Preface

This report, prepared for the PHC, is an attempt to chart a way forward in the development of strategies for the primary prevention of asthma.

All over the world asthma has become of increasing importance. Considerable advances have been made in the medical management of people with a tendency to asthma, both in the treatment of acute attacks, and, where necessary, maintaining control of the disease process.

Airborne allergens such as pollens have long been recognised as triggers of some people's asthma attacks. With the development and introduction of preventive agents such as sodium cromoglycate and inhaled steroids, medication became available that was capable of reducing or preventing acute attacks of asthma. However, to be effective the medication must be taken regularly and continuously as it suppresses rather than reverses the disease process.

While it is not practicable to maintain the environment free of all the allergens that may trigger asthma, the discovery that many asthmatic people may be sensitive to house dust mites raises the possibility of maintaining an indoor environment as free as possible of at least one important allergen. This would benefit some asthmatics, in whom it may reduce the need for medication. This report describes the possible strategies, and current research designed to assess the practicality and effectiveness of indoor humidity control in New Zealand.

Different attitudes and customs concerning housing may affect the practicality of these strategies. As asthma rates appear to be of particular concern to Maori and Pacific Islands people, their input and comments are particularly sought.

Any strategy aimed at influencing the indoor environment will affect a wide range of people in many activities and businesses, particularly building construction, maintenance and housekeeping. The PHC is circulating this report as widely as possible. Comments on the practical, economic or technical feasibility of these strategies will be welcome.

Comments, enquiries and submissions should be sent, preferably by 31 August 1995, to:

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Summary

The last thirty years have seen a considerable growth in interest and concern about asthma, with increased mortality, increased severity and rising prevalence, reported from many countries. New Zealand has been at the forefront of these changes with an epidemic of mortality, high morbidity and rising prevalence.

These dismal statistics despite an enormous increase in the use of pharmaceuticals to manage asthma, have prompted an examination of the role of environmental factors in 'causing' asthma and maintaining symptoms. The development of sophisticated techniques for quantifying allergens in the environment has led to studies, that for house dust mite allergen, have defined levels associated with the risk of sensitisation and levels associated with an increased risk of asthma exacerbations.

Many different strategies have been employed to reduce domestic allergens and these strategies and their efficacy are reviewed in this report. What is known about domestic allergens in New Zealand is reviewed together with studies that are currently in progress.

Lastly recommendations are made concerning the need to increase the awareness of health professionals and the public about allergies and the need to consider further environmental intervention programmes in New Zealand for the secondary and ultimately the primary prevention of atopic asthma.

Background

In order to appreciate the growing concerns over the many aspects of asthma and allergic disease, the stimulus they have given to studies of the environment, and the role that New Zealand has played in these developments, it is necessary to review some of this background.

Epidemics of Asthma Mortality

An epidemic of asthma mortality - 1960s

Concern about asthma mortality began suddenly in the mid 1960s in Australia and the United Kingdom, with case reports, and then time trend data, linking a dramatic increase in asthma mortality, with sales of a new, potent synthetic adrenaline-like, bronchodilator - isoprenaline (Inman and Adelstein, 1969). The epidemic was particularly associated with increasing over the counter sales of a high dose 'forte' preparation by metered dose inhaler and the increase in mortality was most marked in the 14 to 19 age group (Inman and Adelstein, 1969). Mortality increased suddenly in six countries, but was not observed in any country where the high dose preparation was not sold (Stolley, 1972).

In the United Kingdom this dramatic rise in fatal asthma disturbed a hundred years of stable asthma mortality statistics (Speizer and Doll, 1968). Considerable debate at the time, and the lack of formal case control or cohort studies, left many unanswered questions and between the late 1960s and early 1980s a considerable 'rationalisation' took place, along two lines of reasoning. The first hypothesis suggested that the epidemic was spurious and due to diagnostic transfer from other respiratory diseases and incorrect coding of death certificates. The second was that if the increase was real it had arisen because the underlying severity of asthma had been underestimated and death had occurred because of severe asthma, and not as a consequence of therapy (Pearce et al, 1991).

The issue was hotly debated and never satisfactorily resolved. Nevertheless this episode in the late 1960s served to inform physicians that asthma could prove fatal contrary to contemporary dogma, and sowed the first seeds of concern about asthma therapy. This led to the development of more beta 2 selective bronchodilators without the pronounced cardiovascular effects of isoprenaline. With their introduction, and with warnings and restrictions on sales of isoprenaline, the epidemic disappeared as rapidly as it had begun (Inman and Adelstein, 1969).

The New Zealand mortality epidemic

The recognition in 1982 of a second asthma mortality epidemic, confined to New Zealand and commencing in 1976, had a significant impact on asthma research and subsequently on asthma management both in New Zealand and internationally (Jackson et al, 1982). As in the 1960s the epidemic led to considerable national and international interest and speculation about the likely causes, and led New Zealand and many other countries to examine similar trends in asthma prevalence and morbidity. A two year national case study showed that the epidemic was real and not a spurious increase from altered International Classification of Diseases (ICD) coding or diagnostic transfer (Sears et al, 1985). This case study became the touchstone for similar studies internationally and the subsequent demonstration of more gradual rising asthma mortality in many countries (Buist and Vollmer, 1990). In New Zealand a series of case control studies subsequently confirmed the most likely cause of the epidemic was the excessive use of the beta agonist fenoterol during severe asthma attacks (Pearce et al, 1990; Grainger et al, 1991; Crane et al, 1989). Interestingly fenoterol shared many features with isoprenaline in that it was marketed as a high dose 'forte' preparation and had considerably greater cardiovascular effects than other commonly prescribed beta agonists. Increased risks associated with fenoterol were also found in a case control study in Canada (Spitzer et al, 1992) and effective removal of fenoterol from the New Zealand market has been associated with a dramatic decline in asthma mortality and morbidity (Crane et al, 1992).

Rising Asthma Prevalence

During the period between the late 1960s and 1970s reports suggested that asthma was becoming more common (Dawson et al, 1969) and a clear linkage between the pulmonary and allergic aspects of asthma was made. At this stage the increases in prevalence were largely anecdotal as no previous studies were available for comparison. However many small studies of prevalence in children were undertaken around the world, for example, in New Zealand by Milne showing that wheezing illness was now a common problem in childhood (Milne, 1969). Subsequently repeat observations using identical criteria and methods to those first studies, have consistently shown a marked increase in reporting of asthma symptoms (see below).

Associated trends

Changes in the management of asthma in the 1970s

Investigation of cellular events in the airway showed that mast cells could be triggered to release smooth muscle agonists such as histamine by the bridging of cell bound receptors with IgE molecules. These immunological observations linked the atopic or allergic nature of asthma, directly to airway constricting events in the lung (Bhat et al, 1976). The demonstration of smooth muscle contraction by the release of preformed cellular smooth muscle agonists on signals from allergen specific IgE set the course for the regular use of beta agonist bronchodilators as the mainstay of asthma treatment. Through the 1970s the recommendations for using inhaled beta agonists by metered dose inhaler gradually underwent a change from 'as required' to regular use four times or more daily. In the last few years use has reverted to as required following renewed concern over beta agonist bronchodilators.

Worldwide trends

The more gradual worldwide trends of increasing prevalence, severity, and mortality remain puzzling, occurring as they do in the face of large increases in the use of anti-inflammatory asthma treatment and at a time of greater understanding of the pathophysiology of asthma, and when mortality for almost all other common chronic diseases is declining. This asthma paradox has been attributed by some authors to aspects of asthma treatment particularly beta agonist use. These hypotheses have suggested that chronic use of bronchodilators might increase the underlying severity of asthma by increasing exposure to inhaled allergen with the associated symptoms blocked by inhaled bronchodilators, or by beta agonists inhibiting release of potent anti-inflammatory mediators from cells such as mast cells and basophils (Page, 1991).

Increasing prevalence

Increasing asthma prevalence is a more likely explanation for the gradual as opposed to the epidemic rise in mortality and morbidity. There is now considerable evidence that asthma has become more common in children and young adults during the last 20 to 30 years, in countries with widely differing lifestyles and ethnic groups, including New Zealand (see *Table 1*). Although methodological differences in these studies make it difficult to interpret the magnitude of the differences in asthma prevalence between countries, the trend of increasing prevalence amongst similar populations within countries is remarkably consistent, and in some cases of considerable magnitude. As a result, asthma has become one of the most common medical disorders in both developing and western countries.

Table 1: International Trends in Asthma Prevalence in Children and Young Adults

Country	Reference	Period of Observation	"Asthma Prevalence %" † From	To
Australia	Robertson et al, 1991	1964-1990	19.1	46.0
	Peat et al, 1993	1982-1992	12.9	19.3
Canada	Infante-Rivard et al, 1987	1980-1983	3.8	6.5
England	Morrison-Smith, 1976	1956-1975	1.8	6.3
	Whincup et al, 1993	1966-1990	18.3	21.8
	Burney et al, 1990	1973-1986	2.4	3.6
Finland	Haahtela et al, 1990	1961-1986	0.1	1.8
France	Perdrizet et al, 1987	1968-1982	3.3	5.4
Israel	Auerbach et al, 1993	1986-1990	7.9	9.6
Japan	Nishima, 1993	1982-1992	3.3	4.6
New Zealand	Mitchell, 1983	1969-1982	7.1	13.5
	Shaw et al, 1990	1975-1989	26.2	34.0
Papua New Guinea	Dowse et al, 1985	1973-1984	0.0	0.6
Scotland	Ninan et al, 1992	1964-1989	10.4	19.8
Sweden	Alberg, 1989	1971-1981	1.9	2.8
Tahiti	Liard et al, 1988	1979-1984	11.5	14.3
Taiwan	Hsieh et al, 1988	1974-1985	1.3	5.1
USA	Gergen et al, 1988	1971-1976	4.8	7.6
	Weitzman et al, 1992	1981-1988	3.1	4.3
Wales	Burr et al, 1989	1973-1988	4.0	9.0

† Asthma prevalence data for a country is only included if the same method was used on two occasions. Many different methods were used to define asthma or asthma symptoms in studies from the different countries; as a result comparison of the asthma prevalence rates between countries should be avoided.

Studies in New Zealand amongst children have shown an increase in prevalence of asthma over the last 20 years. Perhaps the most striking recent data however, come from children in Australia where current wheezing in eight to 11 year olds has increased from 12.5 percent in 1982 to 25 percent in 1991. Similarly the prevalence of airway hyper-responsiveness (AHR) has increased from nine percent to 18 percent over the same period. Interestingly in this study there was no increase in the number of children showing positive skin test to common allergens. The increase in AHR, however, was predominantly in atopic individuals suggesting greater allergen exposure (Peat et al, 1994).

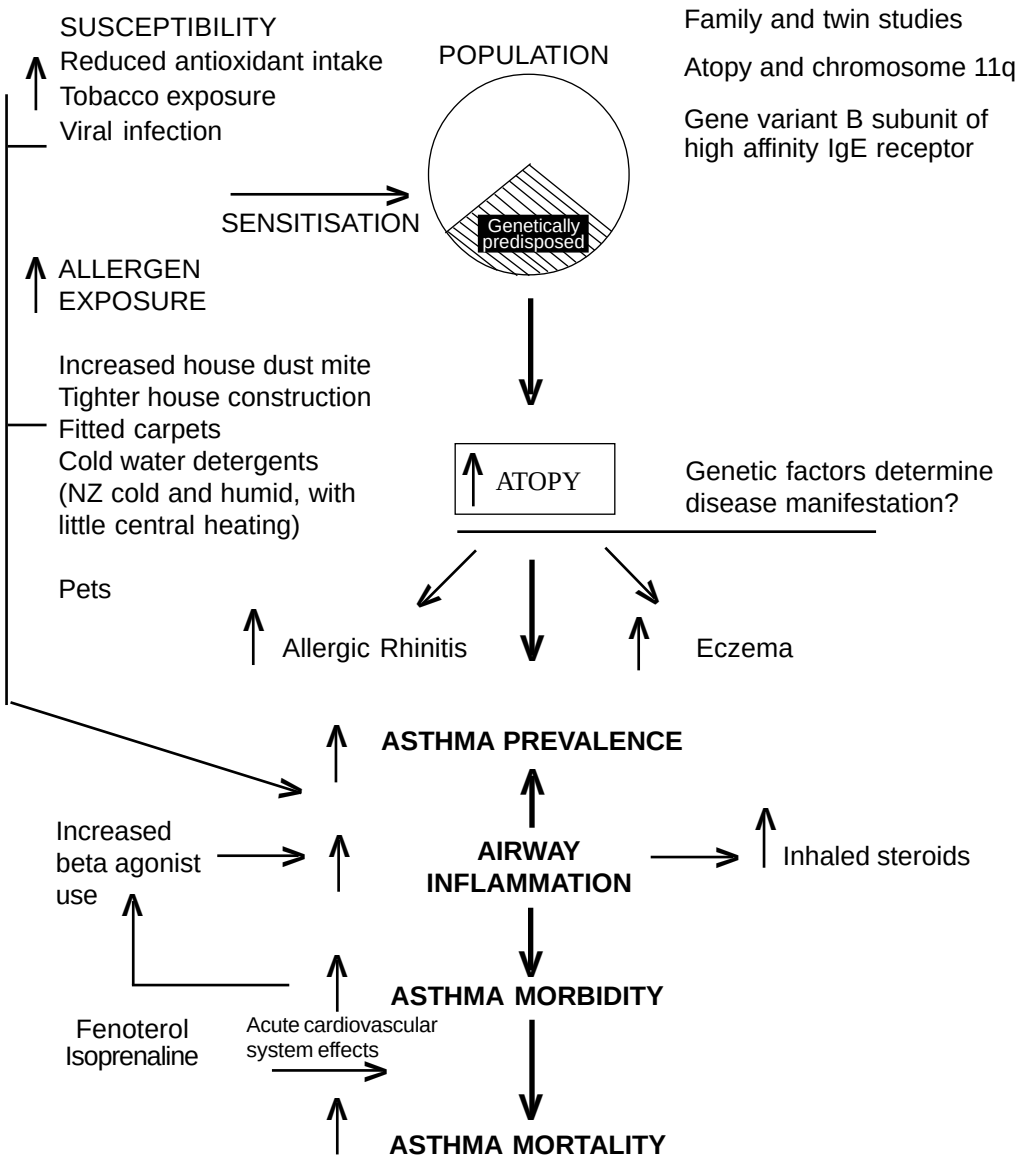
Other studies have examined the prevalence of hay fever and eczema, showing significant increases over recent years (Hsieh and Shen, 1988), and there exists some evidence that the markers of atopy such as positive skin tests to common environmental allergens and IgE levels have also risen over the last 10 to 20 years (Nakagomi et al, 1994).

Potential causes

This evidence suggests much more fundamental changes in the natural history of asthma and atopy over the previous 30 years. It is now possible to construct a tentative schema of some of the factors likely to be important in these changes (see *Figure 1*). Genetic factors are obviously very unlikely to explain the recent increase in prevalence, but are important, in that it is believed that only genetically susceptible individuals are capable of becoming sensitised. Two distinct hypotheses have been put forward:

- increased exposure to allergens, and
- increased susceptibility to allergens.

Figure 1: Factors Associated with Increasing Asthma Prevalence, Morbidity and Mortality



Allergen exposure

Increased exposure to allergens and particularly domestic allergens has been hypothesised. There is limited direct evidence to support this but changes in living conditions over the last 30 years would be expected to have encouraged house dust mite (HDM) proliferation. These include warmer and better insulated houses that have been much more tightly constructed to improve energy efficiency. Such construction means higher internal humidity from the activities of daily living such as showering, washing and drying of clothes, increased use of fitted carpets that cannot be taken outside to air and dry out, and the use of cold water detergents which do not destroy mites. There is certainly evidence for increased tightness of houses in New Zealand with a range of new construction materials and building techniques, since the first oil crises in the early 1970s [Mark Bassett, Building Research Association of New Zealand (BRANZ), personal communication, 1990].

The dramatic rise in asthma symptoms in New South Wales has been associated with a documented increase in mite counts (Peat et al, 1994).

Increased sensitisation

Alternatively (or possibly additionally) western populations may have become increasingly susceptible to environmental allergens. This has recently been suggested by Seaton et al (1994), and is discussed later. Tobacco exposure, particularly passive exposure in infancy and possibly even in utero may enhance sensitisation, by increasing allergen access to submucosal T cells through increased mucosal permeability (Venables et al, 1985) or components of tobacco smoke may act as allergens themselves (Lehrer et al, 1984). Sensitisation may also be increased by increased exposure to oxygen radicals that promote inflammation and are contained within cigarette smoke (Seaton et al, 1994). A significant adjuvant role for tobacco smoke in the development of atopy is certainly suggested by many epidemiological studies and very plausible mechanisms have been suggested.

Increasing airway inflammation

Both increased allergen exposure and increased sensitisation will also act directly on the airway to increase the extent of airway inflammation and hence increase asthma prevalence and asthma severity. A similar action has been proposed for beta agonists and may be one way in which the increased use of these drugs has increased prevalence (see *Figure 1*).

Family size and sibship

Lastly, a number of studies, the most recent from Germany, have shown a clear relationship between sibship and atopy, the risk of being atopic (positive skin prick tests) being inversely related to the number of siblings (von Mutius et al, 1994). The same effect has recently been found in New Zealand school children for reported wheezing and hay fever (Shaw et al, 1994). The cause of this phenomenon is unknown. Von Mutius et al (1994) speculate that it may be related to a protective role for viral infection in early childhood, frequent viral exposure from multiple siblings dampening down IgE production. This sibling effect could partly explain the increasing prevalence of atopy and asthma in association with diminishing family size.

Asthma and the Environment

From the above discussions it is clear that there is now considerable evidence that worldwide asthma prevalence is increasing with many plausible explanations. These changes in prevalence, the failure of current therapy to reduce asthma morbidity significantly, and the concern that drug therapy may even be responsible for increased mortality, has led to growing interest in determining the importance of environmental factors and in trying to reduce their effect.

Outdoor Air Pollution and Asthma

The relationship between air pollution and asthma has been more the subject of speculation than of intensive study. In general while almost any air pollutants will give rise to symptoms of airway narrowing in asthmatic individuals, there is very little evidence that air pollution per se is a risk factor for atopic asthma. In considering the underlying immunology of the airway inflammation there is indeed no reason why this should be so.

Historically, severe air pollution episodes have been associated with increased respiratory mortality as in the well known United Kingdom smogs (from burning fossil fuels) of the early 1950s but these fatalities were rarely in asthmatic patients and were predominantly in patients with severe chronic obstructive pulmonary disease (Scott, 1953). While worsening symptoms have certainly been reported amongst asthmatics in air pollution episodes (Schrenk et al, 1949) there has been no evidence that heavy exposure leads to asthma. The experience of asthma in New Zealand supports this, where asthma represents a major respiratory health problem throughout the country and where air quality is amongst the best in the world. Furthermore in many countries such as the United Kingdom and Taiwan, increases in the prevalence of asthma have been reported during periods of improvement in air quality (Hsieh and Shen, 1988).

The most impressive recent evidence supporting the hypothesis that air pollution is not a risk factor for atopic asthma comes from a study of asthma prevalence in Germany, comparing asthma, bronchitis, and atopy in two primary school populations in Munich, and in Leipzig in the former East Germany. This latter city had significantly greater air pollution in the form of particulates and sulphur dioxide than Munich. Lifetime prevalence of asthma, current wheezing and bronchial hyper-responsiveness to cold air challenge were similar in the two cities. The diagnosis of bronchitis was twice as common in Leipzig as in Munich. Interestingly skin prick tests for common allergens were significantly less common in children in Leipzig compared to those in Munich (von Mutius et al, 1992).

Airborne particulates

Individual air pollutants

Particles with a diameter less than10μm can be inhaled into the lung. Their effect in asthmatics depends partly on the chemical composition and partly on the proportion of respirable particles. They have been shown to lead to exacerbations of asthma (Pope, 1991). Similarly acid aerosols (acid rain) have been shown to give rise to symptoms in asthmatic patients (ATS, 1991).

Pollens and dust

Sudden outbreaks of asthma have been reported from many environments. Often the exact causes are unknown. In Australia studies have revealed outbreaks amongst sensitised individuals following severe rain storms and have been attributed to the release and disintegration of grass pollens (Bellomo et al, 1992). The most intensively researched recent series of asthma attacks occurred in Barcelona and were clearly associated with soya bean dust emitted from faulty silos during unloading from ships in association with temperature inversions over the city. These outbreaks were severe and extensive throughout downtown Barcelona. They ceased abruptly when the lids on the silos were replaced (Anto et al, 1989).

Algal bloom

In 1992 in New Zealand dinoflagellate blooms were associated with an outbreak of acute respiratory symptoms amongst asthmatics and non-asthmatics at Orewa Beach giving rise to wheezing, coughing and eye irritation [M Baker, personal communication, 1994]. Little is known about these outbreaks although similar symptoms have been previously recorded in Florida associated with algal blooms (Baden et al, 1982). Their mechanism of action is unknown but most probably represents direct chemical irritation of airways and mucous membranes.

Sulphur dioxide (SO_2)

Sulphur dioxide arises largely from combustion of fossil fuels containing sulphur impurities and in New Zealand in low concentration from geothermal areas. In the laboratory asthmatics are more sensitive that non-asthmatics and airway narrowing can be induced although this requires quite high ambient concentrations (Ericsson and Camner, 1983). Sulphur dioxide is not a common indoor pollutant except with the use of kerosene heaters.

Ozone (O3)

Ground level ozone is formed from nitrogen oxides, hydrocarbons and sunlight. Exacerbations of asthma have been reported in association with fluctuations in outdoor ozone levels. In the laboratory ozone is capable of inducing bronchial hyper-responsiveness in non-asthmatics and increasing airway hyper-responsiveness in asthmatics and is capable of inducing airway narrowing in both populations. Asthmatic subjects do not appear to be more sensitive to ozone than non-asthmatics (Lipman, 1989). Recently ozone has been shown to increase sensitivity to allergen challenge in asthmatic subjects (Molfino et al, 1991).

Nitrogen dioxide (NO_2)

Outdoors, this pollutant comes principally from car exhausts. It also arises indoors from gas fires and cookers (in higher concentration from unflued appliances). In the home nitrogen dioxide appears to cause irritant respiratory symptoms but does not affect asthmatics any more than non-asthmatics (Neas et al, 1991).

Pesticides

Exposure to pesticides and respiratory health problems have been closely associated in the public mind. Few studies have specifically examined pesticide exposure and asthma. Following case reports of respiratory illness in Saskatchewan, male farmers were surveyed and examined. A significant association was observed between asthma and cholinesterase-inhibiting

insecticides, but not with other types of pesticide (Senthilselvan, 1992). No surveys have been undertaken in New Zealand. Logistic problems of obtaining representative samples of farmers and farm workers and documenting exposures have hampered systematic study in this area.

The New Zealand situation

The contribution of outdoor air pollutants to respiratory disease and asthma in New Zealand has been little studied. Air pollution is likely to be a potential problem only in certain areas such as Christchurch where the burning of indoor fossil fuels combined with local climatic effects may give rise to high levels of particulate and gaseous pollutants.

A pilot study to monitor respiratory symptoms and ambient air quality is due to start in mid-1995 in Christchurch [I Town, personal communication, 1994]. A recent pilot study from the Institute of Environmental Health and Forensic Science examined indoor air quality and gas appliances in 45 homes in Auckland, Taupo and Rotorua. It demonstrated that unflued appliances were capable of raising levels of nitrogen dioxide to the World Health Organization (WHO) levels of concern (greater than 160 parts per billion) especially in houses with relatively low air exchange levels (less than 0.35 air changes per hour). This was recorded in 11 out of 45 houses. Flued gas appliances gave similar levels of nitrogen oxides as houses without gas appliances (Bettany et al, 1993).

The Indoor Environment and Asthma

The domestic environment contains a wide variety of materials and potentially toxic agents for those with asthma and for respiratory health in general. As we are destined to spend a third or more of our lives solely in this environment it is important to consider the respiratory hazards found in the home, and the approaches that might be employed to reduce risk.

Specific investigation of the New Zealand domestic environment in relation to asthma has not been undertaken. This is surprising given the problems associated with asthma in New Zealand and most likely reflects the lack of effective means of co-ordination and communication between health professionals, building scientists, and the building industry. Such communication has been eminently successful in Scandinavia and attempts are now being made to address some of these issues in Australia (Isaacs, 1993).

In the New Zealand domestic environment factors associated with asthma may be classified as follows:

Allergens

- house dust mite faecal pellets
- domestic pets - cats, dogs, pet rodents
- moulds and fungal spores
- cockroaches
- foodstuffs (non allergen - colourings)
- house plants.

Combustion air pollutants

- active and passive tobacco smoke
- oxides of nitrogen from domestic gas appliances
- particulate and chemical emissions from open fires, airtight and non-airtight wood burning stoves.

Domestic chemicals

- cleaning agents
- solder
- spray paints
- sterilising agents.

Construction materials

- formaldehyde from particle board and other sources.

Relationship of domestic environmental factors and asthma

Inhalant chemicals

Within the home, exposure to a wide variety of chemical agents such as those from tobacco, fossil fuel combustion, domestic chemicals and from construction materials have a role to play in the development and maintenance of asthma (Infante-Rivard, 1993).

Worldwide these domestic exposures are only just being systematically quantified and protective standards developed. As yet almost no data are available in New Zealand. A systematic survey of the domestic environment focused on respiratory health with appropriate monitoring would be an important contribution to our knowledge.

Formaldehyde

Formaldehyde is used in manufacturing particle board and is found in many domestic products including gas stoves and as a constituent of tobacco smoke. Its relationship to asthma is not entirely clear. Exposure can lead to irritant airway symptoms but its role in sensitising the airways is uncertain. Chamber studies with inhalation at three parts per million failed to show a decline in lung function in asthmatics or non-asthmatics (Marbury and Kriegler, 1991). Both epidemiological and laboratory data suggest that while non-specific irritant symptoms in asthmatics and non-asthmatics are quite common, formaldehyde induced asthma is not.

Allergens

Studies of asthma prevalence in environments with no requirement for domestic heating and with very different profiles of domestic chemical exposure also show high rates (Hsieh and Shen, 1988). From the available evidence, it appears that domestic allergens and particularly the house dust mite are the most significant risk factors for developing asthma (Sporik et al, 1990).

Inhalant Allergens and Asthma

Inhalant Allergens

For atopic asthma, inhalant allergens represent the most important source of stimulation of the chronic airway inflammatory process, and for the induction of atopy in genetically susceptible individuals (Bjorksten, 1994). The remainder of this report focuses almost exclusively on two of the most common allergens in New Zealand, the house dust mite and the domestic cat, and briefly discusses the role of tobacco smoke in enhancing sensitisation. While many of the other factors may be important in individual cases, the allergens from house dust mites and cats numerically represent an overwhelming predominance in the domestic inhalant allergen load.

Common environmental allergens are important in the development and maintenance of asthma. Initially sensitisation to the allergen occurs, and then continued exposure causes disease manifestations. For the development of atopy, defined as a process of sensitisation to common environmental allergens resulting in the expression of specific IgE class antibodies, a genetic predisposition is thought to be required (Bjorksten, 1994). In addition a genetic predisposition may be responsible for determining specific disease manifestation in atopic individuals (Edfors-Lubs, 1971). Frequently asthma and allergies have been observed to run in families. However, while individual components of the allergic process appear under genetic influence, overall the development of atopy amongst mono and dizygotic twins is less clearly inherited (Bonini et al, 1994). Molecular biological approaches have recently suggested 'atopy' genes residing on chromosome 11 and Shirakawa et al (1994) have identified a candidate gene on this chromosome thought to give rise to an abnormal beta chain for the IgE receptor on various cells.

Sensitisation

Exposure to inhalant allergens in the first year of life has been associated with an increased risk of atopy (Bjorksten et al, 1980). Similar observations have been made in relation to house dust mite exposure and to early exposure to pets (Warner et al, 1991; Warner and Price, 1978). Exposure during this period appears to carry a greater risk than subsequent exposure (Taylor et al, 1973). Some studies have suggested that sensitisation is enhanced by tobacco smoke as discussed briefly above. In utero exposure to tobacco smoke has been associated with high cord IgE levels and in some studies, with increased risk of atopy in later childhood. However these studies are confounded by post natal exposure (Magnusson, 1988). It appears likely that passive exposure to tobacco as well as causing exacerbations of asthma and increased respiratory disease in childhood, may also increase the risk of developing atopic asthma. In terms of air pollution this places passive smoking in a unique position.

Some authors have suggested that chemicals introduced into our environment in agriculture and food preservation over the last 30 to 40 years may also be acting to increase sensitisation and may help to explain the increase in the prevalence of atopy and asthma (Bjorksten, 1994), although there is little evidence in support.

Antioxidants

A recent hypothesis by Seaton et al (1994) has suggested that increased susceptibility to allergens may have increased consequent to a reduction in dietary antioxidants such as vitamin C and beta carotene. A reduction in these oxygen radical scavengers leading to oxygen radical airway inflammation might promote access of allergen to submucosal T lymphocytes, an essential

element in allergen recognition. Seaton et al (1994) cite the reduction in fruit and vegetable consumption that has occurred over the past 20 to 30 years in the United Kingdom as decreasing natural antioxidant intake while exogenous radicals from tobacco smoke and air pollution have continued to add to airway radical loads.

Maintenance of Asthma

Miyamoto, as early as 1968, demonstrated that the inhalation of dust mite extract could induce an attack of asthma (Miyamoto et al, 1968). Allergen challenge studies have shown clinical and inflammatory features indistinguishable from those of natural asthma attacks (Warner, 1976). In the case of the HDM allergen Der p1, it has been proposed that exposure to greater than 2µg/gm fine dust is a significant risk for sensitisation and greater than 10µg/gm increases the risk of acute exacerbations (Platts-Mills and de Weck, 1989). These levels first proposed in 1987 from prevalence studies have continued to broadly define risk levels that have been confirmed in subsequent epidemiological studies (Platts-Mills et al, 1992). While clearly not representing absolute risk levels they have proved extremely useful in defining exposure and goals for eradication.

Allergic Rhinitis and Atopic Dermatitis

No formal epidemiological studies of allergic rhinitis and HDM have been undertaken. However Charpin et al (1988) have shown that 44.5 percent of a volunteer population in Marseilles had positive skin tests to HDM compared to 10 percent in Briançon in the French Alps (1,350 metres). The reported frequency of perennial rhinitis was 12.2 and 4.3 percent respectively.

Patients with atopic dermatitis frequently show IgE antibodies to HDM and positive skin prick tests (Chapman et al, 1983). Patch tests for HDM have shown delayed eczematous type lesions in these patients. Epidemiological studies have also shown an association between levels of HDM and the prevalence of eczema (Beck and Korsgaard, 1989). Thus HDM and other environmental allergens may be increasingly implicated in the development of both allergic rhinitis and eczema.

Cat and House Dust Mite Allergens

The Biology and Distribution of Cat Allergens

A number of cat allergens have been isolated, the most important being Fel d 1, found predominantly in saliva and lacrimal secretions (Milligen et al, 1990). Fel d 1 is distributed by preening where the fur is coated in saliva and ejected into the air in the form of small (less than 2.5μm) diameter sticky particles (Platts-Mills et al, 1986). This cat allergen is much more widely distributed in the environment than HDM allergen. Studies in households with no history of cat ownership have revealed detectable quantities of airborne allergen presumably being distributed on clothing (Warner et al, 1991). Recent analysis of dust from the National Aereonautics and Space Administration (NASA) space shuttle revealed measurable quantities of Fel d 1 in dust obtained from the space vehicle. No mite allergen was found [M Chapman, Christchurch meeting of the Thoracic Society of Australia and New Zealand, August 1994].

The cat saliva particles remain suspended in the air by convection currents for long periods and this may explain the clinical observation that cat sensitive individuals experience symptoms immediately upon entering an environment containing cats. Cats have been shown to shed between 0.05 and 0.17gm of allergen per day resulting in microgram amounts of allergen per cubic meter of air and hundreds of micrograms per gm of fine dust (Luczynska et al, 1990).

Dog allergens have been less intensively studied to date but the recent development of a monoclonal antibody to the main allergen Can f 1 will allow quantification of dog allergens. Can f 1 comes from hair, saliva and dander (Blands et al, 1977). Dog allergens are less likely to cause sensitivity although it is not known if this is due to lower allergenic potential or a less ubiquitous distribution.

For both cat and dog, allergen exposure in the first year of life appears to be particularly important. In a study by Warner et al (1991) almost 90 percent of 21 asthmatic children whose homes contained a cat at their birth were sensitive to cats compared to 36 percent of children without cats or where cats had been introduced after the first year of life. Similar findings have been observed for dogs.

The Biology and Distribution of House Dust Mites

House dust mites belong to the class Arachnida (spiders, scorpions, etc) and were first recognised in 1867 in house dust. The association between respiratory problems and dust goes back at least 300 years when van Helmont, a Flemish physician, reported a monk experiencing severe respiratory difficulty when exposed to airborne dust (van Helmont, 1662).

In the 1960s Voorhorst and Spieksma established the house dust mite as the major source of allergen in dust (Voorhorst et al, 1967). These workers conducted a series of clinical experiments demonstrating improvement in asthma symptoms by isolating asthmatics in dust free environments. The two most important mite species numerically and clinically are Dermataphagoides pteronyssinus and D farinae of the family Pyroglyphidae.

Twelve other members of this family have been found in domestic dust but often in discrete geographical regions. Six species have been found exclusively in association with human habitation, whereas another 26 species live exclusively on birds or in their nests. This has led to the speculation that mites entered the human habitat in association with birds and evolved an exclusive relationship with man. Two other families known as storage mites Acaridae and Glyophagidae have also been implicated in asthma, particularly in people handling grains and cheese (Baker, 1994).

D pteronyssinus is the most common mite found in New Zealand homes (Cornere, 1972). The presence of one of the storage mites, Glycophagus domesticus, has been confirmed in domestic house dust in Wellington.

Mite biology

This was reviewed by Arlian (1989).

Habitat

Mites are distributed throughout the home. The highest levels have been reported from mattresses, pillows, carpets, upholstered furniture, bed covers, clothes, and soft toys. Studies of almost 160 homes in Wellington have shown consistently higher levels in bedding compared to floors (unpublished observations).

Mite population maintenance

Mites will ingest a wide variety of organic matter. Their principal food source when in association with humans is skin scales often colonised by microfungi. This food source is present in super abundance and is not a limiting factor to their growth. Mites have a well developed intestinal system. In the posterior gut the food pellets are enveloped in a fluid that solidifies into a surrounding membrane when the faecal pellet is excreted. This membrane contains the cysteine proteases that constitute the principal allergen, Der p 1.

The survival, proliferation and feeding of mites is dependent on a sufficient direct water intake from the environment and hence they are particularly susceptible to changes in humidity. Humidity is the most important limiting factor for their survival, as mites will only be active and proliferate when the relative humidity is sufficiently high to allow water to be gained from the surrounding air. Thus mites are unlikely to survive if populations are exposed to relative humidities (RH) of less than 50 percent. A higher house dust mite infestation and sensitisation rate is observed in subjects living in humid and coastal areas than is observed in dry areas of the world and excessive growth of house dust mites has been estimated to occur at winter absolute indoor humidities of 7g of water per kg or more of air. Mites are susceptible to desiccation and as humidity declines they will cluster together between the fibres and fabrics of beds, carpets and furnishings in order to maintain a high local humidity. These niches may provide a micro climate which differs from the humidity and temperature in the room.

Humidity requirements of mites are affected greatly by temperature as higher temperatures are more dehydrating. The activity and proliferation of mites is restricted to a narrow range of temperatures. A temperature of 25^{o} C is the optimum for mite colony proliferation; at this temperature mites require about one month to complete a life cycle. At 15^{o} C, one year is required to complete a life cycle. At temperatures greater than 25^{o} C development time is more rapid but fewer mites will survive (Kinnaird, 1974). Mites will not survive freezing for more than six hours and only a few mites will survive six hours at 50^{o} C (at 60% RH) (van Bronswijk and Koekkoek, 1972).

Active dust mites can survive for about 24 hours and the quiescent protonymphal stage can survive for months at 0 percent RH because of the impermeable exoskeleton and minimal loss associated with body processes like excretion, reproduction and feeding in this life cycle stage. This desiccation-resistant protonymph, formed during dry periods of the year, provides the major source of breeding mites for the population growth when the conditions become more favourable (Arlian, 1989).

This sensitivity to ambient water content is thought to be the principal reason for reduced HDM populations found at increasing altitude in Europe (Charpin et al, 1988), (and lower rates of reported asthma symptoms in alpine versus maritime populations) and has been exploited in mite avoidance strategies using domestic heat recovery/ventilation systems.

Quantifying house dust mites and their allergens

There are three principal methods for quantifying mites and their allergens in the environment, quantities being expressed as mites or allergen per gram fine dust or per unit area:

- direct counting of mites (live, dead and eggs) in reservoir dust samples
- measurement of mite allergens by immunological (ELISA) assays in reservoir dust samples
- measurement of mite allergen in airborne samples using air samplers or in settle dust.

Direct counting

Mites can be counted in dust samples, and by using a standard technique comparisons of the degree of infestation can be made between different environments. However, this is a slow labour intensive process and requires skill in recognition. It is not possible to count faecal particles which are the principal source of allergen related to asthma. It does, however, allow the different species to be identified and permit recognition of the life cycle stages of the mite. Live migratory mites can also be directly examined using adhesive tape placed on furniture or bedding overnight. Mites will stick to the tape and can then be counted and characterised. This method is based on the "mobility test" developed by Bischoff et al (1992). The sample adhesive tape may be covered with plastic wrap for viewing on a stereo microscope at 20 to 30 times normal size. Mites can be counted and then removed, and have their species and life cycle stage determined by use of a higher powered microscope.

Allergen measurement

The development of monoclonal antibodies and ELISA assay techniques to allergen measurement has revolutionised the study of allergens and allergen avoidance. The allergens associated with mite faeces can now be measured directly and this allows a quantitative determination of the material that is actually giving rise to airway inflammation. Major and minor allergens have been identified from complex allergenic extracts of D pteronyssinus and D farinae. Two major allergens; Der fI from D farinae (Dandeu et al, 1982) and Der p 1 from D pteronyssinus have been purified and extensively studied (Chapman and Platts-Mills, 1980). The role of other mite allergens (Groups 2, 3 and 4) have been shown to elicit IgE responses but for allergen quantification purposes have been much less widely studied. The importance of their role will become increasingly apparent over the next few years.

The inhalant allergen Der p 1 is a cysteine protease excreted in the faeces of D pteronyssinus. IgE antibodies directed against this major allergen Der p 1 dominate the response of most mite allergic patients (Chapman and Platts-Mills, 1980). It is therefore, an ideal molecule for study as it represents one of the major allergic components of house dust. A two site monoclonal antibody (Mab) ELISA has been developed to measure group I allergens from Dermatophagoides spp; Der p 1 from D pteronyssinus and Der f I from D farinae (Luczynska et al, 1989). These group 1 allergens have been used extensively for measuring allergen levels.

Airborne sampling

Airborne dust mite allergen can most easily be measured using a settle technique. Small open petri dishes are left side by side, one to two metres above floor level with no overhanging shelves and where the dishes are unlikely to be disturbed by children or pets. This technique has the particular advantage of measuring airborne allergen and therefore most closely reflects the degree of disturbance of surface dust, and reflects the allergen available for inhalation [E Tovey, personal communication, 1993]. A few investigators have also examined personal and environmental air samplers for measuring airborne allergen levels. However most investigators have continued to advocate comparisons based on reservoir sampling on furniture, carpets etc, as airborne methods usually only detect allergen following dust disturbance.

The Relationship Between Asthma and Cat and House Dust Mite Allergens in New Zealand

Every asthma prevalence survey in New Zealand that has included skin prick test to common allergens has shown house dust mite and grasses to be the most common positive reactions, followed by cat dander. The relationship of these responses to asthma has been assessed by Sears et al (1988) in the Dunedin cohort study.

At age 13, 30 percent of the children had positive reactions to rye grass and HDM and 13 percent to cat dander. By examining allergen specific relative risks adjusted for sensitivity to other allergens Sears et al (1988) were able to demonstrate for asthma symptoms and airways hyper-responsiveness (AHR) combined, relative risks of 6.7 for HDM, 4.2 for cat, but only 1.3 for rye grass. These findings suggest that while rye grass was the most common sensitising allergen it was not independently associated with asthma symptoms or AHR. They also found high relative risks associated with the mould aspergillus but believed that this was most likely to represent secondary sensitisation following the development of asthma rather than a predisposing factor. This pattern is commonly found in adult asthmatics. No other mould sensitivity was associated with an increased risk. The authors concluded that both cat and HDM had similar high risk associations with asthma but as sensitivity to HDM was twice as prevalent as sensitivity to cat, HDM was clearly the single most important allergen in New Zealand (Sears et al, 1988).

HDM allergen would be by far the most common inhalant allergen worldwide having been found in almost all human environments (Platts-Mills and de Weck, 1989).

Cat and house dust mite allergen levels in New Zealand

No published data are available on these levels in New Zealand. However, cat ownership is extremely common in New Zealand. Sears et al (1988) noted that 78.5 percent of children by age nine had had a cat in their household at some time. Burr et al (1994) recently reported in a four country comparative study of childhood asthma, cat ownership by 63 percent of New Zealand children compared to 28 percent in Wales, 42 percent in South Africa and 20 percent in Sweden.

New Zealand studies

The Wellington Asthma Research Group has recently commenced a series of studies both examining HDM allergen levels in the domestic environment in Wellington, and studying two methods of HDM reduction. Preliminary results from these studies have indicated high levels of Der p 1 in bedding, bedroom carpets and living room carpets. Levels in bedding from a random sample of households with asthmatic children have revealed the highest Der p 1 levels ever recorded (Geo mean 80µg per gm fine dust), and are similar to those found in coastal New South Wales. Almost 25 percent of children in these studies are regularly exposed to 10 times the level of 10µg/gm fine dust associated with risk of exacerbations of asthma and over 93 percent to levels greater than10µg/gm.

Controlling Mites and their Allergens

The allergens associated with HDM have been studied in considerably more detail than any other allergen. Similarly there have been a large number of studies of avoidance using a wide variety of methods.

As mentioned previously the first studies of HDM avoidance in asthma were undertaken in the 1960s by Voorhorst and Spieksma (1967) using specially constructed allergen free chambers. In 1982 Platts-Mills and others admitted a group of severe mite sensitive asthmatics to a special low allergen hospital environment for a prolonged period. They were able to demonstrate

a marked reduction in symptoms and AHR (Platts-Mills et al, 1982). Studies in Europe where some of the old alpine tuberculosis sanatoria have been re-opened as asthma summer camps have shown similar results associated with very low mite counts in the alpine environment (Vervloet et al, 1979). Children have shown dramatic improvements in their asthma with a recurrence of symptoms upon returning to lower altitudes.

Methods of mite control fall broadly into three groups, chemical, barrier (separating mites from the environment), and physical.

Chemical control

A wide variety of agents have been shown to be effective acaricides in the laboratory, ranging from caffeine to organophosphates (Colloff, 1990). However, acaracides have been less effective in the domestic environment, where great variability in efficacy has been observed even with identical protocols in similar premises. These differences are thought to relate to problems in delivering acaricide in sufficient concentration, for example, to the base of carpets (Platts-Mills et al, 1992). Very high kill rates are required to maintain low allergen levels as mite populations can recover very quickly because of their short life cycle. Regular treatment is required raising the possibility of accumulation of residual acaricide over time. Some studies have failed to remove allergen after acaricide treatment. This requires vigorous vacuuming and is essential if clinical outcomes are being measured.

Studies with acaricides have produced equivocal results with regard to clinical improvement in asthma. Tannic acid, which denatures allergens but does not destroy mites, has also been used (Green et al, 1989). There remains also a concern regarding the introduction of further chemicals into the domestic environment of the atopic individual.

Barrier protection

A number of studies have shown considerable reduction in mites on mattresses covered with porous plastic zippered covers. Most clinical studies have shown clear clinical benefit, although only three had control groups (Murray and Ferguson, 1983). Mite levels within the mattress have been shown to increase five-fold so regular inspection for tears in the fabric are essential [E Tovey, personal communication, 1993]. These methods are usually combined with regular bedroom cleaning and the use of washable bedding material that is hot washed regularly. Mites are destroyed by washing in hot water (at or over 55^{o} C).

Physical methods

Humidity

More recently, some investigators have advocated alterations in humidity in the domestic environment as a means of controlling mite counts. In a controlled study, improvement in clinical asthma has been demonstrated using mechanical ventilation systems (Harving et al, 1988). Some investigators regard reduction in humidity as essential for controlling mites, because of the clear sensitivity of the house dust mite to humidity. There are advantages in this method of control, being considerably less invasive than the use of acaricides, and it may obviate the need for excessive cleaning, or removal of carpets and soft furnishings from the home.

The most extensive studies dealing with ventilation systems as a preventive measure in house dust mite allergy have been carried out on a housing estate in Denmark. These studies evaluated a healthy-building project which included a mechanical ventilation and heat recovery system, securing a high ventilation rate in the dwellings. Air inlets were placed in all the living areas

and bedrooms while exhaust air was taken from the most damp rooms, that is, kitchen and bathroom. The total observation lasted one to one and a half years, and incorporated:

- mite counts
- humidity recording
- the clinical improvement of asthmatics in the dwellings.

It was concluded that the high ventilation rate due to the mechanical ventilation system was the major cause of low indoor humidity and thus, in 11 out of 16 households, the disappearance of the house dust mites formerly found in elevated numbers. Marked clinical improvement was registered in asthmatics residing in these dwellings which was highly correlated with changes in the house dust mite counts (Harving et al, 1991).

New Zealand studies: The Wellington Asthma Research Group has set up a smaller scale project along similar lines in conjunction with Electrocorp New Zealand, Capital Power and BRANZ. Similar in-built ventilation and heat recovery systems have been installed in ten Wellington City Council houses that have also had their insulation and dampness upgraded. There are two sets of controls, a further ten houses with the insulation alone and a further ten with no interventions. HDM allergen levels have been monitored for the previous six months and the houses are being monitored for a further 8 to 12 months following installation of the systems. All houses are also having temperature and humidity continually monitored. Results from this study should be available towards the middle of 1995.

This approach is clearly a challenge for the New Zealand environment with much higher winter humidities than Denmark. However, RH levels between 40 to 50 percent have been achieved in most houses for most of the time, although on particularly humid days levels rise briefly above 50 percent. A further option will be to incorporate de-humidification systems into the ducting if RH cannot be maintained below 50 percent. Similar studies are being undertaken in Europe and the United Kingdom.

Portable dehumidifiers: No large scale controlled trials of portable dehumidifiers have been undertaken. While this approach seems a logical one, in practice in the domestic environment moist air is continually exchanged with the drier air from the dehumidified room unless the humidifier is used in a room completely sealed from the rest of the house. Dehumidifiers also become relatively inefficient around 50 percent RH and have considerable difficulty in reducing humidity below that level. A trial in the bedrooms of children in Denmark also revealed that they were unacceptably noisy and caused uncomfortable increases in temperature of around 5^{o}C [J Korsgaard, personal communication, 1993].

Freezing with liquid nitrogen (N_2)

Treatment of carpets, mattresses and soft furnishings with liquid nitrogen has been shown to kill HDM very effectively and help to lift dust which can then be removed with intensive vacuum cleaning. One hundred fold reductions in mite densities have been achieved (Colloff, 1986). Only one clinical trial has been undertaken and this demonstrated clinical benefit to asthmatics (Dorward et al, 1988). How frequently this treatment has to be used has not been studied. The application is difficult and needs to be undertaken by trained operators as liquid nitrogen can cause severe burns and can potentially lead to suffocation when applied in large volumes to floors. Approximately 30 to 40L is required to treat a house. A commercial operation has been started in Scotland but no large scale trials have been undertaken. Most fabrics and carpet materials appear to suffer no permanent damage from this snap freezing (Colloff, 1986).

High pressure, high temperature steam cleaning

This method has been studied recently although the results have not yet been published. Such steam cleaning was shown to kill mites effectively, and to denature effectively both group 1 HDM allergens and also the cat allergen Fel d 1 as both are heat labile [M Colloff, personal communication, 1994]. Appropriate steam cleaning equipment is currently being developed with a carpet cleaning firm in Wellington. The Wellington Asthma Research Group is hoping to pilot this method in a controlled study of 40 asthmatics combined with barrier mattress covers and hot washing of synthetic bedding material. This study is planned for late 1995.

Dry vacuum cleaning

Intensive vacuum cleaning has been shown to remove dead mites and allergen effectively, but not live mites. Live mites attach to fabric fibres with suckers and cannot be removed to any great extent by vacuuming. Vacuum cleaners with small particle filters (trapping particles greater than 1μm) have been shown to reduce respirable allergen in their exhaust (Kalra et al, 1990). The clinical relevance of these measures has not been studied. In the United States recently double skinned conventional collection bags have also been shown to reduce allergen particle exhaust and United States companies are now making double thickness bags [M Chapman, personal communication, August 1994]. Some conventional vacuum cleaners have been shown to increase significantly aero-allergen concentrations following vacuum cleaning.

Wet vacuum cleaning

This has been little studied to date. Der p 1 is water soluble and might be expected to be removed by this process. One recent study showed an increase in D pteronyssinus following this treatment probably due to removal of predator mites and increased carpet humidity from the cleaning process (Wassenaar, 1988). Simple shampooing of carpets has not proved any more effective than intensive dry vacuum cleaning (Boer, 1990).

Sun drying

Tovey found that exposure of carpets to sunlight significantly reduced mite populations secondary to the associated increase in temperature and fall in humidity (Tovey and Woolcock, 1994). Mites are also sensitive to ultra-violet light although this has not been used in eradication strategies.

Air filtration devices and ionisers

Electrostatic or mechanical high-efficiency particulate air filters (HEPA) have shown equivocal results in small clinical trials. In one placebo controlled cross over study slight improvement was noted in asthma and allergic rhinitis after four weeks' treatment (Reisman et al, 1990). Ionisers have usually failed to show improvement in placebo controlled trials, despite a significant reduction in airborne Der p 1 in one study. In this same study an increase in nocturnal cough was noted possibly due to the irritant effects of positively charged ions or ozone on the airway (Lipin et al, 1984).

Conclusions and Recommendations

Large Scale Asthma Prevention

The first step in managing asthma and allergic diseases environmentally is to increase allergy awareness amongst the population and amongst health care providers, and to include evaluation of atopic status and environmental allergens as a routine part of management.

Primary prevention

Primary prevention of asthma is the ultimate goal of allergen avoidance and longitudinal studies of allergen avoidance in high risk infants need to be developed. At present there are no research data to support such interventions on a wide scale. For many research groups working in this area this is now a major priority, and small scale studies are already in the field.

Secondary prevention

In reviewing the rapidly expanding literature it is clear that there is a need to develop a large scale closely monitored programme of secondary asthma prevention.

In developmental terms the prototype 'product' - allergen avoidance - has now completed extensive field trials. The next stage should be to scale up these studies to a closely monitored public health intervention strategy with effective allergen, clinical and economic evaluations. Current asthma pharmaceuticals cost $103 million per annum, 16 percent of the total drug expenditure [PHARMAC, personal communication, 1994]. In economic terms alone, this warrants a well monitored public health intervention. Such a proposed intervention should incorporate multiple avoidance strategies that could easily be adopted nationwide, along the lines outlined in this report.

Promoting allergy awareness

New Zealand has lagged behind many other countries in promoting this aspect of asthma management. There is an urgent need to educate health professionals and the public about allergen avoidance in asthma. Few general physicians, or even respiratory physicians, would regularly determine the atopic status of their patients with skin tests, or specific IgE tests. Such tests should be routinely undertaken in atopic asthma. The New Zealand Asthma Foundation is preparing a campaign to educate both health professionals and the public about allergies and avoidance strategies in 1995. A Therapeutic Note for health professionals and information sheets were made available to the public during Asthma Awareness Week 1995. This increased awareness would allow individuals who can afford it, to undertake their own mite reduction strategies.

Monitoring allergen levels

Monitoring of domestic allergen levels needs to be encouraged, and assays should become clinically available as well as being a research evaluation tool. Such measurements are essential in determining the efficacy of interventions. This will require laboratories to develop the specific ELISA assays which would present very little problem for most clinical laboratories as they do regularly use the ELISA techniques for many other routine clinical measurements. High levels of HDM allergen and the likelihood of similarly high levels of cat allergen in New Zealand underline the importance of promoting these strategies urgently.

Control

Some methods of control have clearly proved more effective than others, although in general HDM and allergens have been reduced, and this has resulted in significant improvements in asthma symptoms. More well controlled and larger studies are now required.

Avoidance may be undertaken at two levels. The first level involves lifestyle changes designed to reduce mite populations. These include:

- regular cleaning
- removal of carpets
- using mattress covers
- the replacement of bedding with synthetic materials that can be regularly hot washed
- the elimination of pets, or strict removal of them from bedrooms.

The second level requires both:

- discrete mite and allergen reduction using either physical or chemical methods
- minimising damp within the home and ensuring adequate ventilation.

Conclusions

This report concentrates on the domestic environment and specifically on HDMs and their allergens. There is now overwhelming evidence for the intimate association of house dust mite allergens with all stages of the development and maintenance of atopic asthma. There is clear evidence of the prime importance of these allergens in New Zealand and universal exposure to sensitising levels in Wellington homes has recently been demonstrated. Any systematic environmental intervention in New Zealand would have to start with attempting to reduce exposure to house dust mites. A wide range of intervention strategies studied in small projects, with varying degrees of scientific rigour, many showing improvement in asthma symptoms, have been presented. Small scale intervention studies are now underway in Wellington and further studies are planned in collaboration with the Christchurch respiratory research group. This is important because the New Zealand outdoor and indoor environments are different from many overseas, and the very high levels of allergen in New Zealand may require different or more intensive strategies.

Such interventions are now entirely feasible, are supported by considerable research data, and require a national effort and should involve public and individual health professionals, the Ministry of Health, voluntary agencies such as the Asthma Foundation, and regional health authorities.

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