CHILDHOOD ASTHMA IN BRISBANE:
EPIDEMIOLOGICAL OBSERVATIONS

E. H. DERRICK

From the Asthma Foundation of Queensland and the Queensland Institute of Medical Research

SYNOPSIS

Analysis of hospital admissions for 20 years suggests that there has been an increase in childhood asthma in Brisbane. The characteristic seasonal pattern of asthma with waves in autumn and spring is evident from the second year of age and continues into adult life. It has not been explained, although respiratory infections, allergens and cold changes probably contribute to it. Unlike adults, children show an increase in asthma in February-March, ascribed to infections spread at school. Maximal asthma is associated with a mean temperature of 20-21°C. This may be optimal for the production of allergens. Further viral studies of asthmatic attacks are desirable.

The aim of this paper is to study epidemiological features of asthma based on admissions to the Royal Children's Hospital, Brisbane, and from them to endeavour to draw conclusions about the etiology. The conclusions may be compared with those of a similar study of asthma at ages 12 years and over (Derrick, 1972).

ANALYSIS OF ADMISSIONS

In the 20 years July 1949 to June 1969, there were 3,645 admissions to the Royal Children's Hospital, Brisbane, for asthma and asthmatic bronchitis (Table I). These comprised 2.37% of the total admissions for the period. Cases in which the diagnosis indicated inflammation in the bronchi or lungs as well as asthma, were included with asthmatic bronchitis.

Asthma and Asthmatic Bronchitis. In infancy the distinction between asthma and bronchitis or bronchiolitis may be difficult. At this age infection and wheezing are frequently associated. It is common for the earliest episodes in a patient to be designated asthmatic bronchitis, and after repeated recurrences the diagnosis of asthma becomes definite.

This sequence is exemplified in the present series by Leanne, who was admitted 15 times between the ages of 2 and 5 years. At the first 7 admissions and 2 of the next 4, the diagnosis was asthmatic bronchitis; with the other 6 it was asthma.

Over the whole period the proportion of "asthmatic bronchitis" to "asthma" admissions was 1:2. In individual years it varied considerably.

Age. Admissions for "asthmatic bronchitis" were most numerous in infancy (Figure 1) and at this age exceeded those for "asthma". With increasing age they steadily declined. "Asthma" admissions, relatively few in infancy, showed one peak at 4 years of age and another at 8-10 years. When the 2 conditions were added, the greatest number of admissions was at 3-4 years. The second peak, both in the "asthma" and combined curves, was due to multiple admissions of a number of severe cases, and the peak disappears when each individual is counted not more than once in

| TABLE I |
|---------|
| Admissions 1949-50 to 1968-69: Diagnosis and Sex |
|          | Male | Female | Totals |
| Asthmatic bronchitis | 743  | 500    | 1,243  |
| Asthma            | 1,398| 1,004  | 2,402  |
| Totals           | 2,141| 1,504  | 3,645  |

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1. Director, Research Bureau, Asthma Foundation of Queensland; Honorary Fellow, Queensland Institute of Medical Research.
Figure 1. Distribution of admissions, 1949-50 to 1968-69. AB: Asthmatic bronchitis by sex and age. AS: Asthma by sex and age. C: Both conditions and sexes combined by age. The broken line indicates the number of admissions when no patient is counted more than once in any year.

ANNUAL VARIATION

The annual admissions for asthma (including asthmatic bronchitis) varied considerably—from 101 to 279, and the curve is undulating (Figure 2). In general, admissions increased until 1958-59, decreased until 1961-62, and then increased again. On the whole there was a highly significant increase over the 20 years of the study, both in the number of admissions and in their proportion of total admissions to the hospital. As is apparent in Figure 2 and Table II, the increase involved mainly older children. The increase in the 6-11 years group was highly significant (P < 0.001); the slight increase in the 1-5 years group was not significant (P > 0.1). Both sexes and all seasons participated in the increase.

The annual admissions to the hospital for all disorders varied from 7,730 in 1949-50 to 8,856 in 1955-56. The significant increase in the proportion of these admissions which were for asthma shows that the increase cannot be ascribed to differing availability of beds in different years.

The overall increase in admissions of the older children since 1949 may therefore be accepted as definite. It suggests strongly that there has been an increase, in Brisbane, in the number of severe cases of asthma in this age-group. Cases which are admitted are, of course, drawn from those with the more severe grades of asthma in the community.
TABLE II

Annual Variation of Asthma Admissions 1949-50 to 1968-69:
Correlations as Time Series

| Admissions in: | 1-5 | Age Groups, Years | 6-11 | 1-11 | 0-11 |
|---------------|-----|------------------|------|------|------|
| July-September| 0.20| 0.78***          | 0.59**|
| October-December| 0.10| 0.62**          | 0.40  |
| January-March  | 0.10| 0.84***          | 0.63**|
| April-June     | 0.12| 0.78***          | 0.57**|
| July-June      | 0.18| 0.86***          | 0.66**|
| " " , Males    | 0.01| 0.84***          | 0.58**|
| " " , Females  | 0.37| 0.80***          | 0.69**|
| " " , percentage of all admissions |       |                   |      |      | 0.74***|

*The measure of time used for determining the coefficients of correlation with the admissions for the corresponding year was 1 for 1949-50, 2 for 1950-51 and so on.
No child was counted more than once in any quarter.
**P < 0.01  ***P < 0.001

Comparison with other countries

The annual variation in Brisbane may be compared with that in two overseas reports.

In the Los Angeles Children's Hospital Richards et al. (1967) concluded that there had not been a statistically significant increase in the proportion of total admissions due to asthma over the 35 years 1937 to 1964. It varied from 0.66 to 2.21% with an average of 1.13%. Although it had been high in the last 4 years of the study, it had also been high from 1940 to 1943.

In a survey of school children in Birmingham, England, Smith et al. (1971) found that the prevalence of definitely diagnosed asthma increased from 1.8% in 1956-57 to 2.3% in 1968-69.

Contributory Factors

Many factors probably contributed to the annual variation. The significant increase in admissions over the 20 years suggesting an increase in severity has been mentioned above. Otherwise little evidence of the factors has emerged.

One to be considered is the advance in therapy in recent years. This would be expected to reduce the number of admissions. Corticosteroids have been used increasingly since about 1952 and aerosol bronchodilators since 1958, with a considerable increase since 1963. However, it is difficult to relate these to the undulations in Figure 2.

Sodium cromoglycate, introduced in 1967, did not come into general use in Queensland in time to influence the graphs.

It is noted below that infection, like asthmatic bronchitis, is more frequent in younger children. The increase in the number of admissions from 1954-55 to 1958-59 was more evident in younger children (Figure 2) and included a higher proportion of "asthmatic bronchitis". This suggests that infection made a greater contribution than usual in those years.

It seems likely, from the studies of seasonal variation described below, that some of the annual variations depicted in Figure 2 were related to the weather, but no significant correlation of them with weather elements could be demonstrated.

Figure 3. Monthly admissions, 1949-50 to 1968-69, ages 1-11 years. A similar seasonal pattern with waves in the spring and autumn is shown by asthma (AS) and asthmatic bronchitis (AB), and (with the two diagnoses combined) by males (M) and females (F). In Figures 3 and 4 no patient has been counted more than once in any month.
Figure 4. Monthly admissions for asthma in relation to age. Admissions at age 0 years show one wave — in winter — at all other ages two waves — in spring and autumn. At ages 2-3 years, there were numerous admissions in March. In this and subsequent figures asthmatic bronchitis has been included with asthma.

SEASONAL VARIATION

In the average of the 20 years the monthly distribution of admissions (Figure 3) showed a minor wave in spring peaking in November, and a major wave in autumn peaking in May. Because of the uncertainty of the diagnosis in children less than 12 months old, these cases were omitted from Figure 3.

The curves were similar for males and females and also for those diagnosed as "asthma" and as "asthmatic bronchitis". The latter similarity and the sequence of the diagnoses in such cases as Leanne noted above, is in accord with the conclusion of Williams and McNicol (1969) that cases of "asthma" and "wheezy bronchitis" belong to a single population with the same defect. The two diagnostic groups, as well as the two sexes, have therefore been combined for further analyses.

Spring and autumn waves were present in each year of age except the first (Figure 4). Those under one year showed only one wave, which was in winter and peaked in June; this type of curve reflects the close association noted above between asthma and respiratory infection in the first year. The high admissions in March for children 2 and 3 years old are noteworthy.

There was considerable variation from year to year in the seasonal pattern. Asthma was very prevalent in December, 1958 (Figure 5). This month had the highest number of admissions for any month in the 20 years, even surpassing any autumn month. 1960-61 (Figure 6) was notable for a series of minor

Figure 5. Weekly admissions, 1958-59, of children for asthma for ages 1-5 and 6-11 years, and for upper respiratory tract infections (including pharyngitis), bronchitis and measles. Continuous lines for URTI and bronchitis 0-11 years, broken lines 6-11 years. Of URTI cases 76% were 0-5 years, of bronchitis 77%. Also admissions for asthma to Royal Brisbane Hospital, ages 12 years and over. In Figures 5 and 6 the curves have been smoothed by plotting a running total of three weeks. February 29 and August 31 have been omitted so that successive seven-day periods fall on the same dates each year.

Figure 6. Weekly distribution of admissions for asthma for four years to illustrate the variation from year to year. Continuous lines 1-11 years, broken lines 6-11 years. 1960-61 had a series of outbreaks, 1961-62 little seasonal variation, 1964-65 no spring wave. 1967-68 had the highest annual admissions for the 20 years. In three of these years sharp outbreaks occurred in February or March. The lowest graph shows for comparison casualty attendances for asthma at the Royal Brisbane Hospital for 1967-68; there were waves in spring and autumn but no increase in February-March.
waves with the highest prevalence in March, 1961-62 for a low prevalence and only slight seasonal variation, 1964-65 and 1967-68 for outbreaks in February. In 1964-65 there was no spring wave, and the autumn wave was delayed and peaked early in June. Most of the outbreaks affected both younger and older children; however, those in September and December 1958, and February 1965, affected mainly younger children.

Relation to Weather

Certain relationships to the weather have been reported in the seasonal variation of night attendances for asthma at the Casualty Department at the Royal Brisbane Hospital (RBH) (Derrick, 1972). A comparable study of childhood asthma has been made over the same 10 years, 1959-1968.

Figure 7 shows that the seasonal distribution of admissions at ages 1-5 and 6-11 years — that is, of pre-school and school children — was similar throughout the average year except for occasional weeks. There was also a general similarity to the RBH attendances, except during February and March when there was much more asthma in the children.

Figure 7 shows also the two-fold relation of asthma to temperature. Asthma is minimal both when the temperature is high in summer and low in winter, and maximal when it is intermediate in autumn and spring. The year was therefore divided, according to prevailing temperature, into 2 equal sections for correlation studies. The age-groups 1-5 years and 6-11 years were dealt with separately.

Other weather elements analysed are included in Tables III and IV. The dew point, a measure of absolute humidity, shows little diurnal variation. Its curve is almost parallel to that of the temperature. The relative humidity at 3 p.m. is close to the daily minimum. The rainfall varied considerably, and in the average year it was greater than the weekly mean of 2.2 cm in most weeks from November to March, and less than the mean in most weeks from April to October. The weekly totals were adapted for calculation as in Derrick (1972). Soil moisture was estimated as in Derrick (1966).

| TABLE III |
| 26 Cooler Weeks: Correlations of Weekly Asthma Prevalence with Weather Elements, and their Time Relations: Mean of Ten Years, 1959-68 |

| Children, Admissions | RBH Attendances |
|-----------------------|------------------|
| **Mean temperature**  | **Dew point, 9 a.m.** | **Relative humidity, 3 p.m.** | **Rainfall** | **Soil moisture** | **Hours of sunshine** | **Mean of Ten Years, 1959-68** |
| 0.82                  | 0.80              | 0.72               | 0.69        | 0.75            | 0.57               |
| Lag, Weeks: 5 (2-11)  | Lag, Weeks: 4 (2-11) | Lag, Weeks: 2 (2-4) | Lag, Weeks: 19 (13-19) | Lag, Weeks: 10 (9-13) | Lag, Weeks: no lag |
| Highest r: 0.945      | Highest r: 0.951  | Highest r: 0.82    | Highest r: 0.83   | Highest r: 0.82   | Highest r: -0.74   |
| Lag, Weeks: 5.6 (2-16) | Lag, Weeks: 5 (1-16) | Lag, Weeks: 6.7 (1-16) | Lag, Weeks: 16 (9-21) | Lag, Weeks: 16 (6-20) | Lag, Weeks: no lag |

These columns show the lag at which r was highest and, in brackets, the range for which P < 0.001 (r at least 0.61).

P < 0.01.
TABLE IV
26 Warmer Weeks:
Correlations of Weekly Asthma Prevalence
with Weather Elements of Same Weeks:
Mean of Ten Years, 1959-68

|                | 26 Weeks | 18 Weeks† |
|----------------|----------|-----------|
|                | Children, Admissions | RBH Attendances | Children, Admissions | RBH Attendances |
| Mean temperature | -0.50** | -0.89*** | -0.82*** | -0.90*** |
| Dew point, 9 a.m. | -0.39* | -0.80*** | -0.83*** | -0.74*** |
| Relative humidity, 3 p.m. | -0.26 | -0.62*** | -0.64** | -0.65** |
| Rainfall | -0.44* | -0.75*** | -0.54* | -0.84*** |
| Soil moisture | -0.64*** | -0.58** | -0.79*** | -0.67** |
| Hours of sunshine | -0.14 | 0.07 | 0.07 | -0.02 |

†Omitting the eight weeks February 5 to April 1.
*P < 0.05  **P < 0.01  ***P < 0.001.

Asthma in the 26 Cooler Weeks. This period (April 23 to October 22) includes most of the autumn wave, the winter recession and the first part of the spring wave. The main conclusions were:

1. The similarity in seasonal patterns of children's admissions and RBH attendances in this half of the year, seen in Figure 7, was confirmed by correlation of weekly totals. Between the 2 children's age-groups, r = 0.75 (P < 0.001); between each of these and R.B.H. attendances, r = 0.70 and 0.66 (P < 0.001).

2. Warm humid weather may be a factor in promoting asthma after a short lag. There were highly significant correlations (P < 0.001) between the weekly prevalence of asthma in the children and the temperature, dew point and relative humidity a few weeks earlier (Table III). There were also highly significant correlations with rainfall and soil moisture some months earlier; their relevance is more remote. As there was very little difference in the weather relations of the 2 children's age-groups, they were combined in the table.

3. Factors unrelated to weather may contribute more to asthma in childhood than in adults. The weather correlations with the children showed in general a similar pattern to those for the adults, but with somewhat lower coefficients. With temperature and dew point, the coefficients were significantly lower.

4. Asthma is maximal a few weeks after the mean temperature is about 20-21°C and decreases as the temperature varies either up or down. In the children the highest prevalence of asthma in autumn was in the 7 weeks, April 23 to June 10. For this period the average temperature was 18.3°C. In spring the highest prevalence was in the 8 weeks, September 25 to November 19, when the average temperature was 21.3°C. As the temperature is falling in autumn and rising in spring, the mean temperature would have been 20°C about 2.5 weeks before the midpoint of each period. With the RBH attendances, the weeks of highest prevalence were not quite the same as with the children, and a similar calculation gave 21°C as the mean temperature 2.5 weeks before the midpoints. As noted above, the highest correlation of asthma with temperature was with a lag of some weeks. The dew point that corresponds to maximal asthma is about 12.5-13.5°C.

Asthma in the 26 Warmer Weeks. This period (October 23 to April 22) includes February and March when, as shown in Figure 7, there was much more asthma in children than in adults. The main conclusions were:

1. The seasonal pattern was similar in the 2 children's age-groups; for the weekly admissions, r = 0.45 (P < 0.05). The 2 age-groups were combined for further studies; separately they gave closely similar results.

2. Correlation of the RBH attendances with the children's admissions was greater, but not significantly so, when the 8 weeks, February 5 to April 1, were omitted (r = 0.77, P < 0.001) than over the whole 26 weeks (r = 0.43, P < 0.05).
3. In the warmer and wetter half of the year, both child and adult asthma decreased significantly, with no lag, as warmth and wetness increased (Table IV).

4. The higher prevalence of asthma in children than in adults in February and March was not apparently related to the weather. Over the 26 weeks, the correlations with temperature, dew point, relative humidity and rainfall were lower with children than with adults; with the first 2 the differences were significant. When the 8 weeks were omitted the differences mostly disappeared.

The two-fold relation of asthma to temperature, based on the correlations in the cooler and warmer weeks, is illustrated in Figure 8.

The Significance of 23°C. The ending of the spring wave and the beginning of the autumn wave occurred at times when the mean temperature was approximately 23°C. This relationship is shown in Figures 7, 9 and 10. In the autumn it is partly obscured by the February-March asthma in the children, but is clearly defined in the RBH attendances.

Some of the asthma increases in the warmer months corresponded with a temporary fall in the mean temperature below 23°C; an example is the increase in mid-March 1961 shown in Figure 6. Other increases did not correspond

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**Figure 8.** The twofold relation to temperature of admissions of children 1-11 years for asthma. Left: Significant positive correlation (P < 0.001) between admissions in the 26 cooler weeks and the mean temperature five weeks earlier. Right: Significant negative correlation (P < 0.001) between admissions in 18 warmer weeks with the mean temperature of the same weeks. The 8 weeks February 5 to April 1, indicated by crosses, have been omitted in the calculation for the regression line. The weekly values in both graphs are averages for the ten years, 1959-1968.

**Figure 9.** Weekly asthma admissions in relation to mean temperature and rainfall, 1956-57. It is noteworthy that a. The spring wave appears unrelated to rainfall; there was no substantial rain for months before it, during or immediately after it. b. Its abrupt ending early in November coincided with a sharp and sustained rise in mean temperature above 23°C. c. In this respect the "school-opening" wave of asthma in February-March was different; this occurred while the temperature remained above 23°C. d. In general the autumn wave occurred after the temperature fell below 23°C. e. The high asthma in May coincided with a sharp fall in temperature.

**Figure 10.** Weekly asthma admissions in relation to mean temperature, rainfall and pollen counts, 1963-64. a. In contrast with 1956 (Figure 9) the temperature did not rise above 23° until near the end of December; the spring asthma wave was correspondingly prolonged. b. Also in contrast with 1956, the spring wave occurred during a period of ample rain. c. In 1964 there was little "school-opening" asthma and the autumn wave began in March at about the time when the temperature fell below 23°. d. From late December until February when grass pollens in the air were maximal, asthma was minimal (Pollen counts by courtesy of Miss Janice Moss).
— those in December 1958 (Figure 5) and in February 1961, 1965 and 1968 (Figure 6).

It is conceivable that sensitivity to a temperature above about 23°C limits the production of some important allergen; this could be a clue to its recognition.

The 23°C level does not sharply divide two distinctive temperature regions. They merge gradually. On a day when the mean temperature is 23°C, the temperature would be lower than this for 12 hours and higher for 12. Even in an average midsummer day the temperature would be below 23° for eight hours. One would therefore expect the influence, if any, of a rising temperature on the prevalence of asthma to be manifest gradually. As a rule the ending of the spring wave is gradual. Only in the occasional year, when the temperature rises sharply and the high level is sustained, is the decrease in asthma sudden.

Asthma in February-March

It was noted above that the higher prevalence of asthma in children than in adults in February-March was not apparently related to the weather. The likely explanation (Derrick, 1968) is the spread of infection in families after the opening of the school year. The spread of respiratory infections in schools is well recognised. Lidwell and Sommerville (1951) concluded that, in the Wiltshire village they studied, the school was the principal source of infection with colds and school children had a major role in introducing infection into their households. Jackson (1971) recorded that one of the three waves of common colds usual each year in U.S.A. occurred in the autumn a few weeks after the opening of schools; the others were in midwinter and spring.

December 1958 Outbreak

This outbreak invites analysis because of its size and unseasonal occurrence. It began gradually about November 16, reached its peak from December 14 to 22 and subsided irregularly to end about January 19, 1959 (Figure 5).

It involved 67 children aged 1-11 years, with 76 admissions. The sex distribution (41 boys, 26 girls) was similar to that in the rest of the year. The proportion of children 1-5 years of age in the outbreak (69%) was significantly higher than in the rest of the year (54%) (P < 0.05). An increase in admissions for asthma to the Royal Brisbane Hospital occurred in December-January; it was later and smaller than the increase in children's admissions.

There was no increase in admissions for asthma in December 1958 to the Royal Alexandra Hospital for Children, Sydney, or to the Adelaide Children's Hospital (data by courtesy of the Medical Superintendents of these hospitals).

Etiological factors were sought in weather associations and evidence of infection.

On all but 2 days in December 1958, the mean temperature exceeded 23°C; and it was mostly over 24°C. This suggests that the etiology of the cases then was different from that of the autumn and spring asthma waves.

October and November 1958 were abnormally dry. In December the rainfall totalled 28 cm (11.07 inches) — the highest December total in the 20 years (normal 13.6 cm — 5.37 inches). It is possible, but unlikely, that the absence of the usual asthma wave peaking early in November and the high December asthma may have been related to the abnormal rainfall pattern. In 1968 there was a brisk spring wave in October in spite of 2 previous dry months.

Another unlikely possibility is that the December outbreak was induced by a wet-air type of fungal spore. These have not been shown to be important allergens in Brisbane (Derrick, 1972). However, in other years there were occasional clusters of admissions during warm wet periods.

The unusually high proportion of younger children in the outbreak suggests, as noted later, an infective etiology. This is supported by the high proportion of admissions in which a diagnosis of "asthmatic bronchitis" was made (40 of 76). The simultaneous increase in admissions for upper respiratory tract infections and bronchitis, although slight, indicates that some infective agents were circulating then (Figure 5). A similar correspondence is noticeable in the preceding
September. However, infections were more prevalent at other seasons without corresponding increases in asthma.

The epidemic of measles which peaked in October 1958 seems too remote to have precipitated asthma in December.

**REVIEW OF ETIOLOGICAL FACTORS**

In a discussion of the etiology of asthma, age, sex and seasonal distribution call for consideration. Salient factors include respiratory infections, allergens and cold changes.

Although the increase of asthma in autumn has been on record since the time of Hippocrates (Aphorisms III, 19, 22), it has not been explained. It is noteworthy that the characteristic seasonal pattern of asthma with waves in autumn and spring is already established by the second year of life. It continues to be manifest throughout childhood and youth and to at least middle age.

**Respiratory Infections**

The age and sex distributions of respiratory infections and asthma have much in common. In 2 Australian surveys the maximum incidence of respiratory infections was found at about one year (Bridges-Webb, 1972). They are usually more common in boys than girls, in adult females than adult males.

Infection is a frequent precipitating cause of childhood asthma, particularly in the younger ages. (It may also supervene as a complication when an attack persists.) Ford (1963) observed that the first attack of wheezing usually appeared together with or after a frank infection. Infection was the dominant precipitating cause in 65% of attacks in those who developed asthma in the first year, in 43% at 1-4 years, 33% at 5-9 years and 23% at 10-14 years.

Williams and McNicol (1969) noted that respiratory catarrhal symptoms were commonly associated with earlier wheezing episodes, but with increasing age other factors — emotional disturbances, seasonal change, inhalant allergens — became important precipitants. Halpern (1972) found that infection was responsible for 90% of attacks at two years, the proportion falling to about 20% at ten years.

Wittig et al. (1959) found that 32% of children, mostly infants, who had had bronchiolitis subsequently developed asthma. With the infants of Zweiman et al. (1966, 1971), the proportion was about half. An infection in infancy had apparently precipitated the first episode of asthma in those with an allergic diathesis. As the children grew older infection continued to be a common precipitant, but attacks were also set off by non-specific stimuli such as exertion, sudden weather changes and, later again, by inhalants — dust, danders and pollens. The proportion of the children that displayed sensitivity to extrinsic allergens by skin tests increased progressively with age.

The decreasing role of infection with increasing age is in accord with the decrease in the relative frequency in Brisbane of a diagnosis of asthmatic bronchitis.

As infection precipitates asthma frequently in younger, but infrequently in older children, it seems unlikely that it can explain the seasonal waves, which are similar in both age-groups. It is noted, however, that rhinoviruses may become very prevalent in autumn and that cold changes in autumn may perhaps reactivate latent respiratory infections (Andrewes, 1967). The occurrence of unseasonal outbreaks of asthma from time to time, as in September and December 1958 (Figure 5 or September 1963 (Figure 10), might well be explained by epidemics of infection.

**Viral Studies.** A number of investigations have been made into the possible role of specific viruses and bacteria. The most productive relate to infancy — the commonest age for asthma to begin — and to respiratory syncytial virus (RSV) — the commonest cause of lower respiratory illness at this age (Jackson, 1971). Characteristic features of RSV infection are bronchiolitis, fever, dyspnoea, cough and wheezing; there may be pneumonia. The occurrence of expiratory wheezing in bronchiolitis makes its differentiation from asthma difficult. Before 12 months of age, 35% of children in U.S.A. have been infected by RSV, by 5 years 95%. Stuart-Harris in Britain (1971) defines the age-range of RSV disease as 2-18 months.

Rooney and Williams (1971) followed up 62 Melbourne children who had had bron-
chiolitis in infancy and from whom RSV had been isolated. Of them, 56% had subsequent attacks of wheezing, 43% on more than 5 occasions. Significantly more of those who subsequently wheezed than those who did not had an allergic diathesis, as shown by the concurrence of other allergies and by asthma in first degree relatives.

In Denver, McIntosh et al. (1972) carried out viral, bacterial, and serological studies on 32 asthmatic children, aged 18 months to 5 years, hospitalized for prolonged observation. Of 137 wheezing episodes, 42% were associated with identifiable virus infections. RSV was the most virulent; it was associated with 13 episodes of pneumonia and with wheezing in 23 of 24 infections. Next came para-influenza virus and coronavirus. Positive cultures of bacteria from nasopharynx or throat were not statistically associated with wheezing.

There is evidence that an allergic mechanism contributes to RSV bronchiolitis. Chanock (1970) pointed out that in the first 6-8 months of life, when RSV most often causes severe disease, infants possess maternally-derived serum antibodies against it, but they lack protective amounts of secretory antibody in the respiratory mucosa. He postulated an interaction of viral antigens and serum antibodies in the lungs, that is, a Type III allergic reaction. Gardner et al. (1970) suggested that the bronchiolitis is the result of a second invasion of the infant by RSV, and that a Type I allergic reaction follows between the virus and reagins produced by the first attack. The eosinophilia in many young asthmatics (Wright, 1973) points to an allergic mechanism, although there may be no convincing evidence of an external allergen.

Gregg (1972) has isolated rhinoviruses more frequently than any other type of virus in episodes of both upper and lower respiratory illness. In the majority of subjects liable to wheeze — that is those who have inherited or acquired bronchial hyperreactivity — rhinoviral infection was associated with an acute onset of wheeze or an exacerbation of chronic wheeze.

**Allergens**

The high correlation in the cooler months between the weekly prevalence of asthma and the temperature and humidity several weeks earlier would accord with the development of airborne allergens. A further point is that there is little if any seasonal variation in the “intrinsic” asthma (for which such allergens are not responsible) of those past middle life.

However, the seasonal prevalence of asthma does not conform with the seasonal prevalence of the commonly recognised allergens — house mites (Domrow, 1970), fungi (Rees, 1964, 1966), pollens (Moss, 1965, 1967) — review (Derrick, 1972). The nonconformity with grass pollens is illustrated in Figure 10. Further, in very young children, only a small proportion of asthma is caused by airborne allergens. In Adelaide, Ford (1963, 1969) found that extrinsic causes were dominant in 29% of those with the onset at 0-4 years (foods accounted for most of the extrinsic cases in infancy), in 56% at 5-14 years and 83% at 15-29 years. He noted that extrinsic causes were less common in hospital than in private patients. In Brisbane, Wright (1973) found that 26% of asthmatic children under 5 years of age had evidence of sensitivity to house dust by skin prick tests plus clinical confirmation, and 55% of those between 5 and 9 years. In French children, Halpern (1971) found the proportion caused by exogenous allergens was very low at 2 years and steadily rose to about 50% at 10 years.

These observations suggest that the definite seasonal variation in admissions for asthma of quite young children in the present study can only in small part be attributed to airborne allergens. Nor would the food allergy of infants be likely to contribute to the seasonal variation.

**Cold Changes**

Many asthmatics are unduly sensitive to cold. They appear to over-react to a normal reflex. Broncho-constriction is a protection against the penetration of cold air or other irritant into the lungs. With some patients it seems to be the application of cold to the skin which initiates the reflex.

In the average year in Brisbane, the autumn wave begins when the environmental temperature starts to fall and it peaks when the
temperature is falling most rapidly (Derrick, 1972). But the response to cold is not a major factor. If it were, attacks would largely be concentrated on days with cold changes. Indeed the spring wave occurs while the temperature is rising, and not every asthmatic is sensitive to cold.

Factors in General

On the evidence presented here, the seasonal distribution cannot be explained by infections, allergens or cold changes. It could perhaps depend on a combination of them, and of other factors not yet identified.

Williams and McNicol (1969) found it impossible to isolate groups of children who had one sole precipitating cause for their wheezy episodes. This did not support a simple division of asthma into infective, allergic, emotional or other types.

From his observations on Perth children, Hobday (1966) concluded that the high incidence of asthma in autumn and spring was not associated with increased incidence of respiratory infection. Infection was present in 22% of those admitted in April-June and in 14% of those in October-December. In the latter period he considered the main precipitating factor was airborne allergens.

In Los Angeles, Richards et al. (1967) were unable to explain the seasonal distribution of asthma. Hospital admissions of children showed in the average year a major increase in autumn (October-November) and a minor increase in spring-summer (May-June). In individual years there was much variation from the average. The period of the autumn increase did not correspond fully with that of any environmental factor. It corresponded partly with the end of the peak weed-pollinating season (August-November), with decreasing temperature and humidity (October-January) and with the incidence of para-influenza (October-December). There was little relation to absences due to respiratory infections in schools, which peaked in December-February, and to the incidence of RSV infections (February-April).

Further investigations are obviously needed to explain the seasonal variation. These include viral studies of asthmatic attacks with the newer techniques and a search for as yet unidentified allergens the production of which is favoured by the Brisbane type of climate. A clue to their recognition, if they exist, would be the temperature relations noted above.

CONCLUSIONS

In the 20 years, 1949-1969, admissions for asthma (including asthmatic bronchitis) to the Royal Children's Hospital, Brisbane, comprised 2.37% of all admissions.

Over this period the annual admissions significantly increased. The increase involved both sexes, all seasons and particularly older children.

Admissions for asthma in children under one year of age peaked in winter. In those aged one year and over, as in adults, there was a major wave in autumn and a minor wave in spring. This two-fold seasonal pattern, unexplained at present, may depend on a combination of respiratory infections, allergens, cold changes and other factors not yet recognised. An increase in asthma in February-March is attributed to the spread of infection in young families after the opening of the school year.

The association of infection with asthmatic attacks is greatest in infancy and decreases with age. Conversely, that of allergens is least in infancy and increases with age.

Asthma is maximal in autumn and spring several weeks after the mean temperature is about 20-21°C and minimal both when it exceeds about 23°C in summer or reaches about 15°C in winter. It is suggested that this temperature range is related to allergen production.

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Queensland Institute of Medical Research, Herston Road, Herston, Queensland, 4006.

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