

Review article

Is the increase in asthma prevalence linked to increase in allergen load?

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All epidemiologic studies of childhood asthma have shown an increasing prevalence, even in countries thought to have little asthma. Fig. 1 shows published studies that have administered the same questionnaires to study populations of children and adolescents at an interval of 5 or more years. In the Finnish study, the measurement was of current asthma, while the other studies all measured diagnosed asthma. Table 1 summarizes data from a number of other studies in which the prevalence of asthma, wheeze, airway hyperresponsiveness (AHR), atopy, and hay fever has been measured. The data about adults are fewer and more difficult to interpret (1, 2), but there is some indication that the prevalence of asthma is increasing in young adults in affluent countries.

The cause of the increase in childhood asthma is probably multifactorial. Firstly, there may have been diagnostic transfer and changes in the use of terms such as "wheezy bronchitis". In Australia, however, the increase in prevalence has occurred steadily over 20 years and cannot be entirely explained by changes in diagnostic labelling practices because AHR has also increased, as shown in Table 1. Furthermore, the prevalence of atopy, as measured by skin prick tests, has not increased to any great degree in Australia over the last 10 years. About 88% of children with current asthma were allergic in both 1982 and 1992, and a similar proportion of asthmatic children

Increases in Diagnosed Asthma in Children / Adolescents
(Same population and same method within each country)

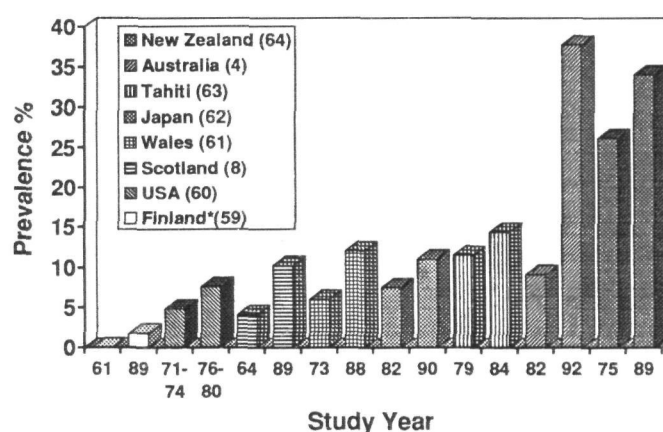


Fig. 1. Increased prevalence of diagnosed asthma in children/adolescents of eight countries using similar questionnaires over 5 or more years. * Army recruits.

in New Zealand are allergic (3). In Australasia, it appears that the increase in asthma is real and has occurred within the allergic population. Since the increase appears to be a worldwide phenomenon, a ubiquitous environmental factor (or several factors) is probably responsible. These factors are discussed.

There are hardly any studies of allergen levels in the literature which have documented changes in load. However, it is generally agreed that house-dust mites have increased in number. Green et al. measured the number of mites in a small sample of

Table 1. Wheeze, airway hyperresponsiveness, and atopy in populations of children

Country	Study (year)	Number	Age (years)	Current asthma	Diagnosed asthma	Wheeze ever	AHR	Atopy (SPT)	Hay fever	References
Australia	82	718	8–11	4.5	9.1	10.5	9.1 (H)	27.7	20.5	(4)
	92	873	8–11	12.0	37.7	27.6	19.8 (H)	29.3	34.0	(4)
New Zealand	81	813	9	11.1	18	–	22 (M)	–	–	(5)
	88	1084	7–10	9.1	14.2	27.2	20 (H)	–	27.5	(6)
	91	873	12	11.1	16.8	26.6	12.2 (E)	–	21.0	(7)
Wales	91	965	12	9.1	12.0	22.3	7.7 (E)	–	16.0	(7)
Scotland	89	3403	8–13	–	10.2	–	–	–	11.9	(8)
Germany	90	5697	9–11	4.2	7.9	–	13.3 (C)	–	–	(9)
Denmark	87	527	7–16	5.3	–	–	16 (H)	21	–	(10)
Sweden	89	1276	12	2.8	4.0	21.7	2.1 (E)	–	14.0	(11)
Indonesia	81	317	7–15	3.7	3.8	14.5	2.2 (H)	36	–	(12)
China	88	3067	11–17	1.9	2.4	6.3	4.1 (H)	30	–	(13)
Japan	92	10 137	6–12	2.9	5.0	–	–	–	12.9	(14)
Papua New Guinea	85	257	6–20	0	0	1.7	1.0 (H)	17	–	(15)

Current asthma=AHR+wheeze in last 12 months; diagnosed asthma=asthma ever diagnosed; AHR=airway hyperresponsiveness; H=histamine; M=methacholine; E=exercise; C=cold air hyperventilation; SPT=skin prick test.

household dust from homes in two Australian towns (one with a humid, coastal climate, and one with a drier, inland climate) on two occasions, 10 years apart, and found a large change (16). House-dust mite infestation has also been shown to have increased in Stockholm (17–19). It is known that in climates in which house-dust mites exist, dust-mite allergen exposure is the most important risk factor for the development of asthma, and the literature linking mites causally to the disease has been reviewed (20). Since housing design has changed over recent years to favour mite proliferation, it is easy to attribute the increase in asthma to a supposed increase in allergen load. However, this extrapolation may be premature. The way in which mite sensitivity causes asthma has not been determined because the source of allergen in homes is not known, and because the way in which mite allergen reaches the airways is unknown. While a change in mite allergen load is likely, it is highly probable that some other factor (or factors) has changed over the last 20 years and is responsible for the changed prevalence of asthma – for example, diet or pollution, or both.

There is accumulating evidence that house-dust mite allergen in the environment increases the severity of asthma (21). A dose-response relationship has been demonstrated between the level of allergen and the severity of asthma (22, 23). It is not clear whether the allergenicity of mites lies in their perennial presence or in their ability to release particularly potent allergens. Hospitalizing patients in a house-dust-mite-free environment resulted in an improvement in airway responsiveness and a reduction in the medication requirement of a few adults (24). Moreover, removal of house-dust-mite-sensitized children to a low-allergen environment decreased airway hyperresponsiveness and abated asthma (25, 26). How-

ever, the low-allergen environment must be maintained because these children did not maintain their improvement on returning home. Reducing domestic allergen levels also reduces the severity of asthma (27). In terms of prevention, avoidance of house-dust mite allergen at an early age has been shown to reduce the incidence of allergic illness in infants and in young children (28, 29).

It can be argued that there is no reason to believe that the increase in the prevalence of asthma is linked to increases in allergen load, because domestic allergen levels have always been high in humid climates. It is just as likely that some factor which was protecting children from asthma has been lost, making mite-related asthma more common. In rural Australia and parts of the USA, mite-allergen exposure is not the dominant risk factor for the development of asthma because of the dry climate. In these areas, other allergens, such as those of *Alternaria* and ryegrass pollen, are risk factors, and in many areas cat allergen is a problem. There is no evidence of an increase in levels of these allergens in recent years.

Table 1 also shows the prevalence of allergic rhinitis, as documented by questionnaire, in studies in Australia, New Zealand, Scotland, and Wales. There is evidence that it also is increasing. It is assumed that most children whose parents say they have “hay fever” are, in fact, allergic. In Australia (30) and New Zealand (3), the percentage of children with nasal symptoms who were allergic was similar to that found for children with asthma (between 85 and 90%).

There are a number of suggestions in the literature and editorials that the prevalence of asthma, rhinitis, and “allergies” has increased in recent years (31–34), but there are few data in the literature to support this commonly held belief. Our own studies

from Australia suggest that the prevalence of allergy is not increasing and remains at about 40–50% of the population (depending on the age group and the size of the wheal considered positive) (4, 30). However, within the allergic half of the population, an increasing number of subjects have developed symptoms of either asthma or hay fever. It has been suggested that the acquisition of allergy is occurring at a younger age (35), but there are no data about changes in the rate of acquisition of allergy in any population.

The reasons for the increase in symptoms of asthma and hay fever are unknown. Our data, obtained from a number of different climatic areas, suggest that there are three important groups of allergens that are responsible for asthma in children: those of house-dust mites, the mould *Alternaria*, and cats. Large sections of different populations are sensitive to pollens (grasses, weeds, and trees), but these allergens appear to be associated with hay fever and are less important as risk factors for persistent asthma (36, 37). Part of the increase in symptoms of asthma may result from exposure to higher levels of domestic allergen, but it is likely that levels of cat and house-dust-mite allergen have been high in Australian and New Zealand homes for many years and well above disease-inducing levels. Therefore, it seems most unlikely that the increase in prevalence is the result of air pollution since the change has occurred in rural areas and the prevalence of passive smoking has decreased. It thus seems more likely that the observed increases are a result of other life-style changes, including diet (38).

Although there are insufficient data to link the increase in asthma with changes in allergen load, this does not mean that allergen loads can be ignored when considering appropriate management and prevention strategies. It does seem that decreasing the environmental allergen load (especially house-dust mite and cat) can lead to an improvement in the control of the disease. Because exposure to allergens with sensitization in early life is more likely to lead to the later development of illness than sensitization which occurs in late childhood (39, 40), and because children who develop severe sensitization are most likely to have a clinically important illness (4), the reduction of allergen exposure in infancy has the potential to delay sensitization and thus to reduce the likelihood of severe asthma developing in childhood.

Progress in understanding ways to reduce indoor allergens makes possible large public health benefits which are likely to reduce the incidence of early sensitization to common allergens, and this, in turn, is likely to reduce the incidence of asthma. It is now clear that primary prevention will involve a number of strategies, apart from allergen avoidance, includ-

ing reduction of indoor air pollutants and probably changes in diet.

There is some evidence that allergen load can make asthma worse in the individual subject, even if it does not cause an increase in prevalence. In Barcelona, many people developed sensitization to soya beans following intermittent exposure to soya bean dust during the unloading of ships in the harbour. Under certain atmospheric conditions, which occurred only infrequently, the city was exposed to high levels of soya bean dust, resulting in a sharp increase in acute exacerbations of asthma symptoms and the resultant hospitalization of many subjects (41), especially those who had become sensitized (42). However, the installation of efficient silo filters has prevented further exposure and eliminated the episodes of soya bean-induced asthma attacks (43).

Allergens from moulds can be potent inducers of asthma and there is some evidence that pollens can also induce the disease. These are not easily avoided (44–46). There is no reliable information about the level of spores and pollens in the air; however, it is known that they vary with the seasons and from year to year.

Circumstantial evidence suggests that changes in diet may be partly responsible for the recent increases in the prevalence of asthma. Changes in ambient levels of allergens and environmental tobacco smoke are more likely to occur locally and to result in local changes in prevalence. However, asthma has increased in all countries where it has been monitored. Diet has also changed in a fairly ubiquitous manner, particularly in affluent countries, during the last decade. There is a higher consumption of processed foods, omega-6 oils, and salt, and a lower consumption of fresh foods. Our own studies have shown that children who eat fish more than once a week are at reduced risk of having AHR and asthma (4, 47).

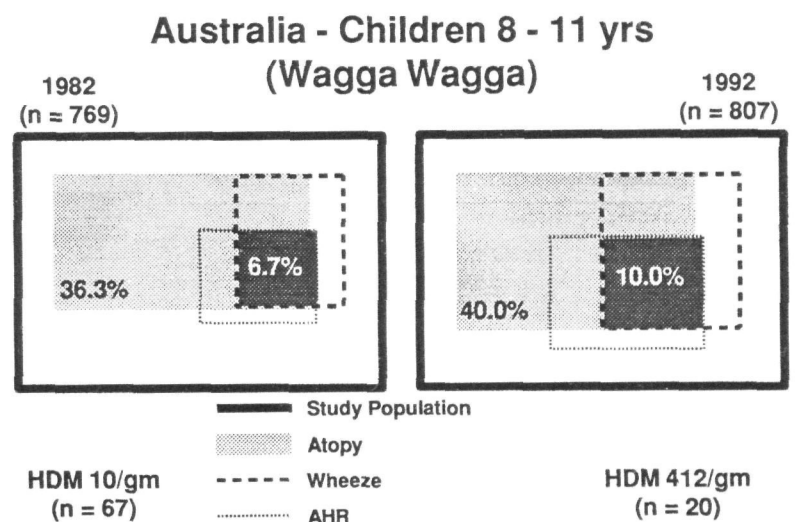


Fig. 2. Prevalence of atopy, recent wheeze, and AHR in Wagga Wagga, Australia, over 10-year interval. Current asthma is presented as area of overlap between wheeze and AHR. HDM = house-dust-mite density in vacuum cleaner dust.

This is a plausible concept because oily fish provides an important source of omega-3 oils, which play an anti-inflammatory role in metabolism (48). Children who eat oily fish more than once a week have one-third the prevalence of asthma (49), suggesting that this dietary intervention alone could reduce the prevalence of childhood asthma.

It has also been suggested that an increased salt intake is associated with increased mortality from asthma and with airway hyperresponsiveness in men (50, 51), and that a diet which is low in antioxidants may promote the development of airway disease (38). Infants who are breast-fed for a prolonged period have less allergic illness in early childhood (52) and are protected from asthma (53, 54), presumably because breast milk protects against infections and prevents early exposure to the foreign proteins present in cow's milk. Changes in the prevalence of breast-feeding worldwide have not been documented, but the duration of breast-feeding may have decreased in recent years.

It may be that some populations are protected from asthma and that the recent increase has resulted, at least in part, from loss of protective factors. Migrants who go from villages to towns have more asthma (55, 56); asthma is less prevalent in crowded conditions (57); and asthma is very rare in populations with traditional, nonaffluent lifestyles (15, 58).

In summary, the evidence that asthma is increasing in children throughout the world is strong, although much of the increase appears to be in mild disease. In Australia, the increase has occurred in atopic children while the prevalence of atopy has hardly changed; therefore, more atopic children are developing symptoms. It is difficult to determine the amount that can be attributed to increasing allergen load because there are few measurements of domestic allergens and it is likely that in most humid countries mite levels have been high enough to induce asthma for many years. It seems more likely that the ubiquitous increase results from a combination of factors that interact with increasing allergen loads. These factors include change in diet (less omega-3 fatty acids and antioxidants, and more "fast" foods), as well as the loss of protective factors which seem to be present in living conditions which are crowded or where washing is infrequent.

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