Asthma

In 1971 John Morrison Smith and his colleague drew attention to a worrying trend. Asthma and wheeze were becoming more common among Birmingham school children. These trends may not have been properly interpretable as a true increase in incidence because a major rebuilding programme taking place at the time caused redistribution of the city's population, which may have been the explanation for the trend. Data collected later suggested that the trend was continuing, but the same problem of interpretation remained. Interest seems then to have been lost in the subject, perhaps as mortality from asthma fell at the end of the 1960s and early 1970s. A similar study in New Zealand came to the same conclusion, and this was also qualified by its authors on the grounds that there had been some changes in the composition of the population between the time of the surveys.

In addition to this less than perfect evidence for an increase in the prevalence of asthma, every modality of treating the condition continued to increase. Hospital admission for asthma increased rapidly in a large number of countries, particularly among the young, treatment of asthma by general practitioners in the United Kingdom increased by almost 100% in selected practices between 1970–71 and 1981–82, and prescriptions for asthma have increased even faster. Though these changes may have been due in part to a broader definition of asthma and a wider recognition of the benefits of its treatment, there is no reason to assume that these were the only causes of the increase. Recognition in the mid-1980s that reported asthma mortality was also increasing, both in the United Kingdom and in a large number of other countries, should have raised an alarm, coming as it did in the face of increased treatment. Apart from a discussion of whether the increase was apparent rather than real, no such alarm was registered in the United Kingdom though task forces were set up in New Zealand, the United States, and Canada to look into the matter further.

The importance of the issues raised by these developments is clear. Though asthma is ill defined, it affects 4–5% of the population at a conservative estimate. If the increase in mortality is real rather than apparent this is either because the disease is becoming more prevalent or more serious, or because the management of the disease is deteriorating in spite of more treatment and perhaps even because of more treatment. Any of these possibilities is of obvious importance.

Since the earlier studies, evidence has begun to accumulate in favour of the view that asthma prevalence, and particularly the prevalence of severe asthma, has been increasing. A further local study from South Wales documented not only an increase in symptoms among 12 year olds over the period from 1973 to 1988, but also showed an increase in the bronchial response to exercise, a more objective marker of asthma in children. Analysis of the records of the National Study of Health and Growth showed an increase in the prevalence of more severe respiratory symptoms over approximately the same period, suggesting strongly that these changes are not simply local effects arising from the study of small and selected communities with changing populations or abnormal local conditions. Similar evidence is also accumulating elsewhere. The findings from New Zealand have already been quoted. Surveys in Taipei in 1974 and 1985 showed an increase from 1.3% to 5.1% in the proportions of 7–15 year olds who had had at least three paroxysmal recurrent attacks of wheezing and dyspnoea in the previous 12 months. In Sweden the proportion of military conscripts found to have asthma increased from 1.9% in 1971 to 2.8% in 1981, and in the United States national surveys have also shown increases in the prevalence of self reported asthma.

If as seems increasingly likely we are experiencing a worldwide increase in asthma prevalence, it is right to ask what might have caused this. Evidence for a coincidental increase in eczema and possibly allergic rhinitis, suggests that the increase may reflect an increase in atopic disease as a whole. Several explanations have been put forward. The first possibility is that the exposure to allergens has changed. This hypothesis has been put forward in particular to explain local increases in asthma in the less developed parts of the world, notably in Papua New Guinea, where the importation of blankets and the consequent importation of dust mites has been blamed for the epidemic increase in asthma prevalence there, and in Kuwait, where the planting of Propolis was blamed for a major increase in asthma. There is however little evidence that allergen exposure as a whole has increased in recent years, and though there is little substantial evidence either way, it is even plausible that exposure to the common allergens may have decreased.

The second possibility is that people are increasingly exposed to unusual and unrecognised allergens derived from new agricultural or industrial processes. This hypothesis is hard to test, and against it is the observation that most asthma in young people is still clearly allergic and associated with positive skin prick tests to common allergens. At least in the younger age groups it seems unnecessary to suppose the presence of novel and unusual allergens. A third possibility is, therefore, that people are becoming more easily sensitised to allergens.

Three hypotheses have been put forward that might explain such an increase in sensitisation. A few studies have now shown increased prevalence of atopy in areas of relatively high atmospheric pollution, and it has been suggested that some forms of air pollution may be responsible for the increase in atopy. This hypothesis gains support from animal models showing an increase in sensitisation of animals exposed to some air pollutants. The epidemiological studies are however, weak, being mostly ecological studies with small numbers of ecological units or districts. Several studies of the effects of air pollution on lung health in children have also failed to show convincing effects of air pollution on the prevalence of asthma as opposed to atopy. In addition, air pollution as a whole has been greatly diminished. The hypothesis would have to be very much more specific to be convincing as an explanation of the increase in prevalence over the last 20–30 years.

A further hypothesis is that the increase is related to changes in maternal smoking. There is evidence that smoking during pregnancy raises cord IgE and the probability of atopic disease in infancy. This hypothesis could explain much of the increase in asthma mortality between generations, the cohorts experiencing the largest change in asthma mortality being those born around 1920, when women of childbearing years first began to smoke, and around 1945 when women began to smoke again.
increase their smoking substantially. On the other hand there is little evidence for a slowing down in the increase in prevalence of respiratory problems in generations of children born since the time, when the increase in smoking among women began to slow down in the mid-1970s.

Finally it has been suggested that changes in family formation may have altered the chances of atopic sensitisation. An atopic history, at least into early adulthood, was associated with fewer older siblings in the 1958 cohort of the National Child Development Study. The reduction in family size that has occurred over the last century might, therefore, have led to an increase in prevalence. However, the principal hypothesis explaining the finding, that a smaller number of older siblings might lead to a lower exposure to viruses in the early years of life, runs counter to most other speculation on the role of viruses in the pathogenesis of asthma, and is unsupported to date by other data.

Explaining historical trends, with all the difficulties inherent in obtaining accurate and suitable data from the past, is perhaps the least promising of all the avenues of approach to this problem. It is much easier to look to the contemporary collection of data to provide adequate clues to the aetiology of asthma. Here again, however, we are in need of hypotheses, and hypotheses require data on the distribution of the disease. The most striking variations in prevalence have been found in developing countries where there are apparently very large differences between urban and rural groups, some aspect of Westernisation or affluence being apparently important in the development of the condition. It is likely that there are also fairly marked even if much smaller variations in the prevalence of the disease in Western countries. However the lack of good standardised methods for assessing the prevalence of disease has delayed our understanding of how asthma is distributed, and has led to the probably erroneous view that the distribution is more homogeneous than it really is. Some risk factors that are unlikely to explain recent trends in asthma may nevertheless be important in explaining regional or national differences. Among these are several newly recognised nutritional risk factors, including a high sodium diet and a low selenium intake.

Over the next few years it can be expected that standardised surveys will be carried out over fairly extended areas. These, combined with incidence studies over the first part of life, are likely to tell us much more about the aetiology of the disease and, in retrospect, may lead to some understanding of the current and past trends.

Asthma mortality has been increasing since the mid-1970s in a large number of countries. In the United Kingdom at least, a good proportion of this increase can be attributed to a cohort effect, an observation that fits well with the explanation that this is due to the increasing prevalence. However, there is good reason to be cautious of accepting this as the only explanation of the increase. Two recent case-control studies have implicated the use of a particular β agonist in fatal cases of asthma, resurrecting earlier fears from the 1960s concerning the safety of standard asthma medications. In this country "excessive" use of methylxanthines has been implicated in some hospital deaths from asthma. These studies are not easy to interpret and have certainly been controversial, but they raise an important question. Age-period-cohort analysis of data from the United Kingdom suggests that there was an increase in mortality during the period 1981-5, independent of any cohort effect that is likely to be explained by increasing prevalence of severity, and this would be compatible with an increase in the prescription of unsafe medications, though it could equally be associated with a change in the way that deaths are certified. This also requires careful assessment.

Asthma presents a wide ranging problem to epidemiologists, clinicians and health service managers. Under one interpretation of the facts, a greatly increasing prevalence is leading to a substantial increase in mortality, hospital admissions, and use of medication. The origins of the increase remain unknown and important questions remain about the safety of some aspects of treatment. On the other hand the increase has been sudden and geographical variations are probably marked. This implies a strong environmental cause for the changes and an equally strong possibility that the effects could be reversed if the causes were known.

Asthma presents a major problem for patients, clinicians and administrators. If the current problems are to be tackled successfully greater priority has to be given to understanding and reversing the causes of the current increase. These are tasks for epidemiology and public health medicine. Though there is much that we still do not know about the distribution of asthma and though many of our current hypotheses may well turn out to be false trails, only preventive strategies can offer much hope in the long term. Without these the toll on the health of the people and on the health services, particularly in poorer countries, is likely to continue rising for the foreseeable future.