

Air pollution and health

MNW Chan-Yeung

Objective. To review the effects of air pollution on health, with special reference to data obtained locally in Hong Kong.

Data sources. *Medline* literature search (1960-1999), websites of the World Health Organization and Environmental Protection Department, Hong Kong, and a report from the Sub-Working Group on the Review of Hong Kong's Air Quality Objectives, 1999.

Study selection. Key words for the literature search were 'air pollution' and 'health'.

Data extraction. The author reviewed relevant information and data.

Data synthesis. In many parts of the world and in Hong Kong, air pollution is associated with increased mortality and morbidity rates, as reflected by increased hospital admissions and doctor visits, not only for respiratory illnesses but also for cardiovascular illnesses. Some studies have found that air pollution is also associated with an increase in respiratory symptoms in lower lung function in children. In Hong Kong, air pollution is mostly because of traffic emissions. During the past decade, the annual mean levels of respirable suspended particulates and oxides of nitrogen and ozone have increased and exceeded the air quality objectives set by the Environmental Protection Department of Hong Kong.

Conclusion. The medical profession should be concerned about the health effects of air pollution and should call for action to reduce the current levels of air pollution to an acceptable level.

HKMJ 2000;6:390-8

Key words: Air pollution; Environmental exposure; Health status; Lung diseases/epidemiology; Risk factors

Introduction

Air pollution is a major environmental health problem worldwide. The World Health Organization (WHO) considers that air pollution is damaging the resources that are needed for the long-term sustainable development of the planet.¹ The sources of air pollution fall into three broad categories:

- (1) Mobile sources, which include combustion-engine vehicles such as gasoline-powered cars, diesel-powered vehicles, motorcycles, and aircraft;
- (2) Stationary sources, which include rural sources such as agricultural production, mining, and quarrying; industrial sources such as manufacturing; and community sources such as the heating of homes and buildings, municipal waste, and incinerators; and
- (3) Indoor sources, which include combustion, tobacco smoking, and biological sources; and emissions from indoor materials or substances such as volatile organic compounds, asbestos, and radon.

Specific air pollutants

Air pollutants are usually classified into suspended particulate matter (dusts, fumes, mists, and smokes), gaseous pollutants (gases and vapours), and odours.

Suspended particulate matter

Suspended particulate matter (PM) consists of finely divided small particulates with diameters of less than 10 µm (PM₁₀). Suspended particulate matter comprises a wide variety of substances, which include inorganic and organic carbon (containing polycyclic aromatic hydrocarbons), acidic or neutral sulphates and nitrates, fine soil dust, residues of lead and other metals, asbestos, and other fibres. Exposure of laboratory animals to fine particles has been shown to lead to inflammation of the airways and lungs.² Most of these particulates are smaller than 1 µm and remain suspended for hours or days. Particles that are smaller than 2.5 µm in diameter (PM_{2.5}) arise mainly from combustion processes, whereas larger particles are generated by grinding and other mechanical or agricultural processes. Small particles efficiently penetrate indoors, where levels are typically 70% to 80% of outdoor levels in the absence of indoor sources. In locations

Department of Medicine, The University of Hong Kong, Queen Mary Hospital, Hong Kong

MNW Chan-Yeung, FRCP (Edin), FHKAM (Medicine)

Correspondence to: Dr MNW Chan-Yeung

with indoor sources (eg cooking or tobacco smoke), indoor levels may be much higher than those outdoors.

Acid aerosols are a subset of fine particles. Atmospheric oxidation of sulphur dioxide (SO₂) may produce sulphuric acid and partially neutralised sulphate salts. The formation of acid aerosols is hastened by humidity and photochemical processes. When individuals with asthma are exposed to acid aerosols, bronchoconstriction is more likely to develop.³⁻⁵

Sulphur dioxide

Sulphur dioxide is released into the atmosphere primarily as a result of the industrial combustion of coal and oil. A small proportion is produced by vehicular sources due to sulphur contained in the fuel. Sulphur dioxide is oxidized to sulphuric acid in a humid environment. Indoor levels are typically lower than outdoor levels, owing to the reactivity of SO₂ with indoor surfaces. It is an irritant gas that elicits bronchoconstriction; individuals with asthma are more sensitive to this effect.⁶

Oxides of nitrogen

Oxides of nitrogen are most frequently produced by the combustion of fossil fuels. Nitric oxide (NO) may be oxidised to nitrogen dioxide (NO₂), the precursor of ozone in photochemical smog. In addition to NO₂ itself, potentially harmful NO oxidation products include nitric and nitrous acid. In indoor environments without combustion sources, indoor levels of NO₂ are lower than levels outdoors. Homes with unvented gas combustion devices, such as gas stoves, generally have higher NO₂ levels than outdoors during cooking. In experimental exposure studies, no consistent effects on lung function have been documented for NO₂. However, exposure of asthmatic individuals to high levels of NO₂ (0.2-0.5 ppm) has been shown to increase non-specific airway hyperresponsiveness⁷ and an enhanced specific airway response to inhaled allergens such as house dust mite allergens.⁸⁻⁹

Photo-oxidants

Photo-oxidants are produced by photochemical reactions in air containing oxides of nitrogen and reactive hydrocarbons. Ozone (O₃) is the most important photo-oxidant. Photochemical pollution causes eye irritation and small temporary changes in lung function, particularly among children or people exercising vigorously. Because O₃ is highly reactive, indoor concentrations are significantly lower than outdoor levels. In both healthy subjects and asthmatic individuals, exposure to O₃ causes a reproducible decrease in lung function and an increase in non-specific airway hyperresponsiveness.³ The dose-response curve may be non-linear

and there is no threshold effect. Exposure of people with asthma to ozone has been shown to increase the specific response to inhaled allergens.^{10,11} Ozone exposure causes inflammation of the nasal mucosa and bronchoalveolar lining, thereby resulting in a reduction in lung function, which is worse in those with asthma.¹²

Volatile organic compounds

Examples of volatile organic compounds (VOCs) are alkanes, alkenes, alkynes, aromatics, aldehydes, ketones, alcohols, esters, benzene, and some chlorinated hydrocarbons. The major sources of VOCs come from the burning of fossil fuels and industrial processes involving solvents. Benzene is emitted from motor vehicle exhausts and/or from evaporated petrol.¹³ Indoor levels of VOC are usually much higher than outdoor levels, because VOCs are present in many building materials, such as paints, adhesives, and sealants. Volatile organic compounds are also released from laser printers and photocopiers. In the indoor environment, exposure to low levels of VOC may result in headache and irritation of the eyes and nose. Some VOCs have been shown to be carcinogenic.⁶

Carbon monoxide

Carbon monoxide (CO) is produced by the incomplete combustion of fossil fuels. Concentrations in urban areas depend on traffic density, topography, and weather conditions. In the absence of indoor combustion devices, indoor levels may be close to outdoor levels. Unvented combustion devices may produce additional CO indoors. The health hazards of CO exposure are related to the binding of this gas to haemoglobin. An increase in carboxyhaemoglobin of 3.6% over baseline levels reduces the time to angina and leads to electrocardiographic changes in exercising men with coronary artery disease.¹⁴

Health effects of air pollution on humans

In a recent American Thoracic Society statement on the adverse health effects of air pollution,¹⁵ the following were considered adverse health effects:

- (1) Any detectable effects on clinical outcomes such as visits to the emergency department, hospital admissions, and mortality;
- (2) Symptoms related to air pollution associated with diminished quality of life or with a change in clinical status;
- (3) Any permanent loss of lung function;
- (4) All reversible loss of lung function in combination with the presence of respiratory symptoms; and
- (5) Decreased health-related quality of life.

Changes in levels of biomarkers and a transient, small loss of lung function that is by itself related to air pollution were not considered to be adverse health effects.¹⁵ The evidence for some of the adverse health effects of air pollution is briefly discussed below.

Mortality

Sudden large increases in mortality due to episodes of extreme air pollution have been described in the Meuse Valley in Belgium,¹⁶ Pennsylvania, United States (US),¹⁷ and in London, England.¹⁸ Recent studies have shown that mortality is associated with much lower levels of air pollution.¹⁹⁻²¹ In a six-city study in the US, a significantly increased mortality rate ratio of 1.26 was found in the most polluted city, as compared with the least polluted city.¹⁹ The relationship was stronger for fine particles than for other air pollutants. A study that examined mortality in London during the winters of 1958 to 1972, identified a significant relationship between mortality and levels of particulate matter, SO₂, and black smoke.²⁰ A multicentre epidemiological study in Europe²¹ was performed to evaluate short-term effects of air pollution on health using time-series analysis. Similar to US studies, an association was found between mortality and daily concentrations of particles, SO₂, and NO₂, with a time lag of 0 to 5 days.

Associations between air pollution levels and daily mortality counts have been interpreted by some researchers as being due to the effects of air pollution on frail individuals with severe underlying heart or lung disease—the so-called ‘harvesting effect’.²² Some mortality time-series studies, however, have found effects across all ages, and not just among the very young and the very old.^{15,22} Two cohort studies from the US have suggested that life expectancy may be 2 to 3 years shorter in communities with high levels of particulate matter than in communities with low

levels.¹⁵ The time-series studies also seem to indicate that combustion-related fine particles present a greater risk than naturally occurring particles such as dust storms, volcanic emissions, and road dust.¹

It has been shown that the level of PM₁₀, O₃, and sulphate particles are independently associated with hospital admissions for asthma.^{23,24} The relationship between the PM₁₀ level and hospital admissions for all respiratory diagnoses or asthma is a linear one (Table 1).²⁴⁻³² Studies of visits to emergency departments for respiratory diseases in the US^{33,34} and Spain^{35,36} have also demonstrated similar findings. The effects of air pollution on health care utilisation may not occur on the same day and can be delayed up to 5 days. A marked reduction in PM₁₀ concentration in regions in the US has been associated with a 50% drop in hospital admissions of children for respiratory disease.^{37,38} The estimated decrease was 7.1% in all respiratory admissions, with a 10 µg/m³ decrease in PM₁₀ levels. This observation gives an indication of the public health and economic benefits of reducing particulate air pollution.

Impairment of lung function

Long-term exposure to O₃ has been found to be associated with a lower level of lung function and a faster rate of decline in lung function.²⁶ Furthermore, the combination of O₃ and acid sulphate may be more important than the effects of O₃ alone. In Germany, children aged 9 to 11 years living in areas with the greatest amount of urban traffic had significantly poorer lung function than those living in areas with less traffic.²⁷

Asthma and allergies

Air pollution is known to be associated with acute asthma exacerbation.^{26,27,33-35} The relationship is strongest with particles and O₃; the higher the pollution, the higher the number of asthma patients with acute

Table 1. Percentage increase in adverse health effect with each 10 µg/m³ increase in PM₁₀

Health effect	References	Increase (%)	Hong Kong data ²⁵ (% increase)
Respiratory symptoms			
cough	24	1.2	-
lower respiratory tract symptoms	24	3.0	-
upper respiratory tract symptoms	24	0.7	-
Asthma attack	24	3.0	-
Bronchodilator use	24	2.9	-
Health care utilisation			
all respiratory disease admissions	24	0.8	1.6
admissions for asthma	24	1.9	1.5
emergency department visits for asthma	26-29	1.0	-
admissions for cardiovascular disease	30	-	0.6
Mortality			
total mortality	31	1.0	-
respiratory mortality	31	3.4	-
cardiovascular mortality	31	1.4	-

exacerbation. There has been a general increase throughout the industrialised world in the prevalence of asthma and allergies in children.²⁸ The reason for this increase is not known and there is no clear evidence that air pollution is causally related. However, there are now several experimental studies showing that diesel particles, SO₂, and O₃ can act as adjuvants, which enhance the production of immunoglobulin E antibodies and possibly increase the prevalence of atopic sensitisation and asthma.^{29,39,40}

Cancer

Known or suspected carcinogens, such as benzene and other polycyclic aromatic hydrocarbons, are detectable in vehicle emissions. Attempts have been made to quantify the cancer risk from vehicle emissions. In Austria, a level of diesel particles of 5 to 23 µg/m³ in the atmosphere has been estimated to yield 1 to 2.6 additional cases of lung cancer per 100 000 persons per year.⁴¹ Other studies have shown a significantly increased mortality risk ratio for lung cancer of 1.36 (95% confidence interval, 1.11-1.66) for an increase of approximately 20 µg/m³ sulphate particles.^{37,38,42} It should be pointed out that quantitative assessment of risks from epidemiological data alone is difficult. Even well-performed quantitative risk assessment is based on assumptions. Furthermore, there are multiple confounding factors, such as individual susceptibility to exposure and exposure to cigarette smoke. Nevertheless, the fact that these studies have demonstrated a relationship between air pollution levels and cancer mortality provides grounds for concern that air pollution may increase the risks of lung cancer.

Respiratory symptoms

A greater frequency of wheezing has been observed among children aged 9 to 11 years living in areas with the highest flow of urban traffic in Munich, Germany.²⁷ Another study in Germany has found a significantly higher prevalence of asthma-like symptoms and allergic rhinitis in children aged 12 to 15 years living near busy roads, especially roads with a high density of trucks.⁴³ Studies from the US,^{31,44} Netherlands,^{45,46} and Switzerland⁴⁷ have demonstrated an increase in upper (runny nose, sore throat, head cold, and sinusitis) and lower (wheezing, dry cough, phlegm, and shortness of breath) respiratory tract symptoms with increased air pollution.

Health studies in Hong Kong

There have been several health studies on air pollution and health performed in Hong Kong since 1989. As these studies have been reviewed thoroughly by the

Sub-Working Group on the Review of Hong Kong's Air Quality Objectives,⁴⁸ the results will be described only in brief below.

A 1989 study⁴⁹ compared respiratory morbidity among school children living in a heavily polluted district with those living in a less polluted area. These children were similar in terms of age, gender, socio-economic status, atopic status, and parental smoking. The prevalence of sore throat, cough, and wheeze was found to be significantly higher among children living in the more polluted district. When legislation was implemented to reduce fuel sulphur levels in 1990, there was a significant reduction in SO₂ and sulphate concentrations in respirable particles. This reduction in pollution was associated with a significant reduction of respiratory symptoms, especially among those living in the more polluted district.⁵⁰ Moreover, children living in the more polluted district had a significantly higher prevalence of non-specific airway hyperresponsiveness compared with those living in the less polluted district.⁵¹ The degree of airway hyperresponsiveness decreased in children living in the polluted areas after the introduction of the fuel legislation in 1990.⁵² A study of hospital admissions for all respiratory diseases, all cardiovascular diseases, chronic obstructive pulmonary disease, and heart failure in 1994 to 1995 in Hong Kong showed a significant association with concentrations of all four pollutants, with a time lag of 0 to 5 days.²⁵ Thus, the findings of local studies of the health effects from air pollution agree with those in other parts of the world.

Magnitude of adverse health effects

Table 1 shows the percentage increase in adverse health effects with each 10 µg/m³ increase in the PM₁₀ level. The magnitude of the increase in health care utilisation in Hong Kong for each increment in the PM₁₀ level is similar to those reported in other countries. The increase in the PM₁₀ level can be 100 µg/m³ above the usual level during a bad episode of air pollution. The percentage increase in hospital admissions in Hong Kong in such a situation would be 16% for all respiratory illnesses and 15% for asthma, based on local data. Because respiratory and cardiovascular diseases account for the majority of hospital admissions, this increase would be considerable. As there is usually a time lag of up to 5 days, the increase in health care utilisation might not be immediately evident.

The Sub-Working Group on the Review of Hong Kong's Air Quality Objectives has estimated the

number of avoidable hospital admissions and deaths on removal of 30% of the highest levels of pollutants, assuming that the maximum of the separate effects of each pollutant apply.⁴⁸ Using data from The University of Hong Kong for 1996, 1596 to 2218 hospital admissions and 217 to 239 deaths from respiratory and cardiovascular diseases would be avoided each year. Using data from The Chinese University of Hong Kong for 1994 to 1995, there would be 1349 to 2072 avoidable hospital admissions each year. It should be noted that deaths and hospital admissions are only the 'tip of the iceberg' of adverse health effects. Below the 'tip of the iceberg' are visits to emergency departments for treatment of respiratory and cardiovascular diseases, unscheduled doctor visits, increases in drug treatment for the acute exacerbation of asthma or chronic obstructive lung disease, loss of productivity due to absence from work, and impaired quality of life. All these factors should be taken into consideration when calculating the direct and indirect cost of air pollution to health.

Deficiencies in knowledge

There remain some gaps in our knowledge on air pollution and health. Firstly, although acute and chronic effects of air pollutants have been studied, it is not known which specific air pollutant is responsible for adverse health effects, or how the pollutants interact with each other and with other factors such as allergens, diet, and housing. Secondly, there have not been

adequate studies making use of personal monitoring to quantify the total dose received by individuals in their daily lives. Thirdly, some pollutants that pose a particular risk to health, such as dioxins, polycyclic aromatic hydrocarbons, acid aerosols, and toxic metals, have not been sufficiently monitored and studied. Fourthly, the effects of long-term exposure on the development of asthma, allergies, and lung cancer are unknown. Finally, a better understanding is needed of the biological mechanism(s) through which air pollutants may lead to mortality.

Air quality guidelines

Guidelines of the World Health Organization

The 1987 WHO air quality guidelines for Europe were based on evidence from epidemiological and toxicological literature published in Europe and North America.³⁰ In view of the different conditions in developing countries, the WHO revised these guidelines in 1999.¹ The objective of the 1999 guidelines was to help countries derive their own national air quality standards and help protect human health from air pollution. The WHO 1999 guidelines are technologically feasible and have considered socio-economic and cultural constraints.

Since 1987, a new database of time-series studies has become available. These studies relate the daily occurrence of events such as deaths and hospital admissions to daily average concentrations of pollutants,

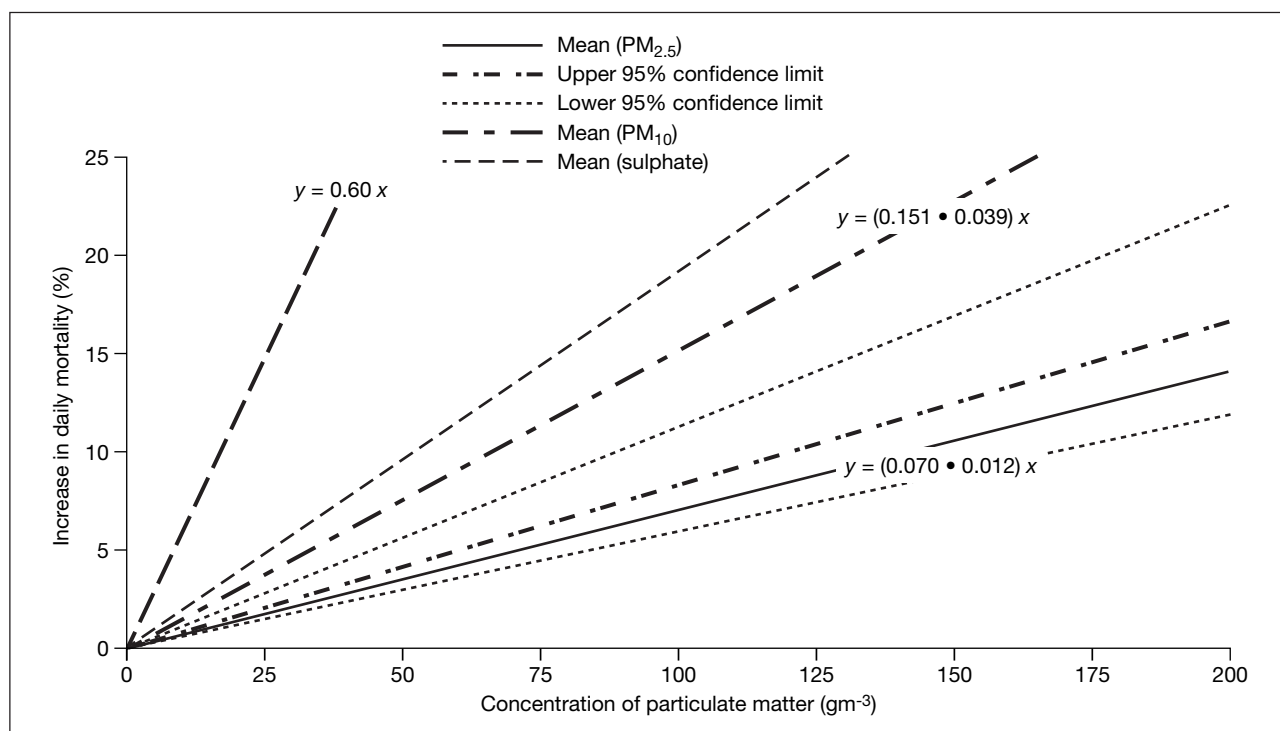


Fig 1. Percentage increase in daily mortality as a function of particle concentration¹

while taking into account confounding factors such as season, temperature, and day of the week. Using powerful statistical techniques, coefficients have been produced, which relate daily average concentrations of pollutants to effects. The results of these studies have been remarkably consistent, demonstrating associations between daily average concentrations of particulates, O₃, SO₂, and NO₂, and health outcomes. An important finding is that these studies fail to show any threshold effect for particles and O₃, because very low concentrations of these pollutants are already associated with health outcomes. Thus, the 'guideline' is a relationship that relates events to airborne concentrations. This concept represents an important departure from the one of a guideline value where the level of exposure at which the great majority of people—even in sensitive groups—would be unlikely to experience any adverse effects.

Figure 1 shows a summary estimate of the relative increase in daily mortality as a function of the concentration of particulate matter. Estimates for morbidity are also available.¹ These data allow the estimation, with caution, of how many subjects would be affected

over a short period of time with increased levels of particulate matter, for a population of a given size and for known mortality and morbidity characteristics. When deriving an air quality standard for PM₁₀ or PM_{2.5}, it has to be decided which curve should be used and what level of risk is considered to be acceptable. Table 2 shows the ambient air quality standards/guidelines for key air pollutants in Hong Kong, some member regions of the Association of South-East Asian Nations, the US, and the WHO.^{53,54} It can be seen that most places have yet to change guidelines regarding respirable suspended particulates, as suggested by the WHO. It should be pointed out that the determination of air quality objectives of various pollutants is based mainly on the health effects of acute exposure. There are no data on the health consequences of long-term low-level exposure, exposure to short-term peaks, and possible synergism among pollutants. There are also no data on the possibility of lower level pollutants inducing genetic mutations, birth defects, and cancer.

Air quality objectives in Hong Kong

One of the aims of the Sub-Working Group on the Review of Hong Kong's Air Quality Objectives was to

Table 2. Ambient air quality standards/guidelines for Hong Kong, the United States, the World Health Organization, and some member regions of the Association of South-East Asian Nations⁵³

Pollutant	Averaging time	Hong Kong	Thailand	Indonesia*	Philippines	Singapore	United States ⁵⁴	WHO [†]
SO ₂ (µg/m ³)	10 min	-	-	-	-	-	-	500
	1 h	800	-	900	850	-	-	-
	24 h	350	300	300	370	365	365	125
NO ₂ (µg/m ³)	annual	80	100	60	-	80	80	50
	30 min	-	-	-	300	-	-	-
	1 h	300	320	-	-	-	-	200
CO (mg/m ³)	annual	90	-	100	-	100	100	40
	15 min	-	-	-	-	-	-	100
	30 min	-	-	-	-	-	-	60
O ₃ (µg/m ³)	1 h	30	50	30	35	40	40	30
	8 h	10	20	10	10	10	10	10
	30 min	-	-	-	200	-	-	-
Suspended matter (µg/m ³)	1 h	240	200	160	-	235	-	-
	8 h	-	-	-	-	-	157	120
	24 h	260	330	230	180	260	-	-
Particulates <10 µm (PM ₁₀) [†] [µg/m ³]	annual	80	100	90	-	75	-	-
	24 h	180	-	-	-	-	150	No guideline value (impact relationship)
Particulates <2.5 µm (PM _{2.5}) [†] [µg/m ³]	annual	55	-	-	-	-	50	No guideline value (impact relationship)
	24 h	-	-	-	-	-	65	No guideline value (impact relationship)
Lead	annual	-	-	-	-	-	15	-
	24 h	-	10	2	-	-	-	-
	3 months	1.5	-	-	20	1.5	1.5	-
	annual	-	-	-	-	-	-	0.5

* Draft standards

† Currently, the US Environmental Protection Agency has a standard for PM₁₀ of 50 µg/m³ (annual average) and 150 µg/m³ for a 24-hour average; for PM_{2.5}, the annual and 24-hour standards are 15 and 65 µg/m³, respectively. In its most recent revision of the air quality guidelines, the WHO elected not to set a threshold value, but instead derived a linear relationship between PM₁₀ or PM_{2.5} concentrations and various health impacts.¹ This revision is based upon the absence of scientific evidence to support a no-effects threshold concentration for airborne particulate matter. These relationships allow each country/region to manage particulate air pollution by assessing the health effects associated with different levels of particulate matter

review the evidence available from Hong Kong studies on pollution and health, with a view to assessing the appropriateness of the current air quality objectives.⁴⁸ In the report published in July 1999, the members of the working group concluded the following:

- (1) In line with other overseas findings, studies from Hong Kong also show that there appears to be no safe level of human exposure to SO₂, respirable suspended particulates, NO₂, and O₃—at least at the levels commonly experienced in most urban environments;
- (2) Excess health risks were noted even on days when the levels of SO₂, respirable suspended particulates, NO₂, and O₃ were below the current set of air quality objectives. Hence, the current set of air quality objectives for these four pollutants are insufficient for the protection of public health; and
- (3) Achieving compliance with lower objective limits of air quality for the four pollutants would provide further protection to public health. This benefit is illustrated by estimating gains in the health of young children and in avoidable hospital admissions and hospital deaths due to respiratory and circulatory diseases.

Trends in air pollution exposure

In most of the cities of western Europe, measures to reduce air pollution levels, such as the regulation of fuel for domestic heating (low sulphur content in oil and coal, and the introduction of natural gas), the building of high chimney stacks for industrial emissions, the introduction of filtering techniques, and the reduction of lead content in petrol, have resulted in a major reduction in levels of SO₂ and lead. In most cities, the annual mean concentrations of SO₂ in residential areas have not exceeded 50 µg/m³. Notable exceptions are several cities in China: SO₂ concentrations in 1994 were 330 µg/m³ in Chongqing and 100 µg/m³ in Beijing.¹ Mexico City has experienced the most dramatic reduction of air pollution, from SO₂ levels between 100 and 140 mg/m³ in 1990/1991 to levels between 32 and 37 µg/m³ in 1995/1996.¹ However, there have been no comparable downward trends in NO₂ concentrations. In fact, in many cities, NO₂ levels have increased since 1960, mainly because of emissions from vehicles.¹ In Europe, road transport contributes up to half of NO₂ emissions.

Diesel vehicles produce much higher emissions of the smallest particles (ultrafine particles that are smaller than 0.1 µm in diameter) than do gasoline-fuelled vehicles. Ultrafine particles have been shown to produce acute inflammatory responses in animal studies,⁵⁵ but

the human epidemiological data are not conclusive regarding the relative toxicity of ultrafine particles versus particles of other sizes. Diesel vehicles not only produce more particle mass, but they also may produce substantially more of the toxic types of particles. They also have traces of adsorbed polycyclic aromatic hydrocarbon, which create potential problems that have not been well studied.⁵⁶

Trends in air pollution in Hong Kong

The trends in air pollution in Hong Kong are similar to those in other countries. The most important achievement has been the reduction in SO₂ levels after the implementation of government fuel regulations that restrict the sulphur content of fuels to 0.5%. Sulphur dioxide levels have fallen in many districts, and the levels of respirable suspended particulates have also been reduced slightly in some areas (Fig 2).⁵⁷

Since 1991, the annual mean roadside measurements of respirable suspended particulates and NO₂ have progressively increased (Fig 2) and exceeded the 1-year air quality objectives that have been recently set by the Environment Protection Department.⁵⁷ The 24-hour or 1-hour measurements have exceeded the air quality objectives many times each year. Thus, the findings in Hong Kong are similar to many of the major cities around the world. Among 36 major cities in the world in 1998, Hong Kong ranked 9th and 15th for the worst levels of respirable suspended particulates and NO₂ pollution, respectively; Singapore ranked 21st and 30th. Of special concern in Hong Kong is the emission of fine particles from diesel vehicles, which are responsible for 79% of the total mileage travelled on the roads.⁴⁸

Conclusion

In Hong Kong, levels of particulates, NO₂, and O₃ are high. The annual means of these pollutants measured by the roadside have exceeded the air quality objectives set by the Environmental Protection Department. In terms of health, local data have indicated that even below the air quality objectives, significant health effects can be detected. It is important that there should be a concerted effort by the medical profession to express concern about the health effects of air pollution and to call for action to reduce the current concentrations of air pollution to an acceptable level.

Acknowledgement

I wish to thank Prof M Brauer of the University of British Columbia for providing information for

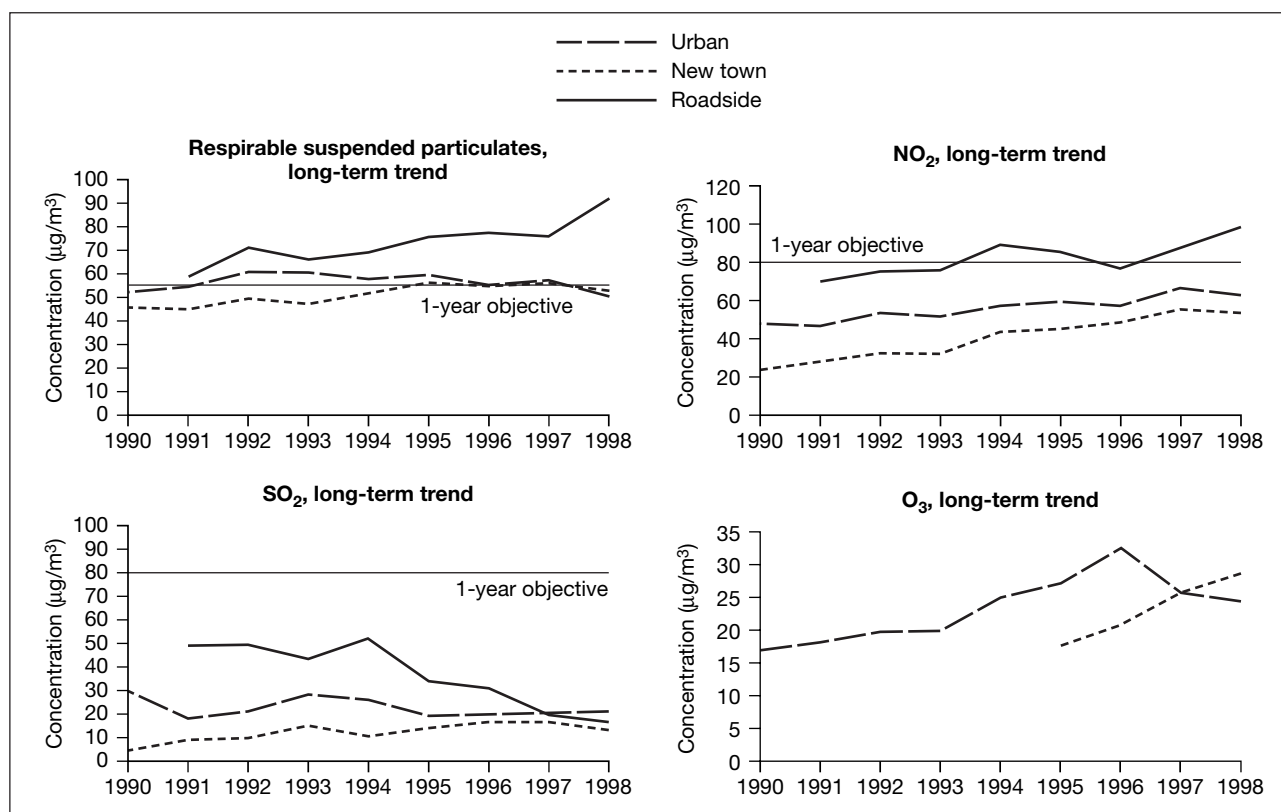


Fig 2. Long-term trends of levels of respirable suspended particulates, NO_2 , SO_2 , and O_3 in Hong Kong, 1990 to 1998⁵⁷

Table 2 and very useful comments, which have been incorporated into the text, and Mr P Ho of the Environmental Protection Department for providing the air pollution data for Hong Kong.

References

- World Health Organization. Guidelines for air quality. Geneva: WHO; 1999. Website: <http://www.who.int/peh/air/airguides2.htm>
- Peden DB. The effects of air pollution in asthma and respiratory allergy. The American experience. *Allergy Clin Immunol News* 1995;7:19-23.
- Folinsbee LJ. Human health effects of air pollution. *Environ Health Perspect* 1992;100:45-56.
- Pierson WE, Koenig JQ. Respiratory effects of air pollution on allergic disease. *J Allergy Clin Immunol* 1992;90:557-66.
- Koenig JQ, Pierson WE, Horike M. The effects of inhaled sulphuric acid on pulmonary function in adolescent asthmatics. *Am Rev Respir Dis* 1983;128:221-5.
- Commission of the European Communities. Directorate General XII for Science, Research and Development. COST 613.2 Report series on Air Pollution Epidemiology. Report No 2. Health effect assessment. Brussels: Commission of the European Communities; 1992.
- American Thoracic Society. Health effects of outdoor air pollution. Part II. *Am J Respir Crit Care Med* 1996;153:477-98.
- Devalia JL, Rusznak C, Herdman MJ, Trigg CJ, Tarraf H, Davies RJ. Effect of nitrogen dioxide and sulphur dioxide on airway response of mild asthmatic patients to allergen inhalation. *Lancet* 1994;344:1668-71.
- Tunnicliffe WS, Burge PS, Ayres JG. Effects of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 1994;344:1733-6.
- Molfino NA, Wright SC, Katz I, et al. Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet* 1991;338:1990-203.
- Jorres R, Norwak D, Magnussen H. The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis. *Am J Respir Crit Care Med* 1996;153:56-64.
- Frischer TM, Kuehr J, Pullwitt A, et al. Ambient ozone causes upper airway inflammation in children. *Am Rev Respir Dis* 1993;148:961-4.
- Wallace LA. Major sources of benzene exposure. *Environ Health Perspect* 1989;82:165-9.
- Allfred EN, Bleecker ER, Chaitman BR, Dahms TE, Gottlieb SO, Hackney JD. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Engl J Med* 1989;321:1326-32.
- American Thoracic Society. What constitutes an adverse health effect of air pollution? Official statement of the American Thoracic Society. *Am J Respir Crit Care Med* 2000;161:665-73.
- Firket J. Fog along the Meuse Valley. *Trans Faraday Soc* 1936;32:1192-7.
- Shrenk HN, Heimann H, Clayton GD, Gafafer WM, Wexler H. Air pollution in Donora, Pennsylvania. Epidemiology of the unusual smog episode of Oct 1948. Preliminary report. *Public Health Bull* 1949;306:1-171.
- Logan WP. Mortality in the London fog incident, 1952. *Lancet* 1953;264:336-8.
- Dockery DW, Pope CA III, Xu X, et al. Association between air pollution and mortality in six US cities. *N Engl J Med* 1993;329:1753-9.
- Schwartz J, Marcus A. Mortality and air pollution in London, a time series analysis. *Am J Epidemiol* 1993;131:185-94.

21. European Commission, Environmental Research Programme. The APHEA Project. Short term effects of air pollution on health: a European approach using epidemiological time series data. *J Epidemiol Commun Health* 1997;50(Suppl):2S-80S.
22. Zeger SL, Dominici F, Samet J. Harvesting-resistant estimates of air pollution effects on mortality. *Epidemiology* 1999;10:171-5.
23. Thurston GD, Ito K, Kinney PL, Lippmann M. A multi-year study of air pollution and respiratory hospital admissions in three New York state metropolitan areas: results for 1988 and 1989 summers. *J Expo Anal Environ Epidemiol* 1992;2:429-50.
24. Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environ Res* 1994;765:271-90.
25. Wong TW, Lau TS, Yu TS, Neller A, Wong SL, Tam W, Pang SW. Air pollution and hospital admission for respiratory and cardiovascular diseases in Hong Kong. *Occup Environ Med* 1999;56:679-83.
26. Bates DV, Szito R. Air pollution and hospital admissions in Southern Ontario. The acid summer haze effect. *Environ Res* 1987;43:317-31.
27. Wjst M, Reitmeir P, Dold S, Wulff A, et al. Road traffic and adverse effects on respiratory health in children. *BMJ* 1993;307:596-600.
28. Burney PG. Epidemiology trends. In: Barnes PJ, Grunstein MM, Leff AR, Woolcock A, editors. *Asthma*. Philadelphia: Lippincott-Raven Publishers; 1997:35-47.
29. Diaz-Sanchez D, Garcia MP, Wang M, Jyrala M, Saxon A. Nasal challenge with diesel exhaust particles can induce sensitization to a neoallergen in the human mucosa. *J Allergy Clin Immunol* 1999;104:1183-8.
30. World Health Organization. Air quality guidelines for Europe, European Series No 23. Copenhagen: WHO; 1987.
31. Pope CA III, Dockery DW. Acute health effects of PM₁₀ pollution on symptomatic and asymptomatic children. *Am Rev Respir Dis* 1992;145:1123-8.
32. Dockery DW, Pope CA III. Acute respiratory effects of particulate air pollution. *Ann Rev Public Health* 1994;15:107-32.
33. Samet JM, Bishop Y, Speizer FE, Spengler JD, Ferris BG. The relationship between air pollution and emergency room visits in an industrial community. *J Air Pollut Control Assoc* 1981;31:236-40.
34. Schwrtz J Slater D, Larson TV, Pierson WE, Koenig JQ. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 1993;147:826-31.
35. Sunyer J, Anto JM, Murillo C, Saez M. Effects of urban air pollution on emergency room admissions for chronic obstructive pulmonary disease. *Am J Epidemiol* 1991;134:277-86.
36. Sunyer J, Saez M, Murillp C, Castellsagne J, Martinez F, Anto JM. Air pollution and emergency room admissions for chronic obstructive pulmonary disease: a 5-year study. *Am J Epidemiol* 1993;137:701-5.
37. Pope CA III. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. *Am J Public Health* 1989;79:623-8.
38. Pope CA III. Respiratory hospital admission associated with PM10 pollution in Utah, Salt Lake, and Cache Valleys. *Arch Environ Health* 1991;46:90-7.
39. Riedel F, Kramer M, Scheibenbogen, Rieger VHL. Effects of SO₂ exposure on allergic sensitization in the guinea pig. *J Allergy Clin Immunol* 1988;82:527-34.
40. Zwick H, Popp W, Wagner C, et al. Effects of ozone on respiratory health, allergic sensitization and cellular immune system in children. *Am Rev Respir Dis* 1991;144:1075-9.
41. Horvath H Kreiner I, Norek C, Preining O, Gerogi B. Diesel emission in Vienna. *Atmospheric Environ* 1988;22:1255-69.
42. Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE. Particulate air pollution as a predictor of mortality in a retrospective study of US adults. *Am J Respir Crit Care Med* 1995;115:669-74.
43. Weiland SK, Mundt KA, Ruckmann A, Keil U. Self-reported wheezing and allergic rhinitis in children and traffic density on street residence. *Ann Epidemiol* 1994;4:243-47.
44. Schwartz J, Dockery DW, Neas LM, et al. Acute effects of summer air pollution on respiratory symptoms reporting on children. *Am J Respir Crit Care Med* 1994; 150:1234-42.
45. Hoek G, Brunekreef B. Effects of low-level winter air pollution concentrations on respiratory health of Dutch children. *Environ Res* 1994;64:136-50.
46. Hoek G, Brunekreef B. Acute effects of a winter air pollution episode on pulmonary function and respiratory symptoms of children. *Arch Environ Health* 1993;48:328-35.
47. Braun-Fahrlander C, Ackermann-Liebrich U, Schwartz J, Gnehm HP, Rutishauser M, Wanner Hu. Air pollution and respiratory symptoms in preschool children. *Am Rev Respir Dis* 1992;145:42-7.
48. Sub-Working Group on the Review of Hong Kong's Air Quality Objectives. Health effects of air pollution. Hong Kong: Sub-Working Group on the Review of Hong Kong's Air Quality Objectives; 1999.
49. Ong SG, Wong CM, Lam TH, Tam AY, Dainel L, Hedley AJ. Studies on the respiratory health of primary school children in urban communities of Hong Kong. *The Science Total Environ* 1991;106:121-35.
50. Tam AY, Wong CM, Lam TH, Ong SG, Peters J, Hedley AJ. Bronchial responsiveness in children exposed to atmospheric pollution in Hong Kong. *Chest* 1994;106:1056-9.
51. Peters J, Hedley AJ, Wong CM, Lam TH, Ong SG, Liu J, Spiegelhalter DJ. Effects of an ambient air pollution intervention and environmental tobacco smoke on children's respiratory health in Hong Kong. *Int J Epidemiol* 1996;25:821-8.
52. Wong CM, Lam TH, Peters J, Hedley AJ, Ong SG, Tam AY, Liu J, Spiegelhalter DJ. Comparison between two districts of the effects of an air pollution intervention on bronchial responsiveness in primary school children in Hong Kong. *Int J Epidemiol Community Health* 1998;52:571-8.
53. Brauer M, Chan-Yeung M. Air pollution and health. In: Zhong NS, Chan-Yeung M, Lam WK, Ip M, editors. *Respiratory medicine: focus on Asia*. In press 2001.
54. Environmental Protection Agency, United States. Offices of Air Quality Planning and Standards. National ambient air quality standards Website: <http://www.epa.gov/air/oaqps/greenbk/criteria.html>
55. Oberdörster G, Ferin J, Felein S, Sonderholm C, Finkelstein J. Role of the alveolar macrophages during lung injury: studies with ultrafine particles. *Environ Health Perspective* 1992;97:193-9.
56. Viegi G, Enarson DA. Human health effects of air pollution from mobile sources in Europe. *Int J Tuberc Lung* 1998;2:947-67.
57. Environmental Protection Department. Air quality in Hong Kong 1998. Hong Kong: Environmental Protection Department; 1998. Website: <http://www.epd-asg.gov.hk/reportsf.html>