The impact of air pollution on population health, health care and community costs

Submission to the chairman and honourable members
of the Legco Environmental Affairs Panel

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Key points

- There is incontrovertible evidence from the worldwide literature that air pollution causes damage to body tissues (especially the eyes, nasal passages, lungs, blood vessels and heart) and causes severe illness episodes and shortening of life expectancy. Longer term effects of pollution cause cardiopulmonary problems and also increase the risk of lung cancer. Long term studies from overseas show that pollution impairs normal development of lung function in young people, so that by the time they reach maturity at around age 18 years old they have suboptimal lung growth – a lifetime defect with major implications for health experience and life-expectancy.

- In Hong Kong analyses of environmental health clearly show that short term effects of pollution have a very large impact on diseases of the lungs, blood vessels and heart. Long term studies of population health overseas give bigger estimates of effects than short term analyses. Most of the current evidence in Hong Kong is based only on short term effects which are likely to under-estimate the total health impact of air pollution on our population.

- There is evidence that pollutants entering the blood circulation of a pregnant mother damage the unborn infant. Effects of pollution on the growth and health of unborn infants, reported from the mainland and overseas, have not yet been investigated in Hong Kong.

- The mechanisms by which air pollution harms population health is being demonstrated by human clinical studies, population surveys, and animal toxicological tests and other experimental analyses. Harmful effects include widespread inflammation in the body, increased susceptibility to infection, development of atherosclerosis in blood vessels and interference with the normal neuro-electrical control of the heart. Illnesses resulting from exposure to pollution include exacerbations of asthma; heart attacks; strokes; lung and other respiratory infections; acute and chronic bronchitic symptoms, and eye and skin irritation.

- Health related community costs of air pollution in Hong Kong are conservatively valued at $2 billion for health care and lost productivity and about $19 billion for intangible costs related to willingness-to-pay to avoid serious illness or death from air pollution.

- There is evidence that pollutants from the combustion of fossil fuels from power generation, road traffic and industrial plants all contribute to these health risks, including particulates, gaseous pollutants, with a wide range of chemicals components including metals.

- Prevention of the health effects of pollution must include a comprehensive approach to pollution abatement from several sources. The World Health Organization Air Quality Guidelines provide a framework for immediate action. They are based on the best evidence worldwide of pollution related health impacts, including those from Hong Kong and other Asian regions. Further delays in initiating and achieving major reductions in air pollutant *concentrations* will be accountable in terms of life long damage to young people, severe losses of health related quality of life and premature deaths.

- We have demonstrated that poor health outcomes are avoidable through interventions which improve air quality. Public health research in Hong Kong has provided one of the very few demonstrations of the large scale population benefits which have resulted from improvements in air quality. The 1990 restriction of sulphur in fuel led to health gains for thousands of school children, and a reduction of 600 deaths per year in those aged 46 and over.

Health effects of air pollution in Hong Kong

Although most studies of air pollution effects on health have been conducted in the US, Europe and other developed regions, there is now a substantial body of high quality research which demonstrates the relationship between concentrations of air pollutants and health outcomes in many Asian countries and regions. Hong Kong public health research on air pollution, from the University of Hong Kong and the Chinese University of Hong Kong, has made an important contribution to this evidence base. This was recognized by the global review process which led to the formulation of the World Health Organization Air Quality Guidelines, launched on October 5, 2006. (Attachment 1)

It is important to acknowledge that the decision analysis in the WHO review was strongly influenced by analyses conducted in Hong Kong and other parts of Asia.

Which pollutants are harmful to health?

In terms of environmental management, reduction of the level of *emissions* is clearly a key measure. However in assessing public health effects we need to focus on the daily *concentrations* of pollutants in the air breathed by individuals in the population.

The pollutants which are commonly used as indicators of the harmful effects of emissions from different sources are the combustion products of fossil fuels and include particulates, nitrogen oxides, sulphur oxides, and the secondary pollutant ozone. Other chemicals implicated in health effects include transition metals and volatile organic compounds. Particulates include elemental and organic carbon compounds, nitrates, sulphates, acids and metals.

The easiest pollutants to measure and those which are used as "criteria pollutants" to estimate health effects include respirable suspended particulates (RSP or PM_{10}), nitrogen dioxide (NO_2), sulphur dioxide (SO_2) and ozone (O_3).

At present there is no conclusive evidence that either one single pollutant or a particular component of particulates is responsible for the observed harmful effects. From a public health viewpoint this emphasizes the need to control *sources* of emissions rather than attempt to focus exclusively on individual pollutant species.

In Hong Kong and other centres in China all four criteria pollutants show significant associations with adverse health effects. However for adverse health outcomes in terms of hospital admissions and deaths risk estimates are consistently higher for nitrogen dioxide and sulphur dioxide than for particulates. Again this emphasizes the need to address sources of pollution in this region rather than place too much emphasis on any one pollutant.
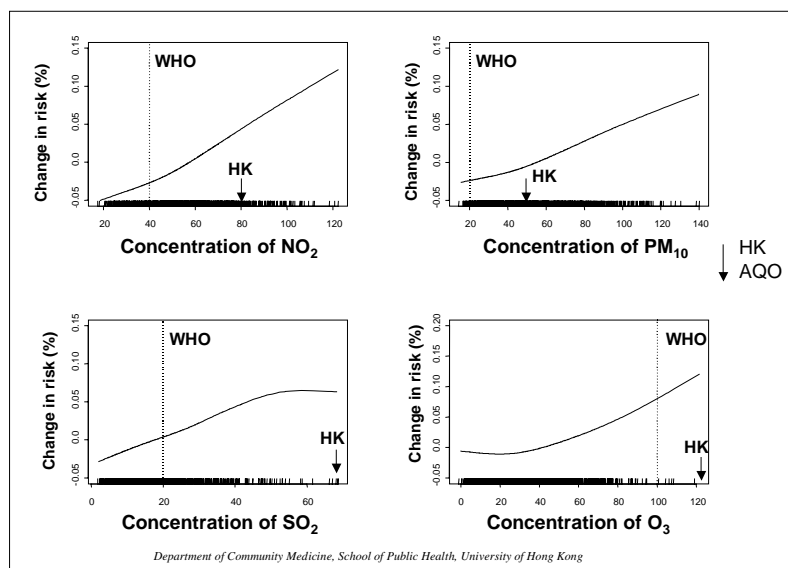
What is the relationship between pollutant concentrations and health effects in Hong Kong?

In Hong Kong all four criteria pollutants (Respiratory Suspended Particulates (RSP) estimated as PM_{10}, NO_2, SO_2 and O_3) are associated with increased daily risks of hospitalization and death at the current prevailing daily levels of pollution.

In studies of the short term (daily) events attributable to pollution, the excess risk estimates for illness episodes, hospital admissions and deaths, for each pollutant, typically lie in the range 0.5% to 1.5% per ten micrograms of pollutant per cubic metre.

It is important to note that most of the estimated bad health outcomes are occurring at levels of pollution which are well *below* the present Hong Kong Air Quality Objectives (AQOs) (Figure 1). The biggest proportion of the harmful effects are determined by the average levels of pollution and not simply the highest pollution days. The risks associated with these levels are not adequately signaled by the Hong Kong Air Pollution Index (API). This is because the index is scaled to the Hong Kong Air Quality Objectives which are far too high. For example a level of pollution at only 50% of the AQO would still be 32% (PM_{10}) to 100% (SO_2) above the WHO guidelines on an annual basis. So even an API as low as 25 would not be a valid indicator of health protection.

- The slope of the curves show the increasing risk with increases in pollutants.
- The "brush border" on the horizontal axis shows the number of days a year at those pollutant levels ($\mu g/m^3$).
- The arrow is the Hong Kong (HK) present AQO; the vertical broken line is the new WHO AQG.

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Figure 1:   Air Quality Standards: do they protect health?
Respiratory Admissions to HA Hospitals

These risks of pollution health effects are estimated by a method known as time-series analysis. In this procedure daily concentrations of pollutants are modeled against daily deaths or hospital admissions. These short-term effects are now widely considered to be under-estimates. It is important for the Panel to note that longer term follow-up (cohort) studies of well-populations in other countries give risk estimates which are at least twice those from time series analyses.

How does air pollution harm body tissues?

A sound evidence base for clean air advocacy is essential if the arguments for urgent intervention are going to be accepted. There is now a large body of evidence which points to fundamental harmful effects of air pollutants on body cells in animals and humans.

Current scientific evidence indicates that the complex chemicals which are common components of air pollution, damage body tissues in a variety of different ways. Probably the most important mechanism is *oxidative stress*. Oxidative stress is a series of reactions which may be caused by smoking, alcohol abuse, radiation, exposure to cold and air pollution. Air pollution may cause oxidative stress because of the presence of several highly reactive *free radicals* in pollutant mixtures.

Free radicals may overcome our anti-oxidant defence mechanisms and cause a widespread inflammatory response, initially in the lungs and then throughout the body as a result of a

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secondary wave of inflammatory cells. Common pollutants in urban air pollution include nitrogen dioxide, ozone and particulates. All of these have been clearly shown to cause or trigger oxidative stress and the inflammatory cell response.

Our lungs have a very large surface area (sometimes approximated to the size of a tennis court) and they are the first target of inhaled environmental pollutants. In apparently healthy subjects the antioxidant defences in the lung may be robust, but there is likely to be a wide range of different degrees of susceptibility. Those people with existing health problems such as asthma may already have deficient lung defences.

Who is most affected by air pollutants?

Air pollution concentrations, which are currently typical of the Hong Kong environment, cause serious health problems in a large minority of the Hong Kong population. It is likely that everyone in Hong Kong is affected to some extent by ambient pollution with increased levels of inflammatory cells in their circulation and body tissues.

We know from experimental studies in humans that when people move to geographical areas with much lower pollution the body markers of inflammation decline.

Typically we find that the health risks are bigger at the extremes of life, in young children and older people from middle age (eg 45 years plus) onwards, and those who have other health risks such as active or passive smoking, poor nutrition, heart or lung disease, or any factor which impairs the immune system.

Studies carried out by the University of Hong Kong and Chinese University of Hong Kong have shown that young children in areas of higher pollution suffer more episodes of bronchitic symptoms and impaired lung function compared with those in less polluted regions. There is clear evidence that the burden of respiratory health problems in children across the whole HKSAR would decline with air quality improvement.

Between January 1996 and December 2002, hospital discharge diagnoses and mortality statistics showed that:

- Acute lung infections in the young, exacerbations of asthma and chronic respiratory problems at all ages were strongly associated with the four criteria pollutants in Hong Kong.

- Cardiovascular diseases (heart attacks and stroke) were also strongly associated with pollutant concentrations when analyzed for hospital admissions and deaths across all ages, not only in the elderly.

Hong Kong has, almost by chance, one of the best pieces of evidence on the health gains which can be achieved by different groups in the population through air quality improvements from analyses of the 1990 fuel restriction on sulphur:

The Hong Kong Air Quality Intervention 1990
On Sunday July 1st 1990 a new Ordinance restricted the sulphur content of fuel to 0.5% by weight. The impact of this very modest intervention on both the environment and population health was immediate and beneficial:

Environmental Impact:
- Levels of sulphur dioxide (SO_2) fell by about 80% in Kwai Tsing district and by about 50% territory wide. These reductions were maintained and eventually improved on until 2000 when an increasing trend in SO_2 is again observed.

  Concentrations of the transition metals Nickel and Vanadium also showed a sharp decline and this was also maintained until 2000-2004 when a significant upward trend is observed.

Health gains:
- Following the intervention there was a marked improvement in the respiratory health of primary school children (and their mothers) with reduction of bronchitic symptoms such as cough, phlegm and wheeze. Tests of lung function showed an improvement over a two year period with children in Kwai Tsing Kwai Chung/Tsing Yi improving to the level of health of children in the less polluted Southern District. Thousands of young children benefited from this reduction in one group of pollutants from sulphur rich fuels.

  There was a marked effect on mortality patterns, mainly from cardiovascular and lung disease, with a 2.2% downturn in the trend in annual numbers of deaths. This was equivalent to 600 deaths avoided each year over the 5 years following the intervention. The reduction in mortality risks was seen at all ages over the age of 45 and the reduction in respiratory causes of death was greatest in the 46-64 year group. (Attachment 2)

The current situation 2006:
Despite claims by the Government there is little if any evidence of substantial and sustained improvements in air quality in Hong Kong in the past 6 years. There are several problems: (a) Any proportional reductions in concentrations are small in relation to the high average levels; (b) Uncertainties in the data (omissions of monitoring data and yearly fluctuations in pollutants are not properly taken into account; (c) Trends in emissions from power generation, marine and civil aviation sources (58% of the total) are upwards and (d) Extrapolation from current trends in pollutant levels indicate that there will be no reasonable health protection from air pollution in Hong Kong in the foreseeable future.

What are the community costs of air pollution?

We can use the risk estimates to calculate the numbers of bad health outcomes and then put a dollar value on the resulting financial stress on the individuals and families affected, the health care system and employers who lose the services of the workforce through illness or premature death. Additional costs, known as intangible costs, can be valued as the willingness-to-pay to avoid daily respiratory symptoms (such as cough), a serious illness episode requiring hospital admission, and a death due to pollution.

We estimate each year that the difference between Hong Kong's *average pollution levels* and much lower levels which are close to the new *WHO Guidelines* is the cause of a large scale epidemic of disease (Figure 2)

- 6.8 million doctor visits for respiratory complaints alone,
- over 60,000 hospital bed days and
- about 1600 deaths.

These translate into annual direct (health care) and indirect (lost productivity) costs of about $2 Billion and a further $19 Billion for the intangible costs (Figure 3). (see also Attachment 3)
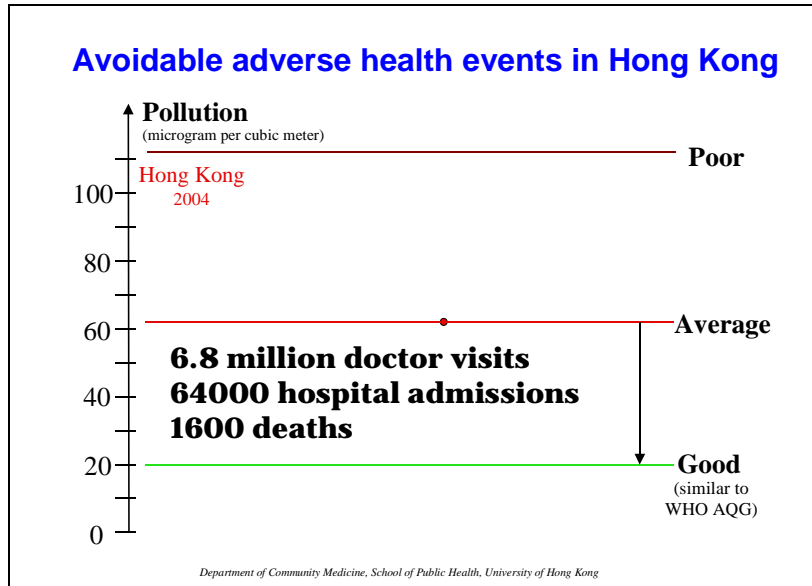
Figure 2: Doctor visits, hospital admissions and deaths avoided with improvement in Hong Kong air pollution from the current average level to a level close to the WHO 2006 Guidelines
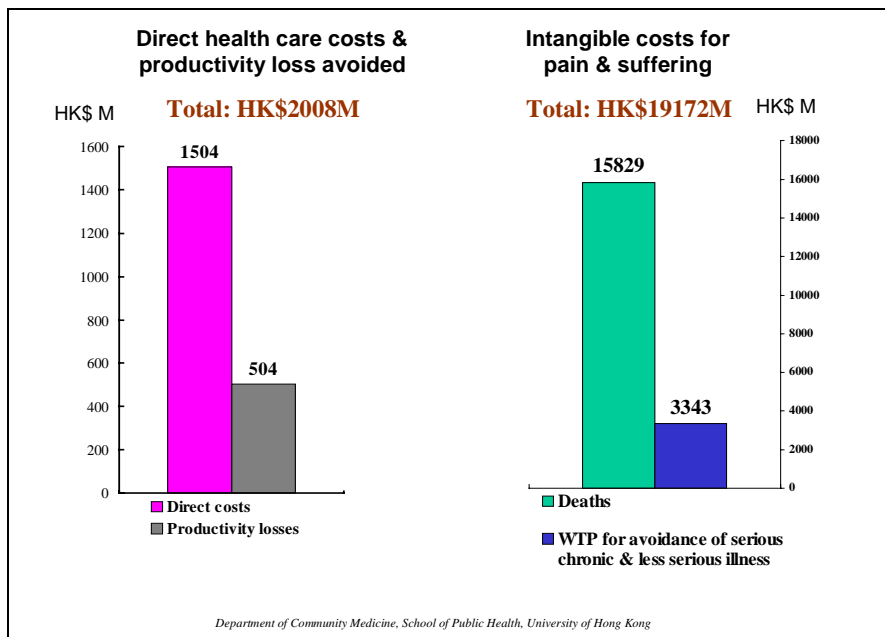
Figure 3:   Values for direct costs and productivity loss and intangible costs associated with the difference between Hong Kong's average pollution and levels similar to the WHO Guidelines

These dollar values are conservative and do not yet include all of the harm from pollution.

What can we gain from the new World Health Organization Air Quality Guidelines?

The recent release of the new WHO Air Quality Guidelines (AQG) provides the most detailed and comprehensive analysis to date of the relationship between air pollutants and population health. The full report is expected to be published in December 2006.

The review of the world's literature, on which the WHO AQG are based is the best summation of available evidence on health effects and the Guidelines should therefore be the benchmark adopted in Hong Kong for environmental management and health protection in the immediate future.

> *While there is always scope for furthering knowledge and developing analytical insights we believe that the new AQG provide a very sound basis for setting Air Quality Objectives in Hong Kong. The strength and consistency of the associations between pollutants and health effects between different regions, on which the Guidelines are based, and the process by which the WHO Working Group handled uncertainties underpin their validity and relevance to Hong Kong.*

On the basis of recent statements from the Environmental Protection Department we believe that the Government may misconstrue the intention of WHO in providing a framework of "interim targets" for each pollutant. As stated by WHO these are intended for use in areas where pollution is high. However the majority of cities which are as badly polluted as Hong Kong do not have the financial resources, technological capability and organizational infrastructure which is possessed by Hong Kong.

> *We would want to strongly re-assert that considerable public health protection from air pollution in Hong Kong and the surrounding region can be achieved on a relatively short timescale providing that there is political will to use the WHO Guidelines as our objectives.*

It is important to acknowledge that the new Guidelines themselves are likely to be regarded as interim guidelines in the longer term. They should not be regarded as *safe*, only *safer* than previously adopted levels of pollutants.

There is general agreement that analyses of health effects do not show any evidence of a threshold concentration below which harm to health does not occur.

Conclusions

In Hong Kong we have already seen, sixteen years ago, that even modest air quality interventions can have a large beneficial effect on population health. We see no reason in principle why this same process cannot be applied across the whole region including the HKSAR and the Pearl River Delta.

Hong Kong should take a resolute lead in this process. Failure to do so will be accountable in terms of lifelong damage to young people, high costs in terms of acute health care, and severe losses to families through premature deaths.

The Hong Kong population urgently needs the application of the precautionary principle to prevent further environmental degradation, health impairment and costs to the whole community.

Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study

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Summary

Background In July, 1990, a restriction was introduced over one weekend that required all power plants and road vehicles in Hong Kong to use fuel oil with a sulphur content of not more than 0·5% by weight. This intervention led to an immediate fall in ambient sulphur dioxide (SO_2). We assessed the effect of this intervention on mortality over the next 5 years.

Methods Changes in trends in deaths were estimated by a Poisson regression model of deaths each month between 1985 and 1995. Changes in seasonal deaths immediately after the intervention were measured by the increase in deaths from warm to cool season. We also estimated the annual proportional change in number of deaths before and after the intervention. We used age-specific death rates to estimate person-years of life gained.

Findings In the first 12 months after introduction of the restriction, a substantial reduction in seasonal deaths was noted, followed by a peak in the cool-season death rate between 13 and 24 months, returning to the expected pattern during years 3–5. Compared with predictions, the intervention led to a significant decline in the average annual trend in deaths from all causes (2·1%; p=0·001), respiratory (3·9%; p=0·0014) and cardiovascular (2·0%; p=0·0214) diseases, but not from other causes. The average gain in life expectancy per year of exposure to the lower pollutant concentration was 20 days (females) to 41 days (males).

Interpretation Pollution resulting from sulphur-rich fuels has an effect on death rates, especially respiratory and cardiovascular deaths. The outcome of the Hong Kong intervention provides direct evidence that control of this pollution has immediate and long-term health benefits.

Lancet 2002; **360:** 1646–52

Introduction

The association between air pollution and health effects including death has been established from reports on high-pollution incidents,[1] time-series analyses,[2] and cohort studies.[3,4] The strongest evidence is for respirable particulates (PM_{10}),[5] but many researchers have reported associations with gaseous pollutants, especially sulphur dioxide (SO_2).[6] Questions remain about the public-health effect of air pollution, particularly about death rates and life expectancy.[7,8] Very few opportunities have arisen to do epidemiological studies of the effects of interventions or of individual components of pollution.

Absence of data from intervention studies means that inconsistencies between studies on the importance of particulates or gases in pollutant mixtures, as causes of health problems and premature deaths, have not been resolved. One difficulty relevant to assessment of the public-health and economic analyses is the issue of mortality displacement or so-called harvesting.[9,10] Do deaths associated with fluctuations in pollutant concentrations arise mainly in sick or highly vulnerable groups of people, who would have died anyway in the short term, or are there longer-term effects from exposures? Time-series and cohort studies have both investigated the relation between pollution and years of life lost, but each has inherent limitations.

SO_2 has been described as a pollutant of public-health concern. The US Clean Air Act Amendments of 1990 proposed a reduction of 10 million tonnes of SO_2 emissions by 2010, with the aim to reduce SO_2, sulphate particulates, and acid precipitation.[11] In the first half of 1990, ambient monthly SO_2 concentrations monitored in Hong Kong ranged from 3 $\mu g/m^3$ to 145 $\mu g/m^3$ between the least and most polluted districts, with a regional mean of 37 $\mu g/m^3$. On July 1, 1990, all power plants and road vehicles in Hong Kong were restricted to use of fuel oil with a sulphur content of not more than 0·5% by weight.[12] This intervention led to an immediate improvement in air quality, which was associated with a fall in SO_2 and sulphate in respirable particulates by up to 80% and 41%, respectively, in the most polluted areas. No great change in any of the other main pollutants was recorded.

In the 2 years after the intervention we showed a reduction of chronic bronchitic symptoms[13] and bronchial hyper-responsiveness[14] in young children. We aimed to assess the immediate and longer-term effect of the air-quality intervention on deaths in the Hong Kong population.

Methods

Procedures

From July, 1985, to June, 1995, we obtained data for deaths per month from all causes, respiratory disease (international classification of diseases 9th revision [ICD9] 460–519), cardiovascular disease (ICD9 390–459), and neoplasms (ICD9 140–239), and other causes (ICD9

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001–009; 140–161; 163–246; 280–294; 320–326; 520–629; 710–719) from the Census and Statistics Department databases.[15] We stratified these data into three groups by age: 15–64 years; 65 and older; and all ages.

Air-pollutant concentrations were obtained from the Environmental Protection Department[16] for the period 1988–95. From five stations with almost complete data (97%), we plotted monthly mean concentrations of SO_2, sulphate in respirable particulates, nitrogen dioxide (NO_2), ozone (O_3; two stations only), and PM_{10} to investigate changes in concentrations of pollutants at each station 2 years before and 5 years after introduction of the fuel regulations. Daily pollutant concentrations measured by the five stations showed an average correlation of $r=0.5$ (range 0·3–0·7). A further three stations had useable data for 1 year before and up to 3 years after the intervention. The average correlation for all eight stations was $r=0.4$ (0·1–0·7) over the period 1990–92.

We used District Board resident populations to estimate the population covered by the different monitoring stations. We estimated that five stations covered 54% of the population, and all eight, 73%.

We assessed the overall change from baseline in average monthly concentration of each pollutant, by corresponding month and station for 5 years after the intervention. We applied a two-tailed t test to establish whether the means of the 5-year differences (maximum n=300) for each pollutant were different from zero.

Statistical analysis
We obtained a measure (λ) of change in death rates, relative to the mean, in the warm (April–September) or cool (October–March) seasons, by three methods. We first obtained monthly expected values from a linear regression model of monthly deaths. Second, we fitted a Poisson regression with observed/expected deaths as the dependent variable and a pair of $\alpha\times$sine and $\beta\times$cosine terms as the independent variables to model one cycle per year, where α and β are coefficients to be estimated from the regression. This model is sufficient to show the major warm to cool season changes. Third, we calculated the value of λ, using the equation shown in the panel, with 95% CIs. We did this calculation for every year after the intervention and for every cause of death by all ages and specific age-groups, and compared the values with those derived from the period before the intervention.[17]

We used the Poisson regression to model monthly deaths as a dependent variable,[18] taking into account

trends, seasonality (by pairs of sine and cosine terms, allowing for one to three cycles per year to capture the main seasonal variations per year), temperature, and relative humidity, with stratification into two 5-year periods, before and after the intervention. We used the coefficient from the regression model to derive the average annual change in number of deaths in each of the two 5-year periods. We calculated the relative change in these estimates between the two periods, by causes of death and age-groups, with an interaction term representing the relative change in trend between the two periods. This term directly indicates the effect of the intervention in terms of average annual reduction in mortality.

We investigated differences in deaths over 5 years between districts with and without sustained reductions in SO_2 versus baseline. These districts were grouped in accordance with their reduction in SO_2 up to 2·5 years after the intervention, as indicated by eight monitoring stations. Over this period, the average change at the four stations with a consistently sustained reduction in SO_2 over 2·5 years was a 52·8% decrease versus an 8·7% increase at the four stations with reductions for shorter periods. These two groups of districts were defined as high and low SO_2 reduction areas. We assessed excess risk of death with Poisson regression on monthly death rates. Covariates included time trend, seasonality (sine and cosine terms), temperature and humidity, and a dummy variable for 5 years before and after the intervention.

We used age at death before intervention (1985–90) to calculate potential years of life lost due to death from all causes and cardiorespiratory disease, on the basis of life expectancy in those years. We calculated years of life that were saved by the intervention by applying the relative change in trend for deaths after the intervention to the years of life lost before the intervention.

We used differences in age-specific death rates, based on the population at the midpoint of two 2-year periods immediately before and after the intervention, to calculate change in life expectancy per year of exposure to the reduced pollutant levels, expressed per 10 $\mu g/m^3$ of SO_2. Furthermore, we estimated gain in life expectancy with the annual relative risk per 10 $\mu g/m^3$ SO_2, for a hypothetical cohort of people age 25–100 years.[8] We obtained numbers of survivors for each 5-year age-group (25–29, 30–34, 35–39, etc) from the 1991 Hong Kong life-table.

All tests of significance are two tailed unless otherwise stated.

Equation used to calculate λ

$$\lambda=\sqrt{\alpha^2+\beta^2}$$

	Baseline*	After intervention								
		1 year†			2·5 years‡			5 years§		
	Mean (SD) ($\mu g/m^3$)	Mean (SD) ($\mu g/m^3$)	Absolute change	Relative change (%)	Mean (SD) ($\mu g/m^3$)	Absolute change	Relative change (%)	Mean (SD) ($\mu g/m^3$)	Absolute change	Relative change (%)	p¶
Pollutant											
SO_2	44·2 (40·1)	20·8 (9·9)	–23·4	–53·0	22·3 (10·8)	–21·9	–49·6	24·5 (12·2)	–19·7	–44·7	<0·0001
SO_4 RSP	8·9 (3·2)	6·9 (2·0)	–2·0	–22·9	7·9 (2·8)	–1·0	–11·7	8·9 (3·1)	0·0	–0·3	0·896
NO_2	54·7 (22·8)	48·1 (12·0)	–6·7	–12·2	52·8 (15·1)	–1·9	–3·5	54·7 (14·7)	0·0	–0·1	0·205
O_3	18·5 (7·5)	21·3 (9·1)	2·8	15·2	22·1 (10·2)	3·6	19·6	23·8 (11·4)	5·2	28·3	<0·0001
PM_{10}	59·8 (17·0)	59·8 (16·9)	0·0	0·0	61·7 (17·4)	1·9	3·2	60·2 (17·6)	0·4	0·6	0·926

SO_4 RSP=sulphate in respirable particulates. *July, 1989, to June, 1990. †July, 1990, to June, 1991. ‡July, 1990, to December, 1992. §July, 1990, to June, 1995. ¶For difference from baseline concentration to corresponding month and stations over the 5 years after the intervention.

Table 1: **Mean (SD) concentration of pollutants based on five stations at baseline and after intervention with mean absolute and relative changes**

Results

In the first year after introduction of the intervention, mean fall in SO$_2$ concentration at five stations was 53% (table 1). Reduction in SO$_2$ concentration was sustained between 35% and 53% (mean 45%) of the mean value before the intervention, over 5 years. At eight stations for which complete data were available for up to 2·5 years, the average reduction in SO$_2$ concentration over this period was 50%.

Mean concentration of sulphate in respirable particulates at five stations for 2 years before the intervention was 8·9 µg/m³. This concentration fell by 15–23% for 2 years but rose again to between 110% and 114% of the concentration before 1990 in years 3–5 after the intervention (data not shown). No significant change in mean concentration of PM$_{10}$ (p=0·926) and NO$_2$ (p=0·205)—but a significant increase of O$_3$ (p<0·0001)—was noted over the 5 years after the restriction on fuel sulphur content (figure 1).

Over the 5 years before the intervention, number of deaths per month showed a stable seasonal pattern for all causes and cardiorespiratory diseases. In the year after the restriction on fuel sulphur content was introduced, the expected cool season peak was absent (figure 2).

The noted seasonal mortality cycle closely fitted the model for the 5 years before introduction of the intervention. In the first 12 months after the intervention, amplitude of the cycle was low compared with that predicted because of a striking reduction in deaths in the cool season (figure 3). This fall was associated with a reduction in the warm to cool season mortality gradient, for every age-group, for all causes, respiratory, and cardiovascular deaths. For example, the seasonal percentage increase for all causes and all ages declined from the average 5-year baseline of 10·3% to 4·2% and respiratory deaths from 20·3% to 5·3% (table 2). In people aged 65 or older, seasonal deaths for all causes declined from 14·7% to 6·1% and respiratory deaths from 22·7% to 5·4%. No consistent change in seasonal pattern of deaths in any age-group for neoplasms or other causes was noted. In the second 12 months a striking rebound in deaths in the cool season deaths arose, followed by a gradual return during years 3–5 to the seasonal pattern before intervention.

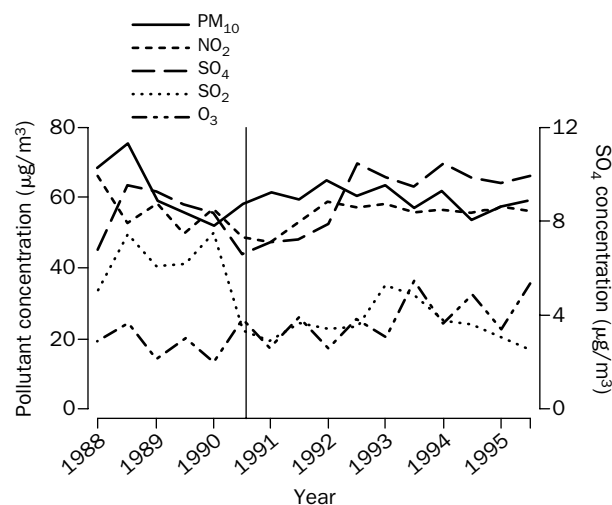The reduction in cool-season deaths in the first year after the intervention showed a consistent pattern across the eight stations, except in one district, which only contributed 1·3% of total deaths covered by air-pollutant monitoring.

The average annual proportional change in number of deaths, for all causes and all ages, was an increase of 3·5% per year in 1985–90, in accordance with the increase in size and ageing of the population. After the intervention
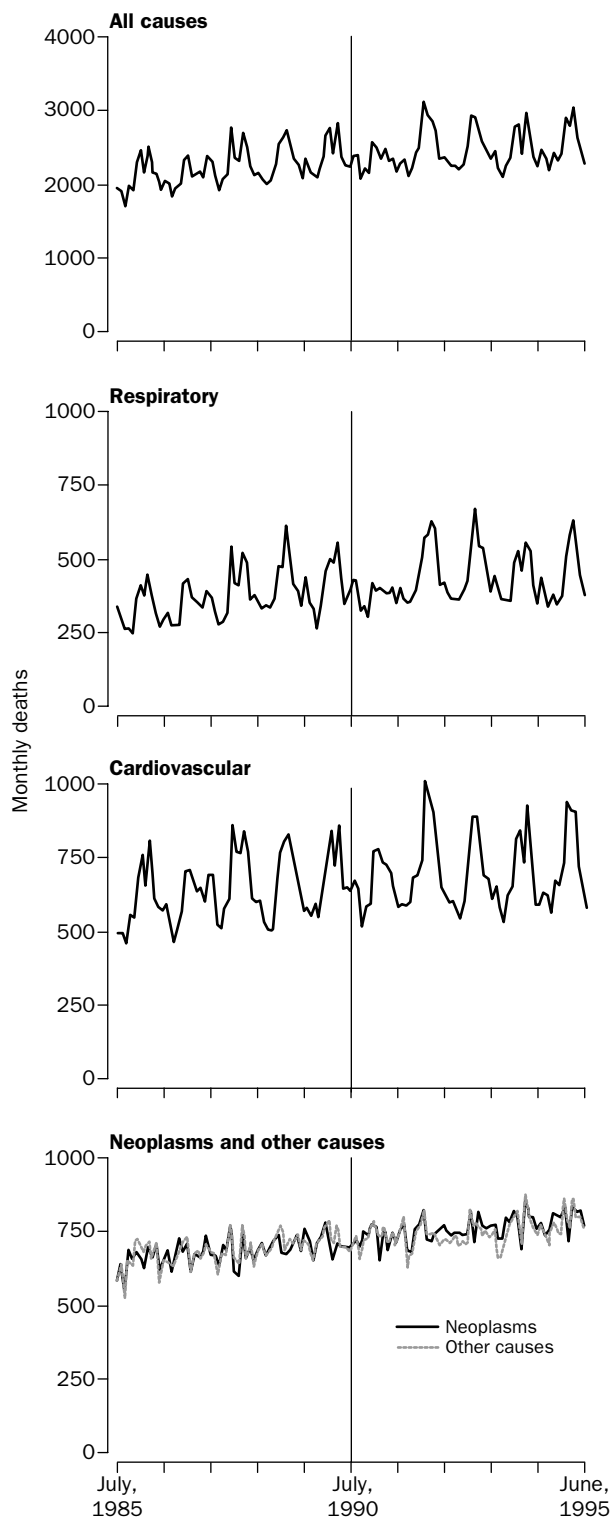

Figure 1: **Average of pollutant concentrations at five monitoring stations**
Vertical line represents date of introduction of fuel regulation.

Figure 2: **Number of deaths per month for all ages from July, 1985, to June, 1995, for all causes, respiratory, cardiovascular, and neoplasms and other causes**
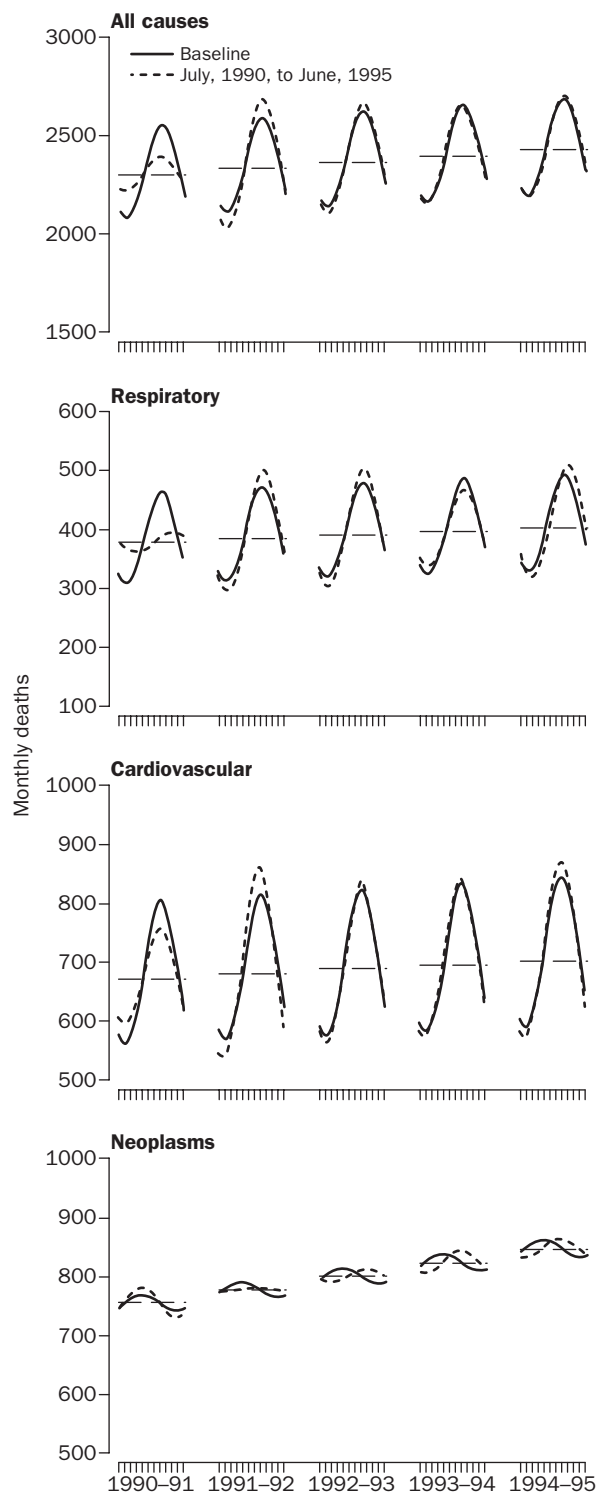Vertical line represents date of introduction of fuel regulation.

Figure 3: Annual cycle of monthly deaths for all ages, for baseline and for each year after the intervention (July, 1990), for all causes, respiratory, cardiovascular, and neoplasms
Mid-point of each period at January. Horizontal lines indicate mean monthly deaths.

was introduced, the average annual percentage change (increase) in deaths for 1990–95 declined for all causes, respiratory, and cardiovascular deaths compared with the 5 years before the intervention (table 3). For all causes and all ages, the average annual percentage change in death rates over 5 years showed a decline of 2·1% (95% CI 0·9–3·3), and for those aged 65 years or older the

reduction was 2·8% (1·4–4·2). The biggest relative change was seen for respiratory deaths for the 15–64 age-group (4·8%; 1·2–8·3), with a smaller but significant change for cardiovascular deaths of 2·0% for all ages and 2·4% for those aged 65 years or older. No significant (p>0·05) post-intervention difference in annual rate of change was found at any age for deaths from neoplasms including lung cancer, or from other causes at age older than 65 or all ages (table 3).

A greater decline in mortality was noted in areas with a higher reduction in SO_2 during the first 2·5 years (–3·27%; 95% CI –7·10 to 0·83) than in areas with less reduction (1·35%; –3·63 to 6·61; p=0·08 one tailed test of significance, for reduction in average deaths between the two districts).

Estimated deaths and mean potential life years lost, for all causes and all ages, in 1985–90, based on the post-intervention decline in annual proportional change in deaths of 2·1%, was 600 deaths per year associated with 10 268 person-years of life per year.

Age-standardised death rates for all causes declined during the 10-year period of the study. The decline was greatest after the intervention, with a corresponding increase in life expectancy. In the 1991 population of Hong Kong, of about 5·8 million, person-years of life gained were 667 095 for males and 308 614 for females over the 2 years after the intervention, which represents an average lifetime gain, adjusted for the baseline trend, of 31 days (0·085 years) for each individual in the population, or 15 days per 10 μg/m³ reduction in SO_2 per year of exposure.

On the basis of age-specific death rates, the estimate of gain in life expectancy (after 15 years of exposure to the lower pollutant concentrations) for men age 25 years and older is 0·73 years per 10 μg/m³ reduction in SO_2. In an alternative approach described by Brunekreef,[8] using the 15-year relative risk of 1·18 obtained from our model of change in death rates, the estimate for a 25-year-old man is 2·58 years per 10 μg/m³ reduction in SO_2.

An appendix with further webtables and figures is available on our website.[19]

Discussion

After introduction of the air-quality intervention in Hong Kong, in addition to the 45% average reduction in SO_2 over 5 years, we noted that sulphate in respirable particulates had sustained reduction up to 2 years, but concentrations rose again and stabilised as part of a regional pattern of sulphate pollution in southern China. No comparable reductions or downward trends in the other main pollutants, PM_{10}, NO_2 and O_3, were recorded. These immediate changes in concentration of sulphur-derived pollutants were associated with the seasonal mortality cycle in the first year, and the estimated change in the proportional mortality trend based on 5-year analysis also suggested that reduction of SO_2 concentration had an important longer-term effect on death rates.

We reported an immediate reduction in cool-season deaths, which suggested that in the first year, many people survived who would have otherwise died. The rebound in cool-season deaths in the second year, followed by a return to the pre-intervention seasonal cyclical pattern, suggested that these later deaths arose in susceptible people whose death had been delayed by the air-quality improvement. This finding, which is the reverse of the postulated occurrence of harvesting and which was closely related to the reduction in sulphur oxides, lends support to the hypothesis that a proportion of the deaths

	Cool season increase in mortality (%) (95% CI)				
	All causes	Respiratory	Cardiovascular	Neoplasms	Other causes
Period					
Baseline					
(July, 1985, to June, 1990)	10·2 (9·5 to 11·0)	20·3 (18·4 to 22·2)	18·0 (16·6 to 19·4)	1·1 (–0·3 to 2·4)	4·2 (2·9 to 5·6)
Year 1					
(July, 1990, to June, 1991)	4·2 (2·5 to 5·8)	5·3 (1·2 to 9·4)	12·3 (9·2 to 15·4)	2·8 (–0·2 to 5·8)	3·7 (0·7 to 6·6)
Year 2					
(July, 1991, to June, 1992)	14·6 (13·0 to 16·2)	27·7 (23·9 to 31·5)	24·1 (21·2 to 27·1)	1·1 (–1·9 to 4·0)	3·6 (0·7 to 6·6)
Year 3					
(July, 1992, to June, 1993)	12·5 (10·8 to 14·1)	26·6 (22·8 to 30·4)	20·1 (17·0 to 23·2)	2·0 (–0·9 to 4·9)	4·9 (2·0 to 7·8)
Year 4					
(July, 1993, to June, 1994)	11·3 (9·6 to 12·9)	17·2 (13·3 to 21·0)	19·8 (16·7 to 22·9)	2·7 (–0·2 to 5·5)	6·8 (3·9 to 9·7)
Year 5					
(July, 1994, to June, 1995)	11·3 (9·7 to 12·9)	24·2 (20·4 to 28·1)	21·4 (18·4 to 24·4)	2·1 (–0·7 to 5·0)	5·1 (2·2 to 7·9)

Table 2: **Cool season increase in mortality and 95% CI for all ages after intervention compared with mean (baseline) for all causes, respiratory, and cardiovascular, neoplasms, and other causes**

associated with air-pollution episodes are in individuals who are frail and already have a short life-expectancy.

In addition to short-term seasonal fluctuations in death rates, we recorded a decline in the average annual proportional increase in deaths in the period after the intervention, which also provides evidence of a longer-term benefit from removal of sulphur. As with the early effect on seasonal deaths, the largest decline over 5 years was for respiratory deaths. Reduction in risk for overall mortality was greater in districts that had large reductions in SO$_2$ than in those that did not.

Differences in age-specific death rates before and after the intervention suggest that it led to an average gain in life expectancy for men aged 25–100 years of 0·73 years for 15 years' exposure per 10 μg/m^3 reduction in SO$_2$. For a man aged 25–29 years, the lifetime gain would be 1·14 years. Brunekreef[8] applied a relative risk of 1·10—derived from US cohort studies, with relative risk per 10 μg/m^3 for PM$_{10}$ ranging from 1·07[4] to 1·17[3]—to the

1992 life-table for people aged 25–100 years to obtain an estimated gain of 1·51 years for a 25-year old Dutch man with 15 years of exposure. This finding indicates the expected difference in life expectancy between populations living in polluted or clean air. On the basis of the relative risk of 1·18 from our Poisson regression, the comparable gain in life expectancy per 10 μg/m^3 SO$_2$ for a 25-year old Hong Kong man is 2·58 years. The short-term analysis of changes in risk of death could have underestimated the benefits, in terms of life expectancy, of the restriction on sulphur in fuel.

Further benefits arising from reductions in the other pollutants, including respirable particulates and the other gaseous pollutants, could be expected in addition to those derived from sulphur sources. The strong association between reduced risk of death and the acute fall in sulphur oxides contrasts strikingly with the conclusions of other analyses—based on time series and cohort studies—of SO$_2$ and deaths in the USA and the Netherlands. Schwartz[20]

	Average annual proportional change (%) (95% CI)*		Relative change (%) (95% CI) per year	
	Pre-intervention	Post-intervention	From pre-intervention to post-intervention period†	Intrapolated to 10 μg/m^3 change in SO$_2$.‡
All causes				
Age 15–64 years	0·65 (–0·01 to 1·31)	–1·16 (–1·83 to –0·48)	–1·75 (–2·98 to –0·50)	–0·89
Age 65 years and older	5·40 (4·93 to 5·88)	2·40 (1·96 to 2·83)	–2·81 (–4·20 to –1·39)	–1·44
All ages	3·50 (3·12 to 3·88)	1·20 (0·84 to 1·56)	–2·11 (–3·32 to –0·89)	–1·08
Respiratory				
Age 15–64 years	2·28 (0·12 to 4·44)	–3·36 (–5·64 to –1·07)	–4·80 (–8·28 to –1·18)	–2·47
Age 65 years and older	7·79 (6·75 to 8·83)	2·91 (1·97 to 3·85)	–4·17 (–6·59 to –1·69)	–2·14
All ages	6·55 (5·62 to 7·48)	1·88 (1·02 to 2·74)	–3·94 (–6·23 to –1·60)	–2·02
Cardiovascular				
Age 15–64 years	–1·33 (–2·78 to 0·12)	–3·12 (–4·64 to –1·59)	–1·64 (–3·95 to 0·72)	–0·84
Age 65 years and older	4·17 (3·36 to 4·99)	1·81 (1·04 to 2·57)	–2·44 (–4·20 to –0·65)	–1·25
All ages	2·79 (2·08 to 3·49)	0·77 (0·09 to 1·45)	–2·01 (–3·66 to –0·33)	–1·03
Neoplasm, without lung cancer				
Age 15–64 years	0·73 (–0·47 to 1·94)	–0·64 (1·90 to 0·63)	–1·34 (–2·95 to 0·30)	–0·68
Age 65 years and older	3·53 (2·16 to 4·91)	4·53 (3·44 to 5·64)	1·06 (–0·64 to 2·79)	0·54
All ages	2·04 (1·03 to 3·05)	2·16 (1·34 to 2·99)	0·17 (–1·08 to 1·44)	0·09
Lung cancer				
Age 15–64 years	–0·48 (–2·54 to 1·63)	–0·52 (–2·62 to 1·63)	0·17 (–2·67 to 3·08)	0·09
Age 65 years and older	5·41 (3·60 to 7·25)	3·00 (1·32 to 4·70)	–2·16 (–4·40 to 0·12)	–1·10
All ages	3·12 (1·82 to 4·43)	1·83 (0·50 to 3·19)	–1·08 (–2·89 to 0·77)	–0·55
Other causes				
Age 15–64 years	0·73 (–0·29 to 1·76)	–1·28 (–2·42 to –0·13)	–1·95 (–3·43 to –0·45)	–0·99
Age 65 years and older	3·99 (2·88 to 5·16)	3·53 (2·46 to 4·62)	–0·50 (–2·04 to 1·06)	–0·25
All ages	2·41 (1·51 to 3·31)	1·55 (0·70 to 2·41)	–0·85 (–2·06 to 0·37)	–0·43

*Estimate obtained from fitting of the Poisson regression model in the stratified pre-intervention and post-intervention period. †Estimated by the intervention by trend interaction term in the Poisson regression model. ‡Estimates derived from column 4, which show reduction in excess risk (relative proportional change) after the intervention. Reduction in excess risk was converted to be associated with 10 μg/m^3 by the log linear assumption.

Table 3: **Average annual percentage change in mortality and 95% CI before and after the intervention, with relative change in annual trend from before to after the intervention**

concluded that there was no association between SO_2 and death rates after modelling SO_2 and total suspended particles separately for season and year, and controlling for climatic and other factors. The estimated unconfounded effect of SO_2 on daily deaths was not significantly different from zero and particulates showed the strongest effect on daily deaths when their association with SO_2 was weakest. In the Netherlands[21] two approaches based on time-series analyses, and trends in death rates over periods when SO_2 concentrations varied, also led to the conclusion that SO_2 is not a causal agent in mortality associated with air pollution. Furthermore, criticisms of the hypothesis that respirable particulates are the main component of pollution mixtures that cause deaths have been extensively reviewed and refuted in view of new analyses of data from US cities, and associations between SO_2 and NO_2 and daily deaths are weak and inconsistent.[5] By contrast, strong associations with respirable particulates and cardio-respiratory deaths were reported. However, results of other time-series analyses have shown strong associations between SO_2 and daily deaths in Europe.[6,22] Associations between SO_2 and deaths[23] and hospital admissions[24] in time-series analyses in Hong Kong are closely similar to those in London, UK.[25,26]

In a time-series analysis on air pollution and deaths,[23] the strongest effects we noted were for gases including SO_2, rather than respirable particulates. Katsouyanni and colleagues[6] also recorded evidence for an independent effect of both SO_2 and particulates in 12 European cities. The effects for SO_2 were similar or stronger than for PM_{10}, and these researchers suggested that this effect might be the result of more complex pollutant mixtures and lower particulate concentrations in Europe than the USA; however in Hong Kong, respirable particulate concentrations are high and about twice those in London, UK.[24]

Public-health policy on air-quality improvement would be strengthened by better data on the effect of pollution on life expectancy.[7] Even if one pollutant greatly affects death rates, do increases in deaths from pollution episodes arise only in susceptible individuals with pre-existing disease, whose life expectancy is already short? The issue of mortality displacement is important for epidemiological studies based on time-series, because if susceptible people die early on in the pollution episode, death rates after the episode will be lower than expected. The resulting average death rate over time could fail to show an association between pollution and death rates.[27]

Time-series analyses have been used to investigate mortality displacement on different time scales after a pollution episode.[9,10] Results of these analyses suggest that although some harvesting takes place for pneumonia, the health effects from a pollution episode continue and actually increase over long periods for respiratory and cardiovascular disease. The conclusion of these analyses was that deaths were displaced by at least 2 months, but that the limitation of the time-series approach does not allow the effects beyond that period to be defined. Results of cohort studies suggest an effect of 1 year or more, and Künzli and colleagues[28] conclude that time-series studies underestimate deaths, and that the effect of air pollution should be based on prospective cohort studies.

The outcome of our study could be challenged on grounds of biological plausibility. The mechanism underlying the immediate health benefits arising from use of low sulphur fuel is unknown. SO_2 is a chemical irritant, but in support of an argument against a role for SO_2, Schwartz[20] cites the finding that 80% of ambient SO_2 is removed by the nose and exhaled. However, scrubbing efficiency could be reduced at low ambient concentrations, and penetration to the lungs is high with oral breathing, and little experimental data on the effects of usual ambient concentrations of SO_2 on healthy people is reported. At high concentrations, SO_2 alters nasal and tracheo bronchial mucociliary clearance rates in both human beings and animals.[29] Bronchoconstriction happens in people with asthma and in those with hyper-reactive airways, but after the air-quality intervention in Hong Kong, the decline in bronchial hyper-responsiveness in children without asthma or wheezing,[14] and chronic bronchitic symptoms in children[13] and adults,[30] provided evidence that important components of ambient pollutant mixtures derived from sulphur were greatly reduced.

However, the apparent benefits of the reduction in SO_2 could have been attributable to other combustion products that are not generated by low-sulphur fuels. Changes in concentration of SO_2 after the fuel regulation was introduced could simply be an indicator of other qualitative changes in fuel and products of combustion, with reduction in another unidentified agent that causes the health effects. Concentrations of PM_{10} were unchanged after the intervention, but SO_2 could be a modifier of the effect of respirable particulates. SO_2 is converted to sulphuric acid, which can be carried into small airways by respirable particulates and impair lung function in children,[31] but no monitoring data are available for free sulphuric acid across the period of intervention.

The benefits to health resulting from the 1990 industrial fuel intervention were achieved with only a moderate effect on overall production costs.[32] As a result of licensing controls, high sulphur fuels have been banned in Hong Kong since 1990, and in 2000 SO_2 concentrations were maintained at below 20 $\mu g/m^3$, more than 50% below pre-intervention concentrations. Use of ultra-low sulphur (0·005%) diesel fuel for public and private transport, conversions to alternative fuels, and tightening of legislation is continuing.

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