CLIMATE AND CHRONIC RESPIRATORY DISEASE IN SYDNEY – THE CASE OF ASTHMA

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Abstract. Asthma remains a major health concern for many Australians. Currently mortality and morbidity rates are very high and have increased substantially over the last 10 years. Within Sydney there is a distinctive pattern of asthma risk with both inner-city and outer western suburbs figuring prominently.

This paper looks at asthma within the Sydney metropolitan area. It pays particular attention to the direct and indirect effects of weather and climate on the disease, as well as the interplay between a number of biophysical and socio-economic factors and the role played by outdoor and indoor air pollution. In particular it discusses the relationship between topography, meteorology, air pollution and asthma in Sydney. Finally, it argues that climate change will greatly influence the prevalence and distribution of the disease.

Introduction

Between 1981 and 1988 almost 14,000 people in the Sydney Metropolitan Area died from respiratory disease. Approximately 5,800 died from chronic obstructive airways disease, 2400 from pneumonia and slightly more than 1200 from asthma. An unacceptable proportion of these deaths were untimely or premature. In the case of respiratory disease as a whole more than 11% of the deaths occurred before the age of 60 years and 48% before the age of 75 years. With respect to asthma 37% of all deaths occurred before the age of 60 and three-quarters before the age of 75 years. Mortality alone, however, tends to disguise the real impact of respiratory disease on the Sydney community. Many respiratory diseases such as asthma do not kill but slowly undermine, debilitate and severely downgrade the life-style and quality of life of the sufferer. Today in Australia respiratory disease remains one of the most important causes of chronic illness and is responsible for a substantial proportion of absenteeism from work and school as well as for considerable human suffering.

Climate and Chronic Respiratory Disease

Respiratory diseases enjoy the reputation of being among the most weather/climate sensitive. Tromp, for example, while arguing that a great majority of human ailments are either triggered, stimulated or even caused by meteorological stimuli classifies respiratory diseases as the most important of the meteorotropic diseases (Tromp, 1980). Despite such confidence the mechanisms of the relationship

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between climate and respiratory disease are still not well understood. Climate would seem to affect the respiratory tract in a variety of ways. In the first place there would appear to be broad seasonal effects which probably represent the combined effect of climatic stimuli on different basic physiological and bio-chemical processes allied to broad climatic-inspired changes in the physical and biologic environment. There have, for example, been reported significant seasonal variations in a wide range of human diseases both chronic and infectious (Table I). In the second place a number of studies have documented the direct effects of climate on the distribution of respiratory disease (see for example, Ayres, 1990). Thunderstorms and sandstorms have often been linked with outbreaks of asthma and periods of very high or low temperatures have been associated with increased morbidity and mortality from a wide range of respiratory diseases. Cold fronts, spring rain and increased cyclonic activity have also been linked with respiratory complaints. The actual mechanisms of some of these effects are complex and still poorly understood. Probably the interaction between a variety of factors for which climate is the initiating mechanism leads to an increase in environmental triggers as

TABLE I: Recognised seasonal effects on respiratory illness

Season	Condition	Possible mechanisms
Winter	acute bronchitis bronchiolitis	colder temperatures increased viral exposure
	acute exacerbations of chronic obstructive airways disease/chronic bronchitis	colder temperatures increased viral exposure
	asthma	viral-induced episodes
	pneumonia	colder temperature
Spring	asthma	spring rains/thunderstorms pollen release agricultural spraying
	hayfever	pollens/atmospheric turbulence
Summer	asthma	pollen release hot still weather/air pollution ozone
	chronic obstructive airways disease/emphysema	hot still weather/photochemical smog
Autumn	asthma	colder temperature/pollen release (e.g. *parietaria*)
	acute bronchitis	viral infections

Source: Ayres, 1990; Tromp, 1980.

well as heightened human susceptibility to viruses and various respiratory disorders. In this way, for example, a prolonged period of hot, humid, still weather in a particular urban locality can lead to increased photochemical smog levels which independently or possibly combined with other environmental factors such as pollens, fungi, moulds and/or smoking behaviour leads to an increased incidence of respiratory disease. A combined increase in temperature with increasing levels of tropospheric ozone could have the same effect, particularly in people suffering from chronic obstructive airways disease or asthma.

The Distribution of Chronic Respiratory Disease in Sydney

Figure 1 illustrates the geographical pattern of high risk areas of respiratory disease mortality in Sydney in 1981–88.[1]

Two distinct areas of high respiratory mortality stand out in the metropolitan area. The first lies within the central-inner part of the city taking in the local government areas of Sydney City, Leichhardt, Marrickville and Ashfield.[2]

The second lies to the southwest of the city encompassing the municipalities of Liverpool and Campbelltown. To the west an extension takes in the municipality of Auburn. Within these broad areas standardised respiratory disease mortality rates

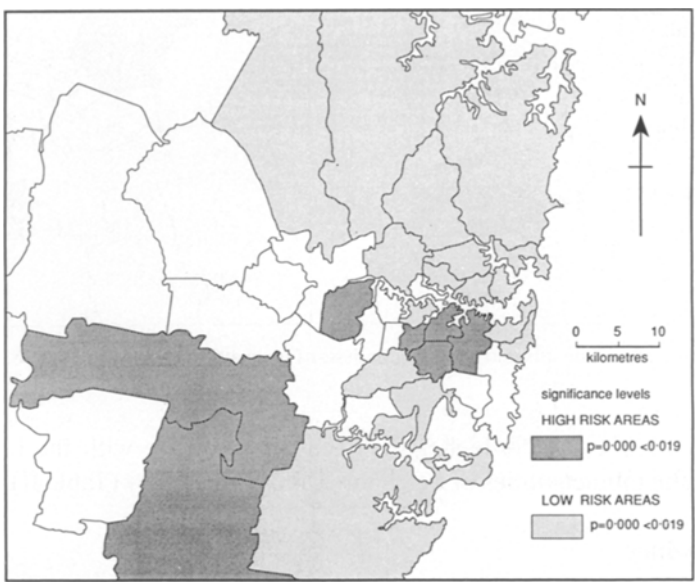

Fig. 1. High and low risk areas – deaths from all respiratory diseases Sydney 1981–88.

[1] Respiratory disease for this purpose includes all diseases categorised under ICD 9th Revision Classification 460–579.

[2] This map and Figure 2 are based on age-standardised mortality rates for 1981–88. A key to Sydney local government areas is appended to Figure 2.

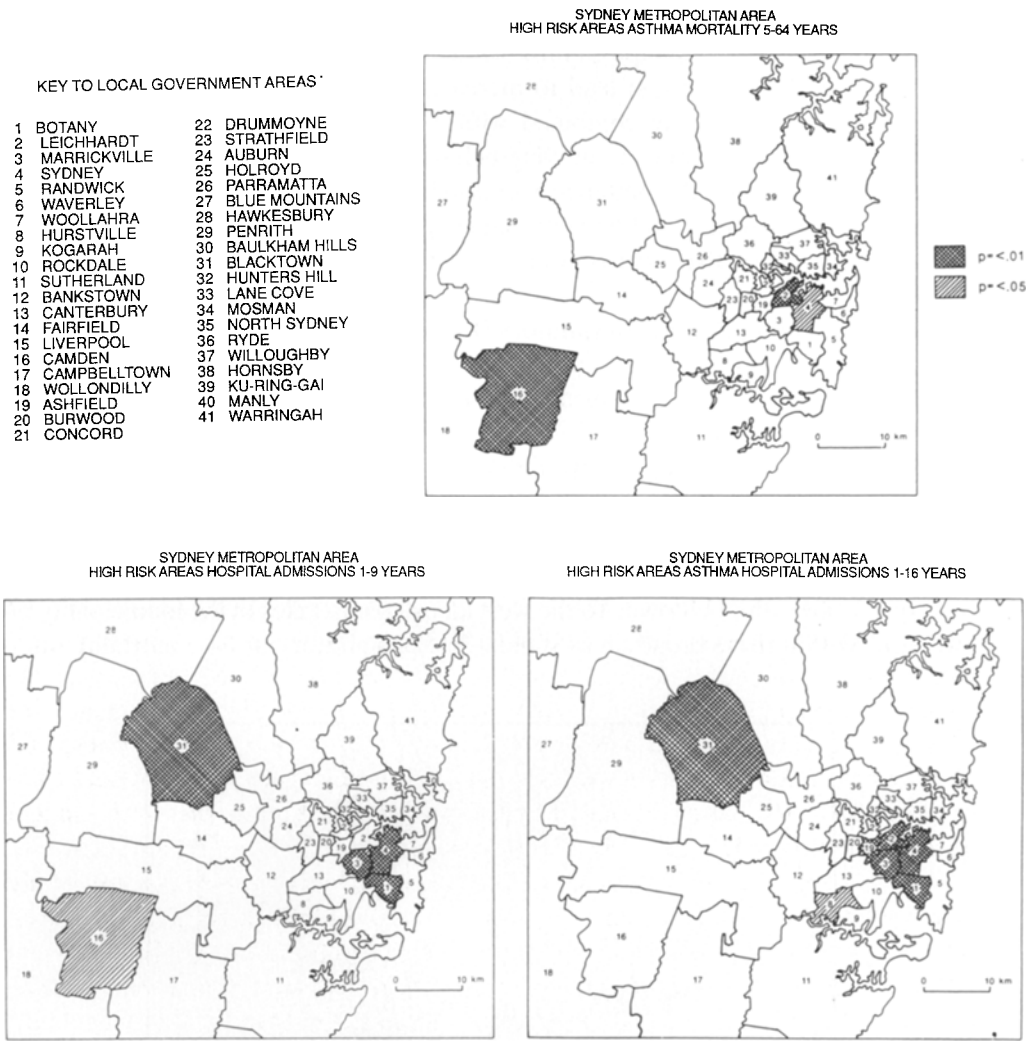

Fig. 2. High risk areas – deaths and hospital admissions from asthma Sydney 1979–88.

are between 18% and 52% higher than the state average with the highest rates occurring in the municipalities of Leichhardt and Sydney City (Table II).

Asthma in Sydney

Asthma is a general term for a heterogeneous chronic respiratory syndrome characterised by periodic attacks of reversible airways obstruction which may or may not be accompanied by wheezing. The disease is variable in frequency of attack, severity and duration and remains a chronic disease of poorly understood aetiology and natural history. Severely incapacitating and sometimes fatal, asthma is

TABLE II: High risk areas respiratory disease mortality, 1981–88

LGA	Standardised mortality rate	Significance level	Confidence interval
Sydney City	138.6	0.000	129–149
Leichhardt	152.6	0.000	141–165
Marrickville	125.5	0.000	115–136
Ashfield	124.1	0.000	111–137
Auburn	125.1	0.000	112–139
Liverpool	127.6	0.000	115–142
Campbelltown	118.8	0.009	105–132

endemic in Australia and possibly affects 15% of young children and 10–12% of adults. The heterogeneous and ubiquitous nature of the disease makes it not surprising that epidemiologic studies have been difficult. Today there still remains an imperfect picture of the disease's distribution in the Australian community despite a growing number of time and place-specific epidemiological studies of incidence and prevalence. Mortality rates from the disease are high and have increased substantially over the last decade. Whether such patterns reflect prevalence is debatable even though there is some evidence that this may be the case in other countries (Woolcock, 1986). Asthma's common occurrence in childhood, its widespread prevalence, chronicity, unknown aetiology and its sensitivity to elements of the physical and biologic environments mean that it is a disease for which climate change holds considerable implications.

There seems to be increasing evidence that asthma is an environmentally acquired disease. While the most striking support for such a proposition comes from studies of populations in developing countries (Godrey, 1975; Anderson, 1978; Van Niekerk *et al.*, 1979; Waite *et al.*, 1980) it would also seem that in countries like Australia and New Zealand the physical and biologic environments play an important role. If this is true then it seems an irony that most of the research into the aetiology of asthma over the last 25 years has concentrated on pathological processes and mechanisms. It is as Burney has remarked, "...as if those wishing to discover the causes of scurvy had concentrated on the pathological processes in the gums of sailors" (Burney, 1988, p. 425).

Since 1981 asthma has killed more than 1200 people in the Sydney Metropolitan Area, approximately 150 deaths per year. At least 60% of these deaths were premature and probably preventable. Today between 350,000 and 500,000 of Sydney's residents may suffer from asthma and although the disease does not kill very many people it is responsible for considerable disability, suffering and human tragedy and often leads to a considerable downgrading in personal life-style and quality of life and a reduced life expectancy. Asthma is also an important cause of hospital admission in New South Wales. It probably remains the most important

reason for hospital admission for young children and in the mid 1980s it was the second most important reason for hospital admission for all males in Australia (Aust. Institute of Health, 1988). In Australia asthma tends to adopt a bimodal distribution during the year with one peak corresponding with Spring and another with Autumn. The Spring peak has usually been attributed to the distribution of pollens and the second to the onset of cold spells.

The Geographical Distribution of Asthma in Sydney

Figure 2 and Table III provide information on the geographical distribution of asthma risk as measured by mortality and childhood hospital admissions. For the purpose of the present analysis, the age range 5–64 years was selected because asthma can be more accurately diagnosed between these ages. With respect to mortality, there are primarily three areas of high risk. Firstly, Sydney City and the adjacent suburbs of Leichhardt and Marrickville. Secondly, the inner western areas of Strathfield and Bankstown, and finally, Camden municipality in the outer west of the urban area. Table III indicates the dimensions of this mortality risk. Camden, for example, has one of the highest asthma mortality rates in the Sydney area with standardised mortality rates between 2.6 and 3.5 times the national average for the selected age groups. For childhood hospital admissions Camden has a rate 29% above the state average. Leichhardt on the other hand is the Sydney local government area with the worst mortality record across a wide range of age groups. Generally mortality rates in this inner municipality were of the order of 2–3 times higher than the national average (Table III). Most people who die from asthma are over 50 years of age. By contrast about 60% of hospital admissions involve children under the age of 16 years. Figure 2 and Table III show the distribution of high risk areas for childhood hospital admissions from asthma. These maps show admission rates to be high not only in the inner parts of Sydney such as Sydney City and Leichhardt but also in the outer western areas of Camden and Blacktown. For young teenagers Blacktown's admission rate is 69% above the state average. In addition Hurstville in the south and Warringah in the north are shown to have high hospital admission rates. It should be noted that hospital admission data are not without their problems. Admissions for asthma are particularly likely to be influenced by social factors. Families of lower socio-economic status may possibly be over-represented in contrast to those of higher status who will tend to make more use of GP services, although in situations of dire emergency this circumstance may not apply. There may also be a distance-decay effect whereby persons far from hospital facilities are forced to cope with or tolerate more asthma attacks than those living closer to hospital facilities.

Finally, there may be other problems of access and utilisation including the fact that the admissions policy of a particular casualty department may to some extent reflect the prejudices of the doctor or doctors on duty.

The reasons for this particular distribution of asthma mortality and hospital

TABLE III: Standardised mortality and hospital admission rates for asthma in high risk local government areas Sydney 1979–86

	Leich-hardt	Marrick-ville	Sydney City	Botany	North Sydney	Banks-town	Holroyd	Ashfield	Hurst-ville	Black-town	Camden
A. Mortality rates (1979–86)											
Total adults 5–64 years	193.1*		166.0+								297.4*
Males 5–64 years	233.5*				217.9+	153.9+					
Total adults 20–64 years	207.9*										258.6+
Total adults 45–64 years	260.0*										352.1+
Males 45–64 years	294.0*	207.0+									
B. Hospital admission rates (1986)											
Children 1–16 years		121.7*	114.9+	129.5*				123.1+		117.8*	129.0+
Children 1–9 years	172.3*	201.0*	198.3*	167.6*				197.8*	129.7+	121.0*	
Children 10–14 years	186.0+			211.5+			152.2+			168.7*	

* significant at < 0.01.
+ significant at < 0.05.
Source of data: NSW Death Records 1979–86 ABS/Reg General; NSW Department of Health.
Note: In the case of asthma mortality the standard population was the Australian population. In the case of hospital admissions the standard population was the NSW population.

admission within Sydney lie in the interplay of a number of biologic, physical and socio-economic factors. While the primary purpose of this paper is to survey the impact of climate on respiratory disease and asthma it should be recognised that in most cases climate does not act independently but synergistically with other elements of the physical, biologic and socio-economic environment. It would also seem that there is a very strong social class factor in the distribution of respiratory disease in Australia. The incidence and mortality for such diseases have been much higher among the more disadvantaged sectors of Australian society. While this has yet to be systematically investigated within the Sydney area, evidence supporting the assertion comes from a study by McMichael who found that males in the lowest socio-economic category had 2.8 times the rate of bronchitis, emphysema and asthma than those in the highest group (McMichael, 1985). Evidence also comes from a study of the epidemiological profile of Western Sydney (Department of Health, 1986). This study while not concerning itself with physical or biologic factors clearly indicates the synergistic relationship that exists between socio-economic, epidemiologic and physical factors. Like inner-Western Sydney, Western Sydney is marked by high levels of socio-economic disadvantage including high levels of unemployment, limited social and family-support services, restricted education opportunities, physical and social isolation of new housing communities and poor public transportation. The area is also a major centre of Sydney's industrial growth. In health terms Western Sydney, like inner parts of the urban area is marked by high levels of premature mortality particularly from heart disease, lung cancer and respiratory diseases. More recently a study of health conditions in Metropolitan Adelaide found a significantly high association between respiratory disease and a variety of indices representing various degrees of disadvantage (Table IV). If this is indeed the case in Sydney it would go some way to explaining the current distribution of high risk asthma areas. In the present study, mortality and admission rates for asthma have only been standardised for age and not for socio-economic status. It is hoped to carry out an analysis which controls for socio-economic status as a follow-up to the present paper.

The Role of Human Behaviour

While there are undoubtedly a wide range of environmental triggers of asthma, the geographical distribution of mortality and acute and life-threatening attacks also reflects human behaviour. While this involves personal health behaviour and the perception of health risk such as smoking and alcohol use it also involves understanding, recognition and management of the disease. It would appear that a large proportion of asthmatics possess a disturbing lack of knowledge about the disease and adopt suboptimal management procedures. A recent study by Bauman *et al.* (1992) has revealed the gap that exists between optimal asthma management techniques and current practice. In families with children with asthma, this study found that only 15% possessed a peak flow meter and only six percent possessed both a

TABLE IV: Significant correlations between respiratory disease and other socio-economic variables, Metropolitan Adelaide, 1982–86

A. Respiratory deaths 15–64 year olds (Local Government Areas)

Variable	Correlation coefficient*
Total unemployed	0.59
15–19 year-olds unemployed	0.53
Dwellings without vehicles	0.58

B. Respiratory diseases hospital inpatients (postcodes)

Variable	Correlation coefficient*
Single parent families	0.50
Unskilled/semi-skilled workers	0.60
Total unemployed	0.54
15–19 year-olds unemployed	0.50
Female work participation	−0.69
Left school age 15 or less	0.67
S.A.H.T. rented dwellings	0.63
General hospital inpatient separations	
General hospital inpatient females	0.80
General hospital inpatient males	0.81
Domiciliary care clients	0.65

Source: South Australian Health Commission, 1990.
* No levels of significance are given in the report.

meter and a coordinated action plan. An earlier study (Rubinfeld *et al.*, 1988) found misconceptions about medication use, crisis recognition and management to be widespread. This manifested itself in the under-assessment of severity, over reliance on bronchodilators and delays in seeking help. There would appear to be a strong social class factor in family/patient comprehension and management of asthma. Rubinfeld found that those possessing the highest levels of education who did not smoke and who had at some time sought both specialist medical advice and educational material were the best prepared to confront the disease (Rubinfeld *et al.*, 1988).

Climate and Asthma

The effects of weather and climate on asthma are well documented (see for example, Tromp, 1968, 1980; Derrik, 1965, 1972). Some studies attribute a direct causative effect to certain kinds of weather, others regard the influence of weather/climate as having an indirect effect working via a chain of interlinkages on the production and dispersal of a variety of airborne irritants and allergens. Whatever the causal path, a wide variety of physical factors such as climate change, air pollution, aeroallergens such as pollens, moulds, fungal spores and dust mites have been recorded as inducing asthma attacks.

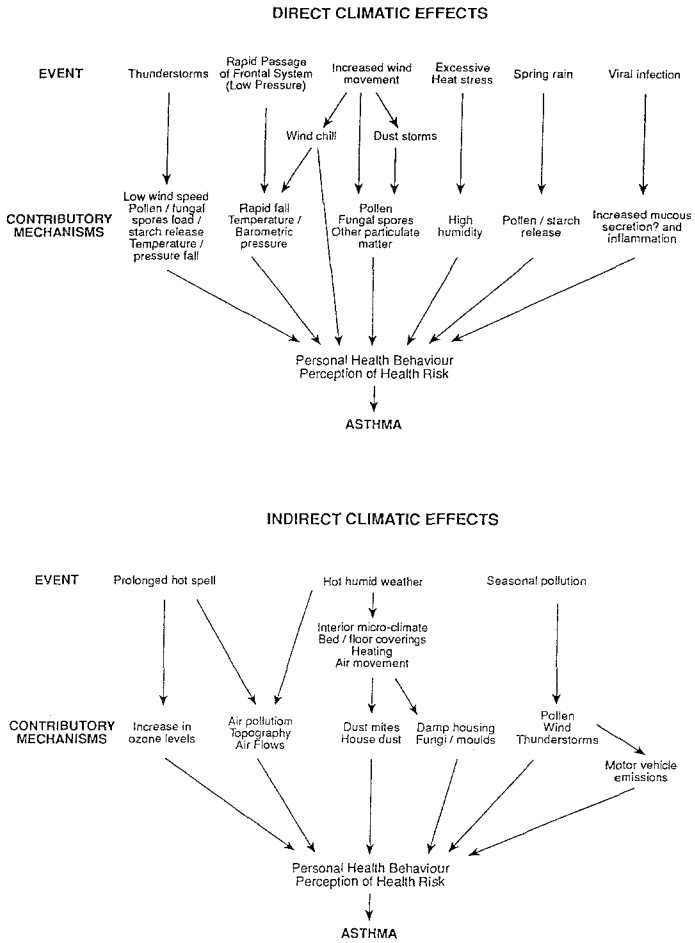

Fig. 3. Direct and indirect climatic effects on asthma.

Direct Effects of Climate

Figure 3 indicates some of the direct effects of climate on the distribution of asthma. Even here, however, it often requires a series of contributory mechanisms including elements of personal health behaviour to interact with the actual climatic event before an asthma outcome is achieved. In the case of climatic variables, rain water has been shown to degranulate rye grass pollen, releasing large quantities of the allergen Lol pIX which has been shown to provoke asthma attacks in susceptible individuals (Suphioglu *et al.*, 1992). Thunderstorms can also induce asthma attacks sometimes producing dramatic short-term epidemics. Such circumstances are not unusual in Australia such as the thunderstorm in Melbourne on 11th November 1984 (Egan, 1985) or the storm in Tamworth on the 1st of November 1990 (Harris and Lewis, 1990). Both events produced an unprecedented number

of people seeking hospital treatment for acute asthma attacks. In both cases, patients' symptoms began at the time of sudden weather changes associated with the thunderstorm. During such storms a unique combination of meteorological events probably interact to increase bronchial sensitivity to which an increase in the pollen/fungal spore load of the air leads to an epidemic of asthma attacks. It may also be that the violent down-draught associated with thunderstorms may bring down to ground level some of the naturally occurring sulphuric acid aerosol that exists at high altitude and this substance may trigger asthma attacks. Whatever the cause, asthmatics have been shown to possess an impaired thermoregulatory mechanism. They possess an inability to adjust to rapid changes in temperature, especially cold changes, which makes them particularly susceptible to the effects of short-term variations in atmospheric temperature. As Tromp has pointed out, such situations are often characterised by great speed of frontal movement, a rapid change in barometric pressure and temperature after the passage of the front, strong precipitation (rain, snow or hail) and considerable disturbances in the electric field of the atmosphere. Such conditions would seem to be a major initiator/provoker of asthma attacks in Western Europe (Tromp, 1980). Atmospheric cooling associated with the passage of a cold front has long been implicated as a major cause of asthma attacks. (Tromp, 1980). Increased wind movement can also play an important role in provoking an attack of asthma. Wind chill can exaggerate the drop in temperature associated with cold fronts and wind acts as an important transportation mechanism of pollens, spores and air pollution.

Although controversial, ionisation has also been shown in some studies to affect the distribution of asthma (Krueger, 1973; Krueger and Reed, 1976). Thunderstorms, winds and a variety of human-induced dielectric fields and airconditioning all influence ion concentrations and balance. Experimental studies indicate that concentrations of small positively-charged ions can produce episodes of asthma and that negative ions have the opposite effect.

Finally, in some cases excessive heat stress often accompanied by high humidity may also induce asthma attacks.

Indirect Effects

On many occasions climate variables interact with other physical, biologic and human factors resulting in what are often marked effects on the incidence of asthma attacks (Figure 3). Asthmatics' respiratory sensitivity renders them especially sensitive to episodes of atmospheric pollution. Oxidant gases such as ozone and sulphur and nitrogen dioxode as well as heavy loads of particulate matter increase airways resistance and have an irritant effect on mucous membranes. These elements are common byproducts of industrial, domestic and motor vehicle activities in cities like Sydney. Under particular climate scenarios these elements can become chemically altered and concentrated. Both prolonged hot spells and hot, humid and still weather can help increase their potency. Temperature and humidity also

impact on asthma in another important way. Many asthmatics are atopic, i.e. they demonstrate some degree of allergic status. Plants and animals provide the greatest store of allergens that commonly affect asthmatics. Pollens, fungal/mould spores and house dust mites are probably the three most important allergen groups as far as asthmatics are concerned. All are intimately affected by temperature, humidity and rainfall. Release of pollen is primarily a spring-early summer phenomenon and generally the warmer and drier the spring/early summer, the worse the asthma. Grass pollens are mainly disseminated by air currents or insects and while the majority of pollen is distributed within a kilometre of its source, some is carried long distances by air currents. The volume of grass pollens in the atmosphere is dependent upon air temperature, rainfall, air turbulence and time of day. Mould and fungi need warm, moist, damp conditions for growth and spore release. They are most active in areas of high humidity and temperature and are particularly concentrated in damp houses.

In coastal Australia particularly on the eastern seaboard, one of the most important allergens for asthma is the house dust mite (*Dermatophagoides pteronyssinus*). Dust mites thrive in warm humid conditions and are particularly prevalent in carpets and mattresses. Their life cycle is very closely tied to changes in temperature and humidity although the microclimate of many houses may have helped modify an otherwise seasonal distribution. Dust mites are extremely allergenic and their faecal pellets produce an intense reaction in atopic subjects.

Asthma and Air Pollution in Sydney

The harmful effects of air pollution on chronic respiratory disease sufferers is well documented in both experimental and epidemiologic studies. The pollutants with the greatest importance for respiratory health are sulphur dioxide, nitrogen dioxide, ozone, and suspended particulate matter. All these agents are known to heighten bronchial sensitivity and impair lung function in asthmatics often at very low levels of concentration. Air pollution acts synergistically with other physical and socio-economic factors such as where sulphur dioxide combines with cold dry air, where high temperatures and sunlight act upon hydrocarbons to produce high levels of photochemical smog or where pollens combine with diesel emissions to produce a much more toxic trigger for the disease.

Living and/or working in a polluted environment and being regularly exposed to high levels of air pollution has been consistently found to produce adverse respiratory effects. In general, there would appear to be more frequent asthma attacks in such areas (Berciano *et al.*, 1989), although it is possible that this association is confounded by the effects of smoking, damp housing, pollens, fungal spores and dust mite exposure. For large urban areas like Sydney the most intractable form of air pollution remains photochemical smog. The nature of air pollution in the Sydney metropolitan area owes much to a complex set of relationships involving the topography and meteorology of the Sydney Basin, the quantity of pollutants emit-

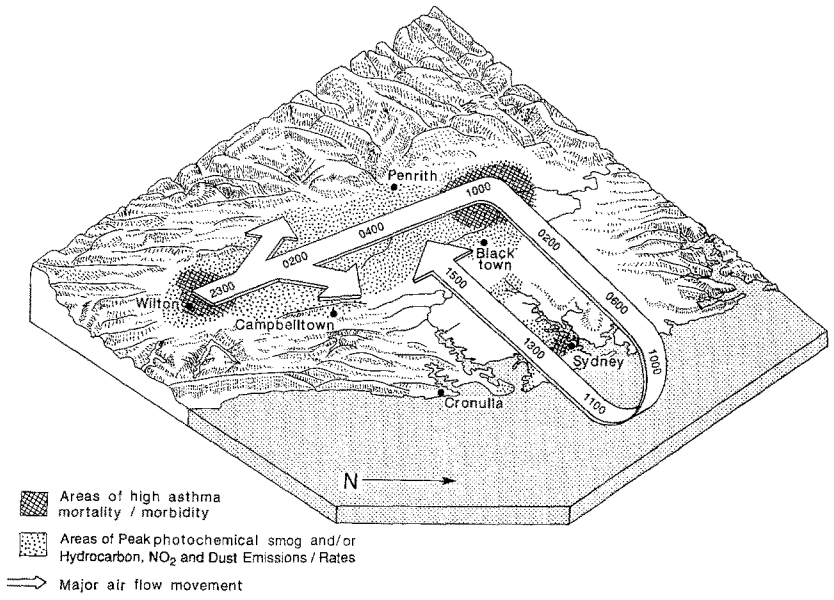

Fig. 4. Sydney's air circulation and the location of areas of peak pollution and asthma.

ted to the air by industry, motor vehicles and domestic activities and the temporal and spatial distribution of such emissions. It would also appear that people who live near motor-ways and major roads may be more at risk from respiratory disease than those living further afield (Hawkes, 1990). In particular the pattern of air flows, particularly sea breezes and drainage flows serves as an important mechanism for the transport and recirculation of polluted air within the urban area (Figure 4). This peculiar pattern of air circulation allied to local emissions can give rise to high pollution episodes especially in so far as air from the central and inner parts of Sydney is directed to and often entrapped within the Hawkesbury basin for lengthy periods. During the early morning this pollution is gathered up by the air flow as it moves down to the Parramatta River valley to the coast. Such effects are also exacerbated by the fact that temperature and sunlight intensities are greater in the western part of Sydney than in other parts of the urban area causing a faster and more concentrated production of photochemical smog. By mid-day this easterly air flow is reversed westwards by the seabreeze taking up on its journey a variety of inner city pollutants en route to the Hawkesbury basin where it arrives in late afternoon. From midnight another drainage flow transports pollutants from the southern end of the Hawkesbury basin northwards to Blacktown with an arm deviating towards Campbelltown and one towards Penrith, Richmond and Windsor. One upshot of this pattern of air circulation has been to displace Sydney's main concentration of ozone pollution into the Camden/Campbelltown area. Not only is there some evidence of unacceptably high pollution levels already in this

area but also in the absence of further pollution controls urban growth in this area over the next two decades will give rise to increases of up to 50% in ozone concentrations (Hyde and Johnson, 1990). Within Sydney most areas of high asthma mortality lie on this trajectory of air movement as do the areas of highest hospital admissions for asthma. The distribution of male deaths from lung cancer also closely corresponds with the inner city and outer western sections of this circulatory system. While cigarette smoking is strongly implicated as a causal factor in lung cancer it seems unlikely that intra-metropolitan variations in lung cancer mortality can be totally explained by variations in the geographical distribution of cigarette smoking. Other environmental influences must play a part both as initiators and promotors of the disease (Curson, 1992). The implications of such a pattern for respiratory morbidity and mortality in the future would appear profound.

Indoor Air Pollution and Asthma

Indoor air pollution in Sydney also presents a considerable threat to respiratory health. The most threatening indoor pollutant especially for asthmatics remains tobacco smoke. It is possible that exposure of a newborn infant or indeed older children to tobacco smoke within the home may trigger bronchial sensitivity which remains for life (Samet *et al.*, 1987, p. 1492). Nitrogen dioxide is also an indoor pollutant of some importance for asthmatics. It is probably present in significant concentrations in many Sydney homes and arises mainly from the use of unflued gas heaters as well as from the use of gas stoves. In addition, there may be a significant problem with nitrogen dioxide from unvented gas heaters in many New South Wales schools (Ferrari *et al.*, 1989). During cooking with a gas stove U.S. studies suggest that peak levels of nitrogen dioxide may reach 200–400 ppb in the kitchen area (Spengler and Sexton, 1983). This is approximately 4 to 10 times the average annual level set by the U.S. Federal Authorities. The nature of domestic heating and its use is not unrelated to the distribution and life-cycle of various biologic triggers for asthma such as dust mites, fungi and moulds. People who live in damp, mouldy homes are more likely to have asthma attacks than those whose homes are dry even despite the fact that awareness of dampness and mould may make people more likely to report respiratory symptoms. Children in particular in such homes have a greater prevalence of respiratory symptoms (Platt *et al.*, 1989). The prevalence of damp housing in Sydney is currently unknown. If New Zealand and Melbourne surveys are any guide, perhaps 30–40 percent of Sydney's homes suffer from some degree of dampness with related mould problems. Damp housing is to some extent a problem associated with older homes where the damp course has deteriorated. Even in new homes, however, dampness can be a problem partly because of condensation and partly because of current forms of heating and ventilation. Today fewer homes in Sydney have someone at home during the day to keep windows open and air circulating. Frequent use of electric clothes driers also

propels damp air into living spaces. In a wet year such as in 1989 or 1990 many Sydney homes would support mould growth.

The Impact of Climate Change on the Distribution of Asthma in Sydney

Under greenhouse conditions Sydney is likely to be a warmer, wetter and less windy environment. Such climate changes will have profound effects on the prevalence of respiratory disease and in particular asthma. In the first place such conditions will greatly influence the life cycle of a wide range of plants and animals that presently influence the distribution of asthma attacks. A warmer more humid climate with milder winters will impact subtantially upon pollens and dust mite populations perhaps even to the extent of modifying the existing seasonal nature of such allergens. A warmer, wetter climate will also have considerable influence on the prevalence of damp, mouldy housing in Sydney with substantial implications for respiratory and general health.

In the absence of any major controls on airborne emissions warmer temperatures and less wind will accelerate photochemical reactions among chemical pollutants in Sydney's atmosphere. There is already evidence that the production of photochemical smog increases markedly with increasing temperature (Johnson, 1984). In this context it is significant that summer day-time temperatures are at least 5 °C hotter in the Hawkesbury basin than elsewhere in Sydney. This temperature effect means that hydrocarbons will produce smog in this area at a rate approximately 25% faster than the same concentrations in the Parramatta River basin or Liverpool basin (Hyde and Johnson, 1990, p. 66). Given the nature of Sydney's air circulation, a temperature rise of 2–4 °C in the annual mean and generally warmer summer conditions with more frequent heat waves it seems highly likely that this western part of Sydney will, failing intervention, experience much higher levels of smog and ozone and by consequence much higher levels of asthma and respiratory disease.

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