

ENVIRONMENT PROTECTION & HERITAGE COUNCIL

Co-operative Studies on Priority Air Quality and Health Related Issues

Asthma Research – A Background Paper

Introduction

The relationship between respiratory illnesses and air pollution has been known for some time (eg Pope, 1989; Pope et al, 1991; Schwartz et al, 1993). Several multicentre studies in the United States (eg National Morbidity, Mortality and Air Pollution Study (NMMAPS)) and in Europe (eg Air Pollution and Health: A European Approach (APHEA)) have examined the effects of air pollutants such as carbon monoxide (CO), nitrogen dioxide (NO₂), ozone (O₃), sulfur dioxide (SO₂) and respirable particulate matter (PM₁₀, PM_{2.5}) on hospital admissions and mortality (Samet et al, 2000; Atkinson et al, 2001). Strong evidence for an association between PM₁₀ levels and hospital admissions for chronic obstructive pulmonary disease (COPD) and asthma was found by both the NMMAPS and APHEA studies.

In Australia, similar results have been found in recent studies of Brisbane, Sydney, Melbourne, and Perth (Petroeschevsky et al, 2001; Morgan et al, 1998; Environmental Protection Authority (Vic), 2001; Department of Environmental Protection (WA), in press). In these studies, significant associations were observed for daily changes in particles and O₃ concentrations and hospitalisations for asthma, COPD, pneumonia and respiratory disease, particularly for children and the elderly.

Asthma and Air Pollution

Respiratory diseases such as bronchial asthma are increasing worldwide. The prevalence of such diseases is greatest in industrialised countries, particularly in urban areas where motor vehicle emissions are the greatest. Although particulate matter and SO₂ have been steadily declining in the Northern Hemisphere, fine/ultrafine particles arising from combustion have increased (Morawska, 2001; Pitz et al, 2001). Such particles remain longer in the air than coarser grade particles and often carry toxins.

Human and animal exposure studies and laboratory-based studies have demonstrated that diesel particles, O₃ and NO₂ induce an inflammatory response, thereby contributing to allergies (Salvi, 2001; Barck et al, 2002; Vagaggini et al, 2002). Air pollutants may not only increase the frequency and intensity of symptoms in allergic patients but may sensitise airways to airborne allergens in predisposed individuals (Bucchieri et al, 2002). One proposed mechanism is for air pollutants to adhere to the surface allergenic pollen grains, thereby changing the morphology of these grains, and inducing airway inflammation (Glikson et al, 1995; D'Amato, 2002). There is evidence for both these

impacts in some Australian studies, especially in Brisbane where fungi are also prominent aeroallergens (Rutherford et al, 2000; Glikson et al, 1995).

Associations have been observed between airborne particle exposure (PM_{10} , $PM_{2.5}$) and restricted activity days and exacerbations of asthma and COPD, while other clinical evidence includes associations with decreases in lung function, increased use of asthma medication, decreased heart rate variability, and the triggering of myocardial infarction (Buckeridge et al, 2002; von Klot et al, 2002; Leikin et al, 2000; Peters et al, 2001).

A recent study of patients suffering from severe asthma found significant associations between PM_{10} and O_3 and asthma attacks (Desqueyroux et al, 2002). In cases of severe asthma, NO_x and O_3 may even cause death. A recent study found that NO_x increased the risk of death in patients with more than one emergency room admission for asthma (Sunyer et al, 2002). In this study, O_3 was also found to increase the risk of death in asthmatic patients during spring and summer. No interactions between air pollutants and pollen or spores were found and there was no significant association between mortality and particles, spores or pollen.

Airborne particulates from biomass burning such as bushfires and controlled burns may also exacerbate asthma. A study of the 1997 haze disaster in Indonesia found that the high concentrations of CO, PM_{10} and polycyclic aromatic hydrocarbons (PAHs) particularly affected individuals with a history of asthma (Kunii et al, 2002). Similarly, studies in the USA and Australia have reported increased respiratory symptoms and asthma hospital admissions as a result of exposure to bushfire smoke (Mott et al, 2002; Johnston et al, 2002). Additional epidemiological research is needed so as to disentangle the effects of air toxics (eg PAHs) from monitored criteria air pollutants (Donaldson et al, 2000; Delfino, 2002).

Childhood Asthma and Air Pollution

Children have relatively greater exposure to air pollutants than adults. Children differ from adults in their activities, their rate of breathing, their lung anatomy and physiology, and their organ maturity. The air pollutants that are of special concern for children include airborne particles, CO, O_3 , NO_x , SO_x , and acid aerosols (Mathieu-Nolf, 2002).

Multicentre studies such as the Central European Study of Air Pollution and Respiratory Health (CESAR) and the Southern California Children's Health Study (CHS) have found increased prevalence of cough and wheeze symptoms (Leonardi et al, 2002; Gauderman et al, 2002). In the Southern California Children's Health Study, these symptoms were associated with NO_2 and acid aerosols, while reduced lung function and deficits in lung function growth rate were found to be associated with $PM_{2.5}$, acid aerosol, and elemental carbon (Gauderman et al, 2002). Recent studies in France and the USA have shown that current ambient concentrations of O_3 , SO_2 , NO_2 and particulate

pollution, although within international air quality standards, have adverse short-term health effects on children with mild-to-moderate asthma (Just et al, 2002; Mortimer et al, 2002). Black smoke and NO₂ were associated with increases in nocturnal cough and respiratory infections in asthmatic children, while O₃ was associated with an increase in asthma attacks and changes in lung function as well as increased bronchodilator use (Just et al, 2002).

Ozone has been shown to affect the lungs of otherwise healthy school children, particularly those who are asthmatic (Lewis et al, 1998; Timonen et al, 2002). Studies in Southern California (Gauderman et al, 2001) have reported a noticeable increase in school absenteeism related to upper and lower respiratory illness which has been associated with relatively small short term changes in levels of ozone. A recent study by Peters et al (1999) found an association between levels of O₃ exposure and decreased pulmonary function in school children and adolescents.

There is a higher prevalence of respiratory symptoms among children living near motorways or freeways, and also a tendency for chronic cough, wheeze, asthma attacks and rhinitis to be more prevalent in areas with higher truck traffic density (Oosterlee et al, 1996; van Vliet et al, 1997; van Der See et al, 1999; Venn et al, 2001; Lin et al, 2002). Other studies have also found a strong association between decreased lung function of children living near motorways and increased air pollution levels from truck and motor vehicle traffic (Brunekreef et al, 1997; Nakai et al, 1999). Findings from the international collaborative study on the impact of Traffic-Related Air Pollution on Childhood Asthma (TRAPCA) confirmed the association between traffic-related air pollution and cough in children under two years of age (Gehring et al, 2002). Exposure to PM₁₀ from diesel emissions has been associated with Sudden Infant Death Syndrome and with mortality as a result of respiratory causes (Woodruff et al, 1997).

Outdoor exercise and air pollution have been shown to contribute to the development of asthma in children (Gauderman et al, 2002; McConnell et al, 2002). A recent study found that the relative risk of developing childhood asthma was greater in children who played three or more sports in communities with high ozone concentrations (McConnell et al, 2002). The incidence of asthma in children has also been associated with traffic-related air pollutants such as NO₂ and PM_{2.5}, particularly for children under one year of age (Brauer et al, 2002; Gauvin et al, 2002; Zmirou et al, 2002).

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