The role of environmental factors in asthma

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Although the everyday experience of asthmatic patients provides ample anecdotal evidence that environmental exposures provoke bronchospasm, it has proved more difficult to assess the impact of air quality on the timing of asthma attacks and the prevalence of asthma in populations.

Spectacular ‘asthma epidemic days’ are sometimes attributable to exceptional outdoor aero-allergen exposures. By comparison, effects of inorganic particles and gaseous pollutants in outdoor air on the incidence of asthma attacks are subtle and poorly quantified.

Environmental tobacco smoke and mould growth are the indoor factors most consistently associated with respiratory morbidity, but their roles in initiating allergic asthma remain uncertain. Evidence relating asthma risk to fumes from gas cooking, and to allergens from dust mites and household pets remains confused and controversial.

It is unlikely that trends in either outdoor or indoor air pollution have contributed substantially to the rise in prevalence of asthma and allergic disease in recent decades.

The role of environmental factors in relation to asthma and allergy has become increasingly topical through the 1990s. There has been widespread public concern that changing patterns of outdoor air pollution underlie the rising burden of asthma, but the professionals are not so sure. The indoor environment, in which people spend most of their time, has received less attention. This chapter summarises current evidence linking both outdoor and indoor air quality to temporal, spatial and individual variations in asthma and allergic sensitisation. The discussion of the indoor environment will focus on domestic exposures, and readers seeking details on occupational causes of asthma are referred to specialised reviews of this field.

Confusion often arises because the links between environment and asthma may operate at various stages in the development of this disease. It is, therefore, useful to distinguish clearly between two questions: (i) what initiates (or induces) the asthmatic state, a tendency to develop episodic airflow obstruction on exposure to a range of environmental and non-environmental stimuli? – part of this process may involve the development of atopic sensitisation to environmental allergens; and (ii) what triggers (or provokes) attacks of asthma among persons who already have the disease?
Table 1 Possible effects of environmental factors on asthma

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The environmental factors which may provoke attacks of bronchospasm include irritant gases, inorganic particles, allergens and infections. These are often identified by patients as triggers of symptoms and may be demonstrated more objectively by chamber experiments. There has been a tendency to implicate the same factors in the initiation of the asthmatic state, but the epidemiological evidence suggests that this simplistic approach to ‘the cause of asthma’ is likely to be misleading. It is more appropriate to seek explanations or ‘causes’ at different levels as summarised in Table 1.

**Short-term variations in the incidence of asthma attacks**

The frequency of asthma attacks varies considerably from day-to-day, as indicated by numbers of hospital admissions or accident and emergency attendances for asthma. In many countries, including Britain, there is a predictable seasonal variation\(^9\), with peak incidence during the early autumn, particularly in children\(^10\). These seasonal fluctuations are thought to be more closely related to the timing of school holidays\(^11\) and circulation of respiratory virus infections\(^12\), than to seasonal variations in air quality or aero-allergen exposure\(^9\).

Superimposed upon the seasonal pattern there are day-to-day variations in attack frequency which have attracted considerable interest in recent years as possible evidence of the adverse effects of outdoor air pollution levels. However, perhaps the most convincing evidence of an environmental influence on the incidence of asthma attacks arises from occasional ‘asthma epidemic days’. Most of the patients affected in these documented epidemics are atopic asthmatics, and unusual aero-allergen exposures have been implicated in several instances.

**Asthma epidemic days**

There have been relatively few reports of noticeable ‘epidemics’ of asthma attacks lasting for 1–2 days, although retrospective analysis of
hospital activity has revealed several epidemics which were not apparent at the time\textsuperscript{13}. A striking example occurred on the night of 24–25 June 1994 when a thunderstorm over southern and eastern England was followed by a 10-fold increase in acute asthma attacks presenting to accident and emergency departments\textsuperscript{14}. Levels of conventionally measured air pollutants were not unusual at the time\textsuperscript{15}. There were earlier reports of thunderstorm-related asthma outbreaks from Melbourne\textsuperscript{16,17} and Birmingham, UK\textsuperscript{18}, and a systematic time-series analysis suggested that a typical English thunderstorm is associated with a 25\% relative increase in asthma admissions on the following day\textsuperscript{19}. This excess is greater following periods of high grass pollen counts, and many of the affected patients were sensitised to grass pollen\textsuperscript{14}. Osmotic rupture or physical disruption of pollen grains during the thunderstorm releases submicroscopic starch granules which carry allergens such as \textit{Lol pIX} and are of a size which could be inhaled into the smaller airways\textsuperscript{17,20}. Thus, these thunderstorm-related epidemics were probably due to ‘aero-allergen pollution’.

Only one-third of asthma epidemic days in England during 1987–1994 were related to thunderstorms\textsuperscript{13}, and the cause of the remainder remains elusive. Nevertheless, the concept of mass outbreaks attributable to unusual aero-allergen exposures is supported by observations of repeated outbreaks of asthma recorded in Barcelona during the 1980s\textsuperscript{21}. Air pollution levels and airborne pollen and spore counts on the epidemic days were below average for the city but subsequent investigation implicated the unloading of soybeans in the harbour as the cause\textsuperscript{22}. Patients affected on epidemic days were found to have circulating soybean-specific IgE\textsuperscript{23}, and no further epidemics have occurred since modifications were made to the procedures for unloading soybeans.

Asthma epidemic days were a recognised phenomenon in New Orleans during the 1950s and 1960s\textsuperscript{24}. Air pollution, possibly related to burning of rubbish dumps, was suspected initially, but the epidemics continued despite cessation of this seasonal activity\textsuperscript{24}. The patients involved in epidemics were predominantly atopic asthmatics, with positive skin prick tests to many common aero-allergens\textsuperscript{25}. Detailed investigations of airborne pollens and fungal spores failed to identify a specific allergen that might be responsible, but it was noted that autumn epidemic days tended to be associated with high total spore and pollen counts\textsuperscript{25}. In this regard, it may be relevant that certain fungal spores, such as \textit{Didymella exitialis} and \textit{Sporobolomyces} are released during summer thunderstorms\textsuperscript{18}. Some patients with late summer asthma may be allergic to \textit{Didymella} spores\textsuperscript{26}, but in general, the role of outdoor fungal allergens as triggers of asthma attacks remains uncertain.
Daily fluctuations in air pollution and asthma

Although inorganic air pollution has not been implicated as a cause of exceptional asthma epidemic days, many studies have examined daily counts of asthma admissions or emergency room visits in relation to short-term fluctuations in measured pollutants. These time-series studies have used increasingly sophisticated statistical methods which can detect subtle daily variations in asthma attack incidence which are far too small to qualify as an 'epidemic'.

The global literature was comprehensively reviewed by the author in 1995\textsuperscript{27}, and subsequently by the author and colleagues in 1998\textsuperscript{28}. Only a minority of the studies published evidence in a form which permitted an estimation of the dose-response relationship to individual pollutants. Quantitative estimates are rarely quoted when no significant correlation was observed between asthma incidence and pollutant levels. Thus, the results of overviews and meta-analyses tend to preferentially include 'positive' associations and may overestimate the dose-response gradient. With this cautionary proviso, the following conclusions may be drawn.

Associations between single pollutants and a health outcome such as asthma are often inconsistent. In our 1998 review of 16 'methodologically sound' studies\textsuperscript{28}, ozone, sulphur dioxide and particles were identified as significant correlates of daily asthma attack rates in no more than half of the reports, and one-quarter or less found a significant effect of particulate or nitrogen dioxide levels. Thus, effects of single pollutants should not be interpreted narrowly, but rather as indicative of complex pollutant mixtures, which may vary by location.

For instance, there is fairly consistent evidence that the type of 'summer haze' affecting southern Canada and the eastern US is associated with a modest short-term increase in the incidence of asthma attacks\textsuperscript{29-32}. The causal agent, however, remains in doubt. Ozone is a possible candidate\textsuperscript{33}, but on the western coast of North America, ozone levels were either uncorrelated with asthma incidence (as in Vancouver\textsuperscript{34} and Seattle\textsuperscript{35}) or inversely correlated with daily asthma attack rates (as in Los Angeles\textsuperscript{36}). In London, ozone levels were positively associated with asthma admissions in summer (possibly indicative of the 'summer haze' mixture) but inversely associated in winter (possibly due to scavenging of ozone by nitrogen oxides in vehicle exhausts)\textsuperscript{28}.

Acid aerosol was considered as a possible agent in southern Ontario, but direct measurements of airborne acid in New York\textsuperscript{31} were no more closely correlated with asthma admissions than were ozone or sulphate levels. Airborne sulphates form a major part of the fine particulate fraction in Northeast US and it has been suggested that the effects of summer haze may be mainly attributable to these respirable particles\textsuperscript{37}. However, the only summer haze study to measure fine particles directly\textsuperscript{30} found no relationship between PM\textsubscript{10} levels and the timing of asthma attacks.
In general, the evidence is less consistent with regard to winter pollutants. This may reflect different emission sources, pollutant 'cocktails' and particle composition in different areas, or a correlation of outdoor conditions with indoor pollutant exposure which varies according to the fuel used for domestic heating. Where winter pollutant effects have been evaluated for different age groups, as in London and Vancouver, the more significant findings are for the older age groups. This may be partly due to diagnostic confusion with other forms of chronic obstructive airways disease.

In December 1991, an episode of unusually high particulate and nitrogen dioxide pollution, mainly from vehicular sources, occurred in London. During the episode week, there was a small (3%) and statistically non-significant increase in asthma admissions in the capital, by comparison with previous years and the surrounding regions. Other respiratory outcomes, notably chronic bronchitis and emphysema among the elderly, were affected to a greater extent than asthma admissions.

Temporary closure of a steel mill in Utah Valley during 1986–1987 offered an unusual opportunity to study the effects of changing levels of 'winter pollutants' on respiratory health at the community level. Although there was no statistically significant reduction in asthma admission rates for all age groups, asthma and bronchitis admissions (combined) among preschool children were halved during the year of the mill closure, suggesting a reduction in 'wheezy bronchitis' rather than 'allergic asthma'.

Outdoor aero-allergen levels and asthma

Fewer studies have applied the time-series approach to study the relationship between asthma attacks and aero-allergen levels on a daily basis. However, apart from the special circumstances associated with thunderstorms, it seems that asthma admissions are not associated with daily variations in airborne counts of grass pollen, or tree pollens. Thus, pollen levels are unlikely to be major confounders in studies of spring and summer air pollutants.

Allergic sensitisation to mould extracts occurs in a minority of asthmatic patients of all ages, although usually in association with other aero-allergens such as pollen, house dust mites and animal danders. Sensitisation to the outdoor mould Alternaria alternata has been implicated as a major risk factor for fatal asthma attacks in Chicago, USA. This may simply reflect a common association with severe asthma, but in Chicago, asthma deaths at ages 5–34 years occur about twice as commonly on days with a high total mould spore count (>1000 spores/m³) as on days with lower spore counts. Panel studies of
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asthmatic patients report inconsistent findings relating symptom severity to mould spore concentrations in outdoor air\(^{45-49}\), but there has been a suggestion that fungal spores may be implicated in some asthma epidemic days\(^{18,25}\). Chamber experiments have suggested a synergistic interaction between prior exposure to a variety of gaseous air pollutants and bronchial responsiveness to allergen challenges\(^{50-53}\). The interaction between ozone and pollen exposure is perhaps the most relevant to the outdoor environment, but in the only time-series study to specifically address this combination, there was no evidence of a synergistic effect in relation to asthma admissions in the London area\(^{28}\).

Outdoor air pollution and the prevalence of asthma

Whereas daily time-series offer some insights into the acute effects of outdoor air quality on asthma attacks, the more fundamental concern is whether long-term exposure to air pollution influences the prevalence of asthma\(^{4-5}\). This has been addressed by area-level studies comparing asthma prevalence in whole towns or cities with differing levels or mixtures of pollution, and by individual or household studies, relating asthma risk to proximity to roads or to other indices of traffic exposure.

Comparisons of areas of high and low pollution

The pollutants which have been most often measured in geographical comparisons relating the prevalence of wheezing illness to air pollution are sulphur dioxide and airborne particles. The evidence relating each of these to asthma and wheezing at the area level is inconsistent\(^{54}\). Comparisons within eastern Europe (often at high levels of smoke and SO\(_2\)) generally support an association with life-time prevalence of wheezing in children, whereas similar studies in elsewhere (often at lower levels of SO\(_2\) and particulate pollution) present a mixed picture\(^{55}\).

Fewer studies have addressed possible hazards due to ‘newer’ pollutants (nitrogen oxides and ozone). In general, NO\(_2\) levels are correlated with SO\(_2\) and particulate concentrations in area comparisons and it is difficult to distinguish their independent effects. Two equally competent studies of wheeze among non-smoking adults yield conflicting findings in relation to photochemical pollutants. In the Swiss SAPALDIA study\(^{56}\), no associations were found between the prevalence of wheezing or current asthma and the annual mean concentrations of particles, ozone or nitrogen dioxide at 8 study centres. However, cough, phlegm and breathlessness were positively associated with particulate pollution among never smokers. Among
Seventh Day Adventists in California\textsuperscript{57}, the incidence of adult-onset asthma diagnosed by physician was significantly and positively associated with cumulative outdoor exposure to particulate air pollution. Unfortunately, ambient particle concentrations were highly correlated with ozone levels so it was not possible to discriminate with certainty the independent effects of each pollutant\textsuperscript{57}.

An unusual opportunity to study the effects of air pollution on health arose from the re-unification of Germany, but comparisons of symptoms related to asthma are complicated by a large difference in prevalence of allergy between East and West Germany\textsuperscript{58}. A consistent finding was a lower prevalence of positive skin prick responses in the polluted areas of East Germany than in West German cities\textsuperscript{59,60}. There appeared to be an excess of irritant symptoms (including 'bronchitis') in the most heavily polluted areas of the former GDR, balanced by a reduced prevalence of 'allergic asthma'\textsuperscript{61}.

Small area variations and exposure to traffic

Although the differences in prevalence of asthma and allergy between East and West Germany could not be explained on the basis of hazards from 'old-fashioned' smoke and sulphur dioxide pollution, exposure to 'modern' pollutants derived from vehicle exhaust was higher in West Germany. Concern that traffic exposure might increase the risk of asthma was raised by several population surveys from Germany and The Netherlands which found a higher prevalence of asthma and allergic disease among children living close to busy roads or heavy truck traffic\textsuperscript{62-65}. The variations in prevalence across extremes of traffic exposure in these studies were generally between 25–50\% in relative terms, but some of this may reflect reporting bias, particularly in studies which rely on self-reporting of traffic exposure.

British studies are based on larger numbers of asthma cases but yield less consistent findings. Within London, no association was found between proximity to major roads and asthma diagnosis, treatment or hospital admissions\textsuperscript{66,67}. However, in Birmingham, young children were more likely to be admitted with asthma from heavily trafficked areas\textsuperscript{68}. A study from Stockholm also found an association of modelled traffic exposure with 'wheezing bronchitis' in infancy\textsuperscript{69}, but it is likely that most of the cases in these studies were 'viral associated wheezing' rather than 'allergic asthma'.

Not only is the published evidence inconsistent, but there is also considerable uncertainty whether any 'positive' associations could be due to air pollution from vehicle emissions\textsuperscript{70}. Place of residence is an imperfect indicator of personal activity patterns, and although mean
nitrogen oxide concentrations decrease with distance from the kerbside, the decline beyond 20 m is small. No correlation between traffic density and ambient NO\textsubscript{2} concentrations was found within Münch, although carbon monoxide, benzene and toluene (vehicle-related pollutants which are not implicated in asthma) were more concentrated in areas of higher traffic flow. Higher traffic density was inversely correlated with levels of ozone, which is formed at some distance from emission sources, and 'scavenged' in city centres by NO from vehicles. Personal exposure to NO\textsubscript{2} and particles is influenced to a small degree by distance from major roads, but to a much larger extent by indoor sources (cooking and heating fuels, and environmental tobacco smoke). It is, therefore, appropriate to turn our attention now to the influence of indoor air quality on asthma.

**Indoor air quality and the prevalence and severity of asthma**

**Gas cooking and indoor nitrogen dioxide**

Nitrogen dioxide has been widely used in studies of outdoor air quality as a marker of pollution related to vehicle emissions. However, the most important source of personal NO\textsubscript{2} exposure is unvented gas appliances, particularly gas cookers. Comparison of disease prevalence between homes with and without gas cookers, therefore, offers a simple method of evaluating whether long-term exposure to NO\textsubscript{2} specifically (but not other motor vehicle emissions) induces the asthmatic state. However, because indoor NO\textsubscript{2} levels are influenced by house design and ventilation, use of a gas cooker is arguably a better marker of peak NO\textsubscript{2} exposure than average levels.

An early meta-analysis of epidemiological studies suggested a pooled relative risk of 1.2 (relative excess of 20%) for a range of respiratory illnesses among children exposed to gas cooking in the home. Many of these studies did not specifically report results for asthma or wheezing. More recent large studies from Europe and North America, among both children and adults, have reported conflicting evidence: some showing up to a 2-fold increase in asthma prevalence in gas cooking homes, others finding no differences. International differences in the strength and direction of the association are apparent even when the survey techniques were similar, and it remains unresolved whether the inconsistencies can be explained by variations in cooker design, use or other factors.

Two studies of adults find no association between gas cooking and allergic sensitisation and atopic asthmatics do not appear to have more severe symptoms or an adverse prognosis if exposed to gas...
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cooking. A causal relationship between nitrogen dioxide exposure and asthma severity has not been disproved, but the balance of evidence suggest it is unlikely.

Environmental tobacco smoke

Many asthmatic patients identify other people's tobacco smoke as an environmental trigger for their symptoms, but there is a surprisingly sparse literature documenting effects of passive smoking on either the prevalence or severity of asthma in adults. Perhaps the most convincing studies are those of lifelong non-smoking Seventh Day Adventists in California, showing a 50% relative increase in the incidence of doctor-diagnosed asthma over 10 years among adults exposed to environmental tobacco smoke.

The literature relating parental smoking to asthma, wheezing and allergic disease in children is much more extensive and has recently been reviewed both qualitatively and quantitatively by the author and colleagues. The results are complex and require careful interpretation, distinguishing between the various wheezing syndromes which may present in childhood.

The most consistent results relate to lower respiratory illnesses in infancy. Smoking by either parent, and particularly by the mother, increases the risk of both wheezing and non-wheezing chest illnesses up to 2 years of age by about 50% (relative risk 1.5), independent of other factors. The medium-term prognosis of these early wheezing episodes is less favourable in smoking households, but as the child grows through adolescence, remission is more likely if the mother smokes.

At school ages there is a higher prevalence of asthmatic symptoms (relative risk about 1.3) among children whose parents smoke, and among children with asthma, markers of disease severity are generally worse in those exposed to smoking in the home. In contrast, allergic sensitisation, at least as measured by skin prick tests, is no more common in the offspring of smoking parents, and the association of wheeze with passive smoke exposure appears stronger among non-atopic children.

One interpretation is that passive smoking is related more closely to transient 'wheezy bronchitis' than to 'allergic asthma'. Its association with symptom prevalence and asthma severity in school age children thus reflects a role predominantly as a trigger of symptomatic episodes (alone, or in combination with viral infection), rather than as a factor initiating the development of allergic sensitisation or atopic asthma.

Dampness and indoor moulds

An association of asthma with 'damp houses and fenny countries' was observed three centuries ago by Sir John Floyer. Studies of the
relationship between home dampness or domestic mould growth and asthma have generally used questionnaire reports of wheeze and dampness/mould to characterise disease and exposure, and most have concentrated on children in order to discount the confounding effects of active smoking, occupation, and selection into different types of housing. The results are fairly consistent, finding that wheeze is about twice as likely to occur in homes reported to be mouldy.

Fewer studies have attempted objective or independent assessment of housing conditions or allergic symptomatology. Those that have been reported generally show little relationship with mould, raising the possibility that at least some of the association in questionnaire data is due to reports of symptoms being increased by awareness of mould in the home (or vice versa). However, one recent study reported an association between asthma severity and independent assessments by surveyor of dampness and mould growth in the patients’ homes.

A large number of potentially allergenic mould species have been isolated from homes and indoor humidity is an important determinant of fungal growth. However, in most cases, the same species of fungi occur in the homes of both affected and unaffected people, often in the same proportions. Allergic sensitisation to indoor moulds is uncommon, even among asthmatic patients. It is, therefore, difficult to attribute the difference in the prevalence of symptoms between mouldy and non-mouldy homes to fungal allergy. A more likely interpretation is that variations in the levels of other airborne allergens, especially from house dust mites, underlie the relationship between damp, mouldy housing and asthma. Cockroach allergen may be a contributory factor in some areas, particularly in the US.

House dust mite allergens

The role of house dust mites in the induction and provocation of asthma has been the subject of a number of international workshops and systematic reviews, but the importance of mite allergen exposure in determining the prevalence and severity of asthma remains controversial. The evidence needs to be considered at various levels.

There is consistent evidence for a correlation between level of exposure and degree of sensitisation to mite allergen in both children and adults. It is now considered unlikely that there is a threshold level below which sensitisation does not occur, as had been suggested by a WHO workshop in 1987. However, the total prevalence of allergic sensitisation is not greatly influenced by mite allergen levels, and by no means all people with demonstrable mite sensitisation have clinical allergic disease. Thus studies of sensitisation in relation to allergen exposure provide only indirect evidence of a health risk.
International variations in the prevalence of asthma do not bear any obvious relationship to the local mite prevalence. Within Australia, widely varying climatic conditions give rise to large regional variations in mite allergen levels, but the prevalence of childhood asthma is similar throughout the country. Mite allergen levels are generally similar in the homes of asthmatic and non-asthmatic subjects, although some studies have reported an association between domestic mite allergen exposure and severity of asthma. A concern in these cross-sectional and case-control studies is that causal associations may have been diluted because of allergen avoidance measures by families of allergic or asthmatic patients. This bias can be reduced, but not totally avoided, by longitudinal studies relating allergen exposure in infancy to subsequent incidence of asthma. So far, few long-term follow-up studies have been published, so the relative importance of perinatal and later exposure remains uncertain.

Intervention studies may offer a more direct evaluation of the importance of current house dust mite exposure. The evidence is reviewed in greater detail elsewhere in this issue. Unfortunately, attempts at mite allergen avoidance have generally been disappointing in terms of their efficacy (reduction in allergen exposure). It remains unresolved whether the more efficacious interventions are clinically effective (in terms of improving asthma symptoms) or of prophylactic value (for primary prevention of asthma).

**Household pets**

Studies of mite allergen exposure in large population samples are expensive and time-consuming, but a simpler model for the hypothesis that allergen exposure induces asthma would relate to pet danders. Presence of a cat or dog in the home is a powerful determinant of exposure to the relevant allergens (Fel d1 and Can f1, respectively), and this can be demonstrated in both dust samples and air samples. However, pet avoidance by allergic families is a potential bias which needs to be addressed in observational studies.

Several large studies have found either no association between asthma and pet ownership, or an inverse relationship, particularly with early dog ownership. On the other hand, inconsistent results emerged from two case-control studies of asthma among teenage children which adjusted for pet avoidance, one showing an association of early pet ownership with more severe asthmatic symptoms, the other finding less asthma and, paradoxically, less cat allergy among children exposed to cats in infancy.

Recent observations of a reduced prevalence of allergic diseases among children of farmers raise the possibility that animal exposure may
reduce the tendency to allergic sensitisation through other mechanisms, perhaps related to infection\textsuperscript{121}. Thus, whereas animal dander may exacerbate asthmatic symptoms among sensitised individuals\textsuperscript{122}, pet ownership in a more general sense may have relatively little effect on the prevalence of pet allergy or associated allergic asthma.

\textbf{Bedding}

For many years, asthmatic patients have been advised to avoid feather bedding on the premise that allergen exposure (from both house dust mites and feathers themselves) would thereby be reduced. More recently, this assumption has been challenged by both exposure measurements and epidemiological studies.

Contrary to expectation, mite allergen levels in dust samples from feather pillows are 5–10 times lower than in dust from synthetic pillows\textsuperscript{123–125}, possibly due to the more tightly woven covers required for feather pillows acting as a barrier to allergen release. Two case-control studies of wheezy children in south London, from 1978 and 1991, found that use of a feather pillow (rather than a synthetic one) was associated with a reduced risk of asthmatic symptoms, even after allowance for deliberate changes to the child’s bedding as a result of asthma or allergy\textsuperscript{126}. Two further studies of British children confirm a strong inverse association of feather pillow use with both mild and severe asthma\textsuperscript{118,127}. Taken together, these four studies suggest that asthma is about twice as common in children using synthetic pillows as those sleeping on feather ones, with a dose-response gradient in relation to symptom severity.

\textbf{Conclusions}

\textit{A look to the past}

The prevalence of asthma and other allergic diseases has been rising in many Westernised countries over the past few decades, at a time when exposure to most measured air pollutants has declined\textsuperscript{54}. Although the epidemiological evidence does not exclude a relatively weak relationship between outdoor air pollution and asthma prevalence, a strong association is unlikely. It is also apparent that indoor air quality is often a more important determinant of personal pollution exposure. For these reasons, it is unlikely that changes in the levels or composition of outdoor air pollution have been a major factor underlying the trends in asthma prevalence in most countries, including Britain\textsuperscript{1–4}.
Few studies have compared measures of indoor air quality over a similar period of time. The sparse evidence in relation to mite allergen levels suggests little change between 1979 and 1989, at least in southern England. Comparison of two studies in south London in 1978 and 1991 found that changes in pet ownership, parental smoking habits, domestic central heating and cooking fuel could not explain the rise in prevalence of wheezing among 8-year-old children over this period. Indeed, the increase in use of non-feather pillows from 44% to 67% over the 13-year period was the only indoor environmental factor studied which potentially explained any substantial part of the increase in prevalence of wheezing from 1978 to 1991.

**A view to the future**

Asthma is a disease characterised by variability of airflow obstruction and episodic occurrence of symptoms in individual patients ('attacks'). It is also a disease which varies in prevalence and severity within populations, between countries, and over time. The environment, defined narrowly as in this chapter, in terms of chemical and biological air pollutants, is much more clearly related to the provocation of asthma attacks and to asthma severity than it is to the induction of the asthmatic state and the prevalence of asthma.

Variations in indoor or outdoor air quality and allergen exposure account for neither the large international variations in asthma prevalence, nor the long-term time trends. In seeking to explain these, and ultimately to prevent asthma, a much broader definition of 'environment' is required. Areas which are currently topical include the intra-uterine environment and the microbial environment, including commensal bowel flora. These, in turn, may interface with components of life-style or medical care. The need for a broad perspective in asthma research during the 21st century is as important today as when it was first suggested 15 years ago.

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