Continued increase in the prevalence of asthma and atopy

S H Downs, G B Marks, R Sporik, E G Belosouva, N G Car, J K Peat

Abstract

Aims—To describe the change in the prevalence of wheeze, diagnosed asthma, and atopy in Wagga Wagga, NSW, Australia, between 1992 and 1997, and to compare this to the increase in prevalence reported between 1982 and 1992.

Methods—A cross sectional study of the prevalence of respiratory symptoms and atopy in schoolchildren aged 8–11 years (n = 1016, response rate 71%) in 1997 compared with studies of similar design in 1992 (response rate 83%, n = 850) and 1982 (response rate 88%, n = 769). Main outcome measures were respiratory symptoms measured by parent completed questionnaire and atopy measured by skin prick tests.

Results—Between 1992 and 1997, the prevalence of wheeze increased by 5.1% (95% CI 1.2 to 9.0), asthma diagnosis by 8.1% (95% CI 3.8 to 12.4), and atopy by 6.7% (95% CI 2.2 to 11.2). Similar increases in prevalence had been found between 1982 and 1992.

Conclusions—The prevalence of wheeze, asthma diagnosis, and atopy in Wagga Wagga has continued to increase. (Arch Dis Child 2001;84:20–23)

Keywords: asthma; atopy; prevalence

Serial cross sectional surveys of children and young adults in western countries have shown that the prevalence of asthma increased in the 1970s and 1980s.1 Increases in the prevalence of allergic diseases such as hay fever have also been reported,2–4 but have rarely been validated by objective measures of atopy. In Melbourne, Australia, an increase of 26.9% in the prevalence of asthma in children was measured between 1964 and 1990.6 Significant increases in the prevalence of both objective and subjective markers of asthma in primary school children were also measured between cross sectional surveys in 1982 and 1992 in the towns of Wagga Wagga and Belmont in New South Wales.7 In Wagga Wagga, parent reported “wheeze in the past 12 months” increased by 6.6% and asthma diagnosis by 17.6%. There was also a small but non-significant increase in the prevalence of atopy. In 1997, we undertook a further cross sectional study in Wagga Wagga to determine whether there had been any further change in the prevalence of wheeze, asthma diagnosis, and atopy since 1992.

Methods

POPULATIONS

The population sampled was primary school children in Wagga Wagga in 1997. The results were compared to those from similar studies in Wagga in 1992 and 1982.8 Wagga is an inland town (population approximately 55 000) in New South Wales, Australia (147°E, 35°S). The cross sectional studies were undertaken during the winters of 1982 (June), 1992 (June), and 1997 (July).

Two thirds of the schools in Wagga Wagga were randomly selected for study in 1982 and two thirds of schools for the study in 1992. Eleven of the 12 primary schools in the western half of Wagga were selected for the study in 1997. The twelfth primary school was excluded because estimated intake of pupils for school years 3, 4, and 5 was less than 20.

Approval for the studies was given by the Human Ethics Committee of the University of Sydney. Consent was obtained from the NSW Department of School Education, the Catholic Education Office, and each school principal. Parents of children from school years 3 and 4 (aged 8–10 years) in 1982 and school years 3, 4, and 5 (aged 8–11 years) in 1992 and 1997 were sent a letter describing the study and requesting consent for their child’s participation. The study includes all children whose parents gave written consent.

QUESTIONNAIRE

Parents completed questionnaires about their child. The study questionnaires were versions of the Children’s Respiratory Questionnaire developed and validated by the Institute of Respiratory Medicine, University of Sydney, PO Box M77, Missenden Road Post Office, Camperdown NSW 2050, Australia. S H Downs G B Marks R Sporik E G Belosouva

New Children’s Hospital, Westmead, NSW, Australia N G Car

School of Biomedical Sciences, Charles Sturt University, NSW, Australia

Methods

Table 1 Change in prevalence (%) of symptoms

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>1992 % (n = 769)</th>
<th>1997 % (n = 1016)</th>
<th>Percent increase (95% CI)</th>
<th>1992–97</th>
<th>1982–97</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ever wheezed</td>
<td>23.9</td>
<td>42.3</td>
<td>13.0 (8.5 to 17.5)</td>
<td>5.3 (0.1 to 9.8)</td>
<td>18.4 (14.1 to 22.6)</td>
</tr>
<tr>
<td>Wheezed in the past 12 months</td>
<td>15.3</td>
<td>44.0</td>
<td>17.6 (13.5 to 21.6)</td>
<td>8.1 (3.8 to 12.4)</td>
<td>12.9 (10.5 to 15.3)</td>
</tr>
<tr>
<td>Four or more wheezing episodes in past 12 months</td>
<td>5.2</td>
<td>16.9</td>
<td>8.5 (3.2 to 11.3)</td>
<td>11.7 (8.8 to 14.5)</td>
<td></td>
</tr>
<tr>
<td>Diagnosed asthma</td>
<td>12.9</td>
<td>38.6</td>
<td>17.6 (13.5 to 21.6)</td>
<td>8.1 (3.8 to 12.4)</td>
<td>12.9 (10.5 to 15.3)</td>
</tr>
<tr>
<td>Ever had asthma medicine</td>
<td>8.5</td>
<td>44.0</td>
<td>26.5 (22.8 to 30.3)</td>
<td>10.9 (6.4 to 15.4)</td>
<td>8.5 (6.2 to 10.5)</td>
</tr>
<tr>
<td>Has ever had hay fever or nasal allergies</td>
<td>22.5</td>
<td>44.0</td>
<td>21.2 (16.6 to 25.8)</td>
<td>−0.3 (−4.3 to 4.8)</td>
<td>21.5 (17.2 to 25.7)</td>
</tr>
<tr>
<td>Natural mother or father has had asthma</td>
<td>25.0</td>
<td>41.9</td>
<td>8.5 (3.1 to 12.0)</td>
<td>11.1 (6.6 to 15.7)</td>
<td>16.9 (12.4 to 21.4)</td>
</tr>
</tbody>
</table>

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Atopic to at least one of

<table>
<thead>
<tr>
<th>Allergen</th>
<th>1982 % (n = 769)</th>
<th>1992 % (n = 850)</th>
<th>1997 % (n = 962)</th>
<th>Percent increase (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>At least one of D. farinae, cat, rye grass, plantain, Alternaria</td>
<td>30.3</td>
<td>34.8</td>
<td>*</td>
<td>4.5 (−0.0 to 9.1)</td>
</tr>
<tr>
<td>* At least one of D. pteronyssinus, rye grass, Alternaria</td>
<td>*</td>
<td>38.7</td>
<td>45.4</td>
<td>*</td>
</tr>
<tr>
<td>Rye grass</td>
<td>*</td>
<td>18.6</td>
<td>30.3</td>
<td>*</td>
</tr>
<tr>
<td>Alternaria</td>
<td>13.0</td>
<td>15.4</td>
<td>20.4</td>
<td>2.4 (−1.0 to 5.8)</td>
</tr>
</tbody>
</table>

*No value because the concentration or source of allergens were not the same between studies.

Table 3 Change in prevalence (%) of symptoms in atopic* and non-atopic children, 1992–1997

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>1992</th>
<th>1997</th>
<th>Percent increase 1992–97 (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atopic % (n = 301)</td>
<td>Non-atopic % (n = 540)</td>
<td>Atopic % (n = 436)</td>
</tr>
<tr>
<td>Wheezed in the past 12 months</td>
<td>37.9</td>
<td>13.3</td>
<td>39.2</td>
</tr>
<tr>
<td>Diagnosed asthma</td>
<td>46.3</td>
<td>21.6</td>
<td>47.4</td>
</tr>
<tr>
<td>Ever had asthma medicine</td>
<td>49.8</td>
<td>26.8</td>
<td>56.0</td>
</tr>
</tbody>
</table>

*Atopic to at least one of D. pteronyssinus, rye grass, Alternaria.

† Proportional increase = ((1997 prevalence − 1992 prevalence)/1992 prevalence) × 100.
of parents who reported having asthma over the whole time period.

There was a significant increase in prevalence of atopy between 1992 and 1997, although the increase between 1982 to 1992 had been non-significant (see table 2). Sensitisation to Alternaria and rye grass pollen increased by 5% and 6% respectively during the 15 year time period 1982–1997 (p < 0.001 for trend).

Comparisons of the increases in prevalence of wheeze and asthma diagnosis between 1992 and 1997 between children atopic to at least one of D pteronyssinus, rye grass, and Alternaria, and in non-atopic children showed that the proportional increases were smaller for the children classified as atopic (see table 3). The same trend was also seen for medication use which increased to a greater extent in non-atopic compared to atopic children.

Discussion

We found that the trend for an increase in the prevalence of asthma and atopy reported for the decade 1982–92 continued to 1997. Importantly, this has been one of the first studies to monitor increases in the prevalence of atopy using objective measurements over a long period of time. In 1997, more than 1 in 4 children were reported to have wheezed in the previous year, almost 4 in 10 had been diagnosed as having asthma, and almost 1 in 2 children had used a medicine for asthma. These figures are surprisingly high and confirm the value of ongoing surveillance studies such as these which incorporate objective measurements to monitor trends in asthma and atopy.

The same standard methods were used to measure symptoms and atopy in the studies. It is unlikely that sample bias substantially affected our estimates of prevalence. In 1982 and 1992, the study participants were from schools selected at random throughout the town. In 1997, only children from schools in western Wagga were selected, but the comparison with the 1996 census showed that this sample was likely to be representative of children in Wagga Wagga. Eleven year old children were not included in the 1982 study, but the prevalence of outcomes in this age group were similar to those found in 8–10 year olds.

The response rates from schools were higher in 1982 and 1992 than in 1997. If parents of children with asthma were more likely to enter their child into the studies, we will have over estimated the size of the increase since 1982. The proportion of parents who had been diagnosed with asthma also increased over the time period. This is consistent with the increase in the prevalence of asthma in young adults which has been reported elsewhere, but it is possible that parents with asthma were more likely to enrol their child into the study. The true prevalence of “wheeze in the past 12 months” in 1997 could lie between the lower confidence limit, calculated on the assumption that all non-participating children did not have asthma (19.2%), and the upper limit, calculated on the assumption that all non-participating children have asthma (48.2%). However, comparison with other studies suggests that bias this extreme is unlikely. The prevalence of “wheeze in the past 12 months” in children was measured in four Australian cities in 1993–94 for the International Study of Allergies and Asthma in Childhood. The prevalence in 10 914 children aged 6–7 years and 12 280 children aged 13–14 years in Melbourne, Sydney, Adelaide, and Perth in 1993/94 was 24.6% (95% CI 23.8 to 25.5) and 29.4% (95% CI 28.6 to 30.2) respectively. These results are similar to the prevalence of wheeze in our study of 27.7%. In addition, the mean annual increase in the prevalence of wheeze in the past year was 0.8% which is within the range of annual increases reported for other comparisons of serial cross sectional surveys.

In this study we were able to standardise the methods used for measuring atopy, and comparison was restricted to allergens manufactured at the same plant to the same concentration. The concentration of D pteronyssinus has been subject to additional quality control procedures and has been licensed as a standardised product in the USA since 1988 (letter from Bayer Australia Ltd, 21/01/2000); the prevalence of sensitisation to this allergen increased by 11.7% between 1992 and 1997. Overall the most significant increase in prevalence of atopy between 1982 and 1992 and a significant increase between 1992 and 1997. In contrast, the prevalence of hay fever increased significantly between 1982 and 1992 but remained the same between 1992 and 1997. There has been one report in the peer reviewed literature of the change in prevalence of atopy measured by skin prick test between cross sectional studies. In this report of two cross sectional studies in Leipzig in former east Germany, the prevalence of atopy in children aged 9–11 years increased from 19.3% in 1991–92 to 26.7% in 1995–96 (p < 0.001). The prevalence of hay fever reported by questionnaire also increased over the time period. These increases were attributed to the introduction of a western lifestyle into Leipzig after unification.

Recent increases in the prevalence of asthma and allergic diseases have been attributed to environmental changes to lifestyle. Although we did not collect information about change in allergen exposure between 1992 and 1997, we found a 4.5-fold increase in the mean numbers of house dust mites in dust samples from homes in Wagga Wagga between 1982 and 1992. Dietary changes in western societies such as the decreased consumption of fresh fruit and vegetables and increased consumption of polyunsaturated fatty acids may affect the immune response and are another possible explanation. It is also possible that the decline in infection rates among infants in western societies has had a detrimental effect on the development of the immune system and increased the likelihood of the development of an atopic response, although it is not a cause for the increase in asthma in non-atopic children.

We found that the proportional increases in wheeze in the past 12 months and asthma...
diagnosis were greater for non-atopic than for atopic children between 1992 and 1997. Increased exposure to early childhood infections has been strongly associated with an increased risk of asthma in non-atopic children. However, there is no evidence that the incidence of early childhood infection changed in Wagga Wagga between 1992 and 1997.

In this further cross sectional study, we were able to show that the increasing prevalence of asthma and atopy that was identified in the 1980s has continued in the past decade, so that a high proportion of children now experience respiratory symptoms. This has important implications for the health of future generations of children, and continues to reinforce the message that prevention programmes are needed.

We thank the children, parents, and school staff in Wagga Wagga for enabling us to undertake the study. We also thank Robyn Paton, Margi Jones, Ann Maine, and Peter Hansen who helped us collect the data. This work was supported by the National Health and Medical Research Council, Australia and Glaxo Allen+Hanburys.

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