Archives of Disease in Childhood, 1989, **64,** 103–107

School holidays and admissions with asthma

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SUMMARY The admission rate for asthma at a children's hospital was studied over an 11 year period. Admissions varied unpredictably over periods of a few days, but there was a repeated yearly pattern of peaks and troughs with an interval of several weeks. The short term variation could be attributed to chance effects alone, excluding any important role for short term influences—for example, weather changes—in precipitating asthma admissions. There was a definite association between the longer term variation and school holidays. The admission rate fell during holidays and there were two or more peaks during terms. The pattern is consistent with a largely viral aetiology for asthmatic attacks throughout the year. We postulate that school holidays disrupt the spread of viral infections in a community, with synchronisation of subsequent attacks. Travel during holidays may facilitate acquisition of new viral strains by the community.

The number of admissions of children with asthma is seasonal,[1 2] with a large peak in autumn and a smaller peak in spring; the spring rise coincides with the hay fever season.[3 4] Before the autumn rise is an August fall,[1] which suggests a possible link with the school summer holidays. The sharp rise in September occurs after mould spore and pollen counts have plateaued,[5] but before the main winter rise in respiratory tract infections. This is probably a combined mechanism of viral infection and increased bronchial hyper-reactivity due to exposure to airborne allergens during the summer months.[6 7]

Admissions with asthma also vary considerably over periods of a few days. Epidemic days are associated with low temperature and humidity,[8] with thunderstorms or sudden rain,[9–11] and with sudden falls in temperature.[12 13] Many parents have reported associations with changes in the weather.

The aims of this study were to determine the significance, if any, of variation in asthma admissions over periods of a few days, and to look for an association between seasonal trends and timing of school holidays.

Material and methods

Date of birth, dates of admission and discharge, sex, and area code were obtained for all admissions to this hospital for the years 1975–1985. The data were obtained from the Hospital Activity Analysis using the International Classification of Diseases code 493. The data were transferred to a microcomputer and checked for accuracy against surviving hospital records. Dates of past school holidays were obtained from the local department of education, and the dates adjusted to include any adjacent weekends. The autumn half term break was treated as a holiday, but the shorter half term breaks were ignored.

To examine seasonal trends, the rate of admissions with asthma was plotted daily as a five day moving average. The association with school holidays was also examined year by year. A pattern linking admissions with asthma to school holidays was first determined by inspection, then subjected to statistical analysis. The combined data for the 11 years are shown in figs 1 and 2: the mean starting dates of holidays and terms are indicated by dotted lines, and the admissions are plotted at their correct relative position within each term or holiday period. The figures therefore show the mean daily admission rate, with the dates of all terms and holidays precisely superimposed.

To examine short term variation in the rate of admissions with asthma, the 11 year period was divided into three day segments starting on 1st January 1975. A prediction for the number of periods within which 0, 1, 2, 3, . . . asthma admissions had occurred was determined from the Poisson distribution. This was calculated separately for each month of the year to exclude any influence of longer term trends. In a similar analysis, data were stratified by area of residence into three broad groups: coast, downs, and other. This was to look for local

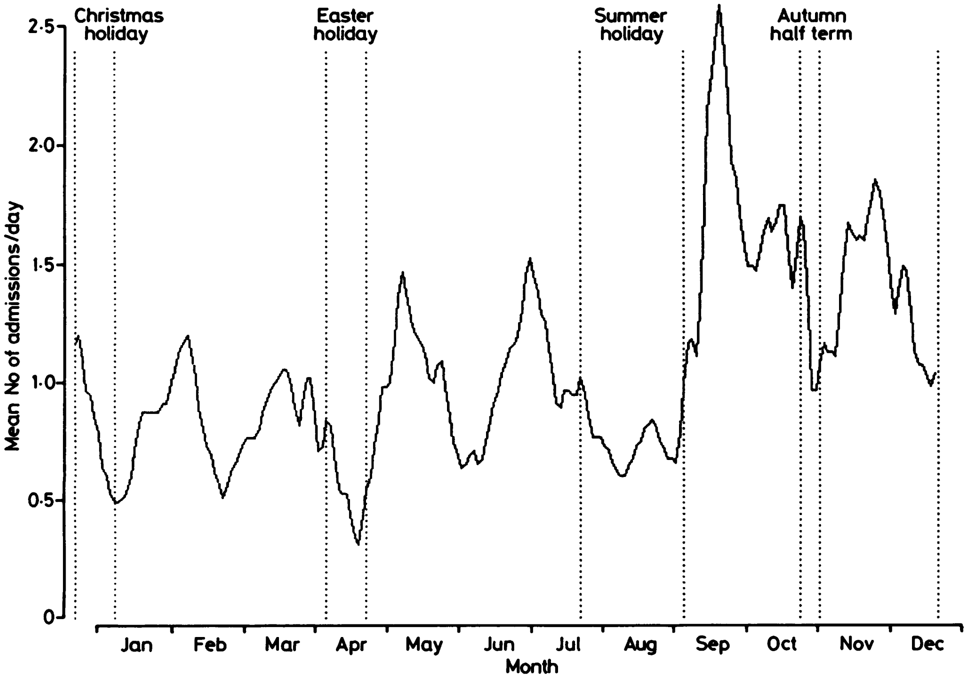

Fig 1 *Cumulative yearly trends (1975–85) in asthma admission rate: all children, 0–16 years.*

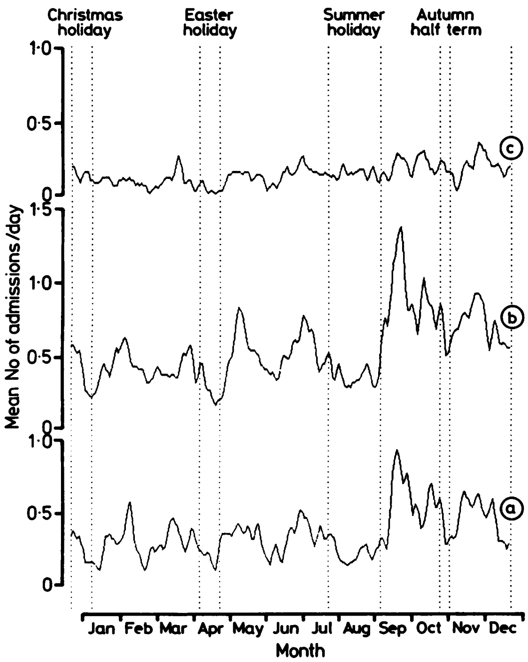

Fig 2 *Cumulative yearly trends 1975–85 in asthma admission rate: (a) 0–3 years, (b) 4–10 years, and (c) 11–16 years.*

influences that might operate in the short term such as the release of fungal spores by a sea mist, or the storm of airborne allergens created by ploughing or harvesting.

The significance of the difference between actual and predicted clustering was determined by the χ^2 test. To avoid random inflation of significance, an appropriately smaller critical p value was used for analyses by area. As there were three areas, the value chosen was one third of $p=0{\cdot}05$—that is, $p=0{\cdot}017$. Differences in rates of admission within holiday periods were assessed by the Wilcoxon signed rank test, and the suitability of a 'two peak/term' model was tested by least squares regression for longitudinal data, and by the χ^2 test for summated data.

Results

The seasonal pattern of admissions with asthma is shown in fig 1 and fig 2. There was a repeated yearly pattern that was not lost by summing the data for the 11 years. Fig 1 shows the pattern for all children, and fig 2 shows that for children in the age groups: 0–3 years (preschool), 4–10 years (primary school), and 11–16 years (secondary school). Older children contributed little to the overall pattern primarily because admissions were fewer, but the pattern is similar though less pronounced. The two younger age groups showed patterns that were remarkably similar both in shape and timing.

The pattern was of a falling admission rate during holidays ($p<0.005$) followed by two major peaks in each term ($p<<0.0001$ in each case). One or two smaller peaks commonly occurred in the autumn term. The largest peak always occurred after the summer holiday.

The distribution of clusters of admissions in three day periods is shown in the table. The distributions were not significantly different from those predicted by the Poisson distribution. Any recurring climatic or other factor operating in the short term should have produced a significant effect, even if only 5–10% of all admissions were precipitated by it. The trend towards significance was greatest in coastal areas ($p=0.04$, critical value of $p=0.017$).

Discussion

The number of admissions of children with asthma in Brighton over an 11 year period showed a repeating pattern of a decrease during school holidays and at least two major peaks during the subsequent terms. The largest peak occurred after the longest holiday. Troughs between peaks during term time corresponded roughly with half term breaks.

The most likely reason for the pattern is that most attacks of asthma in children that require admission to hospital are associated with viral respiratory tract infections. These infections do not seem to be limited to the winter months, and school holidays reduce the opportunity for the spread of viral infections in a community, which may have the effect of synchronising subsequent attacks. Holiday travel may result in the acquisition of new viral strains by the community.

Alternative explanations of the association seem less plausible. The fall in the admission rate during the summer holidays may reflect travel away from Brighton, but as a holiday resort this should be countered by a larger influx, and national data show identical falls.[2 3] One of the commonest allergens, the house dust mite, is more likely to be encountered in the home than at school, and an allergic factor should also exert a greater effect on older children, a larger proportion of whom are atopic.[14] Stress is an important precipitator of asthmatic attacks, but it is not clear why the stress of returning to school should cause attacks one or two weeks later, or why admission rates do not drop sharply on the first day of each holiday. It is equally difficult to

Table *Comparison of clustering in three day periods of asthma admissions, 1975 to 1985, with clustering predicted by the Poisson distribution*

No in three day period	*No of actual and predicted occasions on which a group of particular size occurred*							
	Total		*Coast*		*Downs*		*Other*	
	Actual	*Predicted*	*Actual*	*Predicted*	*Actual*	*Predicted*	*Actual*	*Predicted*
0	186	161	344	321	606	576	1086	1084
1	272	257	392	372	400	426	216	221
2	258	256	259	276	189	206	33	30
3	200	209	141	172	91	84	2	3
4	136	155	82	97	33	31	1	0
5	82	108	60	52	8	10		
6	60	73	22	26	7	3		
7	40	47	12	13	2	1		
8	44	30	9	6	2	0		
9	18	19	12	2				
10	14	11	2	1				
11	6	6	1	0				
12	7	4	1	0				
13	5	2	0	0				
14	5	1	1	0				
15	2	0						
16	2	0						
17	0	0						
18	1	0						
χ^2 (df)	20·9 (13)		17·5 (9)		6·14 (6)		0·19 (2)	
p Value	0·075		0·04		0·4		0·9	
Critical p value	0·05		0·017		0·017		0·017	

see how stress could affect children too young to go to school. A further possibility could be that drugs are taken more regularly during school holidays. Again, this seems an unlikely explanation in preschool children, most of whom are unable to use effective prophylactic drugs. Viral infections, however, are readily transmitted between children during term time, and to preschool children by their elder siblings.

Many studies have shown that viral infection is an important precipitator of asthma in childhood. A community study[15] found that wheezing was a common symptom, predominantly associated with signs of viral upper respiratory tract infection, and with a seasonal pattern identical to that of viral infection in children who did not wheeze. Wheezing is most common in preschool children, but there is an increased incidence at the age of starting school.[16] Viruses have been isolated from 42% of children aged 1–5 with wheezing attacks.[17] Viral infections are not limited to the winter months,[18] and children may be remarkably well between attacks.[17] In some children the wheezing may be a result of sensitisation to the virus by an earlier infection.[19] It is possible that many infections that initiate an attack of wheezing are aborted early and therefore not readily detected.

Our study does not provide evidence that wheezing is a short term response to environmental factors; the only suggestion of abnormal clustering was among children from the coastal region (table). Although this did not reach the set level of significance, it is likely to have been a real effect. As most of the excessive clustering occurred at times of expected high incidence, rapid spread of viral infections in the larger coastal communities remains the most likely explanation.

Any pronounced effect of variables such as changes in the weather should have been readily detectable. Our results are compatible with the findings of Khan, however, who showed that such factors seem to play only a minor part in the aetiology of attacks (5–15% of total variance).[20] More recently, Carey and Cordon suggested that clusters of admissions for asthma are associated with wind direction.[21] Their statistical analysis was comparatively unsophisticated, however, and the association that they reported is meaningless because any two factors that undergo seasonal variation must have a statistical relationship. The potential importance of temporal variation to asthma research is a point worth emphasising.

Most studies of airborne allergens have shown a poor correlation with asthma attacks.[12 20 22–24] Matters have been complicated by the finding that aerosols laden with allergens, with greater ability to penetrate small airways, may only be produced in particular weather conditions, particularly during periods of rainfall.[25] Gross environmental pollution with organic dusts may cause sudden increases in admissions for asthma.[26–28] It seems likely, however, that variations in concentrations of allergens in the atmosphere do not usually produce sufficient day to day variation in asthma to precipitate hospital admissions.

Allergens, particularly the house dust mite, are of considerable importance in asthma. Exposure to allergens produces chronic inflammation of the airways and an increase in bronchial hyperirritability.[29] Not all asthmatic children with viral infections are admitted to hospital, and the chance of this happening may be determined by the degree of pre-existing bronchial hyper-reactivity. Seasonal variations in exposure to allergens may thus contribute to the underlying seasonal variations in asthma. This mechanism is consistent with the otherwise paradoxical finding that in August, a time of high exposure to allergens, asthma admissions are low,[3] whereas deaths from asthma are high.[6]

In conclusion, this retrospective study provides circumstantial evidence that admissions for asthma in children are mainly precipitated by viral infections, and that short term environmental factors seem to be comparatively unimportant.

We thank Douglas Blackmore and Linda Wadey for their help in arranging transfer of the data. JS was supported by a grant from the Royal Alexandra Hospital Centenary Appeal Fund.

References

1 Khot A, Evans N, Lenney W. Seasonal trends in childhood asthma in south east England. *Br Med J* 1983;**287**:1257–8.
2 Ashley JSA. Seasonal trends in childhood asthma. *Br Med J* 1983;**287**:1721.
3 Khot A, Burn R, Evans N, Lenney C, Lenney W. Seasonal variation and time trends in childhood asthma in England and Wales 1975–81. *Br Med J* 1984;**289**:235–7.
4 Sotomayor H, Badier M, Vervloet D, Orehek J. Seasonal increase of carbachol airway responsiveness in patients allergic to grass pollen. Reversal by corticosteroids. *Am Rev Respir Dis* 1984;**130**:56–8.
5 McDonald MS, O'Driscoll BJ. Aerobiological studies based in Galway. A comparison of pollen and spore counts over two seasons of widely differing weather conditions. *Clin Allergy* 1980;**10**:211–5.
6 Khot A, Burn R. Seasonal variation and time trends of deaths from asthma in England and Wales 1960–82. *Br Med J* 1984;**289**:233–4.
7 Carlsen KH, Ørstavik I, Leegaard J, Høeg H. Respiratory virus infections and aeroallergens in acute bronchial asthma. *Arch Dis Child* 1984;**59**:310–5.
8 Salvaggio J, Hasselblad V, Seabury J, Heiderscheit LT. New Orleans asthma. II. Relationship of climatologic and seasonal factors to outbreaks. *Journal of Allergy* 1970;**45**:257–65.
9 Packe GE, Ayres JG. Asthma outbreak during a thunderstorm. *Lancet* 1985;**ii**:199–204.

[10] Salvaggio J, Seabury J, Schoenhardt EA. New Orleans asthma. V. Relationship between Charity Hospital admission rates, semiquantitative pollen and fungal spore counts, and total particulate aerometric sampling data. *J Allergy Clin Immunol* 1971;**48:**96–114.

[11] Anonymous. Asthma and the weather. *Lancet* 1985;**i:**1079–80.

[12] Tromp SW. Influence of weather and climate on asthma and bronchitis. *Review of Allergy* 1968;**22:**1027–44.

[13] Greenburg L, Field F, Reed JI, Erhardt CL. Asthma and temperature change. *Arch Environ Health* 1964;**8:**642–7.

[14] Blair H. Natural history of childhood asthma. 20 Year follow-up. *Arch Dis Child* 1977;**52:**613–9.

[15] Goodall JF. The natural history of common respiratory infection and some principles in its management. III. Wheezy children. *Journal of the College of General Practitioners* 1958;**1:**51–9.

[16] Dodge RR, Burrows B. The prevalence and incidence of asthma and asthma-like symptoms in a general population sample. *Am Rev Respir Dis* 1980;**122:**567–75.

[17] McIntosh K, Ellis EF, Hoffman LS, Lybass TG, Eller JJ, Fulginiti VA. The association of viral and bacterial respiratory infections with exacerbations of wheezing in young children. *J Pediatr* 1973;**82:**578–90.

[18] Mitchell I, Inglis JM, Simpson H. Viral infection as a precipitant of wheeze in children. *Arch Dis Child* 1978;**53:**106–11.

[19] Gardner PS, McQuillin J, Court SDM. Speculation on pathogenesis in death from respiratory syncytial virus. *Br Med J* 1970;**i:**327–30.

[20] Khan AU. The role of air pollution and weather changes in childhood asthma. *Ann Allergy* 1977;**39:**397–400.

[21] Carey MJ, Cordon I. Asthma and climatic conditions: experience from Bermuda, an isolated island community. *Br Med J* 1986;**293:**843–4.

[22] Greenburg L, Field F, Reed JI, Erhardt CL. Asthma and temperature change. II. 1964 and 1965 epidemiological studies of emergency clinic visits for asthma in three large New York City hospitals. *Arch Environ Health* 1966;**12:**561–3.

[23] Girsh LS, Shubin E, Dick C, Schlulaner FA. A study on the epidemiology of asthma in children in Philadelphia. *Journal of Allergy* 1967;**39:**347–57.

[24] Hobday JD, Stewart AJ. The relationship between daily asthma attendance, weather parameters, spore count and pollen count. *Aust NZ J Med* 1973;**3:**552–6.

[25] Hussain R, Norman PS, Marsh DG. Rapidly released allergens from short ragweed pollen. II. Identification and partial purification. *J Allergy Clin Immunol* 1981;**67:**217–22.

[26] Ordman D. An outbreak of bronchial asthma in South Africa affecting more than 200 persons caused by castor bean dust from an oil processing factory. *Int Arch Allergy Appl Immunol* 1955;**7:**10–24.

[27] Antó JM, Sunyer J. A point-source asthma outbreak. *Lancet* 1986;**i:**900–3.

[28] Anonymous. Soy kills in Barcelona. *New Scientist* 1987;**Sept 17:**32.

[29] Cockcroft DW, Ruffin RE, Dolovich J, Hargreave FE. Allergen-induced increase in non-allergic bronchial reactivity. *Clin Allergy* 1977;**7:**503–13.

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Accepted 18 July 1988