TITLE

The prevalence and nature of wheeze in preschool children: a questionnaire study

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THE PREVALENCE AND NATURE OF WHEEZE IN PRESCHOOL CHILDREN: A QUESTIONNAIRE STUDY

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The epidemiology of wheeze, asthma and recurrent cough was studied in preschool children using a parent-answered postal questionnaire. The influences of putative environmental factors and of a family history of wheeze/atopy on the nature and prevalence of wheeze and prevalences of asthma and recurrent cough were investigated.

The study cohort was established from a population-based random sample of Leicestershire children $\leq$ 5 years. The questionnaire was designed for the target population. Questions were derived chiefly from previously tested or standardised questionnaires. Of 1650 questionnaires posted, 1422 (86.2%) were returned.

Wheeze occurred in 15.6% and asthma in 11% of children, both with male predominance. Of the 222 children with reported wheeze, 125 (8.8%) were diagnosed as having asthma. Cumulative prevalence of asthma increased significantly with age. Recurrent cough was reported in 21.8% of children. The characteristics of wheeze were described using six severity variables, volunteered precipitants and seasonal and diurnal variation. Increasing age, number of wheezy episodes in the past year, severity of shortness of breath with attacks and precipitants other than colds were major determinants of the probability of a wheezy child being diagnosed as having asthma.

The proportions of wheezy children who were under current medical review, had received medical attention (ever) or had been admitted to hospital for wheeze were 34.8%, 75.0% and 21.8% respectively. The likelihood of current or past treatment were related to wheeze frequency and exposure to inhaled precipitants whereas hospital admission was more likely where attacks caused shortness of breath and with decreasing frequency of attacks per year.

Predominant factors influencing the prevalence of wheezy episodes in the past 12 months were previous respiratory tract infections, passive smoke exposure and a maternal history of asthma. Recurrent cough was associated with past history of bronchiolitis, bedroom sharing, damp in the home and parental smoking.

This study established a cohort to study further the natural history of wheeze.

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SUMMARY

The epidemiology of wheeze, doctor diagnosed asthma and recurrent cough was studied in a cohort of 1422 preschool children using a parent-answered postal questionnaire. The influences of putative environmental factors - parental smoking, the use of gas cookers, damp and mould in the home, household pets, urban/rural domicile, social background, and of a family history of wheeze and atopy on the nature and prevalence of wheeze and the prevalences of asthma and recurrent cough were investigated.

Preschool children have not been the sole subjects of any previous prevalence study on wheeze. As no validated questionnaire applicable to this age group existed a new questionnaire had to be compiled. Where possible the questions selected were derived from previously tested or standardised questionnaires. Most were either copied unchanged or modified from source questions; a minority were devised anew to suit the target population. In developing the questionnaire every effort was made to comply with the basic principles of questionnaire design, and any medical terms used were defined in language likely to be understood by the respondents. The reliability of the questionnaire was tested by remailing the questionnaire to 100 respondents (75 replied) 6 months after initial posting. Two measures of repeatability were used, percentage agreement and kappa. Questions eliciting wheeze and asthma prevalences were highly repeatable with respective kappa values of 88% and 82%. However the questions on coughing apart from colds showed poor repeatability with kappa value of 19%. Most other questions tested showed good to fair agreement with kappa values above 40%.

The study cohort was established from a population-based random sample of Leicestershire preschool children aged 5 years and under registered on the county health authority's child health index. Sampling was stratified by year of birth (1985-1989) to ensure equal representation of each age group. The study sample was shown to be representative of the source population. Measures used to test this were the study population distributions of gender, month of birth, area of residence (distribution compared to the 1987 Leicestershire birth cohort), and social class (distribution compared to the population census of 1981). As parents of affected children are possibly more likely to respond to a questionnaire concerning their child's specific condition, estimated prevalences may be falsely increased. As no information was available for non-respondents, prevalences of wheeze and doctor diagnosed

asthma were assessed in the respondents for the three mailings (non-respondents were remailed twice to maximise the response rate). These were shown to be similar. Similarly, to assess for bias of the specific parent answering the questionnaire, the same prevalences were estimated in the 3 parent categories (mothers, fathers and both) answering the questionnaire. When both parents answered the questionnaire, wheeze prevalence was significantly higher when compared with those reported by individual parents. As this group constituted only 35 of 1409 tested this is unlikely to have influenced the overall prevalence estimates. The study was conducted in 1990.

Of 1650 questionnaires posted, 1422 (86.2%; 726 boys and 696 girls) were returned. The cumulative prevalence of wheeze overall was 15.6% (95% confidence interval (CI) 13.7% to 17.5%), and higher in boys (17.6%) than in girls (13.5%) (odds ratio (OR) 1.38, $p=0.03$). Overall 11.0% (95% CI 9.4-12.6) of children had been diagnosed as having asthma, the cumulative prevalence in boys (12.7%) being somewhat higher than in girls (9.2%) (age adjusted OR 1.47; $p=0.03$). The cumulative prevalence of asthma increased significantly with age from 7.5% (13/173) in children under 1 year to 15.9% (61/383) in children 4 years and over ($p<0.001$). The overall prevalence of recurrent cough without colds was 21.8% (95% CI 19.6-23.9) with non-significant excess in boys (23.1% versus 20.4%). The overall prevalence of wheezing attacks in the past 12 months was 13.0% (95% CI 11.3-14.8) with a non-significant excess in boys (14.5% versus 11.5%).

The characteristics of wheeze were described using six severity variables, volunteered precipitants and seasonal and diurnal variation. Of the 222 (15.6%) of children reported to have wheezed, 125 (8.8%) had been diagnosed as having asthma. More than 80% of the former had recurrences of wheeze and 40% (72) had 3 or more episodes in the preceding 12 months. Increasing age, number of wheezy episodes per year, the severity of shortness of breath with attacks and precipitants other than colds were the major factors determining the probability that a wheezy child would be diagnosed as having asthma; respective p values 0.006, 0.0006, 0.003 and 0.003. The data also suggest that despite the strong association of symptom-based criteria with the label asthma, asthma was not diagnosed by these same severity criteria in one quarter of the children reporting wheeze.

The proportions of children with reported wheeze who were being followed up medically, had received medical attention for wheeze (ever), or had been admitted to hospital with wheezing illnesses were calculated and the factors important in determining these outcomes were assessed. Among the 222 children with wheeze, 77 (34.8%) were under current medical review, 165 (75.0%) had received medication and 48 (21.8%) reported having being hospitalised. The importance of various wheeze severity, demographic, environmental and family factors in determining these indices of treatment was investigated. Current follow-up was more likely if a child was female (OR 2.22; 95% CI 1.12-4.38), had more than 3 attacks of wheeze in the last year (versus no attacks in the past year, OR 17.44; 95% CI 5.22-58.3), or if the family kept a cat or dog, when inhalants were reported as a precipitant of wheeze (OR 13.65, 95% CI 3.16-58.94). Treatment (ever) became more likely as a child aged [for 4 years or older versus < 1 year (OR 3.91, 95% CI 1.20-12.71)], had suffered more than three attacks in the last year (versus no attacks in the last year OR 4.12, 95% CI 1.53-11.12), reported inhalants as a precipitant of wheeze (OR 4.66, 95% CI 1.08-20.14) or if the family kept a cat or dog (OR 2.28, 95% CI 1.04-5.03). In contrast, hospitalisation was less likely if the child had frequent wheeze [3 or more attacks in the last year versus no attacks in last year (OR 0.30, 95% CI 0.12-0.77), but was more likely if wheeze was associated with shortness of breath with some (OR 4.14, 95% CI 1.39-12.30) or every (OR 17.93, 95% CI 5.70-56.49) attack. No independent relationships with any outcomes were demonstrated for socio-economic status, parental smoking, personal history of atopy, or family history of asthma or atopy. Therefore, in this study, the likelihood of medical follow-up and treatment were related to wheeze frequency and exposure to inhaled precipitants, whereas hospital admission was more likely in those children with attacks causing shortness of breath and with decreasing frequency of attacks per year. Efforts to address the problem of increasing rates of hospital admissions in this age group must therefore include all children with wheeze, regardless of wheeze frequency.

Factors influencing the prevalence of wheezy episodes in the past 12 months (current wheeze) and recurrent cough were assessed by unconditional logistic regression modelling. The predominant influences on current wheeze were previous respiratory tract infections - bronchiolitis (OR 7.80, 95% CI 3.48-17.47), croup (OR 6.18, 95% CI 3.95-9.66) and pneumonia (OR 7.80, 95% CI 1.57-6.10), as well as passive smoke exposure (OR 1.56, 95%

CI 1.01-2.38) and a maternal history of asthma (OR 2.34, 95% CI 1.48-3.73). Recurrent cough was associated with past history of bronchiolitis (OR 2.57, 95% CI 1.83-3.61), bedroom sharing (OR 1.59, 95% CI 1.17-2.16), damp in the home (OR 1.37, 95% CI 1.02-1.84) and parental smoking (OR 1.43, 95% CI 1.06-1.93). Wheeze and cough differed in that genetic factors (family history of asthma) were more likely to influence wheeze and social factors (bedroom sharing and dampness) the prevalence of cough apart from colds. Parental smoking was therefore the predominant home environmental influence on the prevalences of respiratory symptoms in preschool children.

In conclusion, these prevalence findings establish baseline results in an age group hitherto not the sole subject of a questionnaire study on wheezy illnesses. The questionnaire developed for this study was shown to have good to excellent short-term repeatability; long-term repeatability was assessed by the cohort follow-up study. Severity variables and other descriptive characteristics of wheeze which determined the likelihood of asthma being diagnosed were established. Wheeze frequency was an important determinant of on-going follow up and having ever received treatment for wheeze. Shortness of breath with wheezing episodes was the main factor determining admission to hospital; surprisingly wheeze frequency was inversely related to the likelihood of hospitalisation. The implications of these findings reflect the necessity to address all wheezy infants in planning how best to reduce hospital admission rates. The dominant environmental factors in wheeze and recurrent cough prevalences were previous lower respiratory tract infections and parental smoking, the latter having important health education significance. Finally, the study allowed a cohort to be established in which to assess the natural history of wheeze. It seems particularly important to review this cohort during the early school years (5-7 years of age) to determine the factors that predict wheeze persistence and the physiological correlates consistent with the diagnosis of asthma. The usefulness of a diagnosis of asthma made during the preschool years would also be assessable. Finally the long-term repeatability of the questionnaire developed for the present study would also be assessable.

1. INTRODUCTION

INTRODUCTION

The prevalence and nature of wheeze and asthma in childhood has been extensively studied in the developed communities of Europe, North America and Australasia. The results are consistent in showing that most children develop these symptoms in their preschool years, that is before the age of five. Few investigators have included the under-5s in their study populations when exploring the epidemiology of wheeze and asthma, and fewer still have focused solely on this age-group. In their description of wheeze in children under five McNicol and Williams (1973) relied entirely on recall of parents of school-going children. Their study started with school-aged children. Retrospective studies of this nature are potentially inaccurate because of incomplete recollection by parents (Strachan 1985). Results and conclusions about the time period prior to the age at which such studies are undertaken should be viewed with this reservation.

There is evidence from studies in developed communities that the prevalence of wheezing illnesses, particularly asthma, is increasing. The community-based evidence arises from studies repeated in the same geographical area in similar groups of children. However the strongest evidence comes from increased hospital admission rates for asthma in children. These data, which emanate principally from the United Kingdom, New Zealand, America, and other westernised countries, show that the most striking increase in asthma admissions occurred in children aged 4 years and under (Anderson 1978; Mitchell 1985). If, as is suggested, there has been a real increase in the prevalence of asthma and not simply greater readiness by doctors to diagnose asthma and by parents to accept it, it is appropriate to enquire more fully in the age-group where the incidence of new cases is greatest - the under-5s.

Both genetic and environmental influences contribute to the aetiology of wheeze and asthma. Two large twin studies undertaken to establish the magnitude of the genetic component of asthma have shown a heritability of fifteen percent (Edfors-Lubs 1971; Duffy et al 1988). The findings in these studies were consistent despite a difference in the estimated prevalence of asthma in the two study populations of greater than ten percent and a time span between the reports of seventeen years. Therefore the contribution of the genetic component to the

development of asthma has remained constant during the period when the prevalence of asthma doubled (Burr et al 1974; Clifford et al 1989). It would also seem unlikely that during this time the gene pool available has increased at the same rate as the reported prevalences of asthma. Thus, environmental influences must be increasingly implicated in the observed prevalence increases of wheeze and asthma.

Many factors in the home environment are associated with wheeze and asthma. The factors identified in previous epidemiological studies include passive inhalation of cigarette smoke, urban domicile, household damp and mould, nitrogen dioxide exposure from gas heaters and stoves and household pets. All of these, with the possible exception of urban domicile, are relevant primarily to the indoor environment where preschool children spend most time. It is therefore appropriate to investigate the indoor environment in any study of asthma in this age-group.

Cough is a recognised manifestation of asthma, usually in association with wheeze but sometimes as the only presenting symptom (Cloutier and Loughlin 1981; Konig 1981; Hannaway and Hopper 1982). The prevalence of cough and symptoms such as breathlessness have not been well studied in isolation from wheeze (Clifford et al 1989).

Given the present limitations in knowledge in asthma in the preschool age-group when most affected children develop symptoms for the first time, and the striking increase in wheezing illnesses that has occurred at this age, it seemed timely to look afresh at the epidemiology of wheeze and asthma in this age-group.

DEFINITIONS

Terms used in this study are defined below.

1. Wheeze

Defined in the questionnaire as a high-pitched whistling sound from the chest, not the throat.

2. Asthma

Recognised if a parent answered yes to question 15 which stated: 'Has any doctor or hospital told you that he/she (i.e. the index child) has asthma or bronchitis'.

As the terms asthma and bronchitis are often applied interchangeably in children under 5 years old and their treatment is similar, a positive response to this question justified the use of the phrase doctor diagnosed asthma.

The nature of the study did not allow scope to provide support for this diagnosis by scrutiny of doctors' records, clinical examination or challenge studies. In this age-range studies for assessment of reversible airway obstruction would not have been possible.

3. Recurrent cough

Children with recurrent cough were those in whom a 'cough without a cold' was reported, but in addition in whom there was no history of wheeze or asthma.

4. Age-group

Children were grouped by actual age into 5 age-groups i.e. $\leq$ 1 year old, 1-1.99 years, 2-2.99 years, 3-3.99 years, and $\geq$4 years.

5. Non-cold precipitants

Enquiry was made about precipitants of wheezy episodes in Question 9. Answers to sub-question 9b *"Do these attacks occur occasionally apart from colds?"* are referred to as non-cold precipitants.

6. Ingestants

This refers to the precipitants enquired after in the sub-question 9d *"Do these attacks occur with drinking or eating?"*.

7. Inhalants

This refers to the precipitants enquired after in the sub-question 9e *"Do these attacks occur when he/she is near, for example animals, dust, grass and so on?"*

8. Parental smoking

At least one parent smoked.

2. AIMS OF THE STUDY

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The aims of this study were:

1. to develop a questionnaire on respiratory symptoms, especially wheeze, for use in preschool children;

2. to estimate the prevalence of wheeze and doctor diagnosed asthma in children aged 5 years and under in Leicestershire;

3. to describe the nature of wheeze and its relation with doctor diagnosed asthma;

4. to describe the treatment of wheeze using current medical attention, medication (ever) and hospital admissions as the main end points;

5. to identify factor significantly associated with wheeze by assessing family background, environmental factors and social influences; and

6. to establish a subject cohort for further study of wheeze in preschool children.

3. REVIEW OF LITERATURE

INTRODUCTION

There have been many surveys world-wide of the prevalence of asthma in childhood, mostly in children of school-going age (Gregg 1983). These surveys have shown widely differing prevalences of reported asthma within and between countries, with much higher prevalences in developed countries (Anderson 1974; Van Niekerk et al 1979). Migrant studies suggest that these differences may be environmental rather than genetic in origin (Morrison Smith et al 1971; Morrison Smith 1976; Partridge et al 1979; Waite et al 1980; Morrison Smith and Cooper 1981).

In westernised countries, including Britain, where asthma is already the commonest cause of chronic disease in children (Godfrey 1983), there has been evidence from community-based studies and reports of hospital discharge records that the prevalence of asthma has increased in recent years.

PREVALENCE OF WHEEZE AND ASTHMA

Evidence of increase in prevalence

Community studies

The conclusions reached from the community studies are based on the results of repeat studies of similar format in the same population and geographical area 15 to 25 years apart. Three such pairs of studies have been undertaken in Britain.

The first of these was by Morrison Smith et al (1974) who conducted questionnaire studies of the prevalences of wheeze and asthma on children in Birmingham on three occasions over a period of twenty years. These studies, carried out in a "reasonably similar manner", showed an increase in the prevalences of both wheeze and asthma. The authors conceded however that the later questionnaires were broadened and may have identified more children with asthma without there having been a true rise in prevalence.

Burr et al (1989) also repeated an earlier study (1976) in an attempt to establish whether the prevalences of wheeze, asthma, eczema and hay fever were increasing. The two studies, performed 15 years apart on 12 year old children living in a defined area of south Wales,

were based on a short parent-answered questionnaire, peak-flow measurements and exercise provocation tests. In an attempt to distinguish a genuine increase from a greater readiness to diagnose the disease, parents were asked about symptom patterns suggestive of asthma, rather than the diagnosis itself. Their results show that the prevalence of wheeze at any time had increased from 17 to 22 percent, and that of asthma from 6 to 12 percent. There was a concomitant rise in the prevalences of hay fever and eczema.

The most recent repeat study was carried out by Ninan et al in Aberdeen (1992) who performed a questionnaire study similar to that undertaken by Dawson et al (1969) 25 years earlier. The later study reported a two-fold increase in the prevalence of wheeze and a three-fold increase in diagnosed asthma. Unfortunately the methods were not identical and there was evidence of greater awareness and perhaps readiness to report symptoms in the later study. These authors (Ninan et al 1992) felt that these confounding factors could not wholly explain the observed prevalence increases and concluded that there has been a real increase in symptom prevalences during this time.

Geographical variation in the prevalence of asthma, about which there is abundant international evidence, precludes any extrapolation about national trends from these data sets. Burney et al (1990) reported a study of English children aged 4 to 12 years from 22 centers who took part in the national study of health and growth between 1973 and 1986. Data was collected on respiratory conditions by questionnaire, although again with some variation in content. The analysis, which provides the first national estimate of asthma prevalence, showed a definite increase in the prevalences of diagnosed asthma and persistent wheeze. This suggests that the prevalence increases reported by Morrison Smith et al (1976) and Burr et al (1989) were less likely to have been the result of local factors, but of factors more relevant generally. The increase in occasional wheeze was small compared with that of persistent wheeze. The authors suggest several possible explanations: a) if a substantial proportion of the population became slightly more prone to wheeze this would in turn lead to a greater increase in prevalence of severe disease; b) more severe disease resulting from a concomitant increase in atopy might explain the increase in mortality in adults (Burr 1987); c) less severe wheeze may have been overlooked, and d) a greater likelihood of mothers to report persistent symptoms.

Anderson (1989) addressed these issues. Aside from problems of definition and diagnosis, he pointed out that doctors vary in the way they label wheezy illnesses, that families vary in their willingness to accept labels, but that attitudes may change over time. He also noted that the then most recent estimate of the prevalence of wheezing from Nottingham in 1986 (Hill et al 1988) was similar to that in the first Aberdeen survey of 1964 (Dawson et al 1969). This suggests that the changes described over time were methodological rather than actual. He postulated that the significant rise in hospital admissions for asthma in children may be due to an increase in severity rather than a true rise in prevalence.

This evidence suggests that there has been a significant increase in parent reported prevalences of wheeze and in doctor diagnosed asthma. These increases are occurring throughout Britain but may not reflect true increases in prevalence; either an increase in parental awareness or an increased symptom severity could explain these observations.

Hospital-based reports

Increases in hospital admissions in children, based on discharge and death statistics, have been reported in Britain (Anderson 1978; Anderson 1989), New Zealand, Australia (Mitchell 1985) and the United States (Halfon and Newacheck 1986). In Britain, the most striking increase has occurred in children under 5; in the 14 year period from 1972 to 1986 there was an eight-fold increase in asthma admissions in this age-group. Increases for the 5 to 14 year age-group, though less spectacular, were also significant, as admission rates tripled (Burr 1987; Anderson 1989). Possible explanations for these observations include diagnostic transfer, an increase in the prevalence of asthma in the community, an increase in the number of admissions per patient, changes in admission practices with milder cases being admitted, or an increase in the severity of asthma.

Halfon and Newacheck (1986) reported on hospitalisation rates for acute asthma in children from 1970 to 1984. They noted that the increasing trend in hospitalisation for asthma seen between 1970 and 1979 continued following the adoption of the ninth revision of the International Classification of Diseases (ICD-9-CM) in 1979, whereas that of bronchitis which had not changed in the ten years prior to the adoption of the new classification, fell by 22 percent. They also cited evidence from other investigators that the new coding scheme had

resulted in a shift of 35 percent of cases from bronchitis to asthma (Mullaly et al 1984). This suggests that the increase in admissions for asthma is due partly to diagnostic shifts from other lower respiratory illnesses.

The possibility that the increase in admissions reflects a parallel increase in the community cannot be the complete answer as the observed percentage increases in admissions (Anderson 1989) are far greater than increased prevalences reported in the community (Burr et al 1989).

Possibly the best explanation for the rise in hospital admissions is changes in hospital practices with a shift of management from the community to the hospital. This has perhaps been influenced by changes in the management modalities available, e.g. nebulisers, increased parental expectations (Storr et al 1988), and easier access to hospital (Halfon and Newacheck 1986). This has been reflected in a rising trend in readmissions (Anderson 1978). In contrast, Mitchell (1985) has shown that in New Zealand readmissions constitute only 10 percent of the total increase in admission rate.

It has been suggested that the frequency and severity of asthma attacks has increased leading to more admissions to hospital (Mitchell 1985). This was challenged by Halfon and Newacheck (1986) who assessed severity in terms of length of stay in hospital. They showed a 26 percent decrease in admission time from an average of 5 days in 1970 to 3.6 days in 1984.

The increases in admissions to hospital for asthma in children may be influenced by all the factors discussed and not only by increases in asthma prevalence in the community. However admission rates per se provide a poor reflection of asthma prevalence in the community as many asthmatics are never admitted to hospital.

Differences in methodology and definitions

Much of the observed variations in prevalence may relate to differences in the definition of asthma and in study methodologies. Definitions have changed over time and consensus has not been achieved. The methodological differences relate to sample size which will influence the precision of the estimate, the age structure of the population sample studied, the duration

of the estimate - whether prevalence is recent or cumulative and the method of case identification (Usherwood 1987).

Definition of asthma

There is no agreed physiological, clinical or epidemiological definition of asthma that is comprehensive and exclusive, and therefore no uniform practical definition for the identification of cases of asthma in epidemiological studies (Sibbald 1986; Gregg 1986; Samet 1987). This is more relevant in adult populations where one seeks to distinguish between wheezing caused by asthma and that due to other conditions such as emphysema or chronic bronchitis. In children where asthma is the principal cause of wheezing there is less need for absolute precision (Samet 1987).

Expert groups and various individuals (Scadding 1984; Fréour 1987) have proposed definitions of asthma, but none have offered specific criteria for distinguishing asthmatics from non-asthmatics. Asthma is addressed largely in clinical and functional terms, dealing with features like widespread airway narrowing reversible spontaneously or in response to therapy, bronchial hyperreactivity and the clinical manifestations wheezing and breathlessness. Whilst helpful clinically, the usefulness of this approach in epidemiological studies is limited (Tager et al 1987).

This dilemma is illustrated in the following examples. Godfrey (1985) defined asthma as "a disease characterised by wide variations over short periods of time in resistance to flow in intra-pulmonary airways and manifest by recurrent attacks of cough or wheeze separated by symptom free intervals. The airflow obstruction and clinical symptoms are largely or completely reversed by treatment with bronchodilator drugs or steroids". Tabachnik and Levison (1981), state that "any infant with recurrent episodes of wheezing (three or more attacks) should be considered as having asthma, regardless of age of onset, evidence of atopy, apparent precipitating cause of the wheeze or frequency of the wheeze". The former definition is widely accepted but difficult to apply in epidemiological studies. In the latter the symptom wheeze identifies asthma. It is a more practical definition for epidemiological studies but obviously less specific.

Woolcock (1987) has suggested that the problem of defining asthma can be overcome by simply measuring the prevalence of those features known to be most characteristic of the disease; bronchial hyperresponsiveness and symptoms of wheeze and dry nocturnal cough. Breathlessness per se is not particularly helpful as it rarely occurs in asthma in the absence of wheeze.

Wheezy bronchitis vs asthma

A further difficulty in young children is the use of the diagnostic terms wheezy bronchitis and asthma. Many clinicians believe that a distinction should be made between infants who wheeze only in association with respiratory tract infections and those in whom episodes of wheeze are provoked by allergens and other stimuli, as well as by respiratory tract infections (Tabachnik and Levison 1981). The former have been diagnosed as having wheezy bronchitis or various other synonyms like spastic bronchitis, asthmatic bronchitis, asthmatoid bronchitis, obstructive bronchitis and pseudoasthma (Fouchard 1985). These terms have arisen firstly from a reluctance to diagnose asthma in such cases and reflect not only the lack of an agreed definition but also diagnostic denial by clinicians (Hart 1986) as 'wheezy bronchitis' might imply a temporary condition whereas asthma is viewed by both doctors and patients as a chronic and recurring problem (Levy and Bell 1984); and secondly because the traditional paediatric teaching has been that the word "asthma" should not be used lightly when dealing with young children who wheeze so as to avoid needless parental anxiety (Speight et al 1983).

However in their study of 7 year old children in Melbourne, Williams and McNicol (1969) were unable to separate children with wheezy bronchitis from those with asthma. As well as having similar clinical presentations, both 'conditions' were associated with significantly increased incidences of hay fever, skin-test sensitivity and nasal eosinophilia when compared with controls. Furthermore Sibbald et al (1980) showed that these two forms of wheezy illness demonstrate a strong similarity in their family histories. Both studies support the hypothesis that infants with recurrent wheezing, regardless of the precipitating cause, are all from the same population group with a common underlying basic disorder. Therefore it has been suggested that separating these two conditions confuses rather than clarifies knowledge about wheezing in infancy (Tabachnik and Levison 1981), leading to underdiagnosis and inappropriate or inadequate treatment (Speight et al 1983).

This view has recently been challenged. Wilson (1989) suggests that although there is considerable clinical and genetic overlap between these two groups, wheezy bronchitis may still be a separate entity. The failure of Williams and McNicol (1969) to identify it as such could have been related to the age at which their cohort was first seen as many children would have ceased to have episodes of wheeze precipitated solely by upper respiratory infections by seven years of age. The use of this term in older children may restrict the diagnosis of asthma (Speight et al 1983) and influence its treatment, but avoiding it may lead to inconclusive answers to certain questions, for example, whether environmental factors such as parental smoking have a different influence on wheeze precipitated by 'colds' and wheeze provoked by other precipitants. Provided treatment is appropriate for both conditions, an open approach to terminology may be justified at the present time.

Methodology

Age structure of study population

The age structure of the study population may influence the estimated prevalence of wheeze and asthma. In a prevalence study of wheeze in early childhood performed by reviewing general practice records of children at seven years of age, Strachan (1985) found an overall prevalence of wheeze of 31 percent but had explicitly considered episodes of wheezing during infancy. Twelve percent of his population sample had wheezed during their first year of life but only a half of these continued to do so, whereas 63 percent of those presenting after 3 to 4 years of age continued to wheeze. Similarly in the Melbourne study (Williams and McNicol 1969), as many as 30 percent of children with early onset wheeze became asymptomatic by the age of 8 years. Park et al (1986) showed that nearly 75 percent of children who wheezed before the age of 4 years were asymptomatic when questioned at the age of 10 years. These examples demonstrate that point prevalence estimates of wheeze after 5 years of age will significantly underestimate cumulative prevalence.

Furthermore the use of age to separate wheezy bronchitis from asthma might inappropriately exclude younger asthmatics (under 2 and 3 years old) in any estimation of the prevalence of asthma and thereby influence the results obtained. Lenney and Milner (1978) suggested that in children under the age of three years asthma and wheezy bronchitis are two different conditions which merge later into a single entity. Lee et al (1983) state that wheeze that does

not respond to bronchodilator therapy is so common in early infancy that it is best excluded in considering the cumulative prevalence of asthma. The exclusion of such cases might significantly underestimate the true prevalence, as both Blair (1977) and McNicol and Williams (1973) have found that more than half of children with asthma are symptomatic by 2 years and 80 percent by 3 years; in addition, in a general practice audit of all children in their care Levy and Bell (1983) found that 63 percent of children with asthma presented by 1 year of age, 86 percent by 2 years and all but one by 4 years.

Duration of prevalence estimate

The duration of the prevalence estimate, that is current (recent) or cumulative (ever), will influence the prevalence estimate. The studies undertaken in British children that report both current and cumulative wheeze and asthma prevalences are shown in Table 1.

As expected, cumulative prevalence estimates for both wheeze and asthma are higher than current estimates. The cumulative prevalences of wheeze in all, with the exception of a study in south west London (Johnston et al 1987), were approximately double those of current estimates. The pattern was similar for asthma where cumulative prevalences were 50 to 100 percent greater than the current estimates.

Table 1. Prevalences of wheeze and asthma in British children in relation to duration of prevalence estimate.

Year of study	Authors (first)	Geographical Area	Prevalence (%)			
			Wheeze		Asthma	
			Recent	Ever	Recent	Ever
1965	Anderson	National	8.1	18.3	3.1	
1968	Smith	Birmingham	5.5	10.1	2.3	4.3
1969	Peckham	National	4.9	12.3	2.0	3.5
1973	Burr	South Wales	9.8	17.0	4.2	5.5
1978	Anderson	SW London	11.1	18.2	3.3	4.0
1979	Lee	Newcastle	9.3	14.4	1.2	
1984	Johnston	SW London	15.0	17.0	5.0	
1986	Clifford	Southampton*	11.9	19.4	9.5	
1986	Clifford	Southampton+	12.3	18.3	9.5	
1988	Burr	South Wales	15.2	22.3	9.1	12.0

SW - South West. * 7 year-olds; + 11 year-olds.

Methods of case identification

Asthmatic children may be identified in epidemiological studies by questionnaire, by clinical case identification, or by physiological demonstration of reversible airways obstruction.

1. Questionnaire

Epidemiologists prefer to use questionnaires as they have marked advantages over physiological tests. They are cheap particularly if self-administered, do not require skilled staff, and can be delivered through the post to be completed in the subjects own time and convenience. In contrast, tests of lung function are time consuming, require skilled staff and often need patient cooperation. However, a major disadvantage of questionnaires is that the answers they elicit may be strongly dependent on factors other than disease state, e.g.

language and culture (Burney and Chinn 1987). A questionnaire based on symptoms rather than disease labels helps to minimise this problem.

The construction of a questionnaire which is simple and yet specific, reliable and useful is extremely difficult (Woolcock 1987). The criteria by which the questionnaire, or any other diagnostic tests, are assessed, are repeatability and validity (Samet 1978). Assessment of the former in the case of a questionnaire is straight forward; the latter assessment is more difficult and controversial as there is no diagnostic 'gold standard' for asthma. One alternative is to test the questionnaire against non-specific bronchial hyperreactivity (Burney and Chinn 1987).

Validation of respiratory questionnaires in children has been attempted by correlating replies and the results of ventilatory function and bronchial challenge tests. Mitchell and Miles (1983) compared forced expired volume in 1 second (FEV_1) and maximal mid-expiratory flow (FEF_{25-75}) to the answers to key questions on wheeze and productive cough. They were unable to show any difference in FEV_1 between asymptomatic children, children with cough and those with wheeze. However the mean FEF_{25-75} was significantly lower in the group with wheeze than in the other two groups, as was the FEV_1 and the FEF_{25-75} in children with both wheeze and cough compared with those with either or neither symptom. Salome et al (1987) investigated the relation of bronchial hyperresponsiveness (BHR) to inhaled histamine with respiratory symptoms and diagnosed asthma in Australian school-children aged 8 to 11 years. They found a good but incomplete association between symptoms and bronchial reactivity and suggested that inconsistencies are due to many factors including the intermittent nature of both symptoms and bronchial reactivity, symptom denial by parents, the possible detection by BHR testing of pre-symptomatic individuals, and that other conditions can cause bronchial hyperreactivity - including an upper respiratory tract infection per se.

Questionnaire reliability can be tested by administering the questionnaire to the same respondents on two occasions. Mitchell and Miles (1983) tested the reliability of their questionnaire by administering it to a sample of their respondents on two occasions nine weeks apart. There was an agreement of 97 percent for their question on wheeze and 91

percent for the question on cough. Similar degrees of agreement were also found by Salome et al (1987) and Clifford et al (1989) for key questions on wheeze and diagnosed asthma. However, parental recall may not be as perfect as the above suggests. Strachan (1985) compared data from parental questionnaires to General Practitioner (GP) case records, and found that the parental recall of early episodes of wheeze was incomplete and biased by frequency and severity of symptoms.

These studies on validity and reliability of questionnaires on respiratory symptoms confirm their potential utility with the reservation that there is no precise means of validating them, particularly in preschool children who may be unable to cooperate fully with tests of respiratory function and that early symptom recall by parents may be inaccurate (Schenker et al 1983).

Standardisation is a further issue concerning questionnaires. The American Thoracic Society has produced a standardised paediatric respiratory questionnaire (Ferris 1978). There has since been further collaborative work in Britain, Australia and New Zealand to produce an internationally acceptable paediatric respiratory questionnaire (Strachan personal communications 1990) that will hopefully facilitate comparison of results between different studies.

2. Case records

Examination of doctor case records has been used to estimate prevalences of symptoms and of asthma. Consultations with a record of symptoms of cough, wheeze or breathlessness, or of auscultatory signs in the chest, or the diagnosis of wheezy bronchitis or asthma are used to identify cases (Strachan 1985). This method is more sensitive than the use of diagnosis alone in identifying cases of asthma. The latter will underestimate prevalence as doctors will often not make a diagnosis if the presentation is not 'classical' (Marks and Hillier 1983). In their practice audit, for example, Levy and Bell (1983) found diagnosis to be delayed as over half of their asthmatic patients required more than 15 consultations for respiratory problems before being designated as such. In the same practice, with a special interest in asthma, only 11 percent were diagnosed in 5 consultations or less.

3. Bronchial hyperreactivity (BHR)

BHR is said to be the hallmark of asthma (Boushey et al 1980) but epidemiological studies show that this relationship, although significant, is not 'complete'. The prevalence of BHR has been shown to be higher than that of asthma but lower than that of wheeze or respiratory symptoms (Salome et al 1987). BHR as an epidemiological tool lacks specificity for asthma, is ill defined in terms of population distribution, varies (like symptoms) over time, and may be difficult to measure, particularly in young children (Lenney and Milner 1978, Anderson 1986, Enarson et al 1987). Because of these problems, Enarson et al (1987) concluded that 'identifying subjects with asthma in epidemiological surveys by measurement of BHR is not straight forward and that priority should be placed upon designing and validating an asthma questionnaire'.

Most prevalence studies in Britain have been conducted by parent-answered questionnaires, either at interview or by post, and from reports on audits of general practitioners records. Table 2 categorises the surveys by study method.

Table 2. Cumulative prevalences of wheeze and asthma in British children in relation to the study method.

Year of study	Authors (first)	Geographical Area	Subjects Number	Age (yrs)	Prevalence(%) Wheeze	Asthma
Questionnaire interview with parents and children:						
1964	Dawson	Aberdeen	2511	10-15	11.5	4.8
1965	Anderson	National	14571	7	18.5	3.1
1968	Leeder	NW London	2037	5	21.6	3.2
1968	Smith	Birmingham	20958	5-18	10.1	4.3
1969	Peckham	National	13557	11	12.3	3.5
General practitioner's records:						
1983	Den Bak	West Cumbria	655	<16		7.8
1983	Levy	Middelsex	470	1-11	11.0	
1983	Strachan	Edinburgh	437	7	31.0	
1983	Toop	Edinburgh	214	7		8.4
Postal questionnaire to parents:						
1973	Burr	Cardiff	817	12	17.0	5.5
1978	Anderson	SW London	5100	9	18.2	4.0
1979	Lee	Newcastle	2700	7	14.4	1.2
1981	Colver	Newcastle	2668	3-11	13.8	7.0
1984	Johnston	SW London	5287	5-13	17.0	5.0
1986	Clifford	Southampton	1274	7	19.4	9.5
1988	Burr	Cardiff	965	12	22.3	12.0
1989	Ninan	Aberdeen	2511	8-11	21.4	11.8

NW - North West; SW - South West.

The results show that, with the exception of the study by Strachan (1985) who included wheezing in infancy, the reported prevalences of both wheeze and asthma obtained from the three study methods were comparable.

However, the same cannot be said for the precise operational diagnostic criteria employed for determining cases of asthma and wheeze in community surveys. Table 3 lists examples of different diagnostic criteria employed and shows that the more 'relaxed' the criteria used for inclusion the greater the prevalence estimate. This is well demonstrated in prevalence estimates of wheeze and asthma in the same survey, for example, that of Leeder et al (1976) where the prevalence of 'one or more episodes of asthma' was only 3.2 percent compared with the prevalence of 'wheezy, whistling, or chesty episodes without asthma' of 21.6 percent.

Table 3. Cumulative prevalences of wheeze and asthma in relation to exact diagnostic criteria used.

Authors (first)	Diagnostic criteria	Prevalence(%)
Leeder	One or more episodes of asthma reported	3.2
Smith	History of asthma	4.3
Dawson	Confirmed diagnosis of asthma	4.8
Den Bak	Recorded diagnosis of asthma	7.8
Toop	Recorded diagnosis of asthma	8.4
Levy	Recorded diagnosis of asthma	11.1
Strachan	History of wheeze on one or more occasion	14.9
Anderson	Any history of wheeze	18.2
Leeder	Wheezy, whistling or chesty episodes	21.6
Strachan	Any history of wheeze (Case note review)	31.1

Prevalence trends for wheeze and asthma

Any comparison of results from prevalence studies must be carried out in the knowledge of the shortcomings outlined above - the differences in methodology and the limitations of the methods employed. To assess whether an increasing trend in the prevalence of wheeze or asthma in British children has occurred over time, surveys of similar methodology, i.e. cross-sectional questionnaire surveys, comparing like symptoms, i.e. wheeze with wheeze and asthma with asthma, from 1964 (Dawson et al 1969) to the latest survey of 1989 (Ninan et al 1992) are chronologically listed in Table 4.

The data shows that the reported cumulative prevalences of wheeze did not increase with time. One of the earliest studies (Leeder et al 1976) reported a prevalence equal to that of the two most recent reports (Burr et al 1989; Ninan et al 1990), even though there were age differences in the study populations. It appears therefore that there has been no clear-cut major change in symptom prevalence during the past twenty-five years.

However, unlike wheeze, the prevalence data for diagnosed asthma does show an increasing trend over time. The reported prevalences of asthma until 1980 were less than 5 percent, with one exception (Burr et al 1974) where a prevalence of 5.5 percent was reported! Since 1980 there has been a steep increase in the prevalence of asthma with the two latest surveys reporting prevalences of 12 percent, a 2.5 fold increase. As this increase was not seen with wheeze it would suggest that in Britain nationwide there has been a increasing tendency to diagnose children with wheeze as asthma resulting therefore in an increase in the prevalence of asthma but with no change in it's underlying symptom - wheeze. The increases in the prevalences of wheeze and asthma that have been observed from paired community-based studies (Morrison Smith and Cooper 1981; Burr, et al 1989; Ninan et al 1990) might therefore have resulted from changes in methodology rather than in prevalence. The increases in hospital admission rates (Anderson 1989) may reflect this increased tendency to diagnose asthma as the magnitude of the asthma prevalence increase observed of 2.5 times parallels the increase in admission rates for children over 5 years of age (Anderson 1989).

Table 4. Cumulative prevalences of wheeze and asthma in British children.

Year of study	Authors (first)	Geographical Area	Subjects		Prevalence(%)	
			Number	Age	Wheeze	Asthma
1964	Dawson	Aberdeen	2511	10-15	11.5	4.8
1964	Graham	IoW*	3300	9-11		2.3
1965	Anderson	National	14571	7	18.3	3.1
1967	Hamman	Kent	10971	5-14		3.8
1968	Leeder	NW London	2037	5	21.6	3.2
1968	Smith	Birmingham	20958	5-18	9.9	4.3
1969	Peckham	National	13557	11	12.3	3.5
1973	Burr	Cardiff	817	12	17.0	5.5
1975	Golding	National	12977	5	20.8	2.1
1978	Anderson	SW London	5100	9	18.2	4.0
1979	Lee	Newcastle	2700	7	14.4	1.2
1981	Colver	Newcastle	2668	3-11	13.8	7.0
1984	Johnston	SW London	5287	5-13	17.0	5.0
1986	Clifford	Southampton	1274	7	19.4	9.5
1986	Clifford	Southampton	1217	11	18.3	9.5
1988	Burr	Cardiff	965	12	22.3	12.0
1989	Ninan	Aberdeen	2511	8-11	21.4	11.8

*IoW - Isle of Wight; NW - North West; SW - South West.

Gender differences in prevalence

Most studies of early childhood asthma in developed countries (Fergusson et al 1983: Skarpaas and Gulsvik 1985; Holmgren et al 1989) report a preponderance of wheeze and asthma in boys over girls in a ratio varying from 1.2:1 to 2.2:1. In some reports from

developing countries this ratio is reversed with a female predominance (Carswell et al 1977; Van Niekerk et al 1979).

The childhood prevalence surveys conducted in British children which report on the gender differences observed show a consistent male predominance for both wheeze and asthma (Table 5), with ratios for wheeze ranging from 1.1:1 to 1.8:1 and for asthma from 1.2:1 to 2.2:1. The gender ratios for both symptoms are therefore comparable. There is also no evidence that either ratio is changing over time particularly in asthma where there has been a marked increase in reported prevalence. Here a parallel increase in prevalence in boys and girls is seen.

Table 5. Cumulative prevalences of wheeze and asthma in British children - gender differences.

Year of study	Authors (first)	Prevalence(%)					
		Wheeze			Asthma		
		Boys	Girls	M:F	Boys	Girls	M:F
1964	Dawson				6.7	2.9	2.2:1
1968	Leeder	22.5	20.7	1.1:1	3.4	2.9	1.2:1
1968/9	Smith	12.9	7.0	1.8:1	5.6	2.8	2.0:1
1969	Peckham	14.2	11.4	1.2:1	4.5	2.5	1.8:1
1973	Burr	21.3	12.6	1.7:1	7.0	4.0	1.8:1
1979	Anderson	21.1	15.3	1.4:1			
1983	Den Bak				8.6	6.8	1.3:1
1986	Clifford*	22.7	15.9	1.4:1	11.7	7.2	1.7:1
	Clifford+	19.9	16.7	1.2:1	10.7	8.4	1.3:1
1988	Burr	26.7	17.7	1.5:1	14.1	9.8	1.4:1

M:F - Male:Female; * 7 year-olds; + 11 year-olds.

NATURAL HISTORY

The clinical patterns of wheeze in childhood vary considerably and range from mild infrequent attacks with long symptom-free intervals to chronic wheeze with persistent symptoms. This wide range is widely regarded as expressing a continuum of the same disease process, namely, asthma (McNicol and Williams 1973; Godfrey 1983). Alternatively the population of wheezy children may be more heterogeneous. Wilson (1989) has suggested children with milder transient disease may well have wheezy bronchitis (wheeze associated with upper respiratory tract infection) rather than asthma.

Williams and McNicol (1969) observed two main trends in the natural history of wheeze in children. Forty eight percent had wheezing episodes that began between 2 and 4 years of age, recurred twice or thrice yearly over a period of 2-5 years and then stopped spontaneously or became less frequent after the age of 7-8 years. The remaining 50 percent had more frequent severe and prolonged episodes with a minority (about 1 in 40 asthmatics) having persistent symptoms and evidence of airflow limitation between attacks. Their symptoms were less likely to abate with time. One shortcoming of this longitudinal study describing the natural history of wheeze (Williams and McNicol 1973) is that index cases were identified when the children were 7 years of age or older. This could have led to an underestimation of the proportion of milder cases, particularly those presenting earlier in life, with resolution of symptoms before 7 years of age.

To predict the natural history and outcome of wheeze and asthma in children, many associated features should be considered:

1. Age of onset

Early age of onset is characteristic of childhood asthma. There are contradictory reports of its effects on outcome. Early age of onset has been held to have no effect on prognosis (Blair 1977; Park and Golding 1986), to improve prognosis (Strachan 1985; Sporik et al 1991) or to make it worse (McNicol and Williams 1973). In the Melbourne study of McNicol and Williams (1973) most children with mild disease presented after the age of 2 years whilst those with more severe asthma presented earlier. In the latter group 28 percent had wheezed

before the age of 6 months and 60 percent had wheezed by 2 years. However, the cohort was not recruited until the children were 7 years old. It is probable that some mild or infrequent wheezers who presented at an early age and whose symptoms had resolved were omitted because of incomplete parental recall, thereby obscuring the true picture. This potential limitation of studies in school-aged children was observed by Strachan (1985) when he compared parental recall and family-doctor records. Blair (1977) found that 39 percent of asthmatic children presented by one year, 77 percent by 2 years, and 84 percent by 5 years. Early age of onset did not affect prognosis at any stage.

2. Frequency of attacks of asthma

An increased frequency of wheezing episodes, particularly in the first year of life, is associated with a greater risk of persistent symptoms at an older age; that is the more severe the symptoms in the early years the more likely the occurrence of chronic asthma (Williams and McNicol 1973; Martin et al 1982; Anderson et al 1986). Similar findings have been reported in a follow-up study of preschool children aged 1-4 years (Park et al 1986). An increase in frequency before one year was more predictive than frequency data after 1 year.

3. Family history of atopy

Initial severity and final prognosis have been shown to be significantly worse in subjects with a positive family history of atopy. The occurrence of atopic disorders in first-degree relatives was the criterion applied (Blair 1977). In addition, Young et al (1991) demonstrated that this predisposition, as demonstrated by increased airway responsiveness to histamine, was present soon after birth in children with a positive family history of asthma. They suggested that functional differences were present before any symptoms in those with a genetic predisposition. This also appeared to be more strongly associated with a maternal history of atopy and asthma.

4. Effect of gender

In most prevalence studies of childhood asthma in developed countries boys predominate with a male to female ratio of about 3:2. There have been contradictory findings of the effect of gender on the natural history of wheeze and asthma. Blair (1977) found that the gender of asthma patients recruited before 12 years of age and followed up for 20-24 years did not

affect the severity of asthma either at onset or follow-up. However, McNicol and Williams (1973) showed that male predominance increased with increasing severity of asthma at presentation whereas in the milder asthmatics sex incidence was equal or increased in girls. A follow-up of this cohort (Martin et al 1982) showed a trend that has also been observed by others (Åberg and Engström 1990), namely, a drop in the sex ratio to unity in the early teens and then a female preponderance in adulthood. Boys tended to show greater improvement with time than girls and more girls than boys presented for the first time in adolescence.

5. Atopic disease in the patient

Blair (1977) showed that seasonal or perennial rhinitis aggravated asthma severity both at inclusion and completion of his long-term follow-up studies.

Infantile eczema is more frequent in asthmatic children than in non-asthmatic controls. It is predictive of severity of asthma and of persistence of wheeze (Williams and McNicol 1973; Blair 1977; Godfrey 1983; Anderson et al 1986; Åberg and Engström 1990; Sporik et al 1991).

Thus the factors associated with persistence of symptoms in childhood asthma are severity at onset, family or personal history of atopy and female gender.

Influence of puberty

There have been several reports of the influence of puberty on asthma severity. In a prospective study Balfour-Lynn (1985) documented the progress of 38 children with chronic perennial asthma and related disease severity to pubertal status. At the completion of puberty 17 percent of severe cases and 54 percent of the moderate-to-mild group had become symptom-free. The overall decrease in severity of asthma during puberty was preceded by a similar tendency in the three years prior to the onset of puberty in the milder grades only. These results are consistent with those of Martin et al (1980) who reassessed the Melbourne cohort at age 21. Thirteen percent of the severe group and 55 percent of the moderate-to-mild group had become symptom-free. Relapses were seen in both surveys in about a quarter of the cases studied and at average age of 18 years (Blair 1977; Martin et al 1980).

SEVERITY OF WHEEZE AND ASTHMA

The severity of asthma in children has generally been based on the number of attacks of wheeze experienced by the child. McNicol and Williams (1973) classified their asthmatic children when reviewed at 14 years of age into four groups: those who had had no more than 5 wheezy episodes; those with more than 5 wheezy episodes but none within the preceding 12 months; those with a continuing history of episodic asthma over a period of years and who had had symptoms within the preceding 12 months; and those with a current history of very frequent or chronic unremitting asthma, with either prolonged periods of severe symptoms during the last year with remissions of less than one month or more than 10 attacks during the last 3 months. The four groups were assessed for clinical, radiological and spirometric evidence of persisting airways obstruction and pulmonary hyperinflation. These objective assessments of disease severity correlated with the symptom classification, confirming that wheeze frequency was a reliable measure of asthma severity.

In a national cohort study, Park et al (1986) found that the number of attacks of wheeze experienced between the ages of 1 and 4 years was significantly associated with prognosis at 10 years. Thirteen percent of those who had had one attack of wheeze during this period were diagnosed asthma at 10, compared with 33 percent who had had 4 or more attacks. In addition, the greater the number of attacks before a year of age the more likely the child was to have asthma at 10, but this association was not as strong as that seen for wheezing between 1 and 4 years. The age at first attack did not influence outcome at age 10.

In a 20 year follow-up study on the natural history of asthma, Blair (1977) also based the initial assessment of severity on the number of episodes of wheeze reported, the milder group having no more than three attacks of wheeze per year and the severe group more than three. The children entering the study were under 12 years of age. At initial assessment 49 percent were found to have 'mild' asthma and 51 percent 'severe' asthma. The final prognosis was influenced by severity.

In the follow-up of the Melbourne cohort (Martin et al 1982) factors found to be predictive of wheezing at 21 years of age were multiple episodes of prolonged wheezing in the first two years of life, frequent wheezing at 10 years, or persistent wheezing at 14 years of age.

Therefore wheeze frequency during childhood is a valid index of asthma severity (McNicol and Williams 1973) and an accurate predictor of prognosis (Park et al 1986).

In their description of severity of asthma, Williams and McNicol (1973) found the breakdown of their asthma population to be 20 percent, 28 percent, 34 percent and 18 percent respectively in the four groups described. This distribution is similar to that described by Blair (1977), with about half suffering from 'mild' disease and half more 'severe' asthma.

PRECIPITANTS OF WHEEZE OR ASTHMA ATTACKS

Several studies have shown that the most common precipitant of wheezy episodes in infants and young children is viral infections of the respiratory tract (McIntosh et al 1973; Minor et al 1974; Horn et al 1979; Tabachnik and Levison 1981; Foucard 1985). In addition, viral respiratory tract infections are probably the most common cause of attacks of asthma in children of all ages (Ellis 1983).

Viral infections, diagnosed either by culture or serology, have been demonstrated in up to 50 percent of acute wheezy episodes in children. However these figures probably underestimate their true prevalence of viral precipitants as some of the respiratory viruses are difficult to culture (Tabachnik and Levison 1981; Foucard 1985). The viruses most commonly demonstrated in children with acute attacks of wheeze are respiratory syncytial virus in infants (McIntosh et al 1973); and rhino virus in older children (Minor et al 1974), although parainfluenza virus and mycoplasma pneumoniae are also implicated to a lesser degree.

In the two questionnaire surveys conducted 15 years apart in 12 year old school children in South Wales (Burr et al 1974; Burr et al 1989), 'colds' were identified as a cause of attacks of wheeze in over 90 percent of wheezers in the earlier survey and in nearly 80 percent in

the later survey. The corresponding figures of other identified 'precipitants' for the two surveys were 34 percent and 47 percent for running, 10 percent and 16 percent for animal contact and 1 percent and 7 percent for foods. The increased proportion of wheezing episodes attributed to running and animal contact was thought by the authors to be suggestive of a real rise in the prevalence of asthma during this time whereas that attributed to foods might have reflected greater publicity given to food allergy over the same time period.

Wilson (1985) investigated food-related asthma in Asian and non-Asian, mostly Caucasian, asthmatic children aged 3-17 years who had been referred to a hospital asthma clinic, by postal questionnaire and personal interview. She found that either wheeze or cough occurred in response to at least one item of diet in 91 percent of Asian children and 58 percent of Caucasian children, a highly significant difference. More than 13 different items in the diet were identified as causing symptoms, with cola drinks the commonest culprit in Asian children and orange/lemon squash in Caucasian children. This report suggested that food as a trigger of asthma is much more common than has been generally accepted, particularly in Asian children.

WHEEZE AND LOWER RESPIRATORY INFECTIONS

A history of a previous lower respiratory tract infection, particularly bronchiolitis but also croup, pneumonia and bronchitis, is a risk factor for the subsequent development of wheeze and asthma (Tabachnik and Levison 1981; Mok and Simpson 1982; Ellis 1983; Sherman et al 1990).

In a case-controlled follow-up study of children who had had respiratory syncytial virus proven bronchiolitis in infancy, the prevalence of the respiratory symptoms - cough and wheeze - at seven years of age was higher in the index children than controls (Mok and Simpson 1982), an association which has been observed by others (Tabachnik and Levison 1981).

More recently, in a longitudinal study to identify potential risk factors for the development of asthma, Sherman et al (1990) followed a cohort of 770 children aged 5-9 years from 1975 to 1988. They identified a history of pneumonia and a history of bronchitis independently to be associated with the subsequent development of asthma. However in this cohort neither bronchiolitis nor croup were associated with the development of asthma, possibly because the subjects were only studied prospectively from the age of 5 years, long after the peak occurrence of bronchiolitis and croup.

TREATMENT OF WHEEZE AND ASTHMA

In developed countries asthma in children tends to be underdiagnosed and undertreated. This hypothesis was initially tested in Newcastle-upon-Tyne by Speight et al (1983) in a questionnaire survey of 7 year old school-children. A sample of the children who had previously been identified to have wheezed since starting school (Lee et al 1983) were reviewed, noting particularly severity, duration and frequency of wheezy episodes, the diagnostic label, and the treatment the child was receiving or had received from his or her GP. Of the 165 children studied who had visited their GP with chest symptoms suggestive of asthma, the diagnosis asthma had been offered to the parents of only 21 (12 percent), including three of 56 who had experienced 4-12 wheezy episodes a year, and 11 of 31 with more than 12 episodes a year. Six of the latter were diagnosed asthma only after admission to hospital but a further 25 children who had attended hospital with such chest symptoms were not so diagnosed. Among those in whom asthma was not diagnosed, 12 were diagnosed as having wheezy bronchitis, 6 as an 'allergic' chest condition, 60 as recurrent bronchitis or 'chestiness', 23 as recurrent colds or viral infection, and in the remaining 43 no diagnosis was made. Treatment with bronchodilator drugs had never been given to two-thirds of wheezy children, including nearly 80 percent of those who had experienced 4-12 wheezy episodes per year and nearly one third of those with more than 12 episodes a year. This lack of recognition and therefore failure to treat asthma was associated with significant morbidity as 62 percent of children had lost more than 50 days of schooling due to wheeze. As school absenteeism from all causes at this age averages 16 days a year, this constituted more than 3 times the usual amount of school absenteeism.

Several subsequent studies have confirmed that asthma in children is underdiagnosed and undertreated. In a survey of 9 year old school-children in the London Borough of Croydon, Anderson et al (1983) found that only a quarter of children reported to have had a wheezing illness were diagnosed as asthma. The label asthma, as observed by Speight et al (1983), was significantly associated with severity - rising from 22 percent in those with no school absence to more than 50 percent in those children who had missed more than 30 days of schooling because of wheeze. This study did not report treatment received by the children, but does indicate significant morbidity in a large proportion of wheezy children not recognised as having asthma.

The circumstances surrounding hospital admissions for acute asthma were examined by Conway and Littlewood (1985). They found that a third of admissions with previous symptoms of asthma had not been diagnosed as such and had not received adequate medication. In addition, even when asthma was diagnosed, half of such children received ineffective treatment. These results suggest that even among wheezing children requiring hospitalisation, asthma is significantly underdiagnosed and undertreated.

In a more recent report of 5-11 year-old Nottingham school-children Hill et al (1989) reported improvement in the rate of diagnosis of asthma with 47 percent of recurrent wheezers so diagnosed. Although doctors may now be more willing to diagnose asthma in wheezy children the condition is still undertreated as 70 percent of those who had lost more than 10 days from school were receiving inadequate treatment.

Several explanations have been given for the failure to recognise and treat asthma appropriately. Levy and Bell (1984) acknowledge their own resistance to diagnosing asthma. In an audit of their own practice they showed that half of their asthmatic patients were diagnosed after more than 15 consultations and only 11 percent after 5 or less - an experience similar to that of Hart (1986) who found that 30 percent of asthmatics attending his clinic were diagnosed after more than 15 respiratory consultations and only 18 percent after 5 or less consultations. An important explanation for the reluctance to diagnose asthma may be that it is regarded by doctors and patients as a recurring, even chronic, problem whereas the diagnosis 'wheezy bronchitis' implies a temporary illness.

Jones and Sykes (1990) offer an alternative explanation on the basis of a questionnaire study of children with asthma in their practice. They found that even in an 'asthma-aware' practice there was a delay of approximately 40 percent of the total age of the child at the time of diagnosis - a time period that does not change up to the age of 10 years. The ascribe the delay in diagnosis primarily to the nature of the presenting symptoms of the child, with bronchitis more likely to be diagnosed if the dominant symptom is recurrent cough.

Therefore, some ten years after Speight et al (1983) drew attention to the problem of failure of diagnosis and treatment of childhood asthma, there continues to be evidence that this problem has not been resolved.

'AETIOLOGY' OF ASTHMA

Asthma is a multifactorial disease with complex intertwining of genetic and environmental components. This has hampered attempts to study the aetiology of the condition and contributed to the lack of a universally agreed definition that would distinguish it from other conditions characterised by wheeze.

As wheeze and asthma present in most children in the preschool years, the environment to which they have the greatest exposure - the indoor environment - has been extensively investigated. The components of this which have been considered include nitrogen dioxide from gas cookers and heaters, damp and mould, pets, parental smoking and home location. Genetic and environmental elements, including social factors, and their association with respiratory symptoms in children are considered below.

FAMILY FACTORS

Degree of heritability of asthma

In considering the inheritance of asthma, the problems of clinical definition and recognition of asthma are compounded by the clinical manifestations of atopy. This was exemplified in a genetic study by Cookson and Hopkin (1988) who found that among designated atopics, based on immunological criteria, 85 percent had symptoms compatible with atopic disease

compared with 13 percent of non-atopics. However, of the designated atopics with documented symptoms, only 30 percent gave a positive response in a questionnaire to the question 'Have you ever suffered from asthma, hay fever or eczema?'. Thus only a quarter of individuals with an atopic potential report these symptoms (Hopkin 1989).

Several studies have investigated the relative contributions of genes and environment to the development of wheeze and asthma. The genetic influence has been assessed in twins by examining the concordance or degree of similarity in monozygotic twins compared with dizygotic twins. Assuming that the environmental effect on both types of twins is the same, any significant increase in the concordance amongst monozygotic twins is evidence of a genetic influence (Sibbald 1986). In a study of 7000 twin pairs in Sweden, Edfors-Lubs (1971) showed that concordance for asthma was 19 percent for monozygotic twins and 4.8 percent for dizygotic twins, thereby yielding an overall heritability of 15 percent. The population prevalence in this study was 3.8 percent. A study of 3808 twin pairs almost twenty years later by Duffy et al (1990) supported these findings, even though the prevalence of asthma in the Australian study was 4 times that of the Swedish study. Concordance among monozygotic twins was 29 percent compared with 13 percent in dizygotic twins, an overall heritability of 16 percent. The population prevalence in this study was 13.2 percent, a figure similar to that reported in previous epidemiological studies (Salome et al 1987). The genetic correlation for asthma, and in both twin studies for hay fever, was far less than unity. This suggests that additional influences, either environmental or genetic, are present in both diseases. In their report on the identification of a single dominantly inherited gene controlling IgE-responsiveness, Cookson et al (1989) showed that 85 percent of those diagnosed as carrying this gene had an atopic disease such as hay fever and that 20 percent were asthmatics. This suggests that some 80 percent of the variability seen in the expression of asthma in these individuals is controlled either by environmental effects or genes at other loci.

Influence of atopy on the inheritance of asthma

Following the work of Edfors-Lubs (1971) which established the degree of heritability of asthma, it was questioned whether or not asthma is simply a manifestation of atopy. Pepys (1973) showed that asthmatics with greater incidences of multiple skin reactions had first degree relatives with higher prevalences of asthma, eczema and hay fever. The influence of

asthma could not be separated from that of atopy. In a later study Gerrard et al (1976) showed that the prevalences of asthma, hay fever and eczema in children increased when their parents were affected by one or more of these conditions, but that they were more likely to develop the same allergic condition as their parent.

Fergusson et al (1983) supported this hypothesis in a birth-cohort study of 1110 4-year old children where the parent/child relationships for both asthma and eczema were investigated. They found that parental asthma was associated with asthma in boys but not with eczema in either boys or girls; parental eczema was associated with eczema but not with asthma in children of both sexes. In addition there was a tendency for asthma and eczema to occur together in both parents and children.

The influence of atopy on the inheritance of wheeze in children was studied further by Sibbald et al (1980) who examined the family histories of asthmatic, wheezy bronchitic and control children aged 1 to 12 years. They found that significantly more asthmatic and wheezy bronchitic probands than controls had at least one first degree relative with asthma, and that wheezy bronchitis tended to be more prevalent in both wheezy groups than in controls. The family history of wheezy bronchitis was similar to that of asthma. However the prevalences of hay fever and eczema were lower in wheezy bronchitic than in asthmatic probands, differences which may in part have arisen from the slightly lower age of the former; other studies of children of comparable age have also found that atopy and allergy are less prevalent in wheezy bronchitics (Williams and McNicol 1969). These similarities between the two wheezy groups suggest that they share a common genetic defect, but the manifestation of asthma may be influenced by atopy which is not essential for the development of wheezy bronchitis.

Sibbald et al (1980) also compared the prevalences of asthma and atopy in the relatives of children with and without asthma. In the relatives of the asthmatics, the prevalence of asthma was significantly higher in those with atopy; and atopic asthma was more common than non-atopic asthma irrespective of the atopic status of the proband. In contrast, in the non-asthmatic controls atopic status influenced neither the prevalence of asthma in the first degree relatives nor the atopic status of the asthmatic relatives.

These findings support the hypothesis that asthma (or recurrent wheeze) and atopy (eczema/hay fever) are inherited independently but that atopy may enhance the expression of asthma. They might also explain, at least in part, variations in age of onset of symptoms of asthma and changing gender differences in asthma prevalences with age.

Influence of gender on the inheritance of asthma

There have been conflicting reports on sex differences in the inheritance of asthma. Sibbald (1980) studied the prevalence of asthma in male and female relatives of males and females with either atopic or non-atopic asthma (age not specified). In both groups the prevalences of asthma did not differ between male and female relatives of the male and female subjects. This uniformity in the distribution of asthma showed that neither form of asthma is sex-influenced. She concluded that the sex differences observed in many clinical populations must be due to factors other than those associated with the genetics of sex determination.

Fergusson et al (1983) in their cohort study did show that the child's sex influenced the size of the association between parental asthma and early childhood asthma. A parental history of asthma was significantly associated with early childhood asthma in boys while in girls parental asthma and early childhood asthma were unrelated. As these findings are in apparent conflict with those of Sibbald (1980) in older patients, the authors suggest that although the child's sex does not influence the inheritance of tendencies to asthma it does influence the age of onset. Therefore asthma in girls was apparently not related to parental asthma because by 4 years they had not yet started to wheeze. Parental eczema was associated with eczema in boys and girls. There were no parent/child relationships between unlike conditions i.e. asthma-eczema, eczema-asthma.

These findings contrast with those of Davis and Bulpitt (1981). In a study of the relationship between wheeze and atopy in children under 15 they found that parental atopy was associated with an increase in wheeze prevalence only in the boys, and that parental atopy increased atopy only in the girls.

Gender differences in the influence of atopy on the manifestation of wheeze have been reported by Duffy et al (1990). They showed that atopy influenced the prevalence of wheeze

in females and not males. They suggest that there are different patterns of inheritance in men and women, men showing evidence of a non-additive genetic component whereas in women an additive component is evident - thus unlike conditions (e.g. asthma and hay fever) had an influence on the other's inheritance in females (additive) but not in males, whilst like conditions (e.g. asthma-asthma or hay fever-hay fever) influenced the inheritance of the same in males.

It seems clear that for both asthma and atopy there are overlapping genetic components, the nature of which have yet to be fully elucidated. Gender differences may be relevant to the clinical expression of these interactions.

Summary of findings

The findings concerning the inheritance of asthma and atopy can be summarised as follows:

1. Studies of the inheritance of asthma and atopy using questionnaires are limited by the lack of uniform definitions as well as the lack of recognition of their condition by respondents (Cookson and Hopkin 1988).
2. Twin studies have shown an overall heritability of asthma of 15% (Edfors-Lubs 1971; Duffy et al 1990).
3. The contributions of asthma and atopy could initially not be separated when considering their influences on the development of asthma (Pepys 1973).
4. The allergic conditions asthma, hay fever and eczema influence one another's development but individually have a greater influence on their own development - thus the influences of each condition could subsequently be separated (Gerrard et al 1976).
5. Atopic asthma and wheezy bronchitis seem to share a common genetic mechanism although asthma, but not wheezy bronchitis, may also be influenced by atopy (Sibbald et al 1980).
6. In atopic asthmatic children the prevalence of asthma in first degree relatives was higher than in the relatives of non-atopic asthmatics (Sibbald 1980).
7. Gender influences the inheritance of atopy and wheeze, despite earlier evidence (Sibbald 1980) to the contrary. It seems that the influence of unlike conditions may be greater in females than males but that in males there is a strong inheritance of like conditions

(Fergusson et al 1983; Duffy et al 1990). These findings are not however uniform (Davis and Bulpitt 1981).

NITROGEN DIOXIDE

Association of gas cooking with respiratory symptoms

Early studies on British (Melia et al 1979) and American (Speizer et al 1980) primary school children reported associations between respiratory illness and exposure to nitrogen dioxide (NO_2) from the use of gas for cooking. However subsequent investigations, including one by the same British group (Melia et al 1982) have failed to confirm this association.

In a survey to establish the relationships between 12 features of the home environment and respiratory morbidity in 165 seven year old children, Strachan and Elton (1986) were unable to demonstrate any association between the use of unvented gas-fired appliances in the home and respiratory symptoms reported by parents - wheeze or nocturnal cough - or general practice records of lower respiratory tract illness before or after 5 years of age. They suggest that although the earlier studies had shown associations between gas appliances and respiratory symptoms, the relative risks quoted were small and might therefore have been missed in a study of this size. A similar conclusion was reached by Melia et al (1982).

In a much larger study of 4000 children aged 5 to 14 years aimed at identifying home environmental risk factors for childhood respiratory disease, Schenker et al (1983) found that gas cooking stoves didn't constitute an independent risk factor for any of the following chronic respiratory symptoms or illness - cough, persistent wheeze, chest illness within the last year, chest illness before two years of age or doctor diagnosed asthma. Similarly in the American Six Cities study (Ware et al 1984), no association was demonstrated between gas cooking and respiratory symptom prevalences or with tests of respiratory function.

Measured NO_2 levels and respiratory symptoms

None of the above studies quoted the level of NO_2 exposure. This question was addressed in a study of the effects of environmental factors on respiratory health in Dutch school children aged 6 to 12 years (Dijkstra et al 1990). NO_2 levels were measured in most rooms in the houses concerned on two occasions over two years. No association was found between

NO_2 levels and the occurrence of cough, wheeze or asthma, or tests of ventilatory function. Similarly there was no consistent relationship between NO_2 exposure and the development of symptoms over the time of the study. In addition there were no gender differences. In contrast, Melia et al (1983) found that although there was no significant association between gas exposure and symptoms, the effect was consistently positive in the boys and absent in the girls. This suggested a possible gender difference.

Several explanations for these negative findings have been suggested. Exposure might have been too low to cause detectable health effects. In less than 10 percent of the measurements recorded by Dijkstra et al (1990) was the weekly average NO_2 concentrations greater than 60 micrograms per cubic meter ($\mu g/m^3$). As the peak concentrations exceed weekly average concentrations by about 4 to 6 times, it was concluded that only a limited number of homes would have been exposed to peak concentrations greater than the accepted World Health Organisation upper safe limit of 400 $\mu g/m^3$. Alternatively the exposure criteria based on weekly average concentrations may not adequately represent exposure to the potentially harmful short-term peak concentrations of NO_2. Relevant exposure might therefore not have been well characterised. However as most recent studies have consistently failed to show any association between respiratory morbidity in children and exposure to NO_2 from gas stoves, the former explanation is more plausible.

PETS

Association of pet ownership with respiratory symptoms

In Swedish school-children, respiratory symptoms following exposure to animals were reported in approximately 60 percent of asthmatics and only 5 percent of the general population (Kjellman and Petterson 1983). More recent population surveys of healthy school-age children showed no significant association between pet ownership and any respiratory symptom (Andrae et al 1988; Clifford et al 1989) or diagnosed asthma (Horwood et al 1983). Therefore pet exposure does not seem to influence the prevalence of respiratory symptoms in the general population but does significantly influence the development of symptoms in children with asthma.

Influence of pets on symptoms in asthmatics

In asthmatics, allergy to animal danders, particularly to cats, has been shown to increase significantly with age (Vanto and Koivikko 1983; Murray et al 1983). The higher prevalence of cat allergy was not because of greater prevalence of cats in the households as dog ownership was commoner, but because of increased intimacy of exposure - cats were shown to go more commonly into the house and into the bedroom (Murray et al 1983). Furthermore, asthmatic children with allergy to animal danders had higher rates of hospital admissions and medicine intake than those without allergies, perhaps because allergic asthma is more severe or because more severe asthmatics are more aware of their allergies (Kjellman and Petterson 1983).

Magnitude of pet exposure

Pet ownership is common. In Sweden, Kjellman and Petterson (1983) found that 52 percent of homes owned pets, 39 percent in the urban areas and 77 percent in the rural areas. Regular direct contact was slightly more common - 57 percent overall, 44 percent in urban and 84 percent in rural areas. In a study of asthmatic children in Leeds, 55 percent had close pet contact. In addition, in a survey of 150 schools in the same area, 70 percent of these schools kept furry animals (Sarsfield et al 1976). The authors emphasize that school is a source of exposure that must not be ignored. Animal danders, particularly dogs, are therefore so common in the community that sensitive individuals need not keep or have direct contact with a pet to develop allergy (Vanto and Koivikko 1983).

DAMP AND MOULD

Any association between damp and mould in the home and respiratory symptoms or illnesses is complicated by their frequent coexistence with other factors potentially detrimental to respiratory health - parental smoking, overcrowding, types of heating and lower social class. However an association has been demonstrated in studies that controlled for the putative confounding effects of these environmental factors (Strachan and Elton 1986; Andrae et al 1988; Platt et al 1989; Brunekreef et al 1990; Dijkstra et al 1990).

Parental reporting bias

A further problem, particularly in questionnaire surveys, is that of parental reporting bias. Parents who are concerned about the presence of dampness or mould growth in the home might report respiratory symptoms at a lower threshold of severity than others; those with an asthmatic child might be more aware of potential hazards in the home; and others who desire to be rehoused might report respiratory symptoms and damp/mould at a lower threshold (Strachan 1991).

Evidence of a reporting bias has come from studies that have failed to validate symptoms by objective measures. In a study of 7 year old children in Edinburgh the association of mould in the home with parental reports of wheeze was much stronger than with general practitioner consultations for lower respiratory illness in the same children (Strachan 1988). Thus differential symptom reporting might contribute to the observed association between mould and wheeze in questionnaire surveys. In the American Six Cities study of school-children, the observation that more than half of the children with parental reports of persistent wheeze did not report doctor diagnosed asthma may be evidence of the same phenomenon (Brunekreef et al 1989). In another Edinburgh survey the finding that a reduction in FEV_1 after exercise was only weakly associated with mouldy housing despite the reported association between mould and wheezing may be a further reflection of differential reporting (Strachan 1988). The Six Cities study also found little effect of home dampness and mould growth on FEV_1 but there was a significant reduction in maximal mid-expiratory flow rate (FEF_{25-75}) among children from mouldy homes. (Mid-expiratory flow rates were not reported in the Edinburgh study but were subsequently quoted to show the closest association with dampness and mould growth (Strachan 1991)). The American authors (Brunekreef et al 1989) suggest that the lack of association in the Edinburgh study was due to over-reporting of symptoms as a 20 percent prevalence difference was present between affected and unaffected homes compared with only 6 percent in America. Over-reporting of wheeze in affected homes may have diluted the association between wheeze and pulmonary function status.

The validation of parental reporting of housing conditions has been carried out by environmental health officers. In the Swedish study on the effect of home conditions on respiratory symptoms there was 100 percent agreement between parental reporting of damage

due to damp and mould and inspection of homes by experienced health inspectors (Andrae et al 1988). Others have shown a significant and positive association between these subjective and objective evaluations but with disagreement in nearly a third of cases (Platt et al 1989).

Objective humidity and spore measurements and symptoms

Objective measurement of humidity and spore counts and their associations with respiratory symptoms have shown inconsistent results. Higher measured humidity showed a significant association with reported symptoms in one study (Platt et al 1989) but not in another (Strachan and Elton 1989). The latter authors suggest that airborne humidity is a poor indicator of the exposure risk.

Spore counts have also been inconsistently associated with respiratory symptoms. A higher total count in the homes of wheezy children was noted in one study (Platt et al 1989) but not in another (Strachan et al 1990). In addition, a poor correlation was found between spore count and mould growth in homes in three cities in Britain (Platt et al 1989). Furthermore the distribution of the main species of fungal spores appears to be remarkably similar in the homes of wheezy and non-wheezy children (Strachan et al 1990). Thus airborne spore counts may not be the relevant risk indicator to respiratory health.

Association of reported damp and mould with respiratory symptoms

Despite these limitations, reported household damp and mould have consistently been associated with increased prevalences in respiratory symptoms. Damp and mould have been associated with wheeze, persistent cough, exercise-induced cough, nocturnal cough, lower respiratory illness, bronchitis and asthma (Strachan and Elton 1986; Brunekreef et al 1989; Strachan et al 1989, Andrae et al 1988). In the Six Cities study the weakest association was with asthma, possibly because steps have been taken to eliminate damp in families where asthma has been diagnosed (Brunekreef et al 1989).

The associations between damp/mould and upper respiratory symptoms are less consistent. In some studies significant associations with hay fever, (Andrae et al 1988; Brunekreef et al 1989) and sore throats (Strachan et al 1989) were demonstrated. However Strachan (1988)

found no association with ear trouble and sore throats and an inconsistent association with hay fever.

There are several mechanisms whereby dampness might influence the prevalences of respiratory symptoms. It encourages the growth of house dust mites, allergy to which is thought to be of aetiological significance in the development of asthma in mite-sensitive individuals (Burr et al 1980; Sporik et al 1990). Many episodes of wheeze in young children are precipitated by viral upper respiratory infections and relatively high humidity may encourage the spread of the airborne viruses in droplet spray. Dampness also provides the environment conducive to germination of fungal spores.

A causal relationship between airborne moulds or spores and respiratory symptoms in children might operate in different ways. The respiratory manifestations, as with pollen allergy, may be atopic and cause disease only in susceptible individuals. This association would also apply to outdoor mould exposure and perhaps obscure any differences in the proportion of wheezy children coming from mouldy and non-mouldy homes. Alternatively, domestic mould growth might influence the cumulative burden of spores causing sufficient variation in individual exposure to result in differential rates of disease. However, spore counts, as already mentioned, have been inconsistently associated with increases in respiratory symptom prevalences (Strachan 1991).

A dose-response relationship has been observed between damp/mould and respiratory symptoms. Platt et al (1989) examined the dose-response relation between the children's symptoms and increasing severity of dampness. This was significantly associated with wheeze, sore throat and persistent cough. Strachan (1988) reported similar findings. Children with damp or mould in the home had significantly higher prevalences of wheeze and cough than those in dry homes and prevalences were even higher if the child's bedroom was affected.

As with smoking, the association of damp/mould in the home with lower respiratory symptoms meets many of the criteria for an epidemiological association to be considered causal: it is strong, relatively specific when compared with upper respiratory symptoms,

consistent in most studies, shows evidence of a dose-response relationship and is free from substantial confounding by other putative environmental factors.

SMOKING

Influence of smoking on respiratory symptoms

There is now considerable evidence of an association between passive exposure to parental cigarette smoking and lower respiratory tract symptoms and illnesses in children. Increased prevalences of wheeze (Leeder et al 1976a; Wright et al 1991), cough (Andrae et al 1988; Dijkstra et al 1990), asthma (Ware et al 1984; Weitzman et al 1990) and other respiratory illnesses (Leeder et al 1976; Fergusson et al 1981) have been reported in exposed children. There is in addition evidence suggesting that maternal smoking during pregnancy may have deliterious effects on foetal lung growth and subsequent function; effects which are independent of the effects of passive exposure (Magnusson 1985; Collins et al 1985; Young et al 1991).

Evidence of a causal relationship

Even although many studies have now shown an association between lower respiratory illnesses and parental smoking, authors have been reluctant to invoke a causal relationship. However there is accumulating evidence that the relationship is indeed causal rather than coincidental.

1. Resistance to statistical control

Firstly, the association between parental smoking and respiratory illness in children has withstood attempts at statistical control. The correlation has persisted when accounting for family social conditions (Leeder et al 1976), living standards (Fergusson et al 1981), social class (Schenker et al 1983), and composition (Leeder et al 1976; Fergusson et al 1980; Fergusson et al 1981; Strachan and Elton 1986). Lower respiratory illness in the child's siblings (Leeder et al 1976), home environmental factors such as damp and mould (Andrae et al 1988; Strachan and Elton 1986) and nitrogen dioxide (Dijkstra et al 1990), and parental history of respiratory symptoms and atopy (Schenker et al 1983; Ware et al 1984; Strachan and Elton 1986) have been considered.

2. Specificity for lower respiratory tract infections

Secondly, the association appears to be specific for lower respiratory tract illnesses. At least two studies (Leeder et al 1976; Fergusson et al 1980) have shown that a similar association does not exist with upper respiratory illnesses.

3. Dose-response relationship

Thirdly, many studies have shown that the effects of smoking vary with the degree of contact between the smoking adult and the child - maternal and not paternal smoking being significantly associated with lower respiratory illness in children (Ware et al 1984; Weitzman et al 1990; Wright et al 1991). In addition, both Fergusson et al (1980 and 1981) and Ware et al (1984) have shown that the rates of lower respiratory illnesses are approximately linearly related to levels of maternal smoking.

A dose-response relationship between the amount of exposure and the occurrence of respiratory symptoms and illnesses has therefore been demonstrated. Ware et al (1984) found stronger associations between heavier parental smoking history and prevalences of respiratory illness, bronchitis and wheeze. Prevalences were highest in children with current maternal smokers, lower for ex-smokers and lowest for non-smokers - in that order. They were also higher for maternal smokers than paternal smokers and increased in line with current daily maternal smoking. Fergusson et al (1981) showed respiratory illness in the first year of life to be significantly associated with maternal but not paternal smoking. Wright et al (1991) found that both wheezy and non-wheezy respiratory illnesses in infancy were significantly higher in infants whose mothers smoked but not in those whose fathers smoked, and in infants whose mothers smoked more than a pack of cigarettes compared with those whose mothers smoked less than a pack a day. Weitzman et al (1990) showed a significantly higher prevalence of asthma in children under 5 with heavier maternal smoking.

Effect of age on association

The consistency of these associations seems to be age-dependent, with a greater effect in children under 2 years of age. In an earlier study, Colley et al (1976) found the correlation between parental smoking and childhood lower respiratory illness to be most marked in the first year of life and thereafter declined steadily with increasing age. Fergusson et al (1981)

studied prospectively how the effects of parental smoking varied with age in a cohort identified at birth. At one year there were clear differences between children of smokers and non-smokers, by two years the effect was equivocal and by three years absent. These findings are not in keeping with other reports which have shown greater rates of respiratory problems in school-aged children with smoking parents (Ware et al 1984; Cogswell et al 1987; Somerville et al 1988).

However the results of studies in school-aged children are less consistent than in preschool children. Somerville et al (1988), investigated the associations of parental smoking with the respiratory symptoms of wheeze, cough at night, cough in the morning, attacks of bronchitis and attacks of asthma and found a significant association with the presence of one or more of these. They argue that inconsistent findings occur because enquiry is restricted and that investigation of a single symptom may fail to detect an association in school-aged children. Clifford et al (1989) studied the prevalences of wheeze, cough and shortness of breath in 7 and 11 year-old schoolchildren and failed to show any association with parental smoking.

These age differences in associations might be explained as follows. Firstly, they might reflect the strength of the association. In their study in children aged 6 to 10 years of age, Ware et al (1984) found significant associations with cough, wheeze and bronchitis during the previous twelve months of life but the strongest association was that of doctor diagnosed respiratory illness before the age of two. Secondly, exposure might vary as school-aged children are at school during the day and therefore exposed to lower concentrations of household smoke than their preschool counterparts. Wright et al (1991) demonstrated this phenomenon in infants under a year by showing that those infants who stayed at home with mothers who smoked heavily had an increased lower respiratory illness rate, whereas those of comparable mothers who attended a day-care center were no more likely than unexposed children to have lower respiratory illnesses. Thirdly, failure to show associations between respiratory symptoms and parental smoking may represent a degree of denial or lower expectations among parents who smoke (Clifford et al 1989).

Effect on severity of symptoms

As well as effecting prevalences of respiratory symptoms, there is also some evidence that exposure to parental smoking may influence the severity of symptoms. In a study of asthmatic children aged 0-5 years, Weitzman et al (1990) related the likelihood of taking asthma medications and the number of overnight hospitalisations to parental smoke exposure. Children with parents who smoked received significantly more medication but were no more likely to be admitted to hospital.

Effect on age of onset of symptoms

The age of onset is also influenced by parental smoking. Among asthmatics whose parents smoke, a greater proportion develop symptoms in the first year of life compared to those from non-smoking households (Weitzman et al 1990). A similar earlier age of onset in association with exposure to smoke has been seen with lower respiratory illnesses, both wheezy and non-wheezy (Wright et al 1991).

Effect of gender on association

Differing effects on wheeze and asthma prevalences in boys and girls have not been shown. However in asthmatic children exposed to maternal smoking, disease severity was greater in boys than girls. The boys had more severe symptoms and significantly poorer lung function than girls when compared to respective like-gender asthmatic children not exposed to maternal smoking (Murray and Morrison 1989).

Prevalence of parental smoking

The prevalence of smoking among adults is approximately 25-30 percent. In America 26 percent of adults smoke (Time. April 18, 1988 :71-90). Weitzman et al (1990) showed that 25 percent of mothers smoked during pregnancy and Wright et al (1991) found that 25 percent of mothers and 29.6 percent of fathers smoked during the child's first year of life. Mothers who smoke during pregnancy tend to continue to smoke following the birth of the child (Kleinman et al 1988).

HOME LOCATION

Area of residence may be important in the development of respiratory symptoms and asthma. Striking differences have been found in the prevalences of asthma between urban and rural communities (Andrae et al 1988), particularly in underdeveloped regions (Morrison Smith 1976; Van Niekerk et al 1979; Waite et al 1980).

Effect of urbanisation on symptom prevalence

In South Africa, a study comparing the prevalence of asthma in urban Xhosa children with children from the same tribe living in a rural area found a 30-fold increase with urbanisation. The prevalence in the rural areas was 0.14 percent compared 3.2 percent in the urban areas, the latter approaching prevalences observed in some western communities (Van Niekerk et al 1979).

In New Zealand, children who had emanated from the Pacific island of Tokelau had a two-fold higher prevalence than those still living on the island. These children were the first generation to be born into the westernised urban environment after their parents, half of the island's population, had been moved en bloc to New Zealand following partial destruction of the island by a hurricane in 1966. As the gene pool was therefore unchanged, the increase in prevalence observed was most likely to have resulted from the change in environment (Waite et al 1980).

Others have shown similar prevalence increases with migration from underdeveloped largely rural areas to an urban environment. Morrison-Smith and Cooper (1981) compared the asthma prevalences in European, Asian and West Indian children in Birmingham and found that a significantly higher proportion of children of all races developed asthma within the first four years of life than Asian and West Indian children born in their respective countries of origin. This suggested that the observed greater asthma prevalence was environmental rather than racial in origin, and that these environmental factors had a greater influence in the development of asthma early in life (Morrison-Smith 1976).

Possible reasons for observed increase in symptoms

Possible explanations for these prevalence increases with changes in environment are as follows. Firstly, a change in lifestyle in the cities, such as sleeping on beds with mattresses instead of on well aired mats, or shorter periods of breast feeding with an earlier introduction to cow's milk might be responsible (Van Niekerk et al 1979). Secondly, the loss of potential protective factors may be relevant. Frequent intestinal infestations leading to high circulating immunoglobulin E levels may saturate mast cell receptors and thereby prevent the development of asthma (Carswell et al 1977). Thirdly, prevalence studies in migrant populations, particularly questionnaire studies, might be hampered by difficulties in communication so that the higher reported prevalence rates among older residents compared with the new in developed areas might be due to greater facility with English! (Spears 1975). Finally, atmospheric pollution in urban areas might explain the greater observed asthma prevalence. In a population survey in Sweden Andrae et al (1988) found that children living in a district in their survey area in which the air was polluted by a pulp factory had the highest prevalences of prolonged night cough, allergic rhinitis and allergic asthma.

Paired studies support the observation that environmental pollution may be responsible for the greater symptom prevalences associated with urbanisation. The prevalences of wheeze and asthma were estimated 15 to 25 years apart in each of these geographical areas before and after urban development. In Birmingham Morrison-Smith (1976) showed a four-fold increase of wheeze and asthma from 1956 to 1975. Burr et al (1976; 1989) conducted two prevalence studies 15 years apart in 12 year old schoolchildren in south Wales using similar methods. The prevalence of wheeze increased from 17 to 22 percent and that of asthma from 6 to 12 percent. Finally in two studies conducted in Aberdeen 25 years apart (Dawson et al 1969; Ninan et al 1990) the corresponding prevalence changes were two-fold for wheeze and almost three fold for asthma. In each of these areas there had been substantial urban development, particularly in Birmingham and Aberdeen, during the intervening periods.

These studies strongly suggest that factors associated with urbanisation have influenced the development of wheeze and asthma in children, particularly in young children.

SOCIAL CLASS

Many diseases are both more prevalent and more severe in children in families with low socioeconomic status (SES) than in children in better circumstances (Egbuonu and Starfield 1982).

Sources of reporting bias

Differences between social classes in the prevalences of respiratory symptoms and diseases are difficult to interpret for two reasons. Firstly, there are greater frequencies in the lower social classes of the factors known to influence respiratory morbidity such as dampness and mould, overcrowding (Kaplan and Mascie-Taylor 1985), parental smoking (Weitzman et al 1990) and the use of gas cooking stoves (Schenker et al 1983). Secondly, bias in reporting respiratory diseases may occur because of differing educational achievements. For example Leeder et al (1976) found that asthma was reported more commonly in children of upper social classes parents than in those of low SES, whilst wheezing was more common in children of lower social class parents. They suggested that these social class trends reflected differences in reporting behavior. More parents in social classes I and II may report asthma rather than wheezing when confronted with essentially the same illness.

In epidemiological studies over the past twenty years the prevalence of asthma has been shown to be either significantly increased in children of upper class parents (Hamman et al 1975; Leeder et al 1976; Peckham and Butler 1978; Peat et al 1980; Kaplan and Mascie-Taylor 1985) or not associated with social class (Williams et al 1973; Schenker et al 1983; Horwood et al 1985; Mitchell et al 1989). Aside from the study of Williams et al (1973) the surveys which failed to show any association with social class are the most recent and might reflect changes in the use of the diagnostic label with time (Mitchell et al 1989), or in ease of access to doctors by the lower social classes.

Associations between social class and respiratory conditions

Several groups of investigators have reported an excess of severe asthma among children in low SES families. Dawson et al (1969) found more children with severe asthma in the semi-skilled and unskilled manual classes. Peckham and Butler (1978) found an excess of lower social class children among those whose current attacks occurred more frequently than

once a month. McNicol et al (1973) found no significant association between severity of asthma and SES but did find that children with severe asthma came from larger families that often had single-female heads of households. Thus asthma reported by parents may be more prevalent in high SES children but there is evidence that asthma severity (which is less susceptible to reporting bias) is greater in children from lower social classes.

In contrast to the findings for asthma prevalence, many researchers have reported increased respiratory symptom prevalences - wheeze and cough - in children of lower SES (Leeder et al 1976; Schenker et al 1983; Ware et al 1984; Mitchell et al 1989); others have shown no such influence (Peckham and Butler 1978; Schenker et al 1983). Variations in findings also occurs for symptoms within studies. Schenker et al (1983) found that cough was significantly more common in lower social class children whereas there was no association between wheeze and social class.

Lower respiratory illnesses (LRIs) in infancy display the same social class trends as respiratory symptoms. Most studies show that LRIs in infancy are more common in children of low SES (Taylor et al 1982; Schenker et al 1983; Ware et al 1984), although Leeder et al (1976) found no such association.

Categorisation of families by social class is usually achieved by using a classification based on parental, usually paternal, occupations. However this is a somewhat arbitrary classification and other criteria have been used either individually or in combination to categorise families on social grounds.

Taylor et al (1982) used a previously defined 'social index' which is a composite assessment of SES derived from indices of domestic crowding, parental education, father's occupation, type of neighbourhood and tenure of accommodation. They related the prevalence of wheezy bronchitis to this 'index' and found an increased symptom prevalence with low 'social index'.

Bisgaard et al (1987) defined the 'social status' of families using a point system based on the occupation, salary and education of the breadwinner, and living accommodation including room size and the number of persons per room. They studied the association of wheezing

with 'social status' in infants and found a significant association between this symptom and lower 'social status'. However, when social factors other than parental occupation are considered individually, they do not influence the prevalences of respiratory symptoms or of asthma. Horwood et al (1983) found no significant association between early childhood asthma and any family social background factor, including maternal age, maternal education or family size. These findings were consistent for the total population studied, and boys and girls. There was however an increasing trend in asthma prevalence in the boys with increasing family size that was not present in the girls. Strachan and Elton (1986) found no association between wheeze or nocturnal cough and family size, household crowding (more than 1 person per room) or sharing a bedroom.

The findings are therefore not consistent but there is a tendency for individuals of lower SES to report greater prevalences of respiratory symptoms.

CONCLUSIONS

The prevalence of asthma has recently markedly increased but these prevalence changes have not been paralleled by similar increases in wheeze. Possible explanations are increases in wheeze severity, rather than prevalence, producing more asthma, greater willingness to diagnose asthma in wheezy children or methodological differences in studies affecting prevalence estimates. Despite these greater prevalences there remains disturbing tendencies of undertreatment of affected children with consequent suffering.

Both family background and environmental factors contribute towards the development of wheeze and asthma. The degree of heritability has been determined and shown to be incomplete, suggesting that wheezy illness are multigenic and environmentally influenced. Indoor environmental factors implicated are damp and mould, passive smoke exposure and urban domicile.

As wheeze and asthma commonly present in early childhood, in the preschool years, it is desirable that these conditions be further investigated in this age-group. To my knowledge no population-based study has determined the prevalence of wheeze and asthma in this age-

group exclusively. This study aimed to determined the prevalence, nature, treatment and determinants of wheeze as well as the prevalence and nature of doctor diagnosed asthma in preschool children.

4. SUBJECTS AND METHODS

SUBJECTS

1. SOURCE OF SUBJECTS

Leicestershire, a county in the east midlands of England, had at the last census in 1981 a population of about 845 000 people (OPCS 1982). There were just over 54 000 children less than five years old, representing an annual birth rate of about 10 000. The ethnic composition of the population was not determined in the 1981 census. However there is a large Asian community in Leicestershire which comprises approximately 10% of the population in the region and some 40% in the urban areas.

There are three main urban areas in the county; Leicester, which is the biggest and where about a third of the people live, Loughborough and Melton Mowbray. The main industries in the region are those allied to knitwear production, involving primarily knitting and dyeing neither of which is considered to produce significant industrial pollution. Indeed, there is no heavy industry in the area capable of causing any industrial pollution (personal communication - Leicester City Council Environmental Health Office 1991).

The subjects studied were white Caucasian children, aged 5 years and under, living in Leicestershire. They were selected from the Leicestershire Health Authority Child Health Index, a component of the Child Health Computer Database situated at the Leicestershire Health Authority Central Community Unit in University Road, Leicester.

As Leicestershire is a single Health Authority, this index is a computer record of all the children living in the county. It is continually updated as information concerning individual children emerges, for example a change of address. Data for the index is derived from many sources. The first is the Birth notification form which is completed by the midwife at the birth of all children in Leicestershire. Thereafter any additional information is supplied from various sources including community health care staff such as health visitors, school doctors, general practitioners and parents.

The index was accessed and a complete list of all the children born between 1985 and 1989 inclusive was printed. The information requested on each child was: name, date of birth,

gender, home address and ethnic origin. The resulting print-out was sequenced according to the date of birth of each individual in ascending age (Appendix 1). Individuals with the same date of birth were ordered alphabetically first by surname and then by first name. In the print-out, the entry for each child was represented by 4 lines as follows:

line 1:	Surname	First name	Date of birth	Sex	Index number
line 2:	Address with postal code				
line 3:	Place of birth	Live?	Time of birth		Birth weight
line 4:	Ethnic origin.				

If the child had been born outside Leicestershire, line 3 above was omitted and the entry was as follows:

line 1:	Status: Movement in (Date)				
line 2:	Surname	First name	Date of birth	Sex	Index number
line 3:	Address with postal code				
line 4:	Ethnic origin.				

Each page had enough space for 10 individual entries.

This information on each entry is mostly self explanatory, but some data entries need further explanation:

a. Index number. This number identifies each child in the Child Health Computer Database. It was utilised only to print address stickers for posting after the study sample had been selected.

b. Place of birth. The hospital in Leicestershire where the child was born was recorded as the place of birth, e.g. the Leicester Royal Infirmary or St. Mary's Hospital, Melton Mowbray.

c. Live? This referred to whether the child had been born alive. The database also recorded stillbirths.

d. Ethnic origin. The ethnic origin of the child was recorded at birth by the attending midwife and entered into the birth registration form. The following classification was used in the database:

A: British Isles;

B: Other European;

C: African (excluding Asian immigrants from Africa);

D: West Indian;

E: Asian (Indian Subcontinent, wherever born);

F: Middle Eastern Asian;

G: Vietnamese/Chinese;

H: Mixed races;

I: Other;

J: Not known.

These are mutually exclusive. In this context British Isles means white Caucasian.

e. Status: Movement in (Date). This entry identified children born outside of Leicestershire who had now moved into the county and thereby fell under this Health Authority. In the child's details that followed, there was no birth information as the entry into the database was not from the birth records but from one of the other sources mentioned.

2. SAMPLE SIZE DETERMINATION

The primary objective of the study was to estimate the prevalence of wheeze in the population. The cumulative prevalence of wheeze in children in Britain has recently been reported by Johnston et al (1987) as 17.0%; by Hill et al (1987) as 11.5%; by Clifford et al (1989) as 19.4% in seven year-olds and 18.3% in 11 year-olds; by Burr et al (1989) as 22.3% and by Ninan et al (1990) as 21.4%. Therefore a best estimate would be 20%.

The sample size was calculated using standard sample size calculations (Armitage et al 1987). Assuming a prevalence of wheeze of 20%, it was calculated that a sample size of 1000 would be required to be 95% certain of estimating an overall prevalence in the sample within a mean of 2.5% of its true value. Assuming therefore a nominal response rate to the questionnaire mailings of 60%, this sample size would be achieved by sending questionnaires to 1650 children.

3. SUBJECT SELECTION

Subjects were selected from the print-out in a stratified random fashion so that each year of birth between 1989 and 1985 was equally represented. From each of the 5 years, 350 subjects were selected, 50 initially for the pilot study and 300 for the main study. Randomisation was achieved using a set of random numbers derived from a computer based pseudo-random number generator.

4. INCLUSION AND EXCLUSION CRITERIA

Inclusion in the study was determined by the information printed by the Child Health Index on each individual child.

Inclusion criteria

a. Name. The full name of the subject was required.

b. Address. Resident in Leicestershire. However a small number of children from adjacent counties were included. These were children who from their computer details appeared to be resident in Leicestershire. However their questionnaire responses gave a postal address with a postal code in a neighbouring county. An example of such a 'border' town is Market Bosworth.

c. Birth. Born in Leicestershire.

d. Ethnic origin. 'British Isles' only.

All of the above criteria had to be satisfied for inclusion.

Exclusion criteria

a. Name. If no first name was given and the words 'Baby' or 'Child' were used the individual was excluded. This situation arose when the birth notification certificate was incomplete presumably because the parents had not yet named the child.

b. Address. Resident outside Leicestershire.

c. Birth. Born outside Leicestershire.

d. Ethnic origin. Any ethnic origin other than 'British Isles'. If the entry for ethnic origin was entered as 'Not known' or was missing, that child was also excluded.

QUESTIONNAIRE

The questionnaire, preceded by the introductory letter, is shown in appendices 2 and 3.

1. SOURCES OF QUESTIONS.

This study was conducted using a parent-answered postal questionnaire. As there was no suitable complete and well validated questionnaire available for use in a study of asthma in children, particularly young children, a new questionnaire had to be designed. The questionnaire was based largely on those devised by other authors, thereby enabling me to use previously validated questions where possible. These questionnaires were those of:

1. Epidemiology Standardization Project of the American Thoracic Society (ESP-ATS). Ferris BG (1978),
2. Gibson HB, Silverstone H, Gandevia B, Hall GJL (1969),
3. Morrison Smith J, Harding LK, Cumming G (1971),
4. Burr ML, Eldridge BA, Borysiewicz LK (1974),
5. Peckham C, Butler N (1978),
6. Anderson HR, Bailey PA, Cooper JS, Palmer JC, West S (1983),
7. Kjellman B, Pettersson R (1983),
8. Lee DA, Winslow NR, Speight ANP, Hey EN (1983),
9. Mitchell C, Miles J (1983),
10. Anderson HR, Bland JM, Patel S, Peckham C (1986),
11. Britton WJ, Woolcock AJ, Peat JK, Sedgwick CJ, Lloyd DM, Leeder SR (1986),
12. Cookson and Carey (1986 - unpublished),
13. International Union against Tuberculosis and Lung Diseases (Respiratory Diseases Committee) (IUATLD) Questionnaire (1986),
14. Medical Research Council (MRC) Questionnaire on Respiratory Symptoms (1986),
15. Park ES, Golding J, Carswell F, Stewart-Brown S (1986),
16. Salome CM, Peat JK, Britton WJ, Woolcock AJ (1987),
17. Asher MI, Pattemore PK, Harrison AC, Mitchell EA, Rea HH, Stewart AW, Woolcock AJ (1988),
18. Strachan DP (1988),
19. Clifford RD, Radford M, Howell JB, Holgate ST (1989), and

20. Core respiratory questions - personal communications with Strachan (1990).

The questionnaires of the Epidemiology Standardisation Project (1978), Gibson et al (1969), Kjellman and Pettersson (1983), Mitchell and Miles (1983), and Salome et al (1987) were published in full; those of the IUATLD and the MRC were available from the respective bodies; and the questionnaires of Cookson and Carey (1986), Asher et al (1988), Strachan (1988), and Clifford et al (1989) and the Core respiratory questions (Strachan 1990) were obtained by personal communications. In the other articles only the key questions on wheeze and asthma were quoted in full. Therefore only these could be considered when compiling the questionnaire whereas where full questionnaires were available, all questions were considered. The questionnaires available in full were therefore the main sources of questions for this questionnaire.

2. QUESTIONNAIRE DESIGN

In the design and construction of the questionnaire the following specific factors were considered:

a. the age of the children being studied

The questions used had to be appropriate for children from birth to five years of age, as did the answer alternatives offered. It would not be appropriate, for example, to have questions about phlegm or sputum production because young children seldom if ever expectorate; or to have questions about schooling in a preschool population; and so on.

There is no questionnaire available specifically designed for use in this age-group.

b. the questionnaire was self-administered

This necessitated an introductory letter explaining the nature of the questionnaire, a statement of confidentiality, clear instructions on how to complete the questionnaire and a simple example. It also influenced the structure and order of questions posed. Anonymity was not guaranteed as this would have precluded further study of the cohort created by the survey.

c. the basic principals of questionnaire development

These are:

i. Simplicity. The questions should be as simple as possible to ensure accurate answers.

ii. Format. The format of the questions and their answer alternatives should be uniform for ease of completion. Examples of the present questionnaire include questions 3 and 4 under 'Your child'. The same words '...How many attacks has he or she had?', and the answer alternatives cover the same ranges in both questions. Similarly, in the questions under 'Your family', those about the father are repeated in almost the same format for the mother.

iii. Sequence. The questions should follow an order seen by respondents as natural, with smooth movement from item to item. There are two aspects to this. The first relates to continuity and flow and the second to the implications of the sequence. Questions may be put together to establish an association between them, e.g. those on cough with or without colds (questions 13 and 14), or separated to show an established relationship need not always exist. The key questions on wheeze and asthma, for example, have been separated to avoid the implication that you cannot have asthma without wheeze.

iv. Avoidance of leading questions. Questions whose format influences the respondent's answer should be avoided. The question on diurnal variation, for example, asks whether the symptoms are or are not worse at a particular time of day. Other questionnaires simply ask whether symptoms are worse at night. This limits the answer alternative available to the respondent.

v. Fairness. The questions should be fair to the respondents. There are three aspects to this.

1. Applicability. The respondents should be expected to know the answers. For example, respondents are asked to indicate the age of onset of wheezing, particularly when it started before six months of age. As precise recall is not easy, the words 'as near as you can say' have been added.
2. Comprehensibility. The questions should be phrased in language that the respondents can understand. This is of particular importance in any questionnaire where medical

terms are used. Thus, for terms such as 'wheeze', 'eczema' and 'hay fever' a simple clear definition should be added.

3. Suitable alternatives. The answer alternatives should include all possible answers. I refer specifically to the need for the alternative 'don't know' in asking about diagnoses, as the respondent might not have heard of a particular condition or know whether the child has had it. If only 'yes' and 'no' are available, respondents may be placed in an unfair situation.

vi. Brevity. The questionnaire should be as short as possible. This does not mean that one should limit the questions. Brevity is achieved by careful selection of suitable questions, and the minimum of wording without loss of clarity. Multiple-question format should be used, as for 'precipitants' and 'other illnesses' under 'your child' questions 9 and 16 respectively, and for 'conditions suffered' by the parents and siblings.

vii. Position of questions. Delicate or potentially offensive questions should be left to the end of the questionnaire. Those on age, work description and smoking habits of the parents come into this category. If the respondent must get annoyed it should happen towards the end of the questionnaire.

viii. Answer alternatives. The range of responses or answer alternatives should be comprehensive and the categories mutually exclusive.

3. QUESTION FORMAT

Three types of question format were used in the questionnaire. These are 'closed' or 'fixed alternative' questions of which there are two forms, and 'open-ended' or 'free-response' questions. The majority of questions were 'closed' as the answers are easier to analyse. The number of 'open-ended' questions was kept to a minimum as their value is limited on account of difficulties in analysing and interpreting responses.

a. 'Closed' questions

Two types of 'closed' questions were used in the questionnaire. First, those with more than one answer alternative where the answer selected by the respondent is designated by a tick

in the corresponding box ([ ]), and second, those with a written answer which is restricted by the nature of the question, that is 'how many?' or 'how old?', expecting a numerical answer, and by the answer space which designates the way in which the question has to be answered, i.e. in days, months, years and so on. For example: _____ _____
years months

In some 'closed' questions, an 'open' component allows for respondents to explain positive answers more fully. If the parent of a wheezy child, for example, gave a positive reply to the question on whether the attacks of wheeze were precipitated by drinking or eating, the opportunity was then given to specify the implicated foodstuff in an open manner. Similarly, in the question about pet exposure, 'other' pets could be identified. Such questions, by their nature and possible answers, are easier to analyse than completely open questions as there are, for example, a limited number of pets children keep.

The answer alternatives to the closed questions were mutually exclusive but also inclusive of all possible alternatives. Questions which addressed the nature of the wheezing illness, like the number or length of attacks, had answers which were mutually exclusive but open-ended at both extremes to include all possible responses. The remaining 'closed' questions were 'yes' or 'no' questions. However, in questions where the knowledge of diagnosis was required a third answer alternative ('don't know') was included. This ensured accurate and fair answers.

b. 'Open-ended' questions

There were only two 'open-ended' questions in the questionnaire. In designing the questionnaire the number of questions in this format was kept to a minimum because of the potential difficulties in analysing the answers. It is possible in this type of question to get a different answers for each reply received. Such data would clearly be of little value.

The main place of this question format is in exploratory surveys where the answers would indicate the likely replies and provide a guide to the formulation of alternative responses to closed questions. This type of question has been used in only two situations.

First, as a final comment to the section 'Your child' - 'If you wish to make any other comments about your child's health, please do so in the space provided'. This question allowed other, possibly non-respiratory causes of respiratory symptoms to be elicited, for example congenital heart diseases or gastro-oesophageal reflux. Their presence could have resulted in a falsely increased estimate of the reported prevalence of symptoms caused by primary respiratory illnesses.

Secondly, as a final comment to the section 'Your house' - 'If you would like to add any comments about your housing, please do so in the space provided below'. This was important because one of the stated aims of the study was to identify home environmental factors influencing the development of respiratory symptoms. Specific enquiry was made about factors previously identified; if factors were involved but not previously identified an opportunity was given to mention them.

4. DESCRIPTION OF QUESTIONNAIRE

a. INTRODUCTORY LETTER

In postal questionnaire surveys there is no requirement to obtain informed consent formally from each respondent because, by completing and returning the questionnaire consent is implied. However, the respondent needs to be informed about the nature of the study and the introductory letter is the only way of achieving this. It is therefore an essential and important part of the questionnaire.

The main features of my introductory letter were:

i. an introduction to the parents of the topic being studied, and of the interest in the Department of Child Health in chest problems in children;
ii. mention that the survey was approved by the Leicestershire Health Authority;
iii. an outline of the group of children being targeted;
iv. an explanation of the need for all families mailed to complete the questionnaire, even those in whom the selected child was asymptomatic;
v. a request for a rapid reply;
vi. a statement of confidentiality; and

vii. a means of access (by telephone) should they wish to ask about the survey or about specific questions in the questionnaire.

The letter stressed that it was not possible to include all children in Leicestershire in the study, and that the answers should apply to the child being addressed even if there was more than one child under 5 years of age in the family. This was important to prevent parents from answering the questionnaire for a symptomatic sibling in the belief this would be of help.

In the questionnaires that were re-posted to non-respondents, a further letter was attached to the questionnaire in front of the original introductory letter. This was a short letter, again requesting a reply and return of the questionnaire.

b. DEMOGRAPHIC DETAILS

This is the beginning of the questionnaire proper. Following a short introduction including a request to answer the questions as frankly and accurately as possible, an explanation of how to fill in the questions was given, together with a simple example.

The demographic details requested were:

i. Name of the child,
ii. Date of birth,
iii. Home Address,
iv. Date questionnaire was completed, and
v. Person completing the questionnaire, with answer alternatives of 'Mother', 'Father', 'Female guardian', 'Male guardian' or 'Other'.

Details of name, address and date of birth of each child was obtained from the Leicestershire Health Authority Child Health Index. I asked parents to complete these except for the name (which I entered) to check that our records were correct and to update the record if there was any change in address.

Two possible small alterations could have been made, or added to this page - a postal code, like the zip code that was included in the ESP-ATS questionnaire (1978), and a telephone number. When drawing up this page, I felt that parents might regard a request for their telephone number parents as an intrusion, but some offered their number. The availability of a telephone number would facilitate contact for any planned follow-up of the cohort established from this survey.

The format of this page was based on that of the 'Epidemiology Standardization Project' (Ferris 1978).

The remainder of the questionnaire was divided into three sections, 'Your Child', 'Your House' and 'Your family'.

c. QUESTIONS

In this questionnaire some questions were copied essentially unchanged from the source questionnaires, some were modified from these sources and some were newly constructed questions (Table 6-8). Medical terms were defined where used to aid comprehension of the relevant questions (see above under "Questionnaire design") (Table 9).

Table 6. Questions copied essentially unchanged from source questionnaires.

No	Question	Sources+
YOUR CHILD		
1	Has your child ever had attacks of wheezing?	1,2,18,19
2	How old was he/she when the first attack of wheezing occurred?	1,2,12
3	Since the last attack, approximately how many has he/she had?	2
6	How long is it since his/her last attack of wheezing?	2,17,20
7	Do these attacks cause him/her to be short of breath?	1,9
8	Is his/her breathing completely normal between attacks?	1
13	Does he/she usually have a cough with colds?	1
14	Does he/she usually have a cough apart from colds?	1
17	Has he/she ever has eczema?	16,17
21	When your child was born, did he/she need to stay in hospital after his/her mother went home?	1
22	Does he/she attend a day care, nursery school or play school?	1
YOUR HOUSE		
1	How long has he/she lived in the house?	18
2	How many rooms are there in your house? (not counting kitchens, bathrooms, or toilets)	18
3	Which fuels do you use for heating?	18
4	Which fuels do you use for cooking?	18
5	Does your house have central heating?	18
8	Does your child share his/her bedroom with others?	1,18
9	In your child's bedroom, during the winter months:	
	a. is the room heated during the day?	18
	b. is the room heated during the night?	18
	c. is the window left open at night?	18
	d. does condensation ever form on the windows?	18
	e. does condensation ever form on the walls?	18
	f. are there patches of mould or fungus?	18
YOUR FAMILY: FATHER OR MALE GUARDIAN*		
4	Does he smoke cigarettes? If the answer is yes, how many does he smoke each day in the house?	1,18
5	Did he smoke cigarettes during the year in which your child was born?	18
6	At what age did he finish full-time education?	18
YOUR FAMILY: SECTION C		
1	How many adults (16 years or older) usually live in your house?	18
2	Not counting your child, how many children <16 usually live in your house?	18

No - Question number

+ Sources - source questionnaires. Numbers used refer to numbering above under "Sources of questions".

* Similar questions on Mother or Female Guardian derived from same references as father

Table 7. Questions modified from source questionnaires.

No	Question	Sources
YOUR CHILD		
4	During the past 12 months, how many attacks of wheezing has he/she had?	2,12
5	During the past 12 months, on average (as near as you can say) how long do these attacks last (with normal treatment)?	2,9
9a	Do these attacks occur when he/she has a cold?	1
9b	Do these attacks occur occasionally apart from colds?	1
9c	Do these attacks occur when he/she is running or playing?	1,9,11,16,17
10	Do these attacks occur more frequently at any particular time of year?	2,9,19
11	Is his wheezing worse at any particular time of day?	19
12	Has your child at any time in the last 12 months been wakened at night by an attack of coughing when he/she does not have a cold or chest infection?	12,16,18,19
15	Has any doctor or hospital told you that he/she has asthma or bronchitis?	16,17,19
16a-e	Has your child ever suffered from any of the following conditions? - pneumonia, whooping cough, croup, cystic fibrosis, bronchiolitis	1
18	Does your child attend a clinic or see a doctor for wheezing? (or asthma or bronchitis)	16
19	Has you child ever taken medicine for wheezing? (or asthma or bronchitis)	1,16
20	Has your child ever been admitted to hospital: - with wheezing? (or asthma or bronchitis)	18,19
	- with chest trouble other than wheezing?	18
YOUR HOUSE		
6	Do you keep a pet animal or bird?	1,7,12,19
10	Please list any other rooms in your house affected by: (a) condensation or damp? (b) mould or fungus?	18
YOUR FAMILY: FATHER OR MALE GUARDIAN		
1	Is he the: natural father?; step father?; male guardian?; other?	1
3	Has he ever suffered from any of the following conditions?	2
	a. asthma?	1,2,,12,16,17
	b. bronchitis?	1,2
	c. hay fever?	1,2,12,17
	d. eczema?	12,17
	f. other chest infections?	1,2
YOUR FAMILY: SECTION C		
3	Have any of your child's brothers or sisters suffered from any of the following conditions? (a) attacks of wheezing? (b) asthma? (c) bronchitis? (d) eczema? (e) hay fever? (f) other chest infections?	18
4	Are there any other household members who smoke? (not counting those mentioned). If the answer is yes, please state how many	1

Same abbreviation and format as Table 6

Table 8. Newly constructed questions.

No	Question
YOUR CHILD	
9d	Do these attacks occur when drinking or eating?
9e	Do these attacks occur when he/she is near animals, dust, grass and so on?
16f-g	Has your child ever suffered from any of the following conditions? - recurrent ear infections, other chest infections
YOUR HOUSE	
7	Does your child regularly (at least once a week) come into contact with friends' or relatives' pets or animals?
11	Do you think your house is cold during winter?
YOUR FAMILY: FATHER OR MALE GUARDIAN	
2	How old is he?
7	What is his present job? Please give: a. job title b. grade or seniority c. description of work Is he self-employed? If he is unemployed, please tick box [ ] and give nature of last job.

Same format as table 6

Table 9. Definitions of medical terms used in questionnaire.

No	Medical term	Definition	Source
YOUR CHILD			
1	Wheezing	By this we mean breathing that makes a high-pitched whistling sound from the chest, not the throat.	4,8,16,17,18,19
17	Eczema	An itchy, dry rash on the face, arms and legs.	16,17
YOUR FAMILY			
3	Hay fever	Sneezing, runny or blocked nose, sometimes itchy eyes or nose, not associated with a cold.	2,17,18

METHODS

1. QUESTIONNAIRE POSTING

A total of 3 questionnaire postings were mailed over the 3 month period from mid-April to mid-July 1990. The posting dates were April 17 1990, May 31 1990 and July 20 1990. It should be noted that because of the timing of the initial posting, the actual age range of the children studied was 4 months to 5 years and 4 months.

These three postings were the initial posting to all selected subjects, followed about 6 weeks later by the first re-posting to all non-respondents, and then a second re-posting a further 6 weeks later to the remaining non-respondents (Appendix 4). The period of 6 weeks was chosen as it was the time interval after which no further replies for that particular posting were being received. All questionnaires in each posting were mailed simultaneously. No further follow-up was attempted after the third posting.

Envelopes for mailing were addressed using the printed stickers obtained from the Child Health Index computer utilising the index number mentioned above. An addressed postage paid envelope was enclosed with the questionnaires to facilitate their return.

2. PILOT STUDY

A small pilot study was carried out before starting the main study as the questionnaire was new and hitherto untried. Features checked included the comprehensibility of the questions, the presence of ambiguities, the appropriateness of responses and whether the questions' formats provided the information required. In addition, the pilot study tested the effectiveness of the mailing system.

A total of 150 questionnaires, 30 in each year of birth were posted. The methods of subject selection and questionnaire posting were the same as those employed in the main survey. No major problems were identified from the pilot study and the main study was undertaken without any alterations to the questionnaire. The results of the pilot study were therefore added to those of the main study for analysis. These results are presented briefly to demonstrate the level of completeness and response of the pilot study.

a. BRIEF RESULTS OF PILOT STUDY

i. Replies. One hundred and thirty of the 150 questionnaires mailed were returned - a response rate of 86.7%. Of these 116 (89.2%) were returned after the first mailing, a further 11 (8.5%) after the second mailing and 3 (2.3%) after the third and last mailing. There was equal distribution of replies by year of birth. Thirty questionnaires were sent to children born in each of the 5 years from 1985 to 1989, of which 27, 24, 26, 28 and 25 respectively were returned.

ii. Completer. One hundred and sixteen (89.2%) questionnaires were completed by the mother, 10 (6.7%) by the father and 2 (1.5%) by both parents. In 2 no completer was indicated.

iii. Demographic features Of the 130 replies, there were 62 (47.7%) boys and 68 (52.3%) girls. A Male:female ratio of 1:1.1. Sixty two (47.7%) of respondents lived in urban areas and the remaining 68 (52.3%) in rural areas.

iv. Symptom prevalences. Wheeze was reported in 21 (16.2%) children, doctor diagnosed asthma in 19 (14.6%), recurrent cough in 26 (20.3%) and eczema in 32 (25.8%).

The principal aim of the pilot study was to assess whether the questionnaire was comprehensible and therefore the replies received were appropriate, and whether the mailing system was workable. These were regarded to have been satisfactorily achieved and the main study was commenced without any methodological changes.

3. DATA HANDLING

a. Coding of data (Appendix 6 and 7)

A coding schedule was designed and all answers were precoded numerically to facilitate data processing. DKL assumed personal responsibility for coding to ensure accuracy and consistency.

Social class was coded according to the Registrar General's Classification of Occupations (OPCS 1980)

b. Computer entry

The size of this study and the large amount of information it generated necessitated the use of a computer for efficient handling of the data. After coding, the data were entered onto and stored in the University of Leicester VAX Mainframe computer. This was done by Mrs Barbara Sissons.

c. Data checks

The following data checks were performed.

i. Range checks. Although the data had been coded as carefully and accurately as possible prior to entry into the computer, checks were programmed on the computer to provide for detection of any errors. These checks were range checks whereby all entries were limited by a range within which each answer should fall. An attempt to enter an answer outside that range was rejected by the programme. For example, coding for questions with 'yes' or 'no' answers allowed for 1=yes, 0=no, 8=not applicable and 9=missing or uncodable. Any answer other than one of these four alternatives was rejected. Another example is coding the date of birth, with allowable alternatives of 1-31 for 'day', 1-12 for 'month' and 85-89 for 'year'.

ii. Logical checks. The only check of this nature involved the responses on how long the child had lived in the house. There were replies that were of duration longer than the child had been alive. This was obviously a respondent error and referred to the length of time the family had been in the home. In those cases the answer entered was the age of the child indicating that the child had spent his or her entire life in that house.

Any such problems were corrected at coding prior to computer entry.

iii. Data entry. The accuracy with which the data was entered into the computer was checked by re-entering 100 coded questionnaires. These were selected by taking every tenth coding form from a numerically ordered pile. These were the original coding forms. The data had not been recoded. This data re-entry was performed by the same person who entered the original data.

4. REPEATABILITY

In order to test the repeatability or reliability of questionnaire responses, the questionnaire was administered on two occasions six months apart to the parents of 100 randomly selected children. These were selected using a set of numbers produced by a computer pseudo-random number generator. The numbers were 1 to 1650 inclusive, the study numbers assigned to each child. Criteria for selection were first time respondents with a fully completed first questionnaire. Children were not selected on their responses to the key questions on wheeze or asthma.

The same questionnaire with introductory letter in the same mailing format was posted to the selected families. There was in addition a further letter attached to the front of the initial introductory letter which explained the need for reposting, indicating that this was a 'quality control' on the questionnaire itself and not a check on the parents (Appendix 5).

5. STATISTICAL METHODS

Statistical analysis was performed using the SAS statistical software package (1985). All analyses, except where specifically noted, were performed by DKL with the help and supervision of Dr Paul Burton of the Department of Community Health of the University of Leicester.

The analysis of categorical data was based upon contingency tables. Formal tests of association were based upon the Chi-square test for homogeneity. Where appropriate the chi-square test for trends was used to investigate systematic changes in the distribution of a binary variable across the ordered categorical levels of an ordinal variable (Armitage and Berry 1987).

Multiple logistic regression was used to investigate the multivariate relationship between a binary (0,1) variable and a series of explanatory covariates. In essence, this method determines the amount by which each explanatory covariate multiplies the log e (odds) of a positive response, having adjusted for the confounding influence of other covariates in the model. All models were briefly checked for mathematical validity including a search for

necessary interaction terms. The statistical significance associated with the addition or removal of a term from the model was tested using the Wald test (Breslow and Day 1987).

Survival curves were constructed using the product limit estimate originally described by Kaplan and Meier (1958). Survival curves were compared using the log rank test (Mantel 1966).

Repeatability was tested crudely using percentage agreement and more formally using the kappa statistic (Abrahamson 1988).

Where more detailed statistical evaluation was necessary this is described in the appropriate section.

6. ETHICS AND CONFIDENTIALITY

Approval for this study was obtained from the Leicestershire Health Authority Ethical Committee. Consent is assumed in questionnaire studies if the questionnaire is completed and returned.

Confidentiality was achieved by assigning each subject a study number and entering the results into the computer in an anonymised format using these numbers.

5. RESULTS AND DISCUSSION

5.1. QUESTIONNAIRE

Preschool children under 5 years old have not been the sole subjects of any previous population-based questionnaire prevalence study on wheeze. Therefore as no validated questionnaire existed and a new questionnaire had to be compiled. As mentioned under the methods above, questions were either taken essentially unchanged from source questionnaires, were modified from source questionnaires or were newly constructed (Tables 6-8). Three aspects that need reporting on this new questionnaire are repeatability, strengths and limitations.

1. REPEATABILITY

The repeatability (or reliability) of the questionnaire was tested by remailing the questionnaire to 100 respondents 6 months after the initial questionnaire (see Methods). Of these respondents to the first questionnaire, 75 completed and returned the questionnaire the second time. Repeatability results for the key questions on the health of the child and of his or her family are shown in Table 10.

The two measures of repeatability that were used were percentage agreement and kappa. Kappa is a better measure as it measures agreement beyond chance whereas percentage agreement makes no allowance for chance. A kappa value of 75% or more represents excellent agreement, 40% and 74% fair to good agreement and below 40% poor agreement (Abramson 1988).

Repeatability of the key questions on wheeze and asthma in the child and questions on hospitalisation for wheeze and parental present and past smoking habits was excellent. The only question which had a low kappa value and was therefore unreliable was that on 'coughing apart from colds' in the child. All the remaining questions tested had kappa values from about 40% to 74% and therefore had fair to good repeatability.

Table 10. Measures of repeatability of key questions*.

Question		k+	A
Has your child ever had attacks of wheezing?		88	97
Has any doctor told you that (s)he has asthma?		82	96
Does s(he) usually have a cough with colds?		53	78
Does s(he) usually cough apart from colds?		19	82
Has s(he) ever had eczema?		64	84
Does s(he) see a doctor for wheezing?		65	96
Has s(he) ever taken medicine for wheezing?		74	93
Has s(he) ever been admitted for wheezing?		85	99
Has the father ever suffered from	: asthma?	46	88
	: bronchitis?	41	84
	: hayfever?	59	82
	: eczema?	56	87
Has the mother ever suffered from	: asthma?	73	93
	: bronchitis?	65	89
	: hayfever?	79	91
	: eczema?	59	86
Does the father smoke?		90	96
Did he smoke in the year the child was born?		85	90
Does the mother smoke?		89	97
Did she smoke in the year the child was born?		87	93
Have siblings suffered from	: wheeze?	45	71
	: asthma?	44	74
	: bronchitis?	48	78
	: eczema?	39	70
	: hayfever?	38	72

* Results expressed as percentages.
+ The indices used are kappa (k) and percentage agreement (A).

The excellent repeatability of the question on wheeze attacks in the child is comparable with others who found agreements of 88.9% (Clifford et al 1989), 93% (Salome et al 1987) and 97% (Mitchell and Miles 1983) and kappa of 78% (Clifford et al 1989) and 86% (Mitchell and Miles 1983). Validation of the question on wheeze used in the IUATLD questionnaire (1986) was tested in adults by comparing the answers to the question with the bronchial reactivity to 8 μmol of histamine. The question was found to be 86% sensitive and 72% specific with 76% overall agreement (Burney et al 1987). Mitchell et al (1983), attempted to validate this question in children but could only show a difference in the FEF_{25-75} in those wheezers who had more than 10 attacks when compared with asymptomatic children.

The question on the diagnosis asthma had repeatability similar to that of wheeze with values of 82% for kappa and 96% for percentage agreement. This too is comparable with similar assessments in other reports; Salome et al (1987) scored 77% and 94% respectively. Histamine challenge validation of the IUATLD Questionnaire (1986) found the question to be 55% sensitive and 90% specific with overall agreement of 83%.

The questions on cough 'with' and 'apart from colds', particularly the latter were less repeatable than those on wheeze and asthma. Their high percentage agreement yet low kappa values emphasises that the latter is a better measure of repeatability. Kappa values for cough considerably lower than for wheeze and asthma have also been reported by others. Clifford et al (1989) found 80% agreement with kappa of 57%, and the respective indices of agreement for dry nocturnal cough not associated with a cold reported by Salome et al (1987) were 83% and 51%.

The questions on current medical review and receipt of medication (ever) showed good repeatability with kappa values of 65% and 74% whilst the question on past hospitalisation for wheeze had excellent repeatability with kappa of 85%. Similarly high values were attained by Salome et al (1987) who reported a kappa of 81% on past treatment and Clifford et al (1989) who found a kappa of 79% when asking about hospitalisation.

2. STRENGTHS

a. DEFINITIONS

All medical terms were defined in words which are understandable to non-medical individuals.

i. Wheeze

Wheeze is defined thus: 'By this we mean breathing that makes a high-pitched whistling sound from the chest, not the throat'. This definition is based on that used by many other investigators and embraces the important components stressed by others but excludes those inappropriate for the age-group studied.

Gibson et al (1969) and Mitchell and Miles (1983) did not define wheeze as they regarded asthma and wheeze synonymously. In their questionnaires they state 'Note: Please regard "asthma" and "wheezy breathing" as being much the same thing for this survey; we don't ask you to try to tell the difference'. Although Williams and McNicol (1969) stated that they could find no difference between children with wheezy bronchitis and those with asthma. It has subsequently been suggested (Wilson 1989) that Williams and McNicol (1969) may have failed to show a difference because they only studied children from 7 years of age. They could therefore have excluded children who had wheezed at an earlier age and whose wheezing had stopped by this age. Therefore, in a questionnaire designed for preschool children it would not be appropriate to regard wheeze and asthma as the same.

The MRC Questionnaire (1986) designed for use by interviewers and not for self administration, does not define wheeze and simply asks 'Have you had attacks of wheezing or whistling in your chest at any time in the past 12 months?'. In the accompanying instructions it does explain that if the question is not understood vocal demonstration by the interviewer is often helpful, but that the word asthma should not be used. Similarly, the IUATLD questionnaire (1986), also designed to be self-administered and aimed, like the MRC Questionnaire, at an adult population, asked the same question as the MRC Questionnaire. There was no definition or explanation of the word 'wheeze'. The ESP-ATS (1976) questionnaire asks 'Does this child's chest ever sound wheezy or whistling: when .....?' without any further explanation of the term. Anderson et al (1983) asked 'Has he or she ever had an episode of wheeziness in the chest?'. Both questionnaires do however mention that the noise should originate in the chest.

The need for a definition was emphasised by Burr et al (1974) who, in a the pilot study of a larger questionnaire survey, found that the word 'wheeze' was sometimes misunderstood. In his main survey a definition was observed and the question read 'Has a wheeze - that is, a whistling noise (high or low pitched) - ever been heard coming from your child's chest?'.

From about 1983 most questionnaires adapted for children have defined wheezing as in this study. Cookson and Carey (1986) defined it as 'a noisy whistling sound from the chest, not the throat, causing tightness and breathlessness'. As wheezing need not occur with either a

feeling of tightness nor breathlessness, and as chest tightness is a subjective sensation that needs to be vocalised by the subject, this requirement would have been inappropriate in many young children under 5 years of age.

Lee et al (1983) told parents that wheezing meant 'noisy breathing with a whistling quality coming from the chest and not just the throat'. Salome et al (1987) called it 'a whistling noise that comes from the chest', as did Asher et al (1988). Clifford et al (1989) defined it as 'noisy breathing with a whistling sound coming from the chest not the throat'. Strachan (1986) defined wheezing as follows 'By this we mean a high-pitched whistling sound'. He omitted to mention that it was a noise originating from the chest and not the throat.

In summary, this questionnaire has tried to define wheeze in a way that includes all the important points so as to provide a sensitive and specific marker of the prevalence of wheeze in the population under scrutiny. These points are: it occurs with breathing, it is high pitched, it is whistling in nature and it comes form the chest.

ii. Eczema

Eczema is defined as: 'An itchy , dry rash on the face, arms and legs'. Neither Gibson et al (1969), Cookson and Carey (1986) nor the ESP-ATS Questionnaire (1978) defined eczema. The reports by Peckham and Butler (1978) and Anderson et al (1983) make no mention of the presence or lack of a definition. The only questionnaires that did were those of Salome et al (1987) and Asher et al (1988). With the above definition, Salome et al (1987) found the repeatability of the question to be good with percentage agreement of 88% and kappa of 55%. These were similar to the good repeatability achieved in this study.

iii. Hay fever

Hay fever is defined as: 'sneezing, runny or blocked nose, sometimes itchy eyes or nose, not associated with a cold'. The definition used was that of Salome et al (1987) which is basically the same as that of Gibson et al (1969) and Asher et al (1988), except that the words 'not associated with a cold' had been added by Salome et al (1987). This question, with the

definition in full, was found by Salome et al (1987) on repeatability testing to have a percentage agreement of 95% and kappa value of 63%.

b. ACCURATE DESCRIPTION OF WHEEZE

As the study subjects were young, in many instances symptoms were sufficiently recent to ensure accurate parental recall. The questionnaire made use of this to describe wheeze as accurately as possible, for example:

i. age of onset was specified in weeks if less than 6 months,

ii. there were 6 and 5 answer alternatives with narrow ranges for the questions on previous attacks ever and in the past 12 months respectively,

iii. breathlessness or abnormal breathing was enquired after during and between attacks, and

iv. both season and diurnal variation were sought.

Where precipitants of wheeze have been addressed in previous questionnaires, most restricted questioning to exercise (Ferris 1978; Mitchell and Miles 1983; Britton et al 1986; Salome et al 1987; Asher et al 1988; Strachan 1988). The ESP-ATS Questionnaire (Ferris 1978) also asked about wheezing when the child has a cold and occasionally apart from colds. The only source questionnaire which enquired about inhaled (animals, dust or grass) or ingested (drinking or eating) precipitants was that of Burr et al (1989). In this questionnaire all five precipitants were included, again for as accurate as possible a description of wheeze.

c. INVESTIGATION OF THE ENVIRONMENT

The primary aims of the study were to describe the prevalence and nature of wheeze in preschool children. An equally important aspect of such a study is to identify putative environmental, family and social factors associated with greater symptom prevalence. As the study was conducted using a postal questionnaire these factors could only be identified from parental responses and were thereby limited in scope. Therefore house-dust mite levels which may have influenced symptoms could for example not be determined in this study. All relevant putative factors which could be identified by respondents were thoroughly studied, particularly:

i. different types of pets both in and outside the home,

ii. smoking specifically in the home,

iii. mould, damp and cold,

iv. nitrogen dioxide exposure, although not measured, and

v. the socio-economic status.

Pet exposure was covered by two questions, contacts at home and frequent contact outside the home. In the source questionnaires for this question (Ferris 1978; Kjellman and Pettersson 1983; Cookson and Carey 1986; Clifford et al 1989) enquiry was made about home contact. This is not necessarily sufficient as children may have regular contact with pets outside their home - at the nursery, play-school, neighbours or relatives (Sarsfield et al 1976). Exclusion of these sources will underestimate true pet exposure.

The questionnaire specifically asked about the amount of smoking in the home. This was used to assess the effects of passive smoke exposure on respiratory symptoms. This is clearly more relevant than the total daily smoking of individuals, one or more of whom may spend most of their time at work away from the child's environment.

3. LIMITATIONS

a. AGE-GROUP AT WHICH QUESTIONNAIRE WAS DIRECTED

This study investigated preschool children only. None of the questionnaires used in formulating this questionnaire were directed exclusively at this age, nor were the questions therefore validated for preschool children. One cannot presume that questions validated in school-aged children are appropriate for their preschool counterparts. However, as there were no available questionnaires for preschool children, in order to develop a questionnaire, questions from questionnaires for older children had to be used.

b. POSTAL QUESTIONNAIRE FORMAT OF STUDY

The postal questionnaire is an acceptable method of conducting large community-based epidemiological studies. In these studies replies are solely dependent on the interpretation of the questions by the respondents. In this study, the method used (i.e. postal questionnaire) imposes a possible limitation on the study; to assist parental understanding and interpretation in questions where medical terms were used a brief definition was included, albeit short of

the explanation that would have been possible in an interview situation. In addition, to maximise the response rate and to ensure as complete returns as possible, the questionnaire was designed to be as concise and fair as possible. The repeatability of key questions was good and the response rate of 86% was excellent. The questionnaires returned were thoroughly completed. The repeatability, response rate and the completeness of returned questionnaires would therefore suggest that the methods used determined symptom prevalences with acceptable reliability.

c. TREATMENT OF WHEEZE

The questions on treatment asked about current and past (ever) medical treatment for wheeze. No information was sought about the nature of this treatment, specifically which drugs were used. This limitation would have been overcome by simply adding the phrase 'If the question is yes, state which medicines'.

The question on hospitalisation has several limitations. It does not give precise information about the nature of the admission - whether it was an acute exacerbation of wheeze or asthma, or an acute severe chest infection, or an elective admission for the investigation of recurrent symptoms. It does not determine the number of admissions the child has had and it not include information about other reasons for admission that may have been relevant, for example, whether for tonsillectomy and adenoidectomy. An additional question may have been 'Has your child ever been admitted to hospital :for removal of tonsils or adenoids?'.

d. FAMILY HISTORY

The investigation of wheeze and allergy in the child's first degree relatives was comprehensive for individual parents. However when enquiring about the same in the siblings a single answer was accepted for all. The question did not therefore give information about the number of siblings affected, whether the affected sibling was a brother or a sister or whether one sibling was affected with more than one condition or different conditions affected different siblings. This information would have allowed a more accurate analysis of family data, for example, the effects of gender on the prevalence of wheeze and allergy.

e. VALIDATION

Validation of respiratory questionnaires in children has been attempted by correlating replies and the results of ventilatory function and bronchial challenge tests. Questionnaires validation was attempted by Mitchell and Miles (1983) in school-aged children in grades 3 and 7 (mean ages 8.1 and 12.1 years respectively) and by Salome et al (1987) in children aged 8-11 years. These children were older than the children in this study and were able to perform pulmonary function tests. The children in this study were too young to perform pulmonary function tests requiring active co-operation. This study did not include pulmonary function testing and the key questions on wheeze, doctor diagnosed asthma and cough were therefore not validated. However the questionnaire did create a cohort for further study of these children and thereby possible validation of the questionnaire.

CONCLUSION

As a questionnaire is basically an instrument for measuring and collecting data, its yields are subject to error. This study was unique in that it was limited to an age-group which had previously not been investigated in this way. Thus a new questionnaire with questions relevant to this age-group had to be developed. Every effort was made to improve the validity and repeatability of the answers received. As the questionnaire showed good repeatability and a high percentage of completions and returns, and the study population was representative of the community, the information gathered was as relevant, complete and accurate as could be achieved with the methods used. Addressing the limitations described above would enhance the value of this questionnaire in future studies.

5.2. STUDY POPULATION

The demography of the population sample is described in this section. Where possible the appropriateness of the sample for the source population, and hence the sampling method is assessed.

Of the 1650 questionnaires mailed, 1422 completed replies were returned, representing a response rate of 86.2%. As a result of stratification of the sample selection, 330 questionnaires were sent to each year of birth. The respective response rates were 85.2% for those born in 1989, 87.6% for 1988, 85.4% for 1987, 84.8% for 1986 and 87.9% for 1985.

a. Sex distribution

Of the 1422 replies, there were 726 (51.0%) boys and 696 (49.0%) girls - a male:female (M:F) ratio of 1.05:1. These results for each year of birth are shown in Table 11.

Table 11. Sex distribution of population by year of birth.

Year of birth	Number of replies (%)+ Boys	Girls	Total	M:F Ratio
1989	153 (54.4)	128 (45.6)	281	1.10:1
1988	145 (50.2)	144 (49.8)	289	1.01:1
1987	137 (48.6)	145 (51.4)	282	0.94:1
1986	139 (49.6)	141 (50.4)	280	0.99:1
1985	152 (52.4)	138 (47.6)	290	1.10:1

+ Results are numbers of replies for each group. Percentage of year total in parenthesis.

b. Month of birth

There was a comparable distribution of respondents by month of birth throughout the year (Table 12). This distribution was determined by the sampling method used and confirms that there was no bias against the selection of children born in the latter months of the year.

Table 12. Distribution of population by month of birth.

Month of birth	Replies+ number (%)	Month of birth	Replies number (%)
January	108 (7.6)	July	96 (6.8)
February	133 (9.4)	August	121 (8.5)
March	134 (9.4)	September	150 (10.5)
April	112 (7.9)	October	133 (9.3)
May	125 (8.8)	November	104 (7.3)
June	92 (6.5)	December	114 (8.0)

+ Result are numbers in each month of birth. Percentage of whole population in parenthesis.

c. Area of Residence

Leicestershire is divided into 17 primary postal code areas (Figure 1). The percentage of replies from each area is shown in Table 13. Twelve children (0.8%) from neighbouring counties were included in the sample. Replies were compared to live white Caucasian births in Leicestershire during 1987 (LDHA 1987), again to the assess how representative the sample was of the population (Table 13). The distributions were similar.

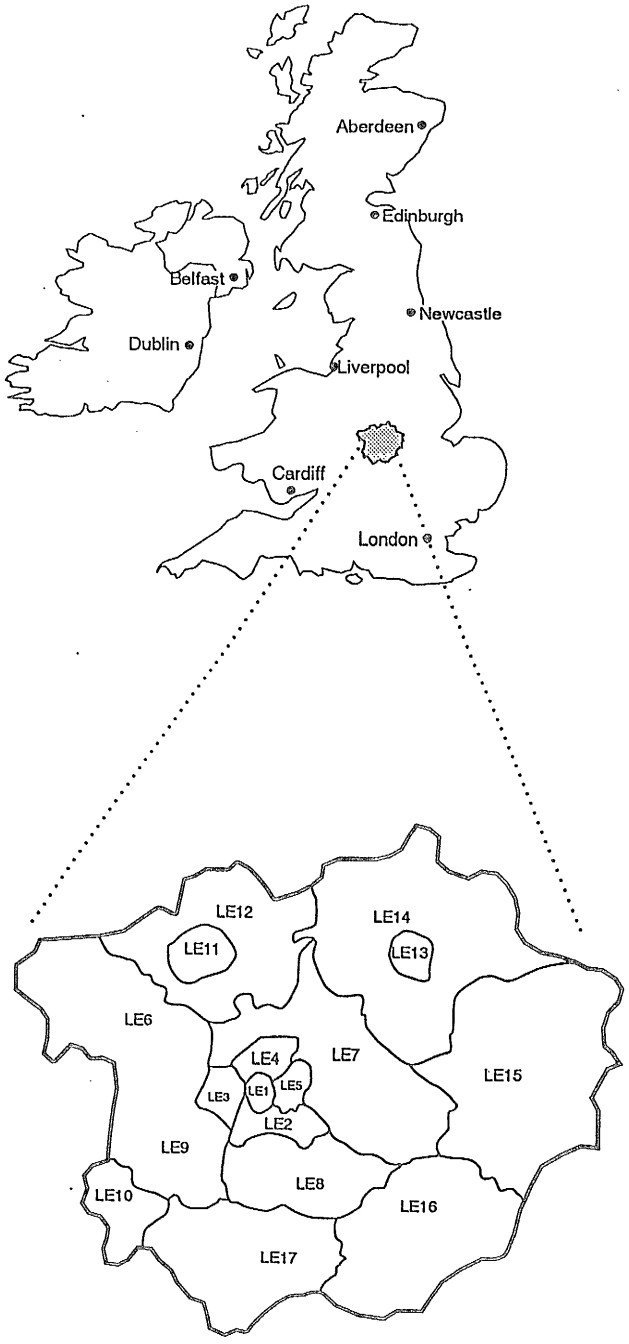

Figure 1. Map of Leicestershire showing primary postal code areas - L1-L17.

Table 13. Distributions of respondents (1990 sample) and 1987 birth cohort by postal code (PC) area.+

PC	'90 sample	'87 cohort	PC	'90 sample	'87 cohort
1*	0.6 (9)	0.2 (138)	10	5.9 (84)	5.1 (506)
2	9.0 (128)	11.1 (1093)	11	7.2 (102)	5.7 (566)
3	9.9 (141)	12.4 (1214)	12	6.5 (92)	5.7 (564)
4	8.3 (118)	9.7 (960)	13	3.9 (55)	3.1 (301)
5	4.6 (66)	5.7 (565)	14	2.2 (31)	1.6 (155)
6	8.8 (125)	9.2 (906)	15	2.9 (41)	3.4 (335)
7	5.2 (74)	4.4 (439)	16	2.7 (38)	2.6 (259)
8	10.9 (155)	8.3 (822)	17	0.9 (32)	2.1 (203)
9	8.4 (119)	8.1 (794)			

+ Results are percentage of population in each group.
Corresponding numbers in parenthesis.
* Numbers 1 to 17 are the primary postal area codes designated LE1 to LE17.

d. Social class

Social class was determined by the father's occupation, the classification used being that of the population census of 1981 (OPCS 1980).

This classification describes the individual classes as shown below. Classes VIII and IX were added for purposes of computer coding. Class VIII is defined as 'Not applicable' indicating a single mother, whereas class IX denoted missing data where the responding parent did not give an occupation.

I - Professional, etc. occupations
II - Intermediate occupations
III - Skilled occupations
IV - Partly skilled occupations
V - Unskilled occupations
VI - Housewives
VII - Students and Armed forces
VIII - Not applicable
IX - Missing answer
0 - Unemployed

The breakdown of responses by social class is shown in Table 14. As this classification is based on the father's occupation, there was only one 'house-husband' - Class VI. However there were 119 families where there was no father present in the home, that is, single mother households.

Table 14. Social class structure of population sample.+

Class	%	(no)	Class	%	(no)
I	6.1	(87)	VI	0.01	(1)
II	23.4	(333)	VII	0.05	(8)
III	43.0	(611)	VIII	8.3	(119)
IV	9.6	(136)	IX	3.0	(42)
V	2.0	(28)	0	4.0	(57)

+ Results are percentage of population in each group.
Corresponding numbers in parenthesis.

These categories were then grouped into 'Upper', 'Middle' and 'Lower' classes with 'Upper' class comprising classes I and II, the 'Middle' class III, and 'Lower' the remaining classes. Class IX - missing data, was excluded from this grouping.

This grouping was compared with similar social class data for Leicestershire from the 1981 Census (OPCS 10% sample 1981), to assess how representative the sample was of the whole population. The limitations of comparing this data with that collected 10 years earlier were recognised, but the census data was the most recent available. The social class distributions were remarkably similar (Table 15).

Table 15. Social class distribution: comparison of 1990 sample with 1981 Census data.

'Class'	1990 Sample (%) (n=1422)	1981 Census (%) (n=24579)
'Upper'	30.4	26.2
'Middle'	44.3	47.8
'Lower'	25.3	25.9

e. Mailing

The response rates to each mailing are shown in Table 16.

Table 16. Response rates to the 3 questionnaire mailings.

Mailing	Questionnaires(no) posted	returned	Response rate(%) per posting	cumulat*
First	1650	1213	73.5	73.5
Second	437	162	37.1	83.3
Third	275	47	17.1	86.2

* Cumulat - Cumulative

It is possible that parents of symptomatic children are more likely to return a completed questionnaire than those without because they have a greater interest in any condition that affects their child. This would overestimate the true population prevalence as the numerator would be falsely increased. How representative the responses were of the whole sample mailed could only be measured from the replies received by assessing whether symptom prevalence were indeed higher in the earlier mailings.

When this was tested (Table 17), there was no significant change in the prevalences in the latter mailings for the symptoms assessed.

Table 17. Symptom prevalences by mailing.

Mailing (no. of replies)		Symptom prevalence+ Wheeze	Asthma#
First	(1213)	16.1	10.8
Second	(162)	13.6	11.1
Third	(47)	10.6	14.9
P value		0.21	0.48

+ Results are percentage in each group.
# Asthma - Doctor diagnosed asthma.

f. Completer

The questionnaire sought the identity of the person or persons completing it - the completer. Nearly 90% were completed by the mother, 8.5% by the father and 2.5% by both parents together (Table 18).

Table 18. Identity of questionnaire completer.

Completer	Replies+	
Mother	88.3	(1255)
Father	8.5	(119)
Both parents	2.5	(35)
Others*	1.6	(13)

+ Results are percentage of population in each group.
Corresponding numbers in parenthesis.
* Others: female guardian (3), grandmother (1), answer missing (9).

The reported prevalences of symptoms may be influenced by the parent who is answering the questionnaire. The father is more likely to underestimate the prevalence, mainly because he does not spend as much time with the child during the day as the mother does (Schenker et al 1983). Affirmation or denial of this can be assessed by comparing the reported symptom

prevalences in those children from whom questionnaires were completed by their respective parents (Table 19).

Table 19. Symptom prevalences by questionnaire completer.

Completer		Symptom prevalence+	
Completer	(Number)	Wheeze	Asthma#
Mother	(1255)	15.4	11.0
Father	(119)	10.9	8.4
Both	(35)	31.4	11.4

+ Results are percentage in each group.
# Asthma - Doctor diagnosed asthma.

The reported prevalences of all three symptoms were higher when questionnaires were answered by mother rather than the father. These differences were not significant. The highest reported prevalence came from questionnaires answered by both parents. This difference was only significant for wheeze ($p=0.04$) but as the group was small, this difference is unlikely to have affected the overall prevalence estimation. Thus reported symptom prevalences were not influenced by whether one or other parent answered the questionnaire.

Therefore by the criteria considered the population sample studied was representative of the under 5 population in Leicestershire. It was also representative of the whole sample mailed and did not depend on whether the answers were given by either or both parents.

DISCUSSION

INTRODUCTION.

Although most childhood asthmatics present with their initial symptoms before the age of 5 years, most epidemiological surveys have not included this age group. Consequently, the descriptions of the prevalence and nature of wheeze and asthma in this age-group have relied on retrospective parental recall, a potentially inaccurate method. The present study addresses this deficiency in the knowledge of childhood wheeze and asthma by targeting this preschool age-group exclusively.

Community and hospital based data have documented evidence of increases in asthma prevalences over the past 20 years. The most notable increases have been seen in children under 4 years of age. Investigation of possible mechanisms for these increases should appropriately target the most affected age-group. Both genetic and environmental influences are implicated in the aetiology of wheeze and asthma. However assuming that the population gene pool is unlikely to change over a period of 20-30 years, except by immigration, a factor controlled for in this study by restricting the study population to white Caucasians, it is reasonable to assume that environmental influences have been responsible for any increase in these symptoms. In recognition of this the study explored environmental factors but was limited in scope by the nature of the study - a parent-answered postal questionnaire study - to the factors which could be reported by the parents. As the study population was under-5, an age when children spend a great deal of time at home, the home indoor environment was specifically targeted. The factors investigated were the fuels used for heating and cooking, household pets, damp and mould in the home, and parental cigarette smoking. Others that required measurement, e.g. level of house-dust mite exposure, were excluded. The environmental factors studied were selected because of their known associations with respiratory symptoms, although at the outset it was not possible to be sure that all relevant variables had been included and whether those actually included were appropriate to the age-group being investigated. Although the genetic contribution to the development of symptoms was unlikely to have changed, family history data was collected and considered in relation to symptom prevalences.

Recurrent cough is a recognised symptom of asthma with or without wheeze. The prevalence of recurrent cough and other symptoms such as breathlessness have not previously been well described. This study identified children presenting with these symptoms with or without wheeze and/or asthma and determined their prevalence not only in the context of wheeze and asthma but also as isolated symptoms.

The database in this study was appropriate for describing the population prevalence of respiratory symptoms because the individuals included were selected in a random manner from a community-based population register. This allowed more accurate assessment of symptom prevalence in the community than previous hospital or practice-based population data. Similarly information obtained about environmental and social factors will be more likely to reflect their importance in the community.

REPRESENTATIVENESS OF DATA SET

Estimation of symptom prevalences and evaluation of risk factors requires reliable and complete data from a representative random sample of the population under study. These requirements were met in this study.

The study sample was selected randomly from a data base of the entire study population in the Health Authority Area (Leicestershire) - the Child Health Index. Subjects were selected primarily on the basis of their ethnic origin coding and without any prior knowledge by the investigator of any factor, for example social class, which might have influenced the individual's response and perhaps have biased selection. Sample selection was thus random and unbiased and as a result representative of the white Caucasian under-5 population in Leicestershire. Sampling was done by one investigator (DKL) thereby ensuring its accuracy and constancy.

The representativeness of the study sample, and hence the sampling method, was tested by evaluation of its distribution by year of birth, sex, month of birth, area of residence and social class. Although the sample was stratified by year of birth, the response rate in each of these five years was assessed and found to be similar. The study sample was therefore equally distributed throughout the five years of birth under study, that is from 1985 to 1989.

Gender distribution was also approximately equal as was the distribution of the sample by month of birth. The relevance of the latter was first, to assess whether the method used biased against selection of children born in the later months of the year; and second, in relation to the association of differing prevalences of respiratory symptoms with birth during particular seasons of the year. If environmental factors influence the development of respiratory symptoms then increased early exposure may 'sensitise' individuals and result in an increase in the incidence of respiratory symptoms - an association between these symptoms and the month of birth might then be expected. The development of respiratory symptoms in a higher than expected proportion of children born during the pollen season between April and September has been observed by some authors (Morrison-Smith and Springett 1979; Bisgaard et al 1987). If such an association occurred in the present study population then the distribution of the sample by month of birth would become important as greater selection from the 'high risk' months would falsely increase the overall prevalence estimates, and vice versa for the 'low risk' months. The even distribution observed confirmed that sample selection was not biased against children born towards the end of the year nor did it lend itself to under or over estimating symptom prevalences for the reasons cited, although in this study no association was found between the month or season of birth and the major symptoms studied. When dividing the year into seasons of birth of summer (April to September) and winter (October to March), symptom prevalences for children born in those seasons were 16.5% (115/696) and 14.7% (107/726) for wheeze and 12.2% (85/696) and 9.8% (71/726) for doctor diagnosed asthma respectively, neither of which were significantly different.

The representativeness of the geographical distribution of the sample in the study area was assessed by comparing it with that of the 1987 white Caucasian birth cohort for Leicestershire. They were found to be similar. Distribution by social class was assessed by comparison with that of the Leicestershire population in the 1981 census, recognising that this census data was ten years old. Again the distributions were similar

Therefore, the assessment of the study sample by year of birth, gender, month of birth, area of residence and social class showed that the sample was indeed representative of the source population and that the sample technique was effective in selecting an unbiased sample.

It is possible that parents with affected children are more likely to return a questionnaire about that specific condition than are parents of unaffected children. This would falsely elevate the prevalence estimate. This potential problem was minimised by remailing two further questionnaires to non-respondents so as to maximise the response rate, and by assessing the symptom prevalence of respondents to each mailing. The overall response rate after three mailings was 86.2% which compares favourably with rates for other questionnaire studies. Many were conducted in school-age children in the class room setting, the child being sent home with a questionnaire and asked to return it completed - a method likely to have a greater yield than the postal method used in this study. Using this method Clifford et al (1989) reported a return rate of 84% in a study of Southampton schoolchildren. Similarly, Salome et al (1987) reported an 85% response rate in a study of Australian schoolchildren, and Hill et al (1989) 80% for schoolchildren in Nottingham. The last authors ascribed their relatively low response rate in part to the fact that, at the request of the head teachers' of the schools involved, reminders were not posted to non-respondents. Lee et al (1983) achieved a remarkably high return rate of over 99% but their questionnaire contained only four questions. In the present study assessment of symptom prevalences for respective mailings was used to evaluate whether parents of symptomatic children replied more promptly. There were no significant change in the prevalences of wheeze or doctor diagnosed asthma with subsequent mailings. As the response rate was acceptable and positive replies were comparable for the three mailings, symptom prevalences were not determined by the timing of the questionnaire return in the present study.

Schenker et al (1983) suggested that replies to the questions set are influenced by the parent who completes the questionnaire, inferring that fathers were likely to underestimate symptom prevalences. In this study 88% of questionnaires were completed by the mother of the child and only 8.5% by the father. Reported symptom prevalences for wheeze and doctor diagnosed asthma were higher in replies from the mother but in neither was the difference significant. However, in the replies returned by both parents - 2.5% of cases - the reported prevalence of wheeze but not doctor diagnosed asthma was significantly greater when compared with maternal or paternal replies. However as this group represents only a small proportion of the total number of replies it is unlikely to have influenced the overall

prevalence estimate. Thus this study showed that the reported prevalence was not influenced by which parent completed the questionnaire.

Correctness of coding was to some extent ensured by range checks inserted into the computer that stored and analysed the data. The accuracy of coded data entered onto the computer was accomplished by utilising these programme checks and by the involvement of only one individual (DKL). Precision of data entry was also tested by re-entry of one hundred randomly selected coded questionnaires. When the original and subsequent entries were compared no differences were detected. The best method of ensuring accurate data entry would have been duplicate entry of the entire database, but this was not possible. As the double entry of the random sample was found to be 100% accurate it seems reasonable to assume that there were few, if any, errors of data entry.

When assessed by the criteria discussed the study sample was shown to be representative of the white Caucasian under-5 population in Leicestershire. The sampling method was also confirmed to be random. Errors in coding and computer entry were avoided thus ensuring that the data was as accurate as possible.

DATA ANALYSIS

In a study of this nature the effect of many factors on the development of symptoms are assessed simultaneously - discussed under the 'aetiology' of asthma. Such factors included genetic background, types of fuels used for cooking and heating, dampness and mould, pets, parental smoking, home location and social class.

The study, as with all observational studies, has the limitation that any observed difference cannot be attributed to a particular risk factor only. This is because one factor is often dependent on or closely linked with another, e.g. the social class of the child has an influence upon the child's exposure to parental smoking.

Therefore evaluation of risk factors from this type of epidemiological data requires an adequate method of analysis. Univariate analysis is essential initially to identify associations but following that multivariate analysis is necessary to unravel the relative contributions of

interdependent factors.

5.3. SYMPTOM PREVALENCES

The overall cumulative prevalences of wheeze and doctor diagnosed asthma were 15.6% (95% confidence interval 13.7% to 17.5%) and 11.0% (95% confidence interval 9.4% to 12.6%) respectively. Wheeze was reported more commonly in boys than girls; respective prevalences were 17.6% (95% confidence interval 16.2% to 19.0%) (128/726) for boys and 13.5% (95% confidence interval 12.2% to 14.8%) (94/696) for girls ($p=0.03$). Asthma had been diagnosed in 12.7% (95% confidence interval 10.2% to 15.0%) (92/726) of boys and 9.2% (95% confidence interval 7.1% to 11.2%) (64/696) of girls ($p=0.03$). Table 20 shows these data by sex and age.

The cumulative prevalence of diagnosed asthma increased systematically and significantly with age ($p=0.0002$). Boys had a consistently higher cumulative prevalence of doctor diagnosed asthma in all age groups with the exception of 3-4 years. A logistic regression model was created by taking the number of diagnosed cases of asthma in each age-sex group as the binomial numerator and the total number of children in the respective groups as the denominator. After adjustment for the effect of age the odds ratio for the diagnosis of asthma (boys:girls) was 1.47 (1.05 to 2.06) ($p=0.03$). After adjustment for the effect of sex the cumulative prevalence of doctor diagnosed asthma still increased with age ($p=0.0001$), the estimated multiplicative increase being 1.27 (1.12 to 1.45) for each additional year of age.

By definition, the cumulative prevalences of wheeze should also have increased systematically with age (Table 20) unless there was a strong birth cohort effect. Overall, however, the cumulative prevalence of wheeze remained remarkably constant with age, being 15.6% (27/173) in children under 1 year, 13.4% (41/306) in those aged 1-2 years, 16.2% (43/265) in those aged 2-3 years, 15.4% (45/293) in those aged 3-4 years, and 17.1% (66/385) in those 4 years and over. As with doctor diagnosed asthma, sex had a strong effect, the odds of wheeze in boys being an estimated 1.38 (1.03 to 1.84) times higher than that in girls ($p=0.03$). These overall data obscured an important interaction: whereas the reported cumulative prevalence of wheeze was reasonably constant (or slowly falling) with age in boys, it increased somewhat in girls, the estimated multiplicative annual increase in the odds ratio was 1.21 (1.03 to 1.44).

Table 20. Cumulative prevalences of wheeze and doctor diagnosed asthma by age group and sex.

Age group (years)	No with symptoms	No in group	Cumulative Prevalence (95% confidence interval) (%)	
		Wheeze		
Boys				
≤1	20	99	20.2	(12.3 to 28.1)
1-1.99	29	160	18.1	(12.2 to 24.1)
2-2.99	23	127	18.1	(11.4 to 24.8)
3-3.99	20	142	14.1	(8.4 to 19.8)
≥4	36	198	18.2	(12.8 to 23.6)
Girls:				
≤1	7	74	9.5	(2.8 to 16.1)
1-1.99	12	146	8.2	(3.8 to 12.7)
2-2.99	20	138	14.5	(8.6 to 20.4)
3-3.99	25	151	16.6	(10.6 to 22.5)
≥4	30	187	16.0	(10.8 to 21.3)
		Doctor diagnosed asthma		
Boys:				
≤1	9	99	9.1	(3.4 to 14.8)
1-1.99	16	160	10.0	(5.4 to 14.6)
2-2.99	17	127	13.4	(7.5 to 19.3)
3-3.99	12	142	8.5	(3.9 to 13.0)
≥4	38	196	19.4	(13.9 to 24.9)
Girls:				
≤1	4	74	5.4	(0.3 to 10.6)
1-1.99	7	146	4.8	(1.3 to 8.3)
2-2.99	10	138	7.2	(2.9 to 11.6)
3-3.99	20	151	13.2	(7.8 to 18.7)
≥4	23	187	12.3	(7.6 to 17.0)

The finding that the proportion of children reported to have ever wheezed was relatively constant with year of birth may indicate that parental perception of significant wheeze is transient and that symptoms that seem relevant to the parent of a young child rapidly fade from the memory as the child gets older. This view is supported by the birth cohort specific Kaplan-Meier functions illustrating the cumulative probability of being perceived to have

developed wheeze with increasing age (Figure 2). Clearly the apparent rate of development of wheeze varies strongly and systematically with age (test for homogeneity: $\chi^2_4=30.5$, $p=0.0001$; test for linear trend: $\chi^2_1=20.6$, $p<0.001$). Thus parents of very young children seemed to be more likely than parents of older children to report that their child had developed wheeze during the early years of life. Interestingly, the age specific Kaplan-Meier survival functions for the cumulative probability of being diagnosed as having asthma also showed an equivalent pattern (test for homogeneity: $\chi^2_4=18.6$, $p=0.0009$; test for trend: χ^2_1, $p=0.002$) although the observed effect was rather less strong (Figure 2).

The prevalence of recurrent cough was 21.8% (95% confidence interval 19.6% to 23.9%). The prevalence of recurrent cough increased significantly with age ($p=0.0003$)(Table 21), the odds increasing by an estimated multiplicative factor of 1.19 (1.08 to 1.31) for each additional year of age. When age was taken into account the odds of recurrent cough in boys was an estimated 1.18 times greater (0.92 to 1.53) than that in girls ($p=0.19$).

Wheezing in the past 12 months (current wheeze) was reported in 12.4% (95% confidence interval 10.7% to 14.1%) of children. Overall, boys had a higher odds of having had a wheezing attacks during the previous 12 months ($p=0.10$), the estimated sex ratio being 1.30 (0.95 to 1.77). Like the cumulative prevalence of wheeze, the overall prevalence remained reasonably constant with age: 15.6% (27/173) in children under 1 year, 13.1% (40/306) in those aged 1-2 years, 12.5% (33/265) in those aged 2-3 years, 11.6% (34/293) in those aged 3-4 years, and 13.2% (51/285) in those aged 4 years and over. There was also a strong interaction between the effects of age and sex ($p=0.02$): in boys the prevalence fell with age at an estimated multiplicative rate of 0.86 (0.74 to 1.00) a year while in girls it was estimated to increase at a multiplicative rate of 1.14 (0.95 to 1.36) a year. These conclusions were unaffected by removing from the analysis children under the age of 1 year, for whom symptoms in the previous 12 months has a modified interpretation.

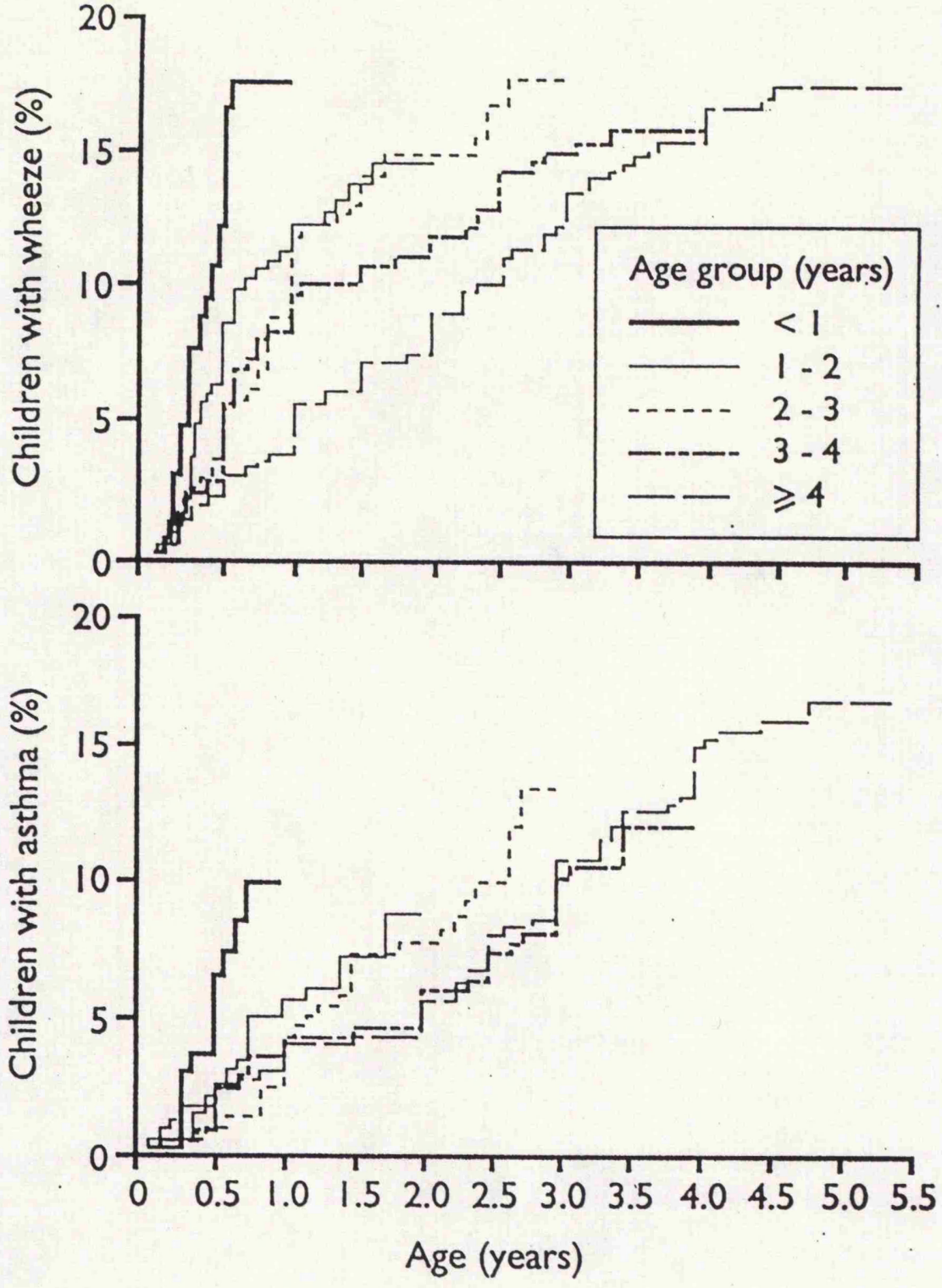

Figure 2. Cumulative probability of being perceived as having developed wheeze and asthma by age-group.

Table 21. Prevalences of recurrent cough and of at least one wheezing attack during the past 12 months by age group and sex.

Age group (years)	No with symptoms	No in group	Cumulative Prevalence (95% confidence interval) (%)	
		Recurrent cough		
Boys				
≤1	17	97	17.5	(10.0 to 25.1)
1-1.99	29	160	18.1	(12.2 to 24.1)
2-2.99	30	127	23.6	(16.2 to 31.0)
3-3.99	35	140	25.0	(17.8 to 32.2)
≥4	55	196	28.1	(21.8 to 34.4)
Girls:				
≤1	15	72	20.8	(11.5 to 30.2)
1-1.99	22	146	15.1	(9.3 to 20.9)
2-2.99	19	137	13.9	(8.1 to 19.7)
3-3.99	35	151	23.2	(16.4 to 29.9)
≥4	50	184	27.2	(20.7 to 33.6)
		Wheeze in the previous 12 months		
Boys:				
≤1	20	99	20.2	(12.3 to 28.1)
1-1.99	28	160	17.5	(11.6 to 23.4)
2-2.99	17	127	13.4	(7.5 to 19.3)
3-3.99	13	142	9.2	(4.4 to 13.9)
≥4	27	196	13.6	(8.9 to 18.4)
Girls:				
≤1	7	74	9.5	(2.8 to 16.1)
1-1.99	12	146	8.2	(3.8 to 12.7)
2-2.99	16	138	11.6	(6.3 to 16.9)
3-3.99	21	151	13.9	(8.4 to 19.4)
≥4	24	187	12.8	(8.0 to 17.6)

Of the 156 children who had been diagnosed as having asthma, 72 (46.1%) had a history of wheeze without recurrent cough, 17 (10.9%) had recurrent cough without wheeze, 53 (34.0%) had both symptoms, and 14 (9.0%) had neither. Most children with reported asthma suffered recurrent cough but only when they had a cold. There was no significant sex difference in the symptomatic pattern of those with doctor diagnosed asthma ($p=0.19$).

However, the proportion of children with wheeze who had been diagnosed as having asthma increased significantly with increasing age (40.7% (11/27), 43.9% (18/41), 48.8% (21/43), 55.6% (25/45), and 75.8% (50/66); p=0.0001), as did the equivalent proportions of children with cough (9.4% (3/32), 15.7% (8/51), 22.5% (11/49), 24.3% (17/70) and 29.8% (31/104); p=0.005).

DISCUSSION

Inferences about the prevalence of wheeze and asthma in preschool children have been based on studies in older age groups (Fleming et al 1987; Ninan et al 1991) and are limited by imperfect recall of symptoms (Strachan 1985). Sound epidemiological data from studies of preschool children are lacking. This study designed, tested and used a postal questionnaire for parents to investigate a wide range of issues pertaining to wheeze in preschool children. The study estimated age and sex specific cumulative prevalences of selected asthmatic symptoms (cumulative and current wheeze and recurrent cough) and doctor diagnosed asthma. Doctor diagnosed asthma is not regarded as or claimed to be the reference standard for true asthma, but may be a useful variable with important implications for health care and epidemiology. One reason for adding doctor diagnosed asthma in the questionnaire was to assess its usefulness over time by following up the cohort established. Hopefully this would demonstrate whether or not the diagnosis 'asthma' applied to wheezing and/or coughing preschool children had long term relevance; it might be that no such diagnostic categorisation should be attached before children are old enough to be diagnosed asthmatic on both clinical and physiological criteria.

This study shows that parental recall of the development of wheeze is inconsistent even within the preschool age range. The fact that parents of very young children report a similar cumulative prevalence of wheeze to the parents of older children strongly suggests that some episodes of wheeze that are perceived as significant by the parents of the younger children are forgotten as the child gets older. It is not just that parents forget when a child starts to wheeze, for that would affect the Kaplan-Meier incidence functions but not the cumulative prevalences themselves, but that parents of older children completely forget episodes of wheeze that the parents of younger children perceive as important. This underlies the

difficulty studying the symptomatic profile of preschool children from retrospective recall of the parents of school age children.

Prevalence comparisons with recent studies

In this study of preschool children in Leicestershire the cumulative prevalence of wheeze was 15.6% and of doctor diagnosed asthma was 11.0%. The current prevalence of wheeze, that is wheeze in the past 12 months, was 12.4%. These figures are similar to those of the more recent reports from questionnaire studies in Britain.

In a study of 7 and 11 year old children in Southampton carried out in 1986 Clifford et al (1989) found cumulative prevalences of wheeze of 19.4% and 18.3% respectively in the two age-groups; and of asthma of 9.5%. In south Wales in 1988, Burr et al (1989) found a cumulative prevalence of wheeze of 22.3% and of asthma of 12.0%, and in Aberdeen in 1989 Ninan et al (1990) found in schoolchildren aged from 8 to 11 years cumulative prevalences of wheeze and asthma of 21.4% and 11.8% respectively. In all these studies the prevalence of wheeze was higher than that recorded in this study, and in addition they all exceeded the upper 95% confidence limit. However the prevalence estimates for asthma were all within the 95% confidence limits of the estimate of this study.

Recent reports of the current prevalence of wheeze are also similar to that of this study. Hill et al (1989), in a study performed in 1985 in Caucasian and Asian schoolchildren aged 5-11 years in neighbouring Nottinghamshire, reported a current prevalence of wheeze of 11.5% whilst Clifford et al (1989) reported one of 12.1%. These prevalence estimates both fall within the 95% confidence limits of the same estimate for this study.

Comparison of results of this study with others in the United Kingdom must take account of age-group and minor methodological differences. The cumulative wheeze prevalence difference noted are not unexpected as a greater percentage of older children will have a lifetime history of wheeze than their younger counterparts on account of a yearly incidence of new cases of wheeze augmenting the cumulative prevalence with time.

The current prevalence of wheeze was similar to that found in the studies quoted for older children. As no significant difference in wheeze prevalence was noted between the 5 age groups in this study, this suggests that current prevalences may be similar from the first year of life up until at least 11 years of age - the age of the older group studied by Clifford et al (1989). It also in part serves to confirm the above observations that the cumulative prevalence should increase with age with the addition of new cases which maintain the current prevalence but in addition that parental recall is poor with many episodes being forgotten.

The similarity between the findings in the present study and that of Clifford et al (1989) for cumulative asthma prevalence is somewhat surprising. One would expect that as the prevalence of asthma increased significantly with age in this study that that trend would continue. The possible explanation for the observed age trend here is that there is a greater reluctance by doctors to diagnose asthma in younger children. If this were indeed so then one would expect a higher prevalence of asthma amongst schoolchildren. However, it is possible that cumulative prevalence increases during the preschool years, then plateaus as fewer cases are added and by 11 years is little different from that seen at 5 years.

Prevalence changes over time.

The trends observed in the cumulative prevalences of wheeze and asthma are consistent with this study. Comparison of these results with those of the other cross-sectional questionnaire surveys (Table 4) show that the prevalence of wheeze has remained fairly constant over the past 25 years at between 15-20%, whilst that of asthma has increased from about 5% to approximately 12% since 1980. The prevalence findings of this study once again confirm the impression that although asthma is being diagnosed more frequently there has not been a comparable increase in the prevalence of the underlying symptom of wheeze.

The community prevalence of recurrent cough was defined for the first time by Clifford et al (1989). As there is no earlier data it is not possible to comment on prevalence changes with time.

This study supports the view that the diagnosis asthma is now more common in wheezy children and that the problems of wheeze have not increased to the same extent. The

increased diagnosis of asthma may be due to better education of both doctor and patients. Doctors diagnose asthma more readily and parents are now better informed through the media. Perhaps changed parental perceptions about the significance of asthma make them more likely to accept this diagnosis.

Age and gender differences in prevalences

The cumulative prevalence of wheeze did not change significantly with increasing age, due largely to the already high prevalence in boys under a year old. However there was a significant increase in the cumulative prevalence of asthma.

The gender differences observed concur with previous British prevalence studies (see Table 5) of higher prevalences of both wheeze and asthma in boys, with comparable male:female ratios of 1.3:1 for wheeze and 1.4:1 for asthma. This male predominance decreased with increasing age. The prevalence of wheeze in girls increased steadily with age approaching the constant prevalence of the boys. The asthma prevalence of both sexes increased with age, but by a greater magnitude in the girls. As this study was restricted to children up to the age of five years, it is not possible to comment on possible prevalence differences between males and females beyond this age range. However, these findings are in keeping with those of studies showing a decrease in the male predominance with age leading eventually to reversal of the ratio at adolescence when females predominate (Dinwiddie 1990).

In, conclusion this study has established baseline age and sex specific results for wheeze, doctor diagnosed asthma and recurrent cough in preschool children. The findings emphasise the importance of studying the age group of interest rather than relying on the recall of parents of school age children. These data are interesting in their own right and should be a valuable comparative baseline for studies in other areas and for investigations of the changes in the incidence and prevalence of asthma over calendar time. The follow up study of the cohort established by this study will hopefully provide answers to the important questions about the development and natural course of asthma and asthmatic symptoms in preschool children.

5.4. DESCRIPTION OF WHEEZE AND ITS RELATION WITH DOCTOR DIAGNOSED ASTHMA

Severity of wheeze

Six variables were employed to describe the severity of wheeze in individual children. These were:

1. the total number of attacks,
2. the number of attacks in the past twelve months,
3. the average duration of attacks during the past twelve months,
4. the length of time since the last attack,
5. shortness of breath with attacks and
6. normality of breathing between attacks.

Eighteen percent (40/221) of wheezy children had only one episode but children in the youngest age group were perhaps too young to have had a large number of subsequent attacks. Overall, 18% (40/221) had 1-2 further attacks, 30% (67/221) 3-5, 17% (38/221) 6-10, and 16% (26/221) had >10 further attacks. One in 10 children had experienced more than 20 attacks of wheeze. There was no significant change in year by year distribution of wheezy episodes ($p=0.4$).

In considering the number of attacks in the preceding 12 months the youngest age group was excluded from analysis. As with the age distribution for total number of attacks the distribution of attacks within the previous year did not differ significantly on year by year analysis ($p=0.6$). Overall 22% (43/195) had had no wheezing episode in the preceding 12 months. Thirty six% (71/195) had 1-2, 24% (47/195) 3-5, and nearly 18% (34/195) >6 attacks (Table 22).

There were 152 replies to the question relating to the length of attacks of wheeze during the preceding 12 months. Overall, 18% (28/152) of attacks lasted up to one day, 48% (71/152) two to three days, 27% (41/152) four to seven days, and 8% (12/152) more than seven days. The distributions were similar in each of the age groups considered ($p=0.4$).

Table 22. Distribution (row percentages (number)) of wheezy episodes in the 12 months before questionnaire by age group.

Age (yrs)	No	Number of attacks 0	1-2	3-5	6-10	>10
1-1.99	41	4.9 (2)	36.6 (15)	41.5 (17)	7.3 (3)	9.7 (4)
2-2.99	43	25.6 (11)	34.9 (15)	20.9 (9)	11.6 (5)	7.0 (3)
3-3.99	45	31.1 (14)	37.8 (17)	17.8 (8)	11.1 (5)	2.2 (1)
≥4	66	24.2 (16)	36.4 (24)	19.7 (13)	13.6 (9)	6.1 (4)
Total	195	22.1 (43)	36.4 (71)	24.1 (47)	11.3 (22)	6.2 (12)

Shortness of breath with attacks of wheeze was reported in two thirds of the whole population, and was an invariable accompaniment in one quarter (Table 23). The reported rate of occasional shortness of breath did not change with age, but shortness of breath with every wheezing attack increased significantly with age ($p=0.04$). Abnormal breathing between attacks was reported in 12.2% (27/221) children.

Table 23. Distribution (row percentages (number)) of shortness of breath with wheeze attacks by age group.

Age (yrs)	No	Shortness of breath with wheeze Never	Occasionally	Always
≤1	27	37.0 (10)	40.7 (11)	22.2 (6)
1-1.99	40	52.5 (21)	35.0 (14)	12.5 (5)
2-2.99	43	27.9 (12)	55.8 (24)	16.3 (7)
3-3.99	45	28.9 (13)	40.0 (18)	31.1 (14)
≥4	66	25.8 (17)	43.9 (29)	30.3 (20)
Total	221	33.0 (73)	43.4 (96)	25.5 (52)

No gender differences were noted for any of the severity variables: that is the total number of attacks ($p=0.3$), the number of attacks in the past twelve months ($p=0.5$), the average duration of attacks during the past twelve months ($p=0.3$), the length of time since the last

attack (p=0.6), shortness of breath with attacks (p=0.9) or abnormal breathing between attacks (p=0.3).

Precipitants of wheeze

Enquiry was made about suspected precipitants of wheezing episodes: colds and "apart from colds" (non-cold precipitants), running or playing (exercise), foodstuffs (ingestants) and aero-allergens such as pollen or animal epithelia (inhalants).

Table 24 shows that about 90% of the children who wheezed had attacks that were reported to be brought on by colds, without significant year by year cohort effect (p=0.9). Wheeze associated with precipitants other than colds was less common particularly in the two youngest age groups. In exercise induced wheeze the extremely low rate in the children under 1 year almost certainly reflected the fact that most of these children were not then ambulant. Attacks of wheeze precipitated by ingestion of food or drinks occurred in only 5.9% of respondents.

Table 24. Prevalences (numerator/denominator) of reported precipitants of wheeze by age group.

Age (yrs)	Cold	Non-cold Precipitant	Exercise	Ingestant	Inhalant
≤1	81.8 (22/27)	33.3 (9/27)	3.7 (1/27)	14.8 (4/27)	7.4 (2/27)
1-1.99	94.1 (36/38)	30.6 (11/36)	8.8 (3/34)	8.6 (3/35)	5.0 (2/40)
2-2.99	88.4 (38/43)	40.0 (14/35)	35.9 (14/39)	8.6 (3/35)	21.4 (9/42)
3-3.99	83.3 (35/42)	30.2 (13/43)	33.3 (14/42)	7.7 (3/39)	20.0 (9/45)
≥4	89.2 (58/65)	63.5 (33/52)	49.1 (28/57)	5.9 (3/51)	32.8 (21/64)

Positive responses to all precipitants were greater for girls than boys when sexes were assessed separately, although the differences were significant only for exercise. Exercise was a reported precipitant for wheeze in 24% (27/113) boys and 38% (33/86) girls (p=0.03).

Respondents were asked to name specific foodstuffs or inhaled allergens thought to be associated with attacks of wheeze. Sixteen children identified eating or drinking as a precipitant of wheeze, and three more than one food type. Seven foodstuffs were identified

with no one single food or drink predominating. Food stuffs implicated as wheeze precipitants were cold drinks in 3 children, milk in 3, eggs in 3, all foods in 5 and in each of the following was reported for one child: preservatives, ice cream, chocolate and strawberries.

Of the 43 children who identified inhaled allergens as precipitants, 17 thought that more than one allergen triggered wheeze. Animals accounted for nearly one third of the allergens identified, with grass and dust nearly a quarter each. Dogs represented 36.8% of the animals incriminated and cats 21%. One respondent reported attacks that were related to inhalation of tobacco smoke. Other reported precipitants were pollens in 3 children, oil seed rape in 2, aerosol in 1 and in 6 the precipitant was not specified.

Positive responses to all precipitants were greater in girls than boys (Table 25), although the differences were significant only for exercise ($p=0.03$) where the prevalence in girls was almost 15% more than in boys.

Table 25. Prevalences (numerator/denominator) of reported precipitants of wheeze by gender.

Sex	Cold	Non-cold Precipitant	Exercise	Ingestant	Inhalant
M	86.4 (125/108)	40.9 (45/110)	23.9 (27/113)	6.6 (7/94)	17.5 (22/126)
F	90.0 (81/90)	42.2 (35/83)	38.4 (33/86)	11.1 (9/81)	22.8 (21/92)

M = males; F = females

Seasonal and diurnal variations

The first year age group was omitted from analysis as many children in it were too young to have exhibited seasonal variation in symptoms. Of 195 children with wheeze in the four older age groups, 33% (64/195) had more attacks at a particular time of year: 86% (55/64) in winter. The patterns were similar for each of the older age groups. There was a significant association between seasonal variation of wheeze and the diagnosis asthma ($p=0.005$).

Murray and Morrison (1988) found that in asthmatic children of smokers, lung function was more severely affected in the winter months. They attributed this to houses being less well ventilated because windows are closed and smoke concentrations higher. The prevalence of winter symptoms in homes where parents did and did not smoke were compared. Of the wheezers whose parents smoked 19.7% (23/117) reported more frequent attacks of wheeze in winter in comparison with 23.8% (25/105) among those whose parents did not smoke. The difference was not significant ($p=0.4$). There was therefore no association between parental smoking and an increased tendency to wheeze in winter.

If, as Murray and Morrison (1988) suggest, symptoms are worse in the winter months when houses are less well ventilated, one might predict presumably then damp and mould in the homes will be worse during this time. Thus attacks of wheeze associated with damp and mould might be more prevalent in winter. During winter, attacks of wheeze occurred in 23.2% (25/108) of wheezers with reported damp in the home and 20.4% (23/113) without ($p=0.6$) and in 20% (6/30) with mould in the home and 21.6% (41/190) without ($p=0.8$). Neither difference was significant.

Sixty-one percent (135/220) of all wheezers reported diurnal variation, of whom 91% (123/135) had nocturnal symptoms. There was a significant association between diurnal variation and the diagnosis of asthma ($p=0.04$) due almost entirely to the association with-night-time wheeze ($p=0.02$).

Characteristics of wheeze and the likelihood of asthma being diagnosed

The contributions of individual measures of wheeze severity to a nominated binary endpoint, doctor diagnosed asthma, were assessed as follows. These statistics were performed by Dr Paul Burton.

Because the individual measures of severity and the responses to different precipitants are likely to be correlated with one another, unconditional logistic regression modelling was used to investigate the multivariate relationship between these various symptom based criteria and the probability that a wheezy child will be formally diagnosed as having asthma. Modelling was performed in S-plus using the 'glm' function (Becker et al 1988) and was restricted to

those children who were known to wheeze and for whom it was also known that the date of diagnosis of asthma did not precede the date of onset of wheezing. Doctor diagnosed asthma was used as the binary response variable (0=without, 1=with) and variables relating to the potentially important symptom based criteria listed in Table 26 entered the model as categorical explanatory covariates. Model construction was based upon the systematic removal of covariates from an initial model containing all main effects. Formal tests for the significance of the deterioration of fit following the removal of the regression terms relating to a specific variable was based upon the likelihood ratio test. In the process of modelling, it became clear that the probability of doctor diagnosed asthma was similar for children with 3-5, 6-10 and >10 attacks of wheeze during the last year and the original five level covariate was therefore collapsed to three levels: 0 = no attacks in the last year, 1 = 1-2 attacks and 2 = 3+ attacks.

Table 26. Factors investigated in the regression modelling of wheeze severity factors and doctor diagnosed asthma.

Gender
Age group
The total number of attacks
The frequency of attacks within the last year
The average duration of attacks
The length of time since the last attack
The presence of shortness of breath with attacks
Colds as a precipitant
Non-cold precipitants
Eating as a precipitant
Dust as a precipitant
Seasonal variation
Diurnal variation
Asthma in first degree relatives

Before drawing definitive conclusions, the final model was subjected to standard model checking procedures, including tests for non-linearity and interaction, the analysis of Pearson residuals and the investigation of leverage. Regression leverage was assessed using the matrix (McCullagh et al 1989). Only one value was high enough to exceed the standard test of 'unusual' leverage (McCullagh et al 1989) and removal of the five children with the

greatest leverage (including the child with 'unusual' leverage) had no important effect upon the coefficients in the model. It would therefore appear reasonable to conclude that the model as presented provides an acceptable representation of the observed data. Because the variable representing 'the number of attacks during last year' has a different interpretation in those children under one year of age, a second model was constructed which excluded the youngest age group. In practice, the second model was so similar to the first that only the latter is presented.

Table 27 details the final unconditional logistic regression model quantifying the multivariate logistic regression relationship between the measures of asthma severity, the pattern of precipitants and the probability that a wheezy preschool child will be diagnosed as having asthma.

The important factors that appear to determine this probability are age, the number of attacks a year, severity of shortness of breath during attacks, and non-cold precipitants. Rather surprisingly, seasonal and diurnal variations of wheeze, and a history of asthma in first degree relatives did not increase the likelihood of asthma being diagnosed when these factors were subjected to multivariate analysis. To take an example, a wheezy child aged 3 who has fewer than one attack a year, no shortage of breath during attacks, and no precipitants other than colds had an odds of $e^{(-3724+1.286)} = 0.086$ which is equivalent to a probability of 8% $(0.086/(1+0.086)=0.08)$ of having been diagnosed to have asthma (e is the base of natural logarithms). On the other hand a child of the same age who has 3 or more attacks per annum, who is always short of breath during attacks brought on by precipitants other than colds has an odds of $e^{(-3.742+1.286+1.651+2.298+2.295)} = 16.25$, which is equivalent to a probability of 94% $(16.25/(1+16.25)=0.94)$ of having been diagnosed to have asthma.

Table 27. Indices of wheeze severity and the likelihood of asthma being diagnosed by a doctor.

Parameter	Coefficient	Odds ratio (95% CI) relative to baseline category	Likelihood ratio test for the removal of variable
Intercept	-3.742	0.024 (0.005 to 0.113)+	
Age group (years)			χ^2_4=14.4, p=0.006
≤1*		1.00	
1-1.99	0.419	1.52 (0.45 to 5.15)	
2-2.99	0.185	1.20 (0.33 to 4.37)	
3-3.99	1.286	3.62 (1.06 to 12.40)	
≥4	2.039	7.68 (2.04 to 28.93)	
Shortness of breath during attacks			χ^2_2=11.9, p=0.003
Never*		1.00	
Sometimes	1.284	3.61 (1.50 to 8.71)	
Always	1.651	5.21 (1.77 to 15.39)	
Number of attacks during the last year			χ^2_2=14.9, p=0.0006
None*		1.00	
1-2	1.805	6.08 (1.92 to 19.29)	
3+	2.298	9.95 (2.79 to 35.53)	
Precipitants other than colds			χ^2_1=9.0, p=0.003
No*		1.00	
Yes	1.295	3.65 (1.54 to 8.63)	

\+ Estimated odds when all covariates at baseline.
* Baseline group
CI - confidence interval

DISCUSSION.

This study described the nature of wheeze in terms of: severity, precipitating events, seasonal and diurnal variation and likelihood of asthma being diagnosed. The diagnosis of asthma, defined on page 7, was based on parental recall of whether this diagnosis had been made by a doctor (general practitioner, community or hospital doctor). The postal questionnaire nature of the study did not provide for the possibility of confirming this parental perception by scrutiny of doctors' records.

Severity of wheeze.

Severity of wheeze was evaluated in this study on the basis of wheeze frequency and duration and the occurrence of associated breathing problems during and between attacks of wheeze. The results should be viewed as being no more than descriptive of what was actually observed. They should be interpreted with care because attained age and the accuracy of parental recall must necessarily influence the responses obtained. High repeatability of the question on wheeze provides some confidence that severity indices may also be accurate. Wheeze frequency has previously been shown to be a reliable indicator of the severity of wheezy illnesses (McNicol et al 1973; Blair 1977; Park et al 1986). In these studies patients were categorised arbitrarily by taking three or five attacks of wheeze as a cutoff point between categories. This may have arisen from a tendency to define asthma in wheezy children only after three or more (sometimes five or more) episodes of wheeze (Tabachnik et al 1981).

In this study the choices of numbers, that is, either frequency or duration, were similarly speculative and arbitrary and categories were based on a previously used questionnaire (Gibson et al 1969). As this study involved only preschool children, among whom it was hoped that parental recall would be more accurate and complete, the response subdivisions were less coarse and required more precise answering. This allowed for a more meticulous evaluation of severity. The age stratification of the study population afforded the opportunity to evaluate changes in severity criteria with age without complete confounding of duration of recall with age.

Nearly 20% of all wheezy children never experienced a second wheezing attack (within the time frame of follow up), a finding that was reasonably constant with age. The highest proportion of children in a single year not experiencing a second attack was seen in the youngest age group. This might indicate that insufficient time had elapsed for further episodes to occur or that single episodes were not uncommon in this age group.

The distribution characteristics of wheezy episodes in the 12 months preceding the study mirrored that for the total number of wheezy episodes. A fifth of children had not had any

wheezy episodes in the past year. This distribution of children in each age group for each category was remarkably similar.

Overall, shortness of breath during attacks affected two-thirds of all wheezers. In two-thirds of these it occurred occasionally and the in remaining one-third with every episode. No age difference was present when comparing 'ever' with 'never' shortness of breath, but the prevalence of shortness of breath with every wheezy episode increased with age. A possible explanation for the age difference is that the more severe the wheezy illness the more likely it is to persist with age. Alternatively, parental perception of shortness of breath may vary with the age of the child affected.

No significant gender difference in severity was observed. In contrast, McNicol and Williams (1973) found that male asthmatics were more severely affected. There were four times as many boys as girls in the most severe group, and more than twice as many in the second most severe group. The proportions were reversed in the mildest group with the predominance of girls. In this study the gender specific distributions were very similar for each of the indices of severity. These findings are in agreement with those of Blair (1977) who showed that the sex of the child did not affect the severity at onset nor the ultimate prognosis.

Precipitants

As in most other studies, the commonest precipitant in this study, affecting nearly 90% of wheezy children, was 'a cold', by implication viral infection of the upper respiratory tract (McIntosh et al 1973; Minor et al 1974; Horn et al 1979; Tabachnik et al 1981; Foucard 1985). Non-cold precipitants, exercise and airborne allergens were also fairly common, being considered responsible for attacks in about 40%, 30% and 20% of children respectively. Many children reported more than one attack precipitant. Ingested allergens affected fewer than 10% of children. In their two cohorts of 12 year old children, Burr et al (1989) found that colds were the commonest wheeze precipitants, but that the frequency with which they precipitated wheezy episodes had dropped over the intervening 15 year period from 90% (Burr et al 1974) to 77% (Burr et al 1989). During the same time period running and animal exposure increased as precipitants. It was suggested that the increase in these precipitants

(running and animal exposure) relative to colds as wheeze precipitants reflected a true increase in asthma prevalence. The high rate of colds as precipitants of wheeze in this study is not in conflict with these observations. Further, when considering the prevalence of precipitants by age, non-cold precipitants, exercise and inhalants increased significantly. In the oldest age group half of the children wheezed with exercise, the same proportion of children as in the latter survey of Burr et al (1989). One interpretation of these findings is that the wheeze at 5 years old is more likely to be due to asthma than wheeze at a year. However, colds remain an important precipitant of wheeze throughout the age range studied.

Gender differences in prevalences of precipitants of wheeze have not been reported previously. The greater prevalence of exercise as a precipitant of wheeze in girls suggests that girls in this age group conform more than boys to 'conventional' asthma characteristics, namely, a tendency to wheeze in response to exercise.

Some 10% of wheezy episodes were precipitated by foods and fluids. This prevalence is similar to that reported in the questionnaire study of Burr et al (1989). Wilson (1985) found a prevalence of nearly 60% in white asthmatic children attending an asthma clinic. In her study, questionnaire findings were supplemented by a personal interview with each respondent. In the present study, and that of Burr et al (1989), all children with wheeze were included rather than a select group attending a hospital outpatient clinic for asthmatic children.

Seasonal and diurnal variation

Seasonal variation or more frequent symptoms at a particular time of year was present in about a third of children with wheeze, three-quarters reporting a winter preponderance. This characteristic was more apparent in older children. Diurnal variation of symptoms, particularly nocturnal worsening, is also a well known characteristic of asthma in older children and in adults. Nearly two-thirds of wheezy preschool children in this study reported that symptoms were worse at a particular time of day, mostly during the night. Population distribution was established early and remained constant with age. For seasonal and diurnal variations and also a family history of asthma there were strong associations with the diagnosis asthma on univariate analysis. This suggests that doctors diagnose asthma in

preschool children when the severity and pattern of wheeze conform with the conventional textbook descriptions. Although this may seem self evident to doctors dealing with wheezy preschool children, we have been unable to find previous population based studies of randomly selected wheezy children in this age range that have focused on these characteristics of wheeze.

Wheeze and the diagnosis of asthma

In preschool children age, wheeze severity, and precipitants other than colds were significantly associated with the diagnosis of asthma. Overall these findings probably reflect diagnostic criteria applied consciously or unconsciously by family doctors when considering whether wheezy preschool children have asthma. It is not intended to imply that all doctors apply these same criteria: some will undoubtedly be influenced by a family history of asthma and/or atopy. In the present study, however, it is clear that many wheezy children with family histories of these disorders were not diagnosed as having asthma. Whether the diagnosis of asthma was justified is uncertain as asthma in preschool children has never been clearly defined. The children studied were too young to have formal assessments of airway reactivity or reversibility to provide supportive evidence for this diagnosis. In this age group there is growing suspicion that many children wheeze for reasons other than asthma, at least where asthma is defined by the criteria applied to older children and adults. Martinez et al (1988) have shown that small airway size may be a determinant of wheeze, usually of limited duration, particularly in male infants. However, the value of investigating which factors are important in arriving at a diagnosis of asthma in this age group becomes apparent upon realising that the chances of receiving appropriate treatment depend strongly upon the receipt of a formal diagnosis (Speight et al 1983). In this study presentations which many would agree are characteristic of asthma at any age, namely frequent wheezy episodes and episodes of wheeze accompanied by shortness of breath, were not diagnosed as such. Indeed, nearly one fifth of wheezers without the diagnosis of asthma had had more than six wheezy episodes, one quarter had had three or more wheezy episodes in the preceding year, and some 60% had had a wheezy episode in the preceding three months. If such children do indeed have asthma the underdiagnosis rate is approximately 25%. Furthermore, a significant number of children with shortness of breath during each attack of wheeziness had not been diagnosed as having

asthma, the underdiagnosis rate being comparable with that described above for frequent wheezing episodes.

Reservations about the diagnosis 'asthma' in this age group have been discussed on page 105. The above comments on 'underdiagnosis' are therefore speculative; it seems more important that wheezy children are adequately treated, whether or not asthma has been diagnosed.

5.5. FOLLOW-UP, TREATMENT AND HOSPITALISATION FOR WHEEZE

In order to investigate the health care received by the 222 children with at least one reported episode of wheeze, three principle binary outcome measures were considered.

1. **On-going medical review.** Children whose parents answered yes to the question: *"Does your child attend a clinic or see a doctor for wheezing (or asthma or bronchitis)?"* were viewed as receiving on-going medical review.

2. **The receipt of asthma medication.** Children whose parents answered yes to the question: *"Has your child ever taken medicine for wheezing (or asthma or bronchitis)?"* were considered to have received asthma medication.

3. **Hospital admission.** Children whose parents answered yes to the question: *"Has your child ever been admitted to hospital for wheezing (or asthma or bronchitis)?"* were viewed as having a history of hospitalisation for a wheezing illness.

Of the 1422 children in the study population, 125 (8.8%) wheezed and had been diagnosed as having asthma, 97 (6.8%) wheezed but had no diagnosis of asthma and 31 (2.2%) had a diagnosis of asthma with cough as the sole symptom. Thus 222 (15.6%) of the study population wheezed and 156 (11%) had a formal diagnosis of asthma. The analysis of patient care (follow-up, treatment and hospitalisation) is based upon the 222 children with reported wheeze. Of these children, just over one third (77/221 = 34.8%, 1 missing observation) were under current medical review at the time of the study, three quarters (165/220 = 75.0%, 2 missing observations) gave a history of having taken asthma medication, and just over one fifth (48/221 = 21.7%, 1 missing observation) reported being hospitalised for asthma or wheezing.

Bivariate relationships between the three outcome measures and the explanatory variables in Table 28 were described using contingency tables.

Table 28. Variables investigated in a bivariate and multivariate analysis of three treatment outcome variables

Variable
Gender
Age group
Total number of subsequent attacks of wheeze
Number of attacks of wheeze in the last year
Length of time since last wheezing attack
Average duration of wheeze attacks
Shortness of breath during wheeze attacks
Nocturnal worsening of wheeze
Seasonal variation of wheeze
Recurrent coughing without a cold
Coughing with a cold
Colds as a precipitant of wheeze
Non-cold precipitants of wheeze
Wheeze precipitated by exercise
Wheeze precipitated by ingestants
Wheeze precipitated by inhalants
Urban/rural domicile
Socio-economic status
Parental smoking
Personal history of eczema
Family history of atopy (including asthma)
Presence of damp or mould at home
Child's bedroom window open at night

Because the various explanatory factors were likely to be correlated with one another, unconditional logistic regression modelling was used to investigate the multivariate relationship between these factors and each of the binary response (outcome) variables. Modelling was performed using GLIM 3.77 (Aitken et al 1989). In each case model construction was based upon the systematic addition of covariates to an initially null model. Formal tests of the significance of the improvement of fit following the addition of the regression terms relating to specific variables were based upon the likelihood ratio test (McCullagh et al 1989). Before drawing definite conclusions, all models were subjected to standard model-checking procedures, including tests for nonlinearity and interaction of leverage. Statistical analyses were performed by DKL with guidance from Dr Paul Burton who checked their accuracy.

The principal analysis included the age of the child at the completion of the questionnaire as a five level categorical variable (taking the values 1 (<1 year), 2 (1 year $\leq$ age $<$ 2 years), 3 (2 years $\leq$ age $<$ 3 years), 4 (3 years $\leq$ age $<$ 4 years), 5 (age $\geq$ 4 years). Further models were constructed using precise decimal age and age^2, but the principle inferences arising from these secondary models were qualitatively similar to those from the original analysis that only the latter are presented. In order to simplify comparisons between the three outcome measures with the results for doctor diagnosed asthma (see 5.4), number of attacks during the last year was used as a marker of wheeze frequency rather than total number of (subsequent) attacks. In any case the two measures were very strongly related to one another and inclusion of total number of attacks in the final models did not qualitatively affect the main conclusions.

Ongoing medical review was more likely in girls ($p=0.006$). Medical review was also positively related to: increasing number of wheezing episodes (total number of attacks and number of attacks in the past year) ($p<0.001$ in both cases), decreasing time since last attack ($p<0.001$), shortness of breath during attacks of wheezing ($p<0.001$), cough without a cold ($p=0.004$), the presence of a cat or dog at home ($p=0.02$) and wheeze precipitated by inhalants, but not to the remaining variables.

The findings for children who had ever received medication for wheeze or asthma were largely comparable, with significant relationships for increasing age ($p=0.03$), increasing number of wheezing attacks (total number of attacks and number of attacks in the past year) ($p<0.001$ and $p=0.003$ respectively), shortness of breath with attacks ($p<0.001$), with cough without a cold ($p=0.02$), when wheeze was precipitated by inhalants ($p<0.001$), and with cat or dog ownership ($p=0.002$). Significant relationships for the number of children admitted to hospital were found only with length of time since the last attack ($p=0.003$) and with shortness of breath during attacks ($p<0.001$).

Table 29 shows the bivariate relationships between the three outcome measures. It is clear from this table that there are strong associations between the perceived need for on-going medical review and a history of having ever received medication for wheeze. The bivariate

relationship between these two measures and hospitalisation for wheeze were all considerably weaker.

Table 29. Bivariate associations (row percentages (number)) between the three principal treatment outcome measures.

History of treatment for wheeze	Under current medical review	
	No	Yes
No	94.6 (52/55)	5.4 (3/55)
Yes	55.8 (92/165)	44.2 (73/165)
	OR = 13.75 (5.15 to 36.75) χ^2_1 = 27.446, p<0.001 2 missing observations	

Ever hospitalised for wheeze	Under current medical review	
	No	Yes
No	68.2 (118/173)	31.8 (55/173)
Yes	54.2 (26/48)	44.2 (22/48)
	OR = 1.81 (0.94 to 3.47) χ^2_1 = 3.263, p<0.07 1 missing observation	

Ever hospitalised for wheeze	History of treatment for wheeze	
	No	Yes
No	27.3 (47/172)	72.7 (125/172)
Yes	16.7 (8/48)	83.3 (40/48)
	OR = 1.88 (0.82 to 4.31) χ^2_1 = 2.274, p<0.132 2 missing observations	

OR = Odds ratio (95% confidence interval)

Table 30 gives the results of the unconditional logistic regression analysis using current medical review as the binary response variable. The important factors identified were gender, the number of attacks in the last year, ownership of a cat or dog and having inhalants (animals, dust or grass) as a wheeze precipitant. Girls were more likely to be receiving on-going medical review. As the number of attacks in the last year increased, so did the likelihood of being under current medical review. There was an important interaction between the factors of pet ownership and inhalants as a precipitant of wheeze. This interaction indicated that pet ownership in the absence of inhalants being precipitants of wheeze does not increase the chance of being under current medical review. However, owning a pet when it has been stated that such inhalants do precipitate wheeze is associated with a high proportion receiving medical follow-up.

Table 30. Unconditional logistic regression analysis of current medical review.

Parameter		Coefficient (standard error)		Odds ratio (95% confidence interval)	
Intercept		-2.946	(0.6046)	-	
Sex					
Male*		-		1.00	
Female		0.7959	(0.3481)	2.22	(1.45 to 4.38)
Number of attacks during the last year					
None*		-		1.00	
1-2		1.080	(0.6314)	2.94	(0.85 to 10.15)
3+		2.859	(0.6157)	17.44	(5.22 to 58.30)
Cat or dog ownership	Animals, dust or grass as a precipitant				
No	No*	-		1.00	
No	Yes	-0.3873	(0.4179)	0.68	(0.30 to 1.54)
Yes	No	0.4485	(0.4961)	1.57	(0.59 to 4.14)
Yes	Yes	2.614	(0.7462)	13.65	(3.16 to 58.94)

* Baseline group

Table 31 shows the results of the unconditional logistic regression analysis for treatment of wheeze. The important factors were age, number of attacks in the last year, cat or dog ownership and inhalants as precipitants of wheeze. Increasing age is associated with an

increased likelihood of being treated. The number of attacks in the last year is positively associated with the likelihood of receiving treatment. Similar positive relationships are demonstrated for pet ownership and the presence of inhalants as wheeze precipitants.

Table 31. Unconditional logistic regression analysis of treatment for wheeze.

Parameter	Coefficient (standard error)		Odds ratio (95% confidence interval)	
Intercept	-0.3471	(0.5678)	-	
Age				
≤1 year*	-		1.00	
1-1.99 years	-0.1891	(0.5660)	0.83	(0.27 to 2.51)
2-2.99 years	0.1109	(0.5755)	1.12	(0.36 to 3.45)
3-3.99 years	0.6593	(0.5947)	1.93	(0.60 to 6.20)
≥4 years	1.364	(0.6014)	3.91	(1.20 to 12.71)
Number of attacks during the last year				
None*	-		1.00	
1-2	0.2714	(0.4304)	1.31	(0.56 to 3.05)
3+	1.416	(0.5065)	4.12	(1.53 to 11.12)
Animals, dust or grass as a precipitant				
No*	-		1.00	
Yes	1.539	(0.7469)	4.66	(1.08 to 20.14)
Cat or dog ownership				
No*	-			
Yes	0.8279	(0.4014)	2.28	(1.04 to 5.03)

* Baseline group

Table 32 details the results of the unconditional logistic regression analysis for a history of hospitalisation for wheeze. The important factors were shortness of breath during attacks and the number of attacks during the last year. Shortness of breath with either some or every wheezing attack was associated with an increased likelihood of being hospitalised. Interestingly there was a strong negative relationship between the number of attacks in the last year and the likelihood of hospitalisation, with an increase in the number of attacks leading to a decrease in the likelihood of being hospitalised.

Table 32. Unconditional logistic regression analysis of hospital admission for wheeze.

Parameter	Coefficient (standard error)		Odds ratio (95% confidence interval)	
Intercept	-1.9731	(0.5118)	-	
Number of attacks during the last year				
None*	-		1.00	
1-2	-1.073	(0.4972)	0.34	(0.13 to 0.91)
3+	-1.200	(0.4809)	0.30	(0.12 to 0.77)
Shortness of breath with attacks				
Never*	-		1.00	
Sometimes	1.42	(0.5560)	4.14	(1.39 to 12.3)
Always	2.887	(0.5852)	17.93	(5.70 to 56.49)

* Baseline group

The main characteristics of preschool children with doctor diagnosed asthma was described in section 5.4. Table 33 details the bivariate relationships between doctor diagnosed asthma and each of the three principal outcome measures relating to follow-up, treatment and hospitalisation. There are significant differences for each outcome measure between those children with, and those without, a formal diagnosis of asthma. Those with a diagnosis of asthma were more likely to be under current medical review, to have ever received medication and to have been hospitalised.

Table 33. Bivariate associations between the three principal treatment outcome measures and doctor diagnosed asthma.

	Percent (number) under medical review	Percent (number) ever treated for wheeze	Percent (number) ever hospitalised for wheeze
Doctor diagnosed asthma			
No	12.5 (12/96)	50.5 (48/95)	11.5 (11/96)
Yes	52.0 (65/125)	93.6 (117/125)	29.6 (37/125)
	OR = 7.583 (3.95 to 14.55)	OR = 14.320 (7.00 to 29.29)	OR = 3.249 (1.51 to 6.64)
	$\chi^2_1 = 37.318$ $p<0.001$	$\chi^2_1 = 53.411$ $p<0.001$	$\chi^2_1 = 10.51$ $p<0.001$
	1 missing observation	2 missing observations	1 missing observation

OR = Odds ratio (95% confidence interval)

DISCUSSION

The determinants of some important aspects of medical supervision of preschool children with wheeze were examined in this study. These are current medical review, receipt of medication (ever) and admission to hospital. These questions were included in the study because it was felt that it was important to attempt to quantify this area of health care in young children, particularly as there have been no validated questionnaires on respiratory symptoms applicable in the preschool age group. Quantification was felt to be particularly important given that the rise in hospitalisations for asthma in this age group may be due to a possible increase in severity of wheeze (Anderson 1978; Mitchell 1985). Where possible, as previously mentioned (see Table 10), questions were used which have been shown to exhibit acceptable repeatability. This meant that the data accrued in response to the questions concerning hospitalisation and receipt of asthma medication, were able to be interpreted with some confidence. However, the question concerning current medical follow-up did not show the same high degree of repeatability as the other two questions. Possible explanations are that

it was limited in scope because the precise nature of follow-up was not defined, and because of the nature of this question (current rather than past treatment) it was more likely to be affected by the six month lag between the initial and repeatability surveys. Furthermore, as both the questions on receipt of medication and that concerning hospitalisation were retrospective in nature, some caution should be exercised in their interpretation. Despite these reservations, and given the repeatability of the questions used, the results obtained are likely to provide important and valuable insights into which factors are important in influencing the management of young children with wheeze, and further how acute hospitalisation is influenced by these same factors

There was a strong association between current medical review and a history of ever having received medication for wheeze. However, the association of both of these variables with hospitalisation was weak and did not reach formal statistical significance.

It is of interest to note in both bivariate and logistic regression modelling the factors associated with the outcomes of current medical review and ever receiving treatment are broadly similar. Furthermore the two regression models presented are themselves similar to the model for doctor diagnosed asthma (see Table 27), in that they indicate the importance of wheeze frequency and precipitants of wheeze other than colds. The number of attacks in the last year appeared to increase the chance of both current medical follow-up and past treatment. The finding that girls are more likely to receive current medical attention may indicate that although wheeze is more common in males, those girls with this symptom are more likely to be severely affected. As current medical review is a measure of point prevalence, it is not surprising that age is not an important determinant. However, when considering ever being treated for wheeze, the association with age is positive since it is formally a measure of cumulative incidence.

Precipitants of wheeze other than colds are important in both regression models although they act in slightly different ways. For children in whom inhalants are not a reported precipitant of wheeze, owning a cat or a dog did not increase the likelihood of being under current medical review. However, in those children for whom inhalants are a precipitant, ownership of a cat or a dog was associated with an increased probability of current medical review. On

the other hand, pet ownership appeared to be associated with an increased probability of ever having required treatment for wheeze, regardless of whether inhalants were precipitants or not. The reason for this is not clear and it may simply reflect inadequate statistical power to resolve an interaction in the case of the treatment outcome. However, it may also indicate that some parents are unaware that the family pet could be a cause of wheeze in their child. This reinforces the need to take a detailed social and environmental history when assessing children with wheeze. Shortness of breath with wheezing attacks does not appear to predict the treatment or follow-up outcomes. This is because information on wheeze severity is adequately described by the number of attacks in the last year.

The factors which are associated with hospital admission contrast with those of medical review and treatment. Shortness of breath during attacks of wheeze greatly increase the likelihood of admission. This is logical as shortness of breath describes wheeze severity during attacks - other variables are either markers of wheeze frequency or precipitants of wheeze. Of interest, the number of attacks in the last year was negatively associated with the likelihood of hospitalisation, as a greater degree of wheeze frequency is associated with a decreasing likelihood of hospitalisation. One interpretation of this is that there is a "training effect" in that the parents of a child with frequent attacks are more used to coping with acute wheezing in their child. Thus it is children who suffer infrequent severe attacks accompanied by short of breath who are at greatest risk of hospitalisation. Although most children with wheeze are never likely to require hospitalisation, an attack leading to admission to hospital could potentially come out of the blue for almost any child, regardless of their overall symptom profile and perceived need for on-going review or treatment. Furthermore, although a significantly greater proportion of wheezy children who had a formal asthma diagnosis required hospitalisation, the absence of such a diagnosis is no guarantee that a severe attack will not occur. The data supports this, for despite the strong association between having a formal asthma diagnosis and both current medical review and ever receiving medication for wheeze, the relationship between asthma diagnosis and hospitalisation was less pronounced, although still statistically significant. However alternative explanations exist. It could be that hospital admissions for wheeze in the youngest age-groups, where wheezing and its various causes are less well defined, might have been subject to spurious reporting. In order to investigate this possibility, the analysis was repeated firstly excluding children

under 1 year of age, and then children under 2. In neither instances were the principal results qualitatively changed. This modelling could only be based upon those children who reported wheeze and therefore we can make no inferences about the factors influencing the management of asthmatic children with cough as the sole symptom. In our population 31/1422 (2.2%) were diagnosed asthmatic on the basis of cough alone.

If this interpretation of the data is correct then there are considerable clinical and public health implications. Given the concern that hospital admissions have increased (Anderson 1978; Mitchell 1985), these results suggest that any intervention to address this problem must be targeted at all wheezy children and not just those who are most severely affected. Thus any strategies to improve the health of childhood asthmatics must include all wheezy children, and not just those with frequent symptoms or a diagnosis of asthma.

5.6. DETERMINANTS OF WHEEZE

In order to investigate the determinants of wheeze and, for purposes of comparison recurrent cough, the variables that were considered were: age, gender, previous history of lower and upper respiratory infections, history of eczema, past exposure risk factors (peri-natal and early childhood), bedroom conditions as well as damp and mould in other rooms in the home, heating and cooking fuels (particularly gas i.e. nitrogen dioxide), cat exposure, parental smoking, urban/rural domicile, socio-economic status, and paternal and maternal asthma and atopy.

Wheeze in the last year was the wheeze outcome measure considered in this analysis. This variable was used to minimise recall bias because, as mentioned previously, parental recall of the development of wheeze is inconsistent even within the preschool age range (see section 5.3). Two outcome measures were therefore considered.

1. **Wheeze in the last year.** Children with a history of wheeze whose parents indicated ≥ 1 attack of wheeze in the question: *"During the past 12 months, how many attacks of wheezing has he/she had?"* There were 114 (8.0%) children who had wheeze in the last year. This will be referred to as current wheeze.

2. **Recurrent cough.** Children whose parents answered yes to the question: *"Does he/she usually have a cough apart from colds?"* Excluded were children with a history of wheeze or asthma (see section 5.3). There were 310 (21.8%) children who had recurrent cough.

Bivariate relationships between the two outcome measures and the putative explanatory factors in Table 34 were described using contingency tables. Unconditional logistic regression modelling was used to investigate the multivariate relationship between these factors and each of the binary response (outcome) variables. Modelling was performed using GLIM 3.77 (Aitken et al 1989). In each case model construction was based upon the systematic addition of covariates to an initially null model. Formal tests of the significance of the improvement of fit following the addition of the regression terms relating to specific variables were based

upon the likelihood ratio test (McCullagh et al 1989). Aside from demographic variables (age group and sex) only factors which remained significant are shown in the results.

Table 34. Variable investigated in a bivariate and multivariate analysis of determinants of wheeze in the last year and recurrent cough.

Variable
Gender
Age group
History of pneumonia
History of whooping cough
History of croup
History of cystic fibrosis (not applicable)
History of bronchiolitis
History of other chest infections
History of recurrent ear infections
Personal history of eczema
Stay in hospital following birth
Day care, nursery school or play school attendance
Sharing child's bedroom
Use of heating in winter months during the day
Use of heating in winter months during the night
Child's bedroom window open at night
Condensation forming on child's bedroom window
Condensation forming on child's bedroom walls
Patches of mould or fungus on child's bedroom walls
Any damp rooms in the house
Gas used for cooking
Gas used for heating
Use of central heating
Cat as pet
Parental smoking
Paternal smoking
Maternal smoking
Urban/rural domicile
Socio-economic status
Manual/non-manual status based on paternal occupation
Paternal history of asthma
Paternal history of hay fever
Paternal history of eczema
Maternal history of asthma
Maternal history of hay fever
Maternal history of eczema

The reported incidences of the individual variables considered (Table 34) not already described are listed below.

Previous histories of respiratory infections were reported as follows: pneumonia in 10 (0.7%) children, whooping cough in 17 (1,2%) , croup in 123 (8.6%), bronchiolitis in 63 (4.4%), previous ear infections in 201 (14.1%) and other chest infections in 221 (15.5%). No child in this study had cystic fibrosis.

Thirty six children remained in hospital after their mothers had gone home. The reasons given were prematurity (22), jaundice requiring phototherapy (10), cardiac defects (3) and tracheo-oesophageal fistula (1). Fifty four percent (765/1422) of children attended a day care center, créche, nursery school or day school.

Eczema occurred in 27.7% (391/1414) of the children. The prevalences in females was slightly higher than that in males - 28% (199/691) and 26.6% (192/723) respectively, but the difference was not significant (p=0.4). A history of atopy (eczema and/or hay fever) was obtained in 51.1% (726) of parent couples, that is either the father or the mother or both, 27.7% (394) of fathers and 30% (427) of mothers. Asthma was reported in 30.7% (437) of parent couples, 16.0% (227) of fathers and 18.1% (258) of mothers.

Five hundred (35.2%) of children shared their bedrooms. Heating of the child's bedroom during the day was reported in 72% (1014//1409) of homes whilst 50.6% (694/1404) of children has their rooms heated at night. Thirteen percent (188/1401) of children slept with a bedroom window open. Eight hundred and eighteen (57.6%) reported condensation on the child's bedroom windows and 27 (1.9%) damp on the walls. Mould was present in 54 (3.8%) of the bedrooms. Six hundred and thirty six (44.8%) reported damp in the rest of the house.

In the 1422 homes gas was used for cooking in 931 (65.6%), for heating in 1006 (71.5%), for both in 736 (51.8%), and for either or both purposes in 1206 (88.9%). Of those using gas for heating, 974 used mains gas i.e. without an open flame in the home. Only a small number of homes (32) were exposed to gas combustion outside the kitchen.

Enquiry about pet exposure was divided into household and other regular exposure. Eight hundred and two (56.4%) children had pets at home, 867 (61.2%) came into contact with other pets at least once a week and 1155 (81.3%) experienced one or other exposure. Exposure to more than one type of pet was classified so that cat exposure, perhaps the most important animal exposure associated with respiratory symptoms, could be identified. One hundred and seventy (12.0%) homes kept only a cat and a further 146 (10.3%) kept a cat and other animals, together representing nearly one quarter of all homes.

Current cigarette smoking by either or both parents was present in 44.4% (631/1422) of homes. In 34.7% (436/1275) of homes the father smoked, in 30.4% (413/1386) the mother smoked, and in 15.6% (222/1422) both parents smoked. During the child's year of birth smoking was present in 44.5% (633/1422). In that year, 37.6% (473/1258) of fathers, 27.5% (374/1362) of mothers, and 15.2% (216/1422) of couples (both) smoked. Since the birth of the child 61 (4.7%) fathers have stopped smoking but 24 (1.9%) have started; the corresponding figures for mothers are 29 (2.1%) and 68 (4.9%). There has therefore been a small increase in smoking prevalence among the mothers and a similar small decrease among fathers. However there has been no real change overall in the prevalence of smoking in the homes when considering either or both parents. As a result no differences in the associations with symptom prevalences were seen when comparing current smoking with smoking during the year of birth of the child. The analysis therefore dealt only with current smoking. There were 40 homes in which 'other' adults smoked. In 20 of these one parent also smoked, so that in only 20 homes in the population were 'other' adults the sole smokers. This small number is unlikely to have influenced the results and has been omitted from further analysis.

The area of residence, or home location, of respondents was recorded from their home address post codes. The post code comprised two components - the first separated the post code area (Leicestershire) into districts, and the second subdivided the districts into, for example, street addresses. The 'district' code was used to distinguish whether the child lived in an urban or a rural area. There are 17 postal districts in Leicestershire, denoted LE1 to LE17, of which 7 representing the main towns have been regarded as urban - Leicester LE1 - LE5, Loughborough LE11 and Melton Mowbray LE13. The remaining districts include

smaller towns and the farming communities within the county. Of the 1422 respondents, 619 (43.5%) resided in urban areas and 803 (56.3%) lived in rural districts.

Social class was determined by the father's occupation and categorised into three classes, 'upper', 'middle' and 'lower' (see Section 5.2.d). Enquiry was made about parental occupations detailing job title, grade or seniority and description of work. This enquiry also determined whether the fathers occupation was manual or non-manual.

Table 35 gives the results of the unconditional logistic regression analysis using current wheeze as the binary response variable. Neither the age nor gender of the child influenced the incidence of current wheeze. Previous history of both lower and upper respiratory infections were identified as important determinants, specifically for lower respiratory tract infections, bronchiolitis and pneumonia and for the upper respiratory tract infections - croup. Amongst these infective conditions, bronchiolitis displayed the strongest association with current wheeze. Similar positive associations with current wheeze were also demonstrated for parental smoking in the home and for a maternal history of asthma.

Table 36 details the results of the unconditional logistic regression for recurrent cough. As with current wheeze, there was no effect for gender when considering the cough end-point but, by contrast, there was a modest association between increasing age and the presence of recurrent cough. The important factors were a history of bronchiolitis, sharing a bedroom, reporting of damp in the home and parental smoking in the home.

Table 35. Unconditional logistic regression analysis for determinants of wheezing in the last year.

Parameter	Coefficient (standard error)	Odds ratio (95% CI) relative to baseline category	Likelihood ratio test for removal of term from model
Intercept	-2.828 (0.335)		
Age group (years)			
≤1*		1.00	
1-1.99	-0.015 (0.366)	0.99 (0.48 to 2.01)	χ^2_4=3.062
2-2.99	-0.365 (0.389)	0.69 (0.32 to 1.48)	p=0.6
3-3.99	-0.548 (0.388)	0.58 (0.27 to 1.24)	
≥4	-0.091 (0.351)	0.91 (0.45 to 1.84)	
Sex			
Male*		1.00	χ^2_1=1.479
Female	-0.266 (0.219)	0.77 (0.50 to 1.18)	p=0.2
History of bronchiolitis			
No*		1.00	χ^2_1=75.77
Yes	1.821 (0.228)	7.80 (3.48 to 17.47)	p<0.001
History of croup			
No*		1.00	χ^2_1=7.709
Yes	0.930 (0.322)	6.18 (3.95 to 9.66)	p=0.006
History of pneumonia			
No*		1.00	χ^2_1=6.389
Yes	2.282 (0.934)	7.80 (1.57 to 6.10)	p=0.01
Parental smoking			
No*		1.00	χ^2_1=4.177
Yes	0.442 (0.217)	1.56 (1.01 to 2.38)	p=0.04
Maternal history of asthma			
No*		1.00	χ^2_1=12.368
Yes	0.854 (0.236)	2.34 (1.48 to 3.73)	p<0.001

* Baseline group
CI - confidence interval

Table 36. Unconditional logistic regression analysis for determinants of recurrent coughing.

Parameter	Coefficient (standard error)	Odds ratio (95% CI) relative to baseline category	Likelihood ratio test for removal of term from model
Intercept	-2.266 (0.269)		
Age group (years)			
≤1*		1.00	
1-1.99	-0.074 (0.286)	0.93 (0.53 to 1.63)	χ^2_4=7.90
2-2.99	-0.122 (0.287)	1.13 (0.64 to 1.98)	p=0.1
3-3.99	-0.294 (0.277)	1.34 (0.78 to 2.31)	
≥4	-0.480 (0.264)	1.61 (0.96 to 2.71)	
Sex			
Male*		1.00	χ^2_1=0.422
Female	-0.098 (0.151)	0.91 (0.67 to 1.22)	p=0.5
History of bronchiolitis			
No*		1.00	χ^2_1=5.337
Yes	0.945 (0.173)	2.57 (1.83 to 3.61)	p=0.02
Child shares a bedroom			
No*		1.00	χ^2_1=8.826
Yes	0.464 (0.155)	1.59 (1.17 to 2.16)	p=0.003
Reporting of damp in house			
No*		1.00	χ^2_1=4.39
Yes	0.316 (0.151)	1.37 (1.02 to 1.84)	p=0.04
Parental smoking			
No*		1.00	χ^2_1=5.62
Yes	0.359 (0.152)	1.43 (1.06 to 1.93)	p=0.02

* Baseline group
CI - confidence interval

DISCUSSION

Introduction

The cause of asthma is clearly multifactorial with influences from both the individual's environment and his or her family background of wheeze/asthma and allergy. The manner with which they influence symptoms might vary with age as reflected, for example, by the shift in male to female gender ratio in asthma prevalence from 3:2 in childhood (McNicol et al 1973; Clifford et al 1989; Burr et al 1989) to 1:2 in adulthood (Martin et al 1980). Tepper et al (1986) presented evidence that male infants have relatively smaller airways than their female counterparts and consequently are more likely to wheeze. The greater frequency and severity of lower respiratory tract infections observed in boys (Glezen et al 1973) supports this concept of anatomical vulnerability in the boys. This vulnerability may diminish with age as the lungs grow and develop. Symptoms in girls may become manifest only when there is an additional atopic component (Duffy et al 1990).

This study examined the association between numerous potential risk factors and the development of wheeze in the last year and recurrent cough. These are divided into: previous risk factors in the child - peri-natal and early childhood (specifically attending pre-school classes); previous upper and lower respiratory infections in the child; background of allergy - in the child of eczema and in the family of wheeze/asthma, hay fever and eczema; indoor environmental factors - sharing of bedroom, damp and mould, gas exposure from heating and cooking, cats and passive smoke exposure; outdoor environmental factors - urban/rural domicile; and socio-economic status.

Previous respiratory infections

A previous history of bronchiolitis, croup or pneumonia were all significantly associated with current wheeze, and bronchiolitis with recurrent cough. These associations may reflect either that such infections are determinants of subsequent recurrent or persistent respiratory symptoms (Wesley et al 1991) or that these conditions present with the symptoms under investigation. In a follow-up to six years of the prolonged after-effects of pneumonia in children, Wesley et al (1991) found that affected children suffered significant sequelae with persistent symptoms in 40%, abnormal radiographs in a half and abnormal lung function in a third. Furthermore this increased risk of permanent impairment of lung function and

development of chronic obstructive lung disease persists to adulthood in children who have had pneumonia (Samet et al 1983). As mentioned, an alternative explanation of the association this study showed is that the reported symptoms may be reflecting the respiratory infections in question. In bronchiolitis, and also pneumonia, wheeze can be the main presenting symptom. In this study 37 of the 63 children in whom bronchiolitis had been reported also reported a history of wheeze, and in only 5 of these 37 was a single wheezy episode reported. The remaining children experienced recurrent wheezy episodes. Recurrent wheeze and cough are not uncommon in the first one or two years of life following bronchiolitis but it is unclear whether these symptoms are the result of bronchiolitis *per se* or whether they reflect an inherent wheezing tendency which increases the likelihood of acute bronchiolitis. The precise impact of bronchiolitis on wheeze cannot therefore be computed and for that reason this study did not exclude such children from the analysis. Thus the estimates of wheeze prevalence and the influences of environmental factors is based on data from all the wheezy infants reported, including the small number with previous bronchiolitis. Both wheeze and recurrent cough are also predominant symptoms in children with cystic fibrosis. There were however no children in the study group in whom cystic fibrosis was reported, but the lack of any such respondent is not surprising as the cohort size was 1422 and the reported prevalence of cystic fibrosis in the United Kingdom is about 1 in 2500 (Dinwiddie 1990).

Parental smoking

Enquiry was made about the prevalence and extent of parental smoking in the home and about other adult smokers present. Fathers and mothers were asked about past smoking habits since the birth of the index child. The questions related specifically to smoking in the home as parents out at work during the day may smoke most away from the child's environment. Thirty percent of mothers and one-third of fathers smoked. This rate of smoking in the population is similar to reported rates of 20-25% for adults in the UK and the USA. The percentage of mother and fathers smoking had changed very little since the birth of the child, which is consistent with previous reports (Kleinman et al 1988). Parental smoking rates of 30-35% have been reported during pregnancy and the child's first year of life (Weitzman et al 1990; Wright et al 1991). When the effects of paternal, maternal and parental smoking on respiratory symptom prevalences were assessed, they were shown to be of similar

magnitude. This might reflect similar degrees of smoke exposure. The average number of cigarettes mothers admitted to smoking in the home was 12 compared with 9 per day by the fathers. Consequently, only the data for parental smoking are shown. Similarly, only current smoking data are shown as past and current smoking prevalences were almost identical. As there were very few other adults who smoked in the home, their results have been omitted from the analysis.

Passive smoke exposure was significantly associated with both wheeze in the last year and recurrent cough. This was the only environmental factor to be associated with wheeze and the only factor to be associated with both symptoms. These findings of positive associations between reported respiratory symptoms and parental smoking at home support previous reports in preschool (Colley et al 1976) and school-aged children (Ware et al 1984; Cogswell et al 1987; Somerville et al 1988). However, in a recent study of 7 and 11 year old children, Clifford et al (1989) failed to show any association between respiratory symptoms and smoking. A possible explanation for this difference in older children is their absence from the home environment for long periods during the day; insults to the lung may be sufficiently reduced to influence its effect on symptoms (Ferguson et al 1981; Wright et al 1991). Alternatively, Somerville et al (1988) have suggested that failure to establish an association between respiratory symptoms and parental smoking in older children is due to limiting enquiry to too few symptoms.

Socio-economic factors, including bedroom sharing

Reported associations between socio-economic influences, including social class and household crowding (as indicated here by bedroom sharing), and respiratory symptom prevalences are inconsistent (McNicol et al 1973a; Leeder et al 1976; Peckham and Butler 1978; Schenker et al 1983; Horwood et al 1985; Kaplan and Mascie-Taylor 1985; Mitchell 1989). One reason for these apparently contradictory findings might be that many of the factors which themselves positively influence greater respiratory symptom prevalences are more commonly encountered in lower socio-economic homes. These factors include damp and mould (Strachan 1991), parental smoking (Weitzman et al 1990; Wright et al 1991) and urban domicile (Colley et al 1970). In this study the above environmental factors were often interrelated with one another and with the social factors assessed. Damp, mould and parental

smoking influences were significantly inversely related to social class in univariate analysis. They also occurred more commonly in urban homes. In turn, save for urban domicile, all of these environmental factors influenced the prevalence rates of at least one of the respiratory symptoms studied. Therefore, any apparent association, for example between lower social class and recurrent cough prevalence, might not be due to social class per se but to the higher prevalence of smoking or household damp with lower SES homes and so on. In addition each environmental factor, although more prevalent in the lower social classes, is not uniformly prevalent and thus their effect, as expressed in the social class analysis, could perhaps be diluted, hence producing disparity in results. Reporter bias is a further possible reason for inconsistencies in reported associations between social factors and respiratory symptom prevalences. For example, respondents in lower social classes report higher wheeze prevalence whereas asthma is reported more commonly by their higher class counterparts, suggesting different perceptions of the same illness by different classes (Leeder et al 1976). In this study recurrent cough, perhaps the least sophisticated symptom, occurred more commonly with bedroom sharing. However, wheeze was unaffected by the social factors studied, a finding in keeping with those of most recent reports (Schenker et al 1983; Horwood et al 1985; Mitchell et al 1985). The conflicting evidence concerning the link between social class status and respiratory symptoms may be that it influences some symptoms and not others. Schenker et al (1983) showed an inverse association between social class and recurrent cough prevalence but no association with wheezing. In this study, as shown above, bedroom sharing was associated with recurrent cough but not with current wheeze. Further explanation for inconsistencies relates to limitations of the SES classification system. In this study families were grouped according to classification used in the National Population Census of 1981 (OPCS 1980). Father's occupation is taken as the measure of class. Clearly this may not reflect home living conditions. As these are more likely to influence respiratory prevalences, attempts to include them in alternative indices of social class have been made (Taylor et al 1982; Bisgaard et al 1989). These indices include some measure of crowding. Despite such modifications, inconsistencies between studies remain with some but not all investigators showing lower social index to be associated with respiratory symptoms. Living conditions are to a considerable extent determined by income. However, families with comparable means are not necessarily equally exposed to the same

environmental factors which may influence respiratory symptoms. This may explain some of the inconsistencies in reported associations.

Damp and mould

The presence of damp or condensation on the windows and walls of the children's bedrooms were listed in separate questions whilst for the remainder of the house parents were asked about unspecified damp. Nearly 60 percent of parents reported condensation on the bedroom windows and almost half the homes had damp elsewhere. Only 2 percent reported damp on the bedroom walls. In a study of Dutch homes, Dijkstra et al (1990) found damp stains in 15% and mould growth on surfaces in 9%. Similar damp:mould proportions have been reported in homes in Scotland (Strachan and Elton 1986) and Sweden (Andrae et al 1988). These data might suggest that household damp in this study was uncommon. If the damp:mould ratio in this region was comparable to those mentioned above (Strachan and Elton 1986; Andrae et al 1988; Dijkstra et al 1990), and together with a mould prevalence in this study of 8%, significant damp could be presumed to have occurred in approximately 14% of homes. However, in the analysis of the effects of damp on respiratory symptoms, a prevalence of damp in the home of 45% was used based on the occurrence of bedroom-wall damp and unspecified damp in the rest of the home. Unfortunately the question was phrased in a manner which did not allow estimation of wall dampness in the home aside from the bedroom. Mould was reported in only 54 (3.8%) of bedrooms and in just under 10% of homes. These small numbers limited the power of the analysis to show statistical differences when real differences may have been present. In addition, the assessment of the influence of damp and mould on respiratory symptoms is compounded by the fact that both may occur together with other factors that increase symptom prevalence i.e. lower social class and urban homes. Recurrent cough was more common where damp was present in the home. This is consistent with other reports of greater respiratory symptom prevalences in the presence of damp (Strachan and Elton 1986; Andrae et al 1988; Brunekreef et al 1989; Strachan et al 1989). As mentioned, the influence of mould in this study may not become manifest for statistical rather than real reasons.

Other environmental factors

Further environmental factors were considered in this analysis but failed to show any significant association with either of the symptom end-points considered. These were exposure to gas (nitrogen dioxide) in the home, exposure to cats in the home and urban/rural domicile. These factors were considered in this study because of previous reports of associations with respiratory symptoms - as discussed below

Nitrogen dioxide

Exposure to nitrogen dioxide (NO_2) is most likely to occur in homes where gas is used for heating and/or cooking. In this study most gas heating came from mains gas which would be a limited source of NO_2 exposure in the home. Neither current wheeze nor recurrent cough prevalences were associated with exposure to NO_2. These findings support reports that no association exists between domestic exposure to NO_2 and respiratory symptom prevalence (Schenker et al 1983; Ware and Dockery 1984; Strachan and Elton 1986; Dijkstra et al 1990). Earlier studies had suggested an association between gas exposure and increased respiratory symptoms (Melia et al 1979; Speizer et al 1980). Perhaps improvements in ventilation since that time have reduced levels of exposure below a critical level necessary for symptom production. Dijkstra et al (1990) recently measured NO_2 in Dutch homes and found that weekly concentrations and peak measurements were within internationally acceptable limits. Comparable data are not available for the UK.

Cats

More than half the families in the study population owned pets and 4 out of 5 children were regularly in contact with animals. This experience is similar to that of Sarsfield et al (1976) who showed that 55 percent of children in Leeds had close contact with pets. These authors emphasised the importance of considering environments other than the home as some 70 percent of schools in their study area had furry animals (Sarsfield et al 1976). Sixty percent of children in this study were exposed to pets outside their home, though many had no pets at home. Similar high exposure rates have been reported from Sweden (Kjellman and Petterson 1983; Vanto and Koivikko 1983). In keeping with other population studies (Andrae et al 1988; Clifford et al 1989) pet ownership or contact was not associated with increased prevalences of wheeze or asthma. Possible explanations for the failure to demonstrate

associations between pet exposure and symptom prevalences include the avoidance of pets by parents (Murray et al 1983), or that animal danders are so widespread in the environment (Vanto and Koivikko 1983) that reported avoidance of the animals does not preclude exposure to their danders.

Urban/rural domicile (Home location)

Reports particularly from underdeveloped communities have shown that the area in which children live may per se influence the development of respiratory symptoms (Morrison Smith 1976). These reports, originating primarily in the southern hemisphere and specifically those of Van Niekerk et al (1979) and Waite et al (1980), have shown that children who move from less developed or rural environments to more developed or urban environments acquire significantly more respiratory symptoms. The factor(s) responsible have not yet been identified but are thought to be associated with industrial pollution that can accompany urbanisation (Andrae et al 1988). In this study, population distribution was identified and subdivided into urban and rural on the basis of postcode area. The urban areas were Leicester, Loughborough and Melton Mowbray, where nearly 45 percent of the study population lived. These areas were not characterised by any particular industry with emissions of known pollutants. Perhaps the major urban pollutant in the area came from motor car exhausts. Possible reasons for the failure of associations between urban dwelling and respiratory symptoms might be the relative 'lack' of industrial pollution in this region or that as the urban areas were relatively small urban exposure to motor car emissions was also limited. However, these reasons remain speculative as the factors in urban dwelling and in pollution that cause greater prevalences of respiratory symptoms remain to be elucidated.

Family background of asthma and atopy

The study was not primarily concerned with the inheritance of wheeze or asthma. However, questions about atopy and wheeze in the natural parents and siblings of the child were included in the questionnaire. Interpretation of responses obtained was limited by the following factors. The study sample was based on the estimated prevalence of wheeze - almost certainly too small to assess genetic associations. Thus, statistical significance may not have been reached because of Type 2 errors. Secondly, where gender differences are apparent, the analysis is limited by lack of information about gender and number of siblings

affected. Thirdly, as shown in the study of atopy by Cookson and Hopkin (1988), clinically documented atopic symptoms may not have been reported in replies to questions of similar format to their study. Finally, in an attempt to avoid misclassifying non-atopic individuals as atopic, hay fever was not included in the enquiry. Coryzal symptoms due to viral upper respiratory infections could be confused with hay fever which is seldom diagnosed with confidence in young children. Direct enquiry about hay fever in this age group could provide misleading results.

Eczema in the child.

Eczema identified atopy in the individual child whereas in parents (and siblings) eczema and/or hay fever were the markers for atopy. Eczema was reported in almost 30 percent of the study population with both sexes equally affected. As eczema was not associated with either respiratory symptom, specifically wheeze, this finding is in apparent conflict with that of Fergusson et al (1983) who showed that the presence of atopy is strongly associated with wheeze and asthma. Therefore the findings of this study are not consistent with the view that the acquisition of atopy may be a major determinant of wheeze in young children.

Parental history of asthma and atopy

The prevalence of current wheeze was influenced by a maternal background of asthma. No other family background variable influenced current wheeze prevalence; parental, maternal or paternal asthma or atopy had no influence on the prevalence of recurrent cough. The predominant influences of factors (past respiratory conditions and environmental factors) other than family asthma/atopy on these symptoms might be explained by failure by respondents to recognise their own and their family's atopy, as shown by Cookson and Hopkin (1988), or the multi-factorial (Edfors-Lubs 1971; Duffy et al 1990) or multi-genic (Cookson et al 1989) nature of wheezy illnesses. In this study 40.5 (90/222) with wheeze had no family background of wheeze and 36.5 (81/222) no background of atopy.

The stronger association between like conditions in parent and child (wheeze-asthma) than unlike conditions (wheeze-atopy) described earlier by Gerrard et al (1976) and subsequently confirmed by Duffy et al (1990) was seen in this study.

There have been conflicting reports of the influence of gender and inheritance of wheeze. In this study the prevalence of current wheeze was influenced by the maternal history of asthma only and not by paternal asthma background. These findings are in apparent conflict with previous reports which have shown either no gender association (Sibbald 1980) and greater parental influences on asthma in boys (Fergusson et al 1983). These studies did however not investigate individual parental influences but rather combined parental effects on individual children.

Conclusions

In designing this study the sample size calculation was determined by the primary objective of the study, namely, to determine the prevalence of wheeze in the population. An estimated 60% response rate would have sufficed to achieve this objective but the very good response rate of 86.2% (1422/1650) increased the power of the study and thereby of the ability to investigate environmental, social and hereditary factors influencing these symptoms. However in a study of this moderate size the small numbers of respondents reporting some of the environmental factors considered (e.g. damp on the walls, mould in the bedrooms, combustion outside the kitchen) could have resulted in false negative associations.

This study has shown a number of associations with respiratory symptoms. The study showed that the determinants of current wheeze in this young group of preschool children were primarily prior respiratory infections together with passive parental smoking and maternal wheeze. In contrast the prevalence of recurrent cough was less influenced by hereditary factors and more by the environmental factors - previous lower respiratory infections, parental smoking, bedroom sharing and dampness in the home. However, it is recognised that association does not imply causation and that to determine cause intervention studies need to be performed.

6. CONCLUSIONS

This study investigated the prevalences of respiratory symptoms in preschool children in Leicestershire. The methodology ensured that the study sample was representative of the source population without selection bias. Accuracy of prevalence estimates was addressed by maximising questionnaire response rates through repeating postings to non-responders, by testing respective prevalences in the responses to the three mailings and by ensuring that computer data capture was as accurate as possible. As children under 5 years old have not been studied in this manner before, that is, a questionnaire prevalence study of respiratory symptoms, a new questionnaire had to be developed. Where possible previously validated questions, from other studies, with good repeatability and reliability were used, however some new questions relevant to the age of the study subjects had to be developed. Although limitations to the questionnaire were identified, good to excellent repeatability of the key questions ensured as accurate prevalence estimates as possible.

Previous studies in school-aged children have interpreted respiratory symptom prevalence estimates (predominantly wheeze) in the preschool years in their study subjects with caution because of imperfect parental recall. This study, which included only preschool subjects, showed parental recall even within this age-group to be flawed. The fact that the parents of very young children reported a similar cumulative prevalence of wheeze to the parents of older children does indeed suggest that some episodes of wheeze which are regarded as significant by parents of the younger children are forgotten as the child gets older. This emphasises the difficulty in interpreting information on the symptomatic profile from retrospective recall and highlights the need for further such studies within this age-group to accumulate more accurate information.

The cumulative wheeze prevalence was lower than that of recent studies in school-aged children whereas that of current wheeze was similar, confirming again that the cumulative prevalence should increase with increasing age with the addition of new cases but in addition that parental recall is poor with many episodes forgotten. However, in contrast, cumulative asthma prevalence is somewhat surprising as it was similar to the older children, reflecting possibly reluctance to diagnose asthma not only in preschool children but also in their older school-aged counterparts. Prevalence estimates were in keeping with observations in other studies over time, that is that wheeze prevalence does increase more slowly than that of

asthma. These findings support notions that more wheezy children than previously are now diagnosed as having asthma.

The description of the nature of wheeze - severity, precipitants, seasonal and diurnal variations - were unique to this study as were the age and gender descriptions. Investigating which factors doctors' used in arriving at the diagnosis of asthma are of potential importance for health-care education. Presentations used in this study population were frequent episodes of wheeze and episodes of shortness of breath. However many presenting with these symptoms were not labelled 'asthmatic' or treated for asthma symptoms.

The treatment of wheeze and factors determining the likelihood of receiving treatment, were investigated. This study showed that only a third of patients were currently under medical review whilst three-quarters had had medication (ever), although factors which determined these treatment variables were comparable - frequent wheeze and inhalant exposure. Only a fifth had been hospitalised for wheeze and, in contrast to the other treatment variables considered, factors descriptive of individual attacks, that is shortness of breath, determined hospitalisation whilst increased wheeze frequency was associated with significantly less hospitalisation. Not unexpectedly perhaps the severity of individual attacks determined whether or not patients were admitted to hospital.

The development of wheeze in this age-group of young preschool children was associated with previous respiratory infections, parental smoking and maternal atopy. The family history or background of wheeze/asthma and allergy playing a lesser role than environmental factors in wheeze and no role in recurrent cough. Wheeze prevalence is high in preschool children. Measures to reduce the rate of development of wheeze need to limit exposure to respiratory infections and parental smoking. The effect of such measures requires further well planned intervention studies.

Follow up of the established cohort during the early school years (ages 5-7 years), a time when most childhood asthmatics will already have presented, and when children can be more fully assessed by physiological studies, will reveal the extent to which preschool wheeze (and recurrent cough) determinants contributed to the development of asthma (diagnosed by both

clinical and physiological criteria). Whether doctors diagnose asthma reliably in the preschool years should also become apparent.

It is recognised that this study has some weaknesses. Firstly, because this study addresses preschool children during the preschool period it was hoped that parental recall would be more complete than in the retrospective studies reported for preschool children based on information from the parents of school-aged children. However even in this study was there evidence that parental recall was incomplete, emphasising the need for future studies of wheeze in preschool children to be prospective in design. Secondly, the questionnaire was not validated for the age-group studied. The questions were derived from previously validated questionnaires designed for an older age-group. Thirdly, there was no confirmation of reported environmental factors. It is possible that the presence of respiratory symptoms might heighten parental awareness and possibly lead to over-reporting of say mould or damp or under-reporting of cigarette smoking. The opposite may be true where symptoms are not present, parents here less likely to recognise potential environmental factors enquired after. In addition, statistical associations do not imply cause and effect relationships, and to determine these interventional studies would be indicated. In this study no attempt was made to try to confirm causality by, after confirming resistance to statistical control (i.e. by unconditional logistic regression analysis), looking for a dose-response relationship or for specificity to the lower respiratory tract. Finally, certain questions in the questionnaire need amplification so as to improve the data yield from the questionnaire. Specifically, the questions on family history of wheeze and atopy could have been more detailed to determine which and how many siblings were affected, and the question on treatment would have been more useful if the respondents had been given the opportunity to identify which medicines had been prescribed.

APPENDIX 1

Example of page of computer print-out from
Leicestershire Health Authority Child Health Index

AS AT 00:10 ON 22-FEB-90

PRINT ALL CHILDREN BORN BETWEEN 1/1/34 AND 31/12/39 (INCLUSIVE)
SORT SEQUENCE : ASCENDING AGE
PRINT FORMAT : BASIC DEMOGRAPHIC,ADDRESS (SINGLE LINE) AND BIRTH DETAILS
SELECTION BY : ALL

HIGGINSON REBECCA M 27-DEC-87 FEMALE 4144732
26 OVERDALE AVENUE, GLENFIELD, LEICESTER, LE3 8GP
LEICESTER ROYAL INFIRMARY LIVE 10:08 PM 3390G 1
ETHNIC ORIGIN : OTHER (I)

HILL CHARLOTTE H3 27-DEC-87 FEMALE 4144686
29 GROSSHEDGE CLOSE, LEICESTER, LE4 2UE
LEICESTER GENERAL HOSPITAL LIVE 04:47 AM 3275G 1
ETHNIC ORIGIN : NOT RECORDED

HOLMES JONATHON 27-DEC-87 MALE 4144740
23 LINLEY AVENUE, SHEPSHED, LEICESTERSHIRE, LE12 9HJ
LEICESTER ROYAL INFIRMARY LIVE 01:50 PM 3570G 1
ETHNIC ORIGIN : BRITISH ISLES (A)

HOLT ROXANNE M 27-DEC-87 FEMALE 4146379
57 EDWARD STREET, HINCKLEY, LEICS, LE10 0DN
NUNEATON MATERNITY UNIT LIVE 10:15 AM 3795G 1
ETHNIC ORIGIN : NOT RECORDED

LONG ROBERT D 27-DEC-87 MALE 4144759
47 MORLEY STREET, MELTON MOWBRAY, LEICESTERSHIRE, LE13 0LG
ST MARYS HOSPITAL LIVE 03:40 AM 3400G 1
ETHNIC ORIGIN : NOT RECORDED

MATTS JAMES W 27-DEC-87 MALE 4144767
43 PINE ROAD, GLENFIELD, LEICESTER, LE3 8DH
LEICESTER GENERAL HOSPITAL LIVE 12:11 PM 2800G 1
ETHNIC ORIGIN : BRITISH ISLES (A)

PARMAR KARAN 27-DEC-87 MALE 4145631
7 VANCOUVER ROAD, LEICESTER, LE1 2SS
LEICESTER ROYAL INFIRMARY LIVE 11:45 PM 3240G 1
ETHNIC ORIGIN : NOT RECORDED

PATEL CHIRAG 27-DEC-87 MALE 4144791
31 HALKIN STREET, LEICESTER, LE4 6JX
LEICESTER ROYAL INFIRMARY LIVE 05:47 AM 2620G 1
ETHNIC ORIGIN : ASIAN (INDIAN SUBCON., WHEREVER BORN) (I)

PICK JOSEPH J 27-DEC-87 MALE 4144805
6 VICARAGE LANE, BARKBY, LEICESTER, LE7 3QN
LEICESTER GENERAL HOSPITAL LIVE 11:00 AM 3300G 1
ETHNIC ORIGIN : BRITISH ISLES (A)

RAJA SUNNI 27-DEC-87 MALE 4144813
1 LINDESFARNE ROAD, SYSTON, LEICESTER, LE7 8QJ
ROUNDHILL MATERNITY HOSPITAL LIVE 03:22 AM 3070G 1
ETHNIC ORIGIN : ASIAN (INDIAN SUBCON., WHEREVER BORN) (I)

APPENDIX 2

Introductory letter to questionnaire

UNIVERSITY OF LEICESTER

School of Medicine

Head of Department:
Professor Hamish Simpson
Tel: 0533 523261/2

Senior Lecturers:
Dr M. S. Tanner — Tel: 523268/9
Dr D. J. Field — Tel: 541414 ext 5522
Dr M. P. Wailoo — Tel: 523264
Dr I. D. Young — Tel: 541414 ext 5636

Honorary Senior Lecturer:
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Tel: 541414 ext 6460

Department of Child Health
Clinical Sciences Building
Leicester Royal Infirmary
PO Box 65
Leicester LE2 7LX

Fax no: 0533 523107

Dear Parent of _____

Coughs, colds and chest problems cause a lot of misery among young children and considerable worry for their parents. In this department we are carrying out a survey of Leicestershire children to find out how many are affected by chest problems and what factors may be related to their ill health.

With the agreement of the Leicestershire Health Authority, this questionnaire is being sent to the parents of a random sample of Leicestershire children aged 5 years and under. For practical purposes, it was impossible to include all children in the study so, even though you may have more than one young child in your family, you are likely to receive only one questionnaire. Please answer the questionnaire for the child whose name appears at the top of this page.

In order to obtain representative results it is important that everybody returns a completed questionnaire. To avoid us having to contact you again, we would be grateful if you would complete the questionnaire today and post it as soon as possible in the pre-paid envelope supplied.

Thank you for helping us with our research into these important matters. You may be assured that all of the information that you give us will be treated in the strictest confidence. If you have any questions about the study please feel free to contact us by post or by phone on Leicester 542982 (6.00 p.m. to 9.00 p.m.).

Yours sincerely

David Luyt

Dr David LUYT
Research Assistant
Department of Child Health

Hamish Simpson

Professor Hamish SIMPSON
Head of Department
Department of Child Health

APPENDIX 3

Questionnaire

This is the questionnaire you have been asked to fill out for your child. Please answer the questions as frankly and accurately as possible about your child and your family.

The questions can be answered by ticking the best answer or by filling in a blank with words or a number.

Example: Do you live in Leicestershire Yes[✓] No[ ]

NAME OF CHILD:_____

DATE OF BIRTH:_____

ADDRESS:_____

_____

_____

DATE QUESTIONNAIRE COMPLETED:_____

PERSON COMPLETING QUESTIONNAIRE:

	Tick box please
Mother	[ ]
Father	[ ]
Female guardian	[ ]
Male guardian	[ ]
Other	[ ]

Please specify:

_____

YOUR CHILD

The questions in this section refer to the health of your child. Several questions are about WHEEZING. By this we mean breathing that makes a high-pitched whistling sound from the chest, not the throat.

1 Has your child ever had ATTACKS OF WHEEZING? Yes[ ] No[ ]

If the answer is YES, please answer from question number 2. If the answer is NO, please leave out questions 2-12 and answer from question 13.

2 How old was he/she when the first attack of wheezing occurred? _____ years _____ months

2a If the first attack occurred before the age of 6 months,please specify the age of onset (as near as you can say) in WEEKS. _____ weeks

3 Since the first attack, approximately HOW MANY has he/she had? Tick one only

None	[ ]
1 - 2	[ ]
3 - 5	[ ]
6 -10	[ ]
11 -20	[ ]
More than 20	[ ]

4 During the past 12 MONTHS, HOW MANY attacks of wheezing has he/she had? Tick one only

None	[ ]
1 - 2	[ ]
3 - 5	[ ]
6 -10	[ ]
More than 10	[ ]

If NONE, please go to question 6.

5 During the past 12 MONTHS, on average (as near as you can say) HOW LONG do these attacks last? (with the normal treatment) Tick one only

About 1 day	[ ]
2 - 3 days	[ ]
4 - 7 days	[ ]
More than 7 days	[ ]

6 How long is it since his/her LAST attack of wheezing? Tick one only

- Less than 1 month[ ]
- 1 - 3 months[ ]
- 4 - 6 months[ ]
- 7 -12 months[ ]
- 13 months or more[ ]

7 Do these attacks cause him/her to be SHORT OF BREATH?

- Yes, always[ ]
- Yes, occasionally[ ]
- No, never[ ]

8 Is his/her breathing completely normal between attacks? Yes[ ] No[ ]

9 Do these attacks occur:(Answer ALL please)

a. when he/she has a cold? Yes[ ] No[ ]
b. occasionally apart from colds? Yes[ ] No[ ]
c. when he/she is running or playing? Yes[ ] No[ ]
d. with drinking or eating? Yes[ ] No[ ]
If yes, please say which food or drink:

_____

_____

e. when he/she is near, for example animals, dust, grass and so on? Yes[ ] No[ ]
If yes, please say which:

_____

_____

10 Do these attacks occur more frequently at any particular time of year? Yes[ ] No[ ]

If the answer is YES, please indicate the 'BAD' months by ticking the appropriate box or boxes. If the answer is NO, please leave blank.

January [ ]	April[ ]	July [ ]	October [ ]
February[ ]	May [ ]	August [ ]	November [ ]
March [ ]	June [ ]	September[ ]	December [ ]

11 Is the wheezing worse at any particular TIME OF DAY? Yes[ ] No[ ]

If the answer is YES,is it WORSE: during the DAY? [ ]
during the NIGHT?[ ]

12 Has your child at any time in the last 12 MONTHS been wakened at night by an attack of coughing when he/she does NOT have a cold or chest infection? Yes[ ] No[ ]

13 Does he/she usually have a cough WITH COLDS? Yes[ ] No[ ]

14 Does he/she usually have a cough APART FROM COLDS? Yes[ ] No[ ]

15 Has any doctor or hospital told you that he/she has ASTHMA or BRONCHITIS? Yes[ ] No[ ]

If the answer is YES, at what age was asthma or bronchitis diagnosed? _____ years _____ months

16 Has your child ever suffered from any of the following conditions? If the answer for any is YES, please state the age at which it was diagnosed.

	Yes	-at age	No	Don't know
a. pneumonia	[ ]	_____	[ ]	[ ]
b. whooping cough	[ ]	_____	[ ]	[ ]
c. croup	[ ]	_____	[ ]	[ ]
d. cystic fibrosis	[ ]	_____	[ ]	[ ]
e. bronchiolitis	[ ]	_____	[ ]	[ ]
f. recurrent ear infections	[ ]	_____	[ ]	[ ]
g. other chest infection	[ ]	_____	[ ]	[ ]

Please give details:_____

_____

17 Has he/she ever had ECZEMA? (An itchy, dry rash on arms, face and legs) Yes[ ] No[ ]

18 Does your child attend a clinic or see a doctor for wheezing? (or asthma or bronchitis) Yes[ ] No[ ]

19 Has your child ever taken any medicine for wheezing? (or asthma or bronchitis) Yes[ ] No[ ]

20 Has your child ever been admitted to hospital:
:with wheezing? (or asthma or bronchitis) Yes[ ] No[ ]
:with chest trouble other than wheezing? Yes[ ] No[ ]

Please give details:_____

_____

21 When your child was born, did he/she need to stay in hospital after his/her mother went home? Yes[ ] No[ ]

If the answer is YES, please give details:_____

_____

_____

22 Does he/she attend day care, nursery school or play school? Yes[ ] No[ ]

If you wish to make any other comments about your child's health, please do so in the space below.

_____

_____

_____

_____

YOUR HOUSE

In these questions, 'YOUR HOUSE' refers to the home where your child usually lives.

1 How long has he/she lived in the house? _____ _____
years months

2 How many rooms are there in your house?
(NOT counting kitchens, bathrooms and toilets)
_____
number of rooms

3 Which fuel(s) do you use for cooking? electricity [ ] gas [ ] other [ ]

4 Which fuel(s) do you use for heating? (Tick more than one box if necessary)
electricity [ ] coal [ ]
paraffin [ ] wood [ ]
mains gas [ ] oil [ ]
bottled gas [ ] other [ ]

5 Does your house have central heating? Yes [ ] No [ ]

6 Do you keep a pet animal or bird? Yes [ ] No [ ]

If the answer is YES, which pet(s)? (Tick more than one box if necessary)
Dog [ ] Bird [ ]
Cat [ ] Horse [ ]
Other [ ], please say which

_____

_____

7 Does your child regularly (at least once a week) come into contact with friends' or relatives' pets or or animals (eg pony)? Yes[ ] No[ ]

IF the answer is YES, say which. _____

_____

In these questions, YOUR CHILD'S BEDROOM is the room in which he/she sleeps.

8 Does your child share his/her bedroom with others? Yes [ ] No [ ]

If YES, please state how many. _____ number of people

9 In your child's bedroom, during the winter months:

a. is the room heated during the day? Yes[ ] No[ ]
b. is the room heated during the night? Yes[ ] No[ ]
c. is the window usually left open at night? Yes[ ] No[ ]
d. does condensation ever form on the windows? Yes[ ] No[ ]
e. does condensation ever form on the walls? Yes[ ] No[ ]
f. are there patches of mould or fungus? Yes[ ] No[ ]

10 Please list any other rooms in your house affected by:

a. condensation or damp._____

_____

b. mould or fungus._____

_____

11 Do you think that your house is cold during winter? Yes [ ] No[ ]

If you would like to add any comments about your housing, please do so in the space provided below.

_____

_____

_____

_____

_____

YOUR FAMILY

We would like some information about the family living with your child, and in particular about the parents or guardians living with your child. (In single parent families, complete only A or B as appropriate). Section C should be completed by all families.

A FATHER or Male guardian

		Tick one only
1	Is he the: natural father?	[ ]
	step father?	[ ]
	male guardian?	[ ]
	other? (eg. grandfather)	[ ]

Please specify_____

2 How old is he? _____ years

3 Has he ever suffered from any of the following conditions?

		Yes	No	Don't know
a.	asthma?	[ ]	[ ]	[ ]
b.	bronchitis?	[ ]	[ ]	[ ]
c.	hayfever? (sneezing, runny or blocked nose, sometimes itchy eyes or nose,NOT associated with a cold)	[ ]	[ ]	[ ]
d.	eczema?	[ ]	[ ]	[ ]
e.	other chest problems?	[ ]	[ ]	[ ]

Please specify_____

_____

4 Does he smoke cigarettes? Yes[ ] No[ ]

If the answer is YES, how many does he smoke each day in the house? _____ number of cigarettes

5 Did he smoke cigarettes during the year in which your child was born? Yes[ ] No[ ]

6 At what age did he finish full-time education? _____ years

7 What is his present job? Please give:

a. job title_____

b. grade or seniority_____

c. description of work_____

Is he self-employed? Yes[ ] No[ ]

If he is at present unemployed, please tick box [ ] and give nature of last job._____

_____

B MOTHER or female guardian

		Tick one only
1 Is she the:	natural mother?	[ ]
	step mother?	[ ]
	female guardian?	[ ]
	other? (eg. grandmother)	[ ]

Please specify_____

2 How old is she? _____ years

3 Has she ever suffered from any of the following conditions?

	Yes	No	Don't know
a. asthma?	[ ]	[ ]	[ ]
b. bronchitis?	[ ]	[ ]	[ ]
c. hayfever?	[ ]	[ ]	[ ]
d. eczema?	[ ]	[ ]	[ ]
e. other chest conditions?	[ ]	[ ]	[ ]

Please specify_____

_____

4 Does she smoke cigarettes? Yes [ ] No [ ]

If the answer is YES, how many does she smoke each day in the house? _____
number of cigarettes

5 Did she smoke in the year that your child was born? Yes [ ] No [ ]

6 At what age did she finish full-time education? _____
years

7 Is she working at present? Yes [ ] No [ ]

If the answer is YES, please give:

a. job title:_____

b. grade or seniority:_____

c. description of work:_____

SECTION C

1 How many ADULTS (16 years and older) usually live in your house? _____ number of adults

2 Not counting your child, how many CHILDREN under 16 usually live in your house? _____ number of children

Please state the AGES of these children (in years and months)

a._____ b._____ c._____

d._____ e._____ f._____

g._____ h._____ i._____

3 Have any of your child's BROTHERS or SISTERS suffered from any of the following conditions?

	Yes	No	Don't know
a. attacks of wheezing?	[ ]	[ ]	[ ]
b. asthma?	[ ]	[ ]	[ ]
c. bronchitis?	[ ]	[ ]	[ ]
d. eczema?	[ ]	[ ]	[ ]
e. hayfever?	[ ]	[ ]	[ ]
f. other chest conditions?	[ ]	[ ]	[ ]

Please specify_____

_____

_____

_____

4 Are there any other household members who smoke?(not counting those mentioned) Yes [ ] No [ ]

If the answer is YES, please state HOW MANY. _____ number of people

APPENDIX 4

Introductory letter to questionnaire for re-mailing of non-respondents

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Tel: 541414 ext 5636

Honorary Senior Lecturer:
Dr U. M. MacFadyen
Tel: 541414 ext 6460

Department of Child Health
Clinical Sciences Building
Leicester Royal Infirmary
PO Box 65
Leicester LE2 7LX

Fax no: 0533 523107

Dear Parent of:_____

We are conducting a survey on coughs and colds in young children in Leicestershire. You have recently received a questionnaire about your child. Our records show that we have as yet not received a reply from you. If you have returned the questionnaire, we would ask you to ignore this and we apologise for any inconvenience. If you have not returned the questionnaire, we request that you please complete the questionnaire and return it to us as soon as possible using the prepaid envelope provided.

Thank you for assisting us with this important health problem.

Yours sincerely,

David Luyt

Dr David LUYT
Research Assistant
Department of Child Health

Professor Hamish SIMPSON
Head of Department
Department of Child Health

APPENDIX 5

Introductory letter to questionnaire for repeatability mailing

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Dr M. P. Wailoo — Tel: 523264
Dr I. D. Young — Tel: 541414 ext 5636

Honorary Senior Lecturer:
Dr U. M. MacFadyen
Tel: 541414 ext 6460

Department of Child Health
Clinical Sciences Building
Leicester Royal Infirmary
PO Box 65
Leicester LE2 7LX

Fax no: 0533 523107

Dear Parent of:_____

Thank you for your help in completing the questionnaire about the health of your child. We have had a very positive response from the Leicestershire parents, for which we are very grateful. We feel sure that we will be able to make an accurate and meaningful analysis of the replies in the hope that this will help to explain the recent worrying increase in wheezing and asthma amongst children in Britain.

In order to undertake this analysis we need to check the questionnaire. This requires a sub-group of the parents originally contacted being asked to repeat the questionnaire. This is a quality control on the questionnaire itself, and not a check on the parents' previous replies. We would be very grateful if you could help us by completing the questionnaire as before, and returning it in the reply paid (freepost) envelope provided.

We apologise for any inconvenience that this may cause and once again thank you for your helpful cooperation in tackling this important health problem.

Yours sincerely

David Luyt

Dr David LUYT
Research Fellow
Department of Child Health

Hamish Simpson

Professor Hamish SIMPSON
Head of Department
Department of Child Health

APPENDIX 6

Questionnaire coding form

'ASTHMA PREVALENCE'

PERSONAL IDENTIFICATION
(SER NO:- ,INITIALS:- 1st and last) serial:[ ][ ][ ][ ] init:[ ][ ]

(DATE OF BITRH), dayb:[ ][ ] monb:[ ][ ] yearb:[ ][ ]

(POSTCODE), pc1:[ ][ ][ ][ ] pc2:[ ][ ][ ]

(MAILING,COMPLETER), mail:[ ] comp:[ ]

(DATE OF RECEIPT), dayr:[ ][ ] monr:[ ][ ]

YOUR CHILD

WHEEZER [0,1] wheeze:[ ]

(AGE OF FIRST ATTACK, IF NON-WHEEZER, TYPE <RETURN>,

year1:[ ] mon1:[ ][ ] week1:[ ][ ]

(DESCRIPTION), totalno:[ ] lastyear:[ ] length:[ ]

last:[ ] SOBA:[ ] SOBN:[ ]

cold:[ ] nocold:[ ] run:[ ] eat:[ ] dust:[ ]

(TIMING), month:[ ]

trim1:[ ] trim2:[ ] trim3:[ ] trim4:[ ]

time:[ ] day:[ ] night:[ ]

(AWAKE WITH COUGH), cowake:[ ]

(COUGHING), ccold:[ ] cnocold:[ ]

(ASTHMA OR BRONCHITIS), asbronc:[ ] yeard:[ ] mond:[ ][ ]

(OTHER ILLNESS?),

pneum:[ ] whoop:[ ] croup:[ ] cystf:[ ]

blitis:[ ] othinf:[ ] earinf:[ ]

(ATOPY?),eczema:[ ] doctor:[ ] drugs:[ ] hospab:[ ] hospoth:[ ]

(BIRTH CHILDHOOD), stay:[ ] nursery:[ ]

(OTHER COMMENTS), other:[ ][ ][ ]

YOUR HOUSE
(SER NO:-) serial:[ ][ ][ ][ ]

(HOUSE DETAILS),

yrlive:[ ] mlive:[ ][ ] rooms:[ ][ ] cookfuel:[ ] heatfuel:[ ][ ]

centheat:[ ]

(PETS), pet:[ ] pettype:[ ] contact:[ ]

(BEDROOM), share:[ ] number:[ ][ ]

(DAMP AND MOULD),

dayheat:[ ] ntheat:[ ] ntopen:[ ]

condwind:[ ] condwall:[ ] mould:[ ]

(NO OF OTHER AFFECTED ROOMS),

ndamp:[ ][ ] nmould:[ ][ ]

(HOUSE COLD?), housecold:[ ]

(OTHER COMMENTS), otherhse:[ ][ ][ ]

MALE GUARDIAN,

(SER NO:-) serial:[ ][ ][ ][ ]

(WHAT RELATION? IF NO MALE GUARDIAN TYPE <RETURN>)m1

malerel:[ ]

(DETAILS ABOUT MALE GUARDIAN),

agem:[ ][ ]

asm:[ ] brm:[ ] haym:[ ] eczm:[ ] othm:[ ]

smokem:[ ] smokeno:[ ][ ] smkmborn:[ ]

endagem:[ ][ ]

(OCCUPATIONAL CODING),

codem3:[ ][ ][ ] codem2:[ ][ ] SEGm:[ ][ ] SCm:[ ] manm:[ ]

FEMALE GUARDIAN

(WHAT RELATION? IF NO FEMALE GUARDIAN TYPE <RETURN>) fmal

fmalrel:

(DETAILS ABOUT FEMALE GUARDIAN),

agef:

asf: brf: hayf: eczf: othf:

smokef: smokeno: smkfborn:

endagef:

(OCCUPATIONAL CODING),

code3f: code2f: SEGf: SCf: manf:

OTHER PEOPLE IN HOUSE:

adults: children:

a: b: c: d: e:

f: g: h: i:

(SIBLINGS ILL HEALTH?),

swheeze: sasthma: sbronch:

seczema: shayf: soth:

(OTHER SMOKERS), smkno:

APPENDIX 7

Coded questionnaire showing coding used

Serial [ ][ ][ ][ ]

This is the questionnaire you have been asked to fill out for your child. Please answer the questions as frankly and accurately as possible about your child and your family.

The questions can be answered by ticking the best answer or by filling in a blank with words or a number.

Example: Do you live in Leicestershire Yes[✓] No[ ]

NAME OF CHILD: _____ init [ ][ ]

DATE OF BIRTH: dayb [ ][ ] monb [ ][ ] yearb [ ][ ]

ADDRESS: _____ pc1 [ ][ ][ ][ ] pc2 [ ][ ][ ]

_____

_____

DATE QUESTIONNAIRE COMPLETED: _____ mail [ ]

PERSON COMPLETING QUESTIONNAIRE:

comp

	Tick box please
Mother	[1]
Father	[2]
Female guardian	[3]
Male guardian	[4]
Other	[5]

Please specify:

_____

Both mother & father [6]

Missing [9]

dayr [ ][ ] monr [ ][ ]

YOUR CHILD

The questions in this section refer to the health of your child. Several questions are about WHEEZING. By this we mean breathing that makes a high-pitched whistling sound from the chest, not the throat.

1 Has your child ever had ATTACKS OF WHEEZING? wheeze Yes[1] No[0]

If the answer is YES, please answer from question number 2. If the answer is NO, please leave out questions 2-12 and answer from question 13.

2 How old was he/she when the first attack of wheezing occurred? year | | | mon | | | years months

2a If the first attack occurred before the age of 6 months, please specify the age of onset (as near as you can say) in WEEKS. week | | | weeks Code 00 if >6 months

3 Since the first attack, approximately HOW MANY has he/she had? totalno

	Tick one only
None	[0]
1 - 2	[1]
3 - 5	[2]
6 -10	[3]
11 -20	[4]
More than 20	[5]
Missing	[9]

4 During the past 12 MONTHS, HOW MANY attacks of wheezing has he/she had? lastyear

	Tick one only
None	[0]
1 - 2	[1]
3 - 5	[2]
6 -10	[3]
More than 10	[4]
Missing	[9]

If NONE, please go to question 6.

5 During the past 12 MONTHS, on average (as near as you can say) HOW LONG do these attacks last? (with the normal treatment) length

	Tick one only
About 1 day	[1]
2 - 3 days	[2]
4 - 7 days	[3]
More than 7 days	[4]
Not applicable	[8]
Missing	[9]

6 How long is it since his/her LAST attack of wheezing? last

Tick one only
Less than 1 month[1]
1 - 3 months[2]
4 - 6 months[3]
7 -12 months[4]
13 months or more[5]
Missing [9]

7 Do these attacks cause him/her to be SHORT OF BREATH? SOBA

Yes, always[2]
Yes, occasionally[1]
No, never[0]

8 Is his/her breathing completely normal between attacks? SOBN Yes[1] No[0]

9 Do these attacks occur: (Answer ALL please)

a. when he/she has a cold? Yes[1] No[0] cold
b. occasionally apart from colds? Yes[1] No[0] nocold
c. when he/she is running or playing? Yes[1] No[0] run
d. with drinking or eating? Yes[1] No[0] eat
If yes, please say which food or drink:

_____

_____

e. when he/she is near, for example animals, dust, grass and so on? Yes[1] No[0] dust
If yes, please say which:

_____ _____

_____

10 Do these attacks occur more frequently at any particular time of year? month Yes[1] No[0]

If the answer is YES, please indicate the 'BAD' months by ticking the appropriate box or boxes.
If the answer is NO, please leave blank.

January [ ]	April[ ]	July [ ]	October [ ]
February[ ]	May [ ]	August [ ]	November [ ]
March [ ]	June [ ]	September[ ]	December [ ]

trim 1 trim 2 trim 3 trim 4

171 yes [1] no [0]

11 Is the wheezing worse at any particular TIME OF DAY? time Yes[1] No[0]

If the answer is YES, is it WORSE: during the DAY? [ ] day yes [1]
during the NIGHT? [ ] night no [0]

12 Has your child at any time in the last 12 MONTHS been wakened at night by an attack of coughing when he/she does NOT have a cold or chest infection? cowake Yes[1] No[0]

13 Does he/she usually have a cough WITH COLDS? ccold Yes[1] No[0]

14 Does he/she usually have a cough APART FROM COLDS? cnocold Yes[1] No[0]

15 Has any doctor or hospital told you that he/she has ASTHMA or BRONCHITIS? asbronc Yes[1] No[0]

If the answer is YES, at what age was asthma or bronchitis diagnosed? yeard [ ] mond [ ][ ]
years months

16 Has your child ever suffered from any of the following conditions? If the answer for any is YES, please state the age at which it was diagnosed.

	Yes -at age	No	Don't know	
a. pneumonia	[ ]	[0]	[7]	pneum
b. whooping cough	[ ]	[0]	[7]	whoop
c. croup	[ ]	[0]	[7]	croup
d. cystic fibrosis	[ ] *	[0]	[7]	cystf
e. bronchiolitis	[ ]	[0]	[7]	bllts
f. recurrent ear infections	[ ]	[0]	[7]	earinf
g. other chest infection	[ ]	[0]	[7]	otinf

Please give details: _____

* If yes: 0-1 yrs [1]
1-2 yrs [2]
2-3 yrs [3]
3-4 yrs [4]
4-5 yrs [5]

17 Has he/she ever had ECZEMA? (An itchy, dry rash on arms, face and legs) eczema Yes[1] No[0]

18 Does your child attend a clinic or see a doctor for wheezing? (or asthma or bronchitis) doctor Yes[1] No[0]

19 Has your child ever taken any medicine for wheezing? (or asthma or bronchitis) drugs Yes[1] No[0]

20 Has your child ever been admitted to hospital:
:with wheezing? (or asthma or bronchitis) Yes[1] No[0] hospab
:with chest trouble other than wheezing? Yes[1] No[0] hospath

Please give details:_____

_____

21 When your child was born, did he/she need to stay in hospital after his/her mother went home? stay Yes[1] No[0]

If the answer is YES, please give details:_____

_____

_____

22 Does he/she attend day care, nursery school or play school? nursery Yes[1] No[0]

If you wish to make any other comments about your child's health, please do so in the space below. other

_____

_____

_____

_____

5

Serial [ ][ ][ ][ ]

YOUR HOUSE

In these questions, 'YOUR HOUSE' refers to the home where your child usually lives.

1 How long has he/she lived in the house? yrlive [ ] years mlive [ ][ ] months

2 How many rooms are there in your house? (NOT counting kitchens, bathrooms and toilets) rooms [ ][ ] number of rooms

3 Which fuel(s) do you use for cooking? cookfuel
electricity [1] gas [2]
other [3]
more than one with gas [4]
more than one without gas [5]

4 Which fuel(s) do you use for heating? heatfuel (Tick more than one box if necessary)
electricity [1] coal [5]
paraffin [2] wood [6]
mains gas [3] oil [7]
bottled gas [4] other [8]
more than one with gas [10]
more than one without gas [11]

5 Does your house have central heating? Yes [1] No [0]
centheat

6 Do you keep a pet animal or bird? Pet Yes [1] No [0]
none [0]

If the answer is YES, which pet(s)? (Tick more than one box if necessary)
Dog [1] Bird [3]
Cat [2] Horse [4]
Other [5], please say which

Pettype
More than one with cats [6]
More than one without cats [7]

7 Does your child regularly (at least once a week) come into contact with friends' or relatives' pets or or animals (eg pony)? Yes[ ] No[ ]

IF the answer is YES, say which.

Contact Animal type coding as for question 6

In these questions, YOUR CHILD'S BEDROOM is the room in which he/she sleeps.

8 Does your child share his/her bedroom with others? share Yes [1] No [0]

If YES, please state how many. number ☐☐ If No = 00
number of people

9 In your child's bedroom, during the winter months:

a. is the room heated during the day? Yes[1] No[0] dayheat
b. is the room heated during the night? Yes[1] No[0] ntheat
c. is the window usually left open at night? Yes[1] No[0] ntopen
d. does condensation ever form on the windows? Yes[1] No[0] condwin
e. does condensation ever form on the walls? Yes[1] No[0] condwall
f. are there patches of mould or fungus? Yes[1] No[0] mould

10 Please list any other rooms in your house affected by:

a. condensation or damp. ndamp ☐☐

b. mould or fungus. nmould ☐☐

11 Do you think that your house is cold during winter? housecold Yes [1] No[0]

If you would like to add any comments about your housing, please do so in the space provided below. otherhse ☐☐☐

YOUR FAMILY Serial [ ][ ][ ][ ]

We would like some information about the family living with your child, and in particular about the parents or guardians living with your child. (In single parent families, complete only A or B as appropriate). Section C should be completed by all families.

A FATHER or Male guardian

Tick one only

1 Is he the: natural father? [1]
step father? malerel [2]
male guardian? [3]
other? (eg. grandfather) [4]

Please specify Not applicable [8]

2 How old is he? agem [ ][ ] years

3 Has he ever suffered from any of the following conditions?

	Yes	No	Don't know	
a. asthma?	[1]	[0]	[2]	asm
b. bronchitis?	[1]	[0]	[2]	brm
c. hayfever? (sneezing, runny or blocked nose, sometimes itchy eyes or nose, NOT associated with a cold)	[1]	[0]	[2]	haym
d. eczema?	[1]	[0]	[2]	eczm
e. other chest problems?	[1]	[0]	[2]	othm

Please specify _____

_____

4 Does he smoke cigarettes? Smokem Yes[1] No[0]

If the answer is YES, how many does he smoke each day in the house? smokeno [ ][ ] number of cigarettes

If no, then smokeno not applicable =88

5 Did he smoke cigarettes during the year in which your child was born? Yes[1] No[0]
smkmborn

6 At what age did he finish full-time education? ☐☐ years
endagem

7 What is his present job? Please give:

a. job title _____ codem3 ☐☐☐

b. grade or seniority _____ codem2 ☐☐

c. description of work _____ SEGm ☐☐

Scm ☐

Is he self-employed? Yes[ ] No[ ] manm ☐

If he is at present unemployed, please tick box [ ] and give nature of last job. _____

_____

B MOTHER or female guardian

Tick one only

1 Is she the: natural mother? fmalrel [1]
step mother? [2]
female guardian? [3]
other? (eg. grandmother) [4]

Please specify Not applicable [8]

2 How old is she? agef ☐☐ years

3 Has she ever suffered from any of the following conditions?

	Yes	No	Don't know	
a. asthma?	[1]	[0]	[2]	asf
b. bronchitis?	[1]	[0]	[2]	brf
c. hayfever?	[1]	[0]	[2]	hayf
d. eczema?	[1]	[0]	[2]	ec2f
e. other chest conditions?	[1]	[0]	[2]	othf

Please specify_____

_____

4 Does she smoke cigarettes? smokef Yes [1] No [0]

If the answer is YES, how many does she smoke each day in the house? smokfno [ ][ ]
number of cigarettes

If no, then smokfno not applicable = 88

5 Did she smoke in the year that your child was born? smkfborn Yes [1] No [0]

6 At what age did she finish full-time education? endagef [ ][ ]
years

7 Is she working at present? Yes [ ] No [ ]

If the answer is YES, please give:

a. job title:_____ code3f [ ][ ][ ]

b. grade or seniority:_____ code2f [ ][ ]

c. description of work:_____ SEGf [ ][ ]

SCf [ ]

manf [ ]

SECTION C

1 How many ADULTS (16 years and older) usually live in your house? adults ☐☐
number of adults

2 Not counting your child, how many CHILDREN under 16 usually live in your house? children ☐☐
number of children

Please state the AGES of these children (in years and months) Code in completed years

a._____ b._____ c._____

d._____ e._____ f._____

g._____ h._____ i._____

3 Have any of your child's BROTHERS or SISTERS suffered from any of the following conditions?

	Yes	No	Don't know
a. attacks of wheezing?	[1]	[0]	[2] swheeze
b. asthma?	[1]	[0]	[2] sasthma
c. bronchitis?	[1]	[0]	[2] sbronch
d. eczema?	[1]	[0]	[2] seczema
e. hayfever?	[1]	[0]	[2] shayf
f. other chest conditions?	[1]	[0]	[2] soth

Please specify_____

_____

_____

_____

4 Are there any other household members who smoke?(not counting those mentioned) Yes [ ] No [ ]

If the answer is YES, please state HOW MANY. Snckno ☐☐
number of people

Code only number of people
ie. if answer no, then snckno = 00

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