allergen load or an increased susceptibility is largely responsible.

There is no evidence that asthma was under-recognised in the children studied in 1992. In 1982 the prevalence of diagnosed asthma and recent use of an asthma drug was lower than that of recent wheeze, but in 1992 this trend has reversed in both towns. The finding of a large increase in the prevalence of diagnosed asthma is consistent with other studies that have examined labelling patterns.³² In 1992 the prevalence of children with diagnosed asthma was extremely high at 38% in Belmont and 30% in Wagga Wagga and was higher than for recent wheeze. This suggests that there has been a large change in awareness and in labelling patterns, which may explain some of the increase in reporting of recent wheeze.

EVIDENCE FOR THE ROLE OF ENVIRONMENTAL FACTORS

The increase in airway abnormality in atopic children occurred in the relatively short period of one decade, which suggests that a change in environment is either directly or indirectly responsible. The children studied in 1982 were born between 1970 and 1972, and the children studied in 1992 were born between 1980 and 1982. Because the prevalence of atopy was very similar in these cohorts it is unlikely that a change in the prevalence of genetic susceptibility had taken place, and it seems more likely that changes in environment or lifestyle were responsible for the changes in airway abnormality. Also, cigarette smoking by adults had decreased and outdoor air quality had tended to improve over this period, suggesting that these factors were probably not relevant. It is also unlikely that an increase in severe respiratory infections in early life,

perhaps as a result of increased care facilities for infants and young children, is responsible because the prevalence of infections that required medical treatment before the age of 2 years did not increase. Although the rate of upper respiratory tract infections at the times of the studies was not measured in a standardised fashion, it is unlikely that they changed significantly in both places or that they could have had a large influence on the findings. The factors that seem most plausible, and which should be investigated in detail in future studies, are increased allergen exposure, dietary changes, treatment patterns, and any factors that are thought to influence the immunological systems of children.

Allergen exposure

Airway hyperresponsiveness in atopic children increased between 1982 and 1992, and populations of house dust mites increased in homes in both regions during this period. The methods used to measure numbers of house dust mites, although rather crude, were the same on both occasions to enable valid comparisons. Because of this, and because the same observer counted the number of mites in both years and there was no overlap in the range of these numbers between years, the finding of greatly increased numbers of house dust mites in dust was unlikely to be solely due to methodological factors and was more likely to be caused by changed climatic or indoor environmental conditions. However, no evidence for this is available, and a study in England found no increase in the levels of house dust mite allergen (*Der p I*) between 1979 and 1989, although the assay that was used was different on different occasions.³³ In Belmont, where more children were sensitised to house dust mites and where numbers of mites were higher, we found that atopy was a significant effect modifier in the increased prevalence of airway hyperresponsiveness. This was not found in Wagga Wagga, where numbers of mites were lower. This evidence, although entirely circumstantial, lends support to the suggestion that increased levels of house dust mite allergen have been involved, either directly or indirectly, in the mechanisms that have caused an increase in airway hyperresponsiveness in atopic children living in humid coastal regions of Australia.

We also have some anecdotal evidence that exposure to mould allergens may have increased in rural regions due to recent changes in agricultural methods. *Alternaria* is an opportunistic mould that proliferates on dead plant material in dry regions. New farming practices, such as increased crop production, leaving crops to die or chemical spraying (so that plant material is dead before harvesting), and leaving crop residue on the land rather than burning or ploughing it, are thought to have resulted in massive proliferation of *alternaria*. Furthermore, modern harvesters blow dust metres high, carrying *alternaria* spores into air currents that can disperse them over large distances. However, no data have been collected to verify that numbers of *alternaria* spores have increased.

Because the number of atopic children did not increase substantially in the interval between studies and the proportion of children sensitised to different allergens did not change significantly it is unlikely that the proportion of children who are genetically predisposed to become atopic has increased. However, the finding of increased airway hyperresponsiveness in towns with different prevalent allergens suggests that a ubiquitous cofactor, such as drug use or a chemical used to produce foods or goods, is primarily responsible for increased susceptibility and that higher allergen levels may have a secondary action in increasing airway hyperresponsiveness in sensitised subjects. Alternatively, it is plausible that allergen levels have increased from being just above the threshold for

Public health implications

- Reporting of symptoms of childhood asthma has increased during the past decade, but it is unclear whether this is due to a true increase in incidence of symptoms or a decrease in under-recognition of asthma
- In this study of prevalence of asthma, conducted in 1982 and 1992 with children aged 8-10, we included objective measurements of airway responsiveness and sensitisation to allergens
- Prevalence of recent wheeze increased 1.5-fold to about 25%, and prevalence of airway hyperresponsiveness increased twofold to almost 20%
- Most of the increase in airway hyperresponsiveness occurred in children sensitised to common allergens
- Increased airway abnormalities may be due to higher allergen levels (numbers of house dust mites increased fivefold during study) or due to new environmental factors

sensitisation to being well above the threshold for increased airway responsiveness.³⁴ If so, this would have been especially effective in increasing airway hyperresponsiveness if it occurred before the subjects in the 1992 cohort were born.³⁵

CONCLUSION

Our study has shown that the prevalence of atopy has not increased but that the frequency of symptoms has increased, perhaps because awareness has increased and underrecognition has decreased. Most importantly, there is strong evidence that airway abnormality has increased dramatically in children who are sensitised to common allergens. At present, research is largely centred on treating asthma, and little attention is being given to trying to prevent this disease from developing in children, although the importance of exposure to allergens, particularly those of house dust mites, is well documented.^{36,37} The challenge for the future is to elucidate the mechanisms by which immune mechanisms in children have changed and by which allergens are involved in the aetiology of childhood asthma. It seems especially important to design interventions which protect children from the early acquisition of atopy and from developing airway inflammation.

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Rates of admission to hospital for asthma

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Abstract

Objective—To describe trends in hospital admission rates for asthma in England and Wales (1976-85), the East Anglian region (from 1976 to 1991-2), and Wales (1980-90).

Design—Descriptive study.

Setting—Hospitals in England and Wales; hospitals in the East Anglian Regional Health Authority; hospitals in Wales.

Main outcome measures—Hospital admissions for asthma as principal diagnosis in England and Wales (Hospital In-patient Enquiry, 1976-85), for the East Anglian region (Hospital In-patient Enquiry, 1976-7; Hospital Activity Analysis, 1978-86; Regional Information System, 1987-8 to 1991-2), and for Wales (Hospital Activity Analysis, 1980-90).

Results—Rates for England and Wales as a whole showed a steady upward trend throughout the period examined. Rates in East Anglia, though they were similar to the national trends in the early years, showed a peak in 1985 (for males and females) with some indication of a decline in rates thereafter. Rates for Wales showed an upward trend until 1988 (for both males and females) after which they showed a decline.

Conclusions—Interpretation of the East Anglian trends is made more difficult by the change in England in 1987 of the system for the collection of hospital admission data. The fact that the rates for the East Anglian region seem to decline before this change and other considerations suggest that the observed trends, although partly reflecting the disruption of the coding during the changeover in systems, may not be entirely artefactual. The possible roles of diagnostic transfer and changes in the delivery of care, asthma treatment, admission and readmission policies, and the severity and preva-

lence of asthma in changing admission rates are considered. The changing trends in admission rates for East Anglia and Wales reflect recently published trends for mortality from asthma in England.

Introduction

There has been considerable interest in hospital admissions attributed to asthma over the past three decades. For England and Wales, Mitchell used data from the Hospital In-patient Enquiry to describe a sixfold increase in admission rates for 0-14 year olds from 1957 to 1981.¹ Similar trends have been described in the United Kingdom,^{2,4} New Zealand,⁵ Australia,⁶ and the United States.⁷ The reasons for these increases are still debated, although there is some evidence to suggest that they may be due to the transfer of cases from other categories of disease (diagnostic transfer)⁶ or to changes in medical practice.⁸ It has also been suggested that there has been a change in the severity³ or prevalence of asthma.^{9,10}

At present, data on hospital admission rates for England and Wales as a whole are available from Hospital In-patient Enquiry reports published up till 1985. The regional system on which the Hospital In-patient Enquiry was based (Hospital Activity Analysis) continued until the end of 1986, but no national summary was published for that year. The Hospital Activity Analysis data have been replaced in England and Wales after the changes introduced in NHS information systems by the Körner committee. Data (now based on the financial year) are collected locally by a regional information system and collated nationally as hospital episode statistics. Data from these statistics are so far available only for the year 1989-90.¹¹ For England and Wales as a whole, therefore, it is not possible to examine admission rates

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