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Developing an Instrument for Assessing the Health and Economic Impacts of Air Pollution in Hong Kong

Final Report

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Submitted by

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Executive summary

In this study, we have developed a tool to assess the health and economic impact of air pollution in Hong Kong using the most up-to-date data on air pollution, health statistics, and health care costs that are available. This is a generic assessment tool which is capable of performing cost/benefit comparison for economic and health impacts of air pollution control measures. The users could make reference to the tool and decide on the parameters/pollutants to be considered and provide updates on some of the data/parameters based on the best available information as appropriate and to suit the purposes of specific tasks. A checklist of parameters to be reviewed/updated can be found in Section 6.3 of the report.

In this study, we first conducted a literature review of *health impact assessment* (HIA) and *economic impact assessment* (EIA). We also reviewed all local epidemiological studies and studies of long-term effect of air pollution on health in other countries to identify the most appropriate unit *relative risks* (RR) for the HIA and EIA. To update cost of visits to private general practitioners (GP), which forms part of the cost of illness attributable to air pollution, we also conducted a survey on GP charges for upper respiratory infections (URI) consultations.

Explanatory Note

1. *Health impact assessment: a combination of procedures, methods, and tools by which a policy, programme, or project may be judged as to its potential effects on the health of a population.*
2. *Economic impact assessment: a combination of procedures, methods, and tools by which the potential costs and values of the anticipated benefits of a proposed programme, policy or regulatory initiative may be compared; it reflects trade-offs inherent in alternatives and has been used in quantifying the hidden costs and benefits of policy options in environmental health and social development.*
3. *In public health, the term 'risk' is defined as the probability of having an adverse health outcome (e.g., a disease, an accident, or dying). A 'risk factor' is an attribute that increases the risk of a disease / death. For example, air pollution is a risk factor for heart and lung diseases and dying from these diseases. In epidemiology (the study of what causes diseases), the magnitude a risk factor is expressed as its relative risk (RR). The relative risk (also known as 'risk ratio') is defined as the ratio of the incidence of (or death from) a disease, in a population group that is exposed to a risk factor compared to the incidence in a group that is not exposed to that risk factor. Excess risk (ER) is defined as RR - 1 and is usually expressed as a percentage.*
4. *A measure of the importance of a risk factor in the population is the 'attributable fraction'. The population attributable fraction is the proportional reduction in population disease or mortality that would occur if exposure to a risk factor were reduced to an alternative ideal exposure scenario (e.g., an improvement of air quality). Mathematically, the attributable fraction is equal to (RR-1) / RR .*

We assumed that the mean annual concentrations of air pollutants (*particulate matter* with an aerodynamic diameter less than 2.5 μm, or $PM_{2.5}$ and nitrogen dioxide, NO_2) can be reduced to a hypothetical level (that we defined as the air quality guidelines promulgated by the World Health Organization in 2005). Taking the difference between these 'guidelines (at 10 $\mu g/m^3$ for $PM_{2.5}$ and 40 $\mu g/m^3$ for NO_2 respectively) and the current air pollutant concentrations experienced in Hong Kong (at 28.6 $\mu g/m^3$ for $PM_{2.5}$ and 52.7 $\mu g/m^3$ for NO_2 respectively in 2014), we estimated the '*attributable fraction'* of the health outcomes using the corresponding RRs.

Based on the annual mortality statistics and data on hospital admissions for cardiovascular and respiratory diseases (diseases that are causally associated with air pollution), and the estimated annual numbers of GP consultations for URI (also associated with air pollution), we then calculated the annual numbers of deaths, hospital admissions and GP consultations that are attributed to air pollution.

Particulate air pollution (from $PM_{2.5}$) is causally associated with 4,316 (95% CI: 1,519 – 7,413) premature deaths annually. An additional 1,993 (95% CI: 1,145 – 2,809) premature deaths are attributable to NO_2, giving a total of 6,308 premature deaths from both air pollutants (assuming a 30% overlap of their effects) every year. Expressed as *life-years*, 36,644 years of life are lost with every 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration. 66,494 life-years would be gained if $PM_{2.5}$ is reduced from 28.6 $\mu g/m^3$ (in 2012) to the WHO air quality guideline of 10 $\mu g/m^3$.

Explanatory Note

5. *Particulate matters are a complex mixture of extremely small particles and liquid droplet suspended in the airs. Particle pollution is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. $PM_{2.5}$ are fine particles with an aerodynamic diameter less than 2.5 micrometers (μm) and cause greater harm to health as they can reach the smallest airways of the lung. The main sources of $PM_{2.5}$ in Hong Kong are motor vehicles especially diesel engines, power plants and industries. $PM_{2.5}$ causes heart and lung diseases (including lung cancer) and deaths from these disease.*
6. *Nitrogen dioxide (NO_2) is an air pollutant that shares its common sources with $PM_{2.5}$. It also causes heart and lung diseases and deaths from these diseases.*
7. *A life-year is a unit that denotes one year of life lived by one person. The sum of years lost due to premature deaths in a population with different age groups can be expressed as x no. of life-years lost.*

$PM_{2.5}$ is also responsible for 2,731 (95% CI: 1,730 – 3,735) admissions for cardiopulmonary diseases (i.e., heart disease and other diseases of the circulatory system, and lung diseases) into public hospitals under the Hospital Authority (HA) in 2012. NO_2 is responsible for 1,882 (95% CI: 1,315 – 2,437) admissions for cardiopulmonary diseases. Based on two estimates of the number of GPs in Hong Kong, we estimate that annually, from 310,159 to 818,081 (mean = 564,120) GP visits for URI are attributable to $PM_{2.5}$, with a slightly lower number (301,373 to 794,906, mean = 548,140) for NO_2. 19,029 and 25,878 visits to general outpatient clinics (GOPC) operated by the HA are attributable to $PM_{2.5}$ and NO_2 respectively. For both hospital admissions and GP / GOPC visits, we use the higher number of estimates derived from the two air pollutants (instead of adding the sums together, because of possible overlap of effects).

We have used the average value of US$2.02 million (=HK$15.7 million) for the *value of a statistical life* (VOSL) of Hong Kong residents. This figure of VOSL is obtained from the average of a low and a high estimate of VOSL, at US$1.17 million (data from China) and US$2.87 million (data from WHO European Regional Office) respectively. The estimated annual economic impact (using the average VOSL estimate) is US$8.73 billion (=HK$67.8 billion), and ranges from US$5.05 billion (low VOSL estimate) to US$12.40 billion (high VOSL estimate), or HK$39.3 billion to HK$96.3 billion for $PM_{2.5}$ (for a 18.6 $\mu g/m^3$ change). After adding the effect of NO_2 (=HK$31.3 billion, per 12.7 $\mu g/m^3$ change), the total annual economic impact is HK$99.1 billion. As about 80% of deaths attributable to air pollution are aged 65 and above, the annual *loss of productivity* estimate from $PM_{2.5}$ (per 18.6 $\mu g/m^3$) is relatively low, at HK$127.0 million. The annual productivity loss attributable to NO_2 is HK$58.6 million, while the combined estimate is HK$185.5 million annually (about 0.2% of the average VOSL estimate).

Similar to findings elsewhere, *the direct costs of illness* (COI), from hospital admissions or GP visits, are much lower compared to the impact based on VOSL. We estimate an annual total of HK$53.81 million is spent on admissions to HA hospitals for cardiopulmonary diseases, with an associated productivity loss of HK$4.37 million. For GP visits, our estimate ranges from HK$77.54 million to HK$204.52 million attributable to $PM_{2.5}$, and HK$75.34 million to HK$198.73 million attributed to NO_2, using a lower and higher estimate of the number of GPs in practice. The mean estimate is HK$141.03 million for $PM_{2.5}$ (per 18.6 $\mu g/m^3$ change), and HK$137.03 million for NO_2 (per 12.7 $\mu g/m^3$ change). For clarity, the mean estimate is presented in Table (i). COI for GOPC visits are HK$7.33 million for $PM_{2.5}$ (per 18.6 $\mu g/m^3$ change), and HK$9.96 million for NO_2 (per 12.7 $\mu g/m^3$ change). Taking the larger estimate of the two pollutants (that for a 18.6 $\mu g/m^3$ change in $PM_{2.5}$), the total annual COI (private and public health care) for URI is estimated to range from HK$84.9 million

Explanatory Note

8. *Value of statistical life (VOSL): In economic terms, the value of a statistical life is the amount of money a person (or society) is willing to spend to save a life. Since there is no formal market for lives, the only way to measure the VSL is through indirect methods (e.g., surveys or observed human behaviour in risky environments). Understanding the value of life is important for government policies where citizens' lives are at risk or where the goal is to save lives. For example, if pollution abatement measures come at a societal "cost per life saved" does the societal benefit of saving a life exceed this cost? [Source: Maxwell School of Syracuse University: http://sites.maxwell.syr.edu/vsl/]*
9. *Loss of productivity is defined as the monetary loss resulting from absence from work due to illness or premature death among the economically productive sector of the population. In this study, we define this group as adults aged below 65 years earning an income equivalent to the median income reported by the Census and Statistics Department of Hong Kong.*
10. *The direct cost of illness is defined as the sum of hospital / outpatient costs (as reported by the Hospital Authority) and the fees charged by a private general practitioner (GP) for hospitalizations or clinical consultations.*

(i.e., lower estimated GP visits of HK$77.54 million + estimate of GOPC visits of HK$7.33 million) to HK$211.9 million (i.e., higher estimated GP visits of HK$204.52 million + estimate of GOPC visits of HK$7.33 million), with a mean of HK$148.4 million (i.e., mean estimate of GP visits of HK$141.03 million + estimate of GOPC visits of HK$7.33 million). The associated productivity losses range from HK$120.55 million (lower estimated GP visits, per 12.7 $\mu g/m^3$ change in NO_2) to HK$327.23 million (upper estimated GP visits, per 18.6 $\mu g/m^3$ change in $PM_{2.5}$) for private GP visits and from HK$7.61 million (per 18.6 $\mu g/m^3$ change in $PM_{2.5}$) to HK$10.35 million (per 12.7 $\mu g/m^3$ change in NO_2) for GOPC visits. The annual productivity loss from GP (mean estimate) and GOPC visits amounts to HK$233.26 million. Overall, the total annual COI and productivity loss from URI attributable to air pollution ranges from HK$216.20 million (lower GP estimate, per 12.7 $\mu g/m^3$ change in NO_2) to HK$546.69 million (upper GP estimate, per 18.6 $\mu g/m^3$ change in $PM_{2.5}$), using central RR and a single pollutant approach). The total COI (at HK$148.36 million, using the mean estimated GP visits) and productivity loss (at HK$233.26 million), is HK$381.62 million (taking the slightly higher value from $PM_{2.5}$). These estimates are about 3 to 4 times the annual COI (HK$53.81 million) and productivity loss (HK$4.37 million) for hospital admissions (HK$58.18 million in total). Table (i) summarises the health and economic impact of $PM_{2.5}$ and NO_2 at current levels against levels recommended by the World Health Organization 2005 Guidelines.

Table (i): Health and economic impact of $PM_{2.5}$ and NO_2 on Hong Kong

Health impact / Economic impact	Attributed to $PM_{2.5}$ (18.6 μg/m^3)	Attributed to NO_2 (12.7 μg/m^3)	Total *
No. of premature deaths	**4,316**$^{\phi}$	**1,993**$^{\phi}$	**6,308**$^{\phi}$
loss of VOSL	HK$67.8 billion	HK$31.3 billion	HK$99.1 billion
Productivity loss	HK$127.0 million	HK$58.6 million	HK$185.6 million
No. of life-years lost	66,494$^{\phi}$	30,703$^{\phi}$	97,197$^{\phi}$
No. of hospital admissions	**2,730**	**1,882**	**2,730***
Hospital cost	HK$53.8 million	HK$37.1 million	HK$53.8 million
Productivity loss	HK$4.37 million	HK$3.01 million	HK$4.37million
No. of GP visits (mean estimate)§	**564,120**	**548,139**	**564,120***
Direct cost of illness	HK$141.03 million	HK$137.03 million	HK$141.03 million*
Productivity loss	HK$225.65 million	HK$219.26 million	HK$225.65 million*
No. of GOPC visits	**19,029**	**25,878**	**25,878***
Direct cost of illness	HK$7.33 million	HK$9.96 million	HK$9.96 million*
Productivity loss	HK$7.61 million	HK$10.35 million	HK$10.35 million*
Total cost of mortality(VOSL) and illness (COI + productivity loss) from air pollution	**HK$68.24 billion**	**HK$31.72 billion**	**HK$99.55 billion***

* Instead of adding the two estimates, we use the higher estimate of two because of overlapping effects of $PM_{2.5}$ and NO_2 in the estimates of hospital and GP visits. For deaths, the overlapping effects have been adjusted, and a total number is presented.

$^{\phi}$ Rounded-off to whole numbers

§ In this table, we have presented the health impact and costs of GP visits based on our annual mean estimate of 564,120 GP visits. The attributable cases based on the lower estimate of GP visits (310,159) are about one third of that derived from higher GP estimate (818,081).

As reported in other studies, the health and economic impacts of air pollution are substantial both in terms of death tolls, hospital illnesses and minor respiratory diseases, and in dollar terms when using the VOSL as a unit of cost of mortality. While the validity of using VOSL has been debated, this is a conventional method that has been widely used in developed countries and in China. By contrast, the COI and loss of productivity approach (especially on mortality) can capture only a limited part of the wide spectrum of costs of ill-health and loss of life which all of us treasure. We consider that the use of VOSL is an appropriate approach, and recommend that the results be used for the assessment of the health and economic burden of air pollution as well as the benefits of air quality improvement. With further improvements and regular revisions (preferably in 5-year cycles) in the light of more comprehensive health and economic data, this study will provide the basis of evidence-based policy formulation.

Chapter 1: Introduction

1.1 Background

Air pollution is an important cause for mortality. In the 2010 study of global burden of diseases, particulate air pollution accounts for 3.1 million deaths and 3.1% of disability adjusted life years lost globally (Lim et al, 2012). This problem is especially serious in East Asia, where ambient air pollution is the 4th highest risk factor responsible for disease mortality. Globally, air pollution ranks as the 9th most important risk factor. In most developed countries in Europe and North America, it is customary to perform an assessment of the health and economic impact of air pollution. A properly conducted, scientifically valid health impact assessment (HIA) and economic impact assessment (EIA) enable policy makers to make the most appropriate choice out of several alternative options. The results the HIA and EIA also help to convince the public to accept and participate in the air pollution strategies promulgated.

In 2013, the Environment Bureau published a policy paper 'A Clean Air Plan for Hong Kong', which outlined a series of strategies to control air pollution and improve air quality in the years ahead. To facilitate the implementation and evaluation of these policies, it is necessary to collect local health and economic data and utilize scientific methods to develop an appropriate tool to quantify the health and economic impact on the community. An objective assessment of the public health improvement and the cost effectiveness of air pollution control measures requires the development of standardized methodologies to quantify the health and economic benefits of air pollution control strategies. The objective of this commissioned study by the Environmental Protection Department (EPD) is to develop an instrument that assesses the health and economic impacts of air pollution in Hong Kong using local health data, including mortality and morbidity statistics, air pollution concentrations and data on health care costs and valuation of illness avoidance.

1.2 Scope of Study

The scope of this study includes the following tasks:

(i) A literature review of the methodology of health impact assessment (HIA);
(ii) A literature review of the health risk estimates associated with short-term and long-term exposure to air pollution from local and overseas studies;
(iii) A literature review of the economic costs of air pollution in terms of health impact;

(iv) An assessment of exposure of the population to ambient and roadside air pollution in Hong Kong;
(v) Health economic analysis of the potential cost of air pollution; and
(vi) Develop a standardized methodology and a user-friendly calculation tool for evaluating and quantifying the adverse health outcomes of air pollution in Hong Kong and their associated cost.

1.3 Service Requirements

The following services will be provided in this study:

(i) A comprehensive review of HIA undertaken by local and overseas health authorities or institutions and international public health agencies including the World Health Organization (WHO), the United States Environmental Protection Agency (USEPA) and the Department for Environment, Food and Rural Affairs (Defra) of the United Kingdom, and the pros and cons of various HIA methodologies, to develop an HIA instrument in accordance with the best international practice;
(ii) A literature review of the health risk estimates of the impacts of air pollution in Hong Kong and overseas studies to determine the most appropriate unit health risk associated with short-term and long-term exposure to air pollution, using the best available evidence;
(iii) A literature review of the economic costs of air pollution in terms of health impact, and the recommendation of an appropriate approach to monetize these costs;
(iv) Based on (i) to (iii) above, to list the adverse health outcomes of air pollution relevant to HIA, and the short-term and long-term benefits to health from a reduction of air pollution for health economic analysis;
(v) Using air monitoring data of the EPD and other on-going studies on roadside air pollution in different districts and altitudes and personal exposure, to provide the best estimate of the exposure to pollutants from roadside and ambient air for HIA;
(vi) Based on the unit health risks in (ii) above and the estimated population exposed to air pollution in homes and work environments estimated in (v), to calculate the health impact to air pollution in Hong Kong;
(vii) Based on (iii), to develop a health economic model to monetize the costs of the health impact of air pollution in Hong Kong, including the costs of public hospital beds and intensive care unit beds per day, the cost of attendance of Accident and Emergency Departments of public hospitals, the unit cost of public outpatient clinic consultations from the Hospital Authority, and to estimate the true cost of

illnesses requiring different levels of care in the public hospitals and public outpatient care; to conduct a questionnaire survey to private doctors (with a sample size not less than 200) to obtain the median doctor consultation charges for general practitioner consultations in the private medical sector; to compute the indirect cost of illnesses in terms of sickness absence and productivity loss; to estimate the value of life using potential loss of income due to premature death; and to study any other non-health related costs of air pollution, including the impact on investment, tourism industry, and damage to buildings, wildlife and vegetations, pollution of potable water and soil through literature review;

(viii) Based on an understanding of the health impacts of air pollution, including on high risk groups such as children with asthma and those with chronic heart and lung diseases, multi-pollutant effects and seasonal effects, to develop a standardized methodology and provide a user-friendly calculation tool to quantify the health impact of air pollution in Hong Kong and their associated costs;

(ix) Using this tool, to estimate the health and economic benefits of air pollution control strategies that improve the air quality in Hong Kong.

1.4 Reference

Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, Amann M et al (2012) A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the global burden of disease study 2010. Lancet 380(9859): 2224–2260.

Chapter 2: Health Impact Assessment (HIA): A Literature Review

2.1 Definition

Health impact assessment (HIA) has been defined as 'a combination of procedures, methods, and tools by which a policy, programme, or project may be judged as to its potential effects on the health of a population, and the distribution of those effects within the population. HIA identifies appropriate actions to manage those effects' (WHO, 2002; Lee et al, 2013; Quigley, 2006). The HIA offers a framework, influenced by policy and regulation, that can be used to assess, justify, and manage projects that may potentially influence public health (Birley, 2011).

2.2 Health Impact Assessment and public policy

Although the modern HIA emerged and was developed in the 1980s and 1990s, the concepts on which it is based could be traced back at least as far as the 1800s, when improving public health – through enhancing living conditions – became an important issue for policy makers (Kemm et al, 2004; NRC, 2011). There has been increasing international attention on the potential for using HIA as a way to mainstream health into sector policies, as evidenced during the World Conference on Social Determinants of Health (October 2011) and the United Nations Conference on Sustainable Development (June 2012). A number of countries have adopted legislative frameworks and governance mechanisms to consider the impact of policies, programmes or projects on health. However, differences in political, socioeconomic and administrative settings lead to substantial variations in the use and institutionalization of HIA. There is limited research on the systematic use of HIA and the institutional processes that support or impede its use. Lee et al (2013) compared the institutionalization of HIA in nine (mainly middle- and high-income) countries and the European Union to gain a better understanding of the enabling and limiting factors that could then contribute to the identification of strategies for wider and more effective implementation of HIA. The following is quoted from Lee's report: 'An analytical framework and sample research questions were developed based on existing HIA literature and case studies. The framework covers five areas: degree of and mechanisms for institutionalization; political setting and context; framing and type of HIA; implementation, resource requirements and structures; and outcomes and conclusions. In-depth interviews were conducted with policy-makers, experts, public health officials and other stakeholders from Australia (South Australia), Canada (Quebec), Finland, Lithuania, the Netherlands, Slovakia, Switzerland, Thailand, the United States of America and the European Commission.

The findings from the interviews showed that *all countries have institutionalized HIA to a certain extent*. The degree of institutionalization varied within and across countries; yet there were similarities in the mechanisms used to achieve it (for example through Public Health Acts or establishment of research centres). Drivers for the institutionalization of HIA included recognition of the importance of and need for intersectoral action; increasing international movement towards health promotion and use of HIA; support from the health sector; experiences with the institutionalization of Environmental Impact Assessment (EIA); and advancement of HIA at the local level. The key factors enabling institutionalization of HIA were legislation (for example inclusion of HIA within Public Health Acts); political willingness; involvement of research communities; awareness of the inadequacy of EIA or other assessments in considering health; capacity and resources; availability of international committal documents and tools; and public participation. Challenges to institutionalization and systematic implementation included lack of clarity around methodology and procedures; narrow definitions of health; lack of awareness of relevance to other sectors; and insufficient funding and tools. Based on their experiences, key informants from countries proposed these core recommendations: embed HIA in national normative systems; clarify definition and operationalization of HIA and develop guidelines and methodological criteria; strengthen and build capacity for HIA practice; and improve cooperation between sectors…. To support progress in the institutionalization and systematic implementation of HIA and to build on the work that is already being done, WHO could continue to advocate the systematic assessment of policies, programmes and projects in countries that have not institutionalized any form of HIA; work to improve the definition of health (determinants and impacts) and cooperate with other agencies, institutions, and organizations to develop methodology and guidelines to strengthen and systematize the coverage of health in other forms of assessments; extend work with more countries to develop governance mechanisms for healthy public policy using HIA in other sectors; and establish a global network of centres to support HIA practice' (Lee et al, 2013)

2.3 Models of Health Impact Assessment

There are many models of health impact assessment (HIA), although to begin with, these largely fall into two camps (Kemm et al, 2004). The biomedical models mainly examine the underlying mechanisms of health and disease, as related to the influence of the environment; they stem from toxicological and epidemiological knowledge, and tend to involve quantitative analysis and modelling. The broader social models, arising as they do from the social sciences, place more stress on topics such as housing and employment, which also can

influence health. By their nature, the analyses used in these models are more qualitative. Nowadays, HIAs incorporate aspects of both kinds of model, and may also look at policy, the economy, and other possible health determinants.

2.4 The Health Impact Assessment Process

The following is a summary of the HIA process (Kemm et al, 2004; WHO, 2005; Birley, 2011).

1. Screening – determining whether a project needs HIA
2. Scoping – setting a framework and goals for the HIA – identifying potential health risks and benefits and affected stakeholders; defining geographical and time boundaries; finding data sources
3. Appraisal / risk assessment – collecting information; finding vulnerable communities; establishing the baseline level of health; assessing and analysing health risks
4. Reporting – prioritising health impacts and suggesting actions to mitigate them; preparing reports and recommendations
5. Dissemination – providing the results of the assessment to stakeholders, including those affected by the proposed project, and supporting the decision-making process
6. Monitoring and evaluation – collecting information and evaluating the impact or outcome of the project

2.5 Health risk assessment of air pollution

Health impact assessment (HIA) plays an important role in the formulation of air pollution policy and control measures. It provides environmental policy makers with quantitative and qualitative information about how any air pollution control policy, strategy, programme or project may affect the health of a community. In respect of the health consequences of air pollution levels, HIA aims to elucidate the effects of air pollution currently experienced by the population, and the improvement in health that might be expected through reductions in air pollution.

In the WHO Air Quality Guidelines Global Update Report, the overall estimated burden of disease due to outdoor air pollution may account for approximately 1.4% of total mortality, 0.5% of all disability-adjusted life years (DALYs) and 2% of all cardiopulmonary diseases (WHO 2006). Lim et al analyzed the global burden of diseases and injuries from 67 risk

factors and concluded that ambient particulate air pollution was one of the major risk factors for mortality, years of life lost and disability adjusted life years lost (Lim et al, 2012). Susceptible population subgroups include young children, the elderly and those with chronic heart and lung diseases. Also, certain regions of the world share a higher burden of disease, such as those heavily dependent on coal for fuel and residents of bigger cities exposed to high concentrations of traffic-related pollution (WHO 2006).

'To analyze quantitatively the impact on health of outdoor air pollution in a specific city, region or country, information is needed on air pollution concentrations and exposure, the population groups exposed, background incidence of mortality and morbidity, and concentration–response (CR) functions. The choice of which health outcomes to include in the assessment may be determined by the strength of available studies, the accessibility of health information, and the importance of the impact from a health and economic perspective. Most analyses conducted to date indicate that effects on mortality, particularly those relating to long-term exposure to air pollutants, tend to dominate the estimated economic effects' (WHO 2006).

2.6 Health risks Estimates and Unit Health Risks associated with Air Pollution

The association between air pollution and health is well established and the amount of literature on such studies is huge. In general, adverse effects on health and survival have been shown on short-term and long-term exposure to different types of air pollutants. Different levels of evidence have been established for different health outcomes. Higher risks of mortality and morbidities, in particular, from respiratory and cardiovascular diseases, have been shown on both short- and long-term exposure to air pollution (WHO 2006). In addition, evidence is accumulating that air pollution is also linked to other illnesses – adverse birth outcomes such prematurity, low birth weight as congenital malformations. There is also emerging evidence that long-term exposure to particulates, a key air pollutant in scientific research, might be linked with neurodevelopment in children and cognitive function, and other chronic diseases such as diabetes (WHO 2013a). Effects on a physiological attribute – lung function, has been well researched in cohort studies in South California (Gauderman et al, 2007, 2015), as well as in many other countries and cities including Hong Kong (Yu et al, 2001; He et al, 2010; Gao et al, 2013). However, lung function loss has not been used as a health outcome in HIA due largely to its uncertain impact on specific diseases and survival.

The following is a summary list of adverse health outcomes from exposure to different air pollutants for both the short- and long-term.

2.6.1 Health Outcomes associated with short- and long-term exposure to air pollution

2.6.1.1 Mortality

Among the 'criteria air pollutants', all have been shown to be associated with premature deaths. Most of the evidence of a link between short-term exposure to air pollution and mortality risk is derived from time series studies. In these studies, statistical models are constructed to study the association between daily changes in the concentrations of air pollutants and the risk of dying in different populations. The earliest time series studies on air pollution and mortality were published in the United States and Europe in the early and mid-1990s. There are many references in the literature. The first European studies were reported in a multi-centre study – the first 'Air Pollution and Health: a European Approach' (APHEA I) study. All these studies were reported in a Supplementary issue of the Journal of Epidemiology and Community Health in 1996. Studies in other countries, e.g., Canada, South Korea and China have been reported at different times. In Hong Kong, similar studies were reported in the early 2000s, separately by Prof. CM Wong of the University of Hong Kong (Wong CM et al, 2001) and the author at the Chinese University of Hong Kong (Wong TW et al, 2002). The most recent study on air pollution and mortality is the Public Health and Air Pollution (PAPA), a multi-city led by Prof. CM Wong (2010). Using air pollution data from the Environmental Protection Department, Wong et al showed that short-term exposure to nitrogen dioxide (NO_2), ozone (O_3), sulphur dioxide (SO_2) and particulate matter with an aerodynamic diameter less than 10 μm (PM_{10}) were all statistical significant risk factors for all-cause mortality in general, and more specifically, deaths from cardiovascular and respiratory causes. The findings in the PAPA study are broadly consistent with the earlier local studies, and with many overseas studies. In general, while most or all the 'criteria air pollutants' have been shown to be associated with air pollution, many US studies only focussed on PM, and many US researchers consider gaseous pollutants like NO_2 to be proxies of PM – the underlying "*true cause*" of mortality.

Explanatory Note:

Causation is a complex issue in epidemiology (the study of the causes of a disease). This is because the epidemiologist is often limited in his/her ability in collecting evidence on what causes a disease. For example, he/she often cannot perform an experiment on humans to prove that air pollution causes a disease, although in some experimental studies on volunteers, exposure to air pollutants have been shown to cause some changes in the respiratory system (which are not synonymous of having developed a disease). Hence, epidemiologists have to review and synthesize all available scientific evidence and determine whether the evidence is strong enough for him / her to conclude a cause-effect relationship, e.g., whether exposure to air

Explanatory Note (continued):

pollution causes a certain disease (e.g., chronic obstructive lung disease). The epidemiologist often has to rely on results from observational studies instead of experimental studies. While both studies might suffer from biases, observational studies have inherent problems with confounding factors that might affect the validity of the observation. Another problem in establishing a cause-effect relationship is that many diseases (e.g., lung cancer) are multi-factorial, i.e., they have more than one cause, so that it is not a straightforward matter to conclude that lung cancer is caused by particulate air pollution.

The association between long-term exposure to air pollution and mortality risk has been established from cohort (follow-up) studies, in which population groups exposed to different concentrations of air pollutants are followed-up for many years and their mortality risks compared. The earliest study was the Harvard Six Cities Study (Dockery et al, 1993). The most widely cited cohort study for HIA is the American Cancer Society Study by Pope et al (2002). After adjusting for confounding factors, the mortality risk (from all causes, cardiovascular and respiratory diseases and lung cancer), in populations with long-term exposure to higher concentrations of $PM_{2.5}$ is higher than those exposed to lower concentrations. Cohort studies provide the strongest evidence of a causal link between long-term exposure to air pollution and mortality risk.

2.6.1.2 Morbidities

Likewise, references on research of the effect of air pollution on different health outcomes are too numerous to be listed. A good account (with references) can be found in the Air Quality Guidelines, a Global Update 2005 (WHO 2006). In Hong Kong, the first comprehensive study on air pollution and hospital admissions was reported by the author (Wong TW et al, 1999). Hospital admissions for all respiratory and all cardiovascular diseases, as well as specific diseases – chronic obstructive pulmonary diseases and heart failure were significantly associated with NO_2, O_3, SO_2 and PM_{10}, while asthma, influenza and pneumonia are significantly associated with three of the four pollutants (i.e., except SO_2). The findings on O_3 were independently confirmed in a short report (Wong CM et al, 1999). Locally, there are many published reports on the impact of air pollution on respiratory illnesses in general, as well as specific respiratory diseases such as bronchial asthma and chronic obstructive pulmonary diseases (COPD) (Ko et al, 2007a; Ko et al, 2007b). Besides hospital illnesses, the author (Wong TW et al, 2006) conducted a time series study that demonstrated a significant association between NO_2, O_3, SO_2, PM_{10} and $PM_{2.5}$ and general practitioner (GP) consultations for upper respiratory tract infections (URTI). Earlier studies have also reported higher prevalence of respiratory illnesses and bronchial hyperreactivity (BHR) in schoolchildren who live in more polluted districts compared with those in less polluted districts (Ong et al, 1991; Tam et al, 1994; Yu et al, 2001).

Besides hospital admissions for cardiopulmonary diseases, several respiratory morbidities have been used in HIA in Europe and Australia (Kunzli et al, 1999; Department of Environment and Conservation (NSW), 2005) – the *incidence* of bronchial asthma attacks, the incidence of acute bronchitis among those aged below 15, the prevalence of chronic bronchitis in adults, and the number of days of restricted activity resulting from air-pollution related illnesses. Other effects such as reduced lung function and increased *prevalence* of respiratory symptoms (e.g., cough) have not been used for HIA and EIA, as their economic impacts have not been quantified.

Explanatory Note :

In epidemiology, incidence and prevalence are terms used to measure the frequency of diseases. Incidence refers to the number of <u>new</u> cases of a disease that occurs within a time period (usually one year). It is commonly expressed as a rate, i.e., how many cases of asthma per 100,000 in one year. The incidence of a disease is a measure of the 'risk' (=probability) of having that disease among the population (the 'population at risk').

Prevalence is a measure of the number of cases (new and old) that are currently present in a population. It is also expressed as a rate. Prevalence rate can be expressed either in terms of 'point prevalence' or 'period prevalence', depending on the time frame of the frequency count – whether the disease is counted "at one point in time" or during a specified time period. For acute illnesses of short duration, incidence is not too different from prevalence. For chronic diseases, the incidence and prevalence differs widely. In general, the two are related by the formula:

Prevalence = Incidence x duration of illness

2.6.2 Unit Health Risks

Unit risks of air pollution on health have been expressed as relative risks (RR) of developing the disease under study per unit increase in the concentration of an air pollutant. For the same diseases and air pollutants, these unit risks often differ quantitatively among studies conducted in different countries / cities, although the ranges of these estimates often overlap. In the performance of HIA, it is logical to use local data on unit health risk wherever available. This is because local characteristics in air pollution profile and exposure patterns determine the magnitude of risk, despite the similarities in human response to air pollution.

2.6.2.1 Relative risks from short-term exposure local time series studies

Table 2.1 shows the relative risks (RR) from short-term exposure to air pollutants that are recommended for HIA in Hong Kong, based on the latest and most comprehensive study on mortality and morbidity (as hospital admissions for cardiovascular and respiratory diseases – the PAPA study, and GP and GOPC consultations for URTI (Wong CM et al, 2010; Wong TW et al, 2006; Tam et al, 2014). [Note: the % excess risks (% ERs) quoted in the the PAPA Study, are converted to RRs: RR = ER (expressed as a decimal) + 1.

Table 2.1: Relative risk (95% confidence interval) of mortalities and morbidities attributable to a 10μg/m^3 increase in air pollutant concentration derived from time series studies in Hong Kong

Air pollutant	All-cause mortality (all ages)*	Cardio-vascular mortality*	Respiratory mortality*	Cardio-vascular diseases	Respiratory diseases	Upper respiratory tract infections	
						GP #	GOPC §
NO_2	1.0103 (1.0069-1.0137)	1.0138 (1.0075-1.0201)	1.0141 (1.0067-1.0215)	*1.0100 (1.0073-1.0126)	*1.0075 (1.0050 - 1.010)	1.030 (1.020-1.040)	1.01 (1.006 * 1.013)
PM_{10}	1.0051 (1.0023-1.0080)	1.0063 (1.0011-1.0116)	1.0069 (1.0008-1.0131)	*1.0058 (1.0036-1.0080)	*1.0060 (1.0040-1.0080)	1.020 (1.016-1.025)	1.005 (1.002 – 1.009)
$PM_{2.5}$	1.004097 (1.001806-1.006394)**	1.014151 (1.009940-1.018380)**	1.010416 (1.005256-1.015602)**	1.0066 (1.0036-1.0097)***	1.0097 (1.0065-1.0129)***	1.021 (1.010-1.032)	not available
SO_2	1.0091 (1.0040-1.0142)	1.0123 (1.0027-1.0221)	1.0131 (1.0021-1.0243)	*1.0098 (1.0057-1.0139)	*1.0013 (-1.0024-1.0050)	0 (0.987-1.013) **NS**	1.004 (1.000 – 1.008)
O_3	1.0034 (1.0002-1.0066)	1.0063 (1.0004-1.0123)	1.0036 (0.9967-1.0105) **NS**	*1.0012 (0.9988-1.0037) **NS**	*1.0081 (1.0058-1.0104)	1.025 (1.012-1.038)	1.009 (1.006 – 1.012)

References quoted in this Table:

*Wong et al, 2010: Wong CM, Thach TQ, Chau PYK, Chan EKP, Chung RYN, Ou C-Q, Yang L, Peiris JSM, Thomas GN, Lam TH, Wong TW, Hedley AJ 2010. Part 4. Interaction between Air Pollution and Respiratory Viruses: Time Series Study of Daily Mortality and Hospital Admissions in Hong Kong. In: Public Health and Air Pollution in Asia (PAPA): Coordinated Studies of Short-Term Exposure to Air Pollution and Daily Mortality in Four Cities. HEI Research Report 154, Health Effects Institute, Boston, MA. (Table 9, p301)
The web version of this report was posted at www.healtheffects.org in November 2010.

** ER of all-cause, cardiovascular and respiratory mortality from Prof. Wilson Tam, 2016, unpublished data, based on time series of $PM_{2.5}$ on all-cause mortality, 2001-2010.

***[ER of hospital admissions for $PM_{2.5}$, derived from Qiu et al. *Atmos Environ* 2013 and Dr. Qiu Hong, personal communication.]

#Wong et al, 2006:Wong TW, Tam W, Yu ITS, Wun YT, Wong AHS, Wong CM. Association between Air Pollution and General Practitioner Visits for Respiratory Diseases in Hong Kong. *Thorax* 2006; 61:585-591.[Available at: http://thorax.bmjjournals.com/cgi/content/abstract/thx.2005.051730v1]

Qiu H, Yu ITS, Wang XR, Tian LW, Tse LA, Wong TW. Differential Effects of Fine and Coarse Particles on Daily Emergency Cardiac Hospitalizations in Hong Kong. *Atmospheric Environment* 2013; 64:296-302.

§ Tam WWS, Wong TW, Ng L, Wong SYS, Wong AHS. Association between air pollution and general outpatient clinic consultations for upper respiratory tract infection in Hong Kong. *PLOS ONE* 2014; 9(1) e86913:1-6. Published 23 Jan 2014. Accessible at: http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0086913

2.6.2.2 Relative risks from long-term follow-up (cohort) studies

A unit health risk of mortality from long-term exposure to air pollution is not available from local studies. For the assessment of the health impact of air pollution, U.S. researchers have focussed on PM as a causative risk factor for all-cause mortality and cause-specific (cardiovascular, pulmonary, and lung cancer) mortalities. There are more data on mortality risk from long-term exposure to $PM_{2.5}$ than PM_{10}. Therefore, HIA using $PM_{2.5}$ is preferred by experts in their HIA studies (Kunzli et al, 1999; Fisher et al, 2002; Department of Environment and Conservation (NSW) 2005; COMEAP 2010). Dockery et al first published an RR of 1.26 (96% CL: 1.08-1.47), adjusted for smoking and other risk factors, for mortality from fine particulates ($PM_{2.5}$) in the Harvard Six City Study (Dockery et al, 1993). Most HIA studies performed in developed countries used the RR reported by Pope et al (2002) in the American Cancer Society cohort study. The RR of all-cause mortality was 1.06 (95% CI: 1.02-1.11). In a more recent 'extended analysis' of the American Cancer Society study (Krewski et al, 2009), a slightly higher RR of 1.078 (95% CI: 1.043-1.115) assuming a linear model for the RR of all-cause mortality from a $10\mu g/m^3$ increase in $PM_{2.5}$, whereas the log

model gives an even higher RR of 1.128 (95% CI: 1.077-1.183) from 5-15 μg/m^3 increase and a RR of 1.079 (95% CI: 1.048-1.112) for an increase from 10-20 μg/m^3. In the HRAPIE Report (WHO 2013b), an RR of 1.062, derived from meta-analysis of 11 cohort studies, was recommended. However, the 2015 London HIA study by King's College (Walton et al, 2015) still used Pope's RR as the unit health risk for assessing the impact of long-term exposure to $PM_{2.5}$. The choice of an appropriate RR of all-causes mortality from long-term exposure to $PM_{2.5}$ is important, as the magnitude of this RR influences the overall health impact assessment. Compared to the other health endpoints, all-cause mortality is the single most important outcome in terms of health and economic impact.

For long-term NO_2 exposure on all-cause mortality, an RR of 1.055 (95% CI: 1.031 – 1.080), derived from a meta-analysis of cohort studies on long-term air pollution exposure and cardio-respiratory mortality (Hoek et al, 2013), was recommended by the HRAPIE (WHO 2013b) experts. In the calculation of total health and economic impact of air pollutants, we have also included the effects of NO_2, assuming a 30% overlap of effects between $PM_{2.5}$ and NO_2, similar to the approach of the London study by King's College (Walton et al, 2015). In the HRAPIE Report (WHO 2013b), it is mentioned that the overlapping effect on mortality is likely to range from 0 to 33%. In our study, the RR was adjusted from 1.055 to 1.039 (95% CI: 1.022 – 1.056) to reflect this overlap.

2.6.3 References

Birley, Martin (2011). Health Impact Assessment: Principles and Practice. Abingdon: Earthscan.

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

DEC 2005. Department of Environment and Conservation (NSW). *Air Pollution Economics: Health costs of air pollution in the Greater Sydney Metropolitan Region*, Sydney 2005.

Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. *New England Journal of Medicine* 1993; 329:1753-59.

Fisher GW, Rolfe KA, Kjellstrom T, Woodward A, Hales S, Sturman AP, Kingham S, Petersen J, Shrestha R, King D. *Health effects due to motor vehicle air pollution in New Zealand, Report to the Ministry of Transport*, Wellington, 2002.

Gao Y, Chan EYY, Zhu Y, Wong TW. Adverse effect of outdoor air pollution on cardiorespiratory fitness in Chinese children. *Atmospheric Environment 2013;* 64:10-17.

Gauderman WJ, Vora H, McConnel R, Berhane K, Gililand F, Thomas D, Lurmann F, Avol E, Künzli N, Jerrett M, Peters J. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *Lancet* 2007; 369(9561):571-577.

Gaudermann WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, Chang R, Lurmann F, Gilliland F. Association of improved air quality with lung development in children. *The New England Journal of Medicine* 2015; 372(10):905-913.

He QQ, Wong TW, Du L, Jiang ZQ, Gao Y, Qiu H, Liu WJ, Wu JG, Wong A, Yu TSI. Effects of ambient air pollution on lung function growth in Chinese schoolchildren. *Respiratory Medicine* 2010*;* 104:1512-1520.

Hoek G, Krishnan R, Beelen R, Peters A, Ostro B, Brunekreef B, et al. Long-term air pollution exposure and cardio-respiratory mortality: a review. Environmental Health 2013; 12(1):43.

Kemm, John; Parry, Jane; Palmer, Stephen (Eds.) (2004). Health Impact Assessment: Concepts, theory, techniques, and applications. New York: Oxford University Press.

Ko FWS, Tam WS, Wong TW, Lai CKW, Wong GWK, Leung TF, Ng S, Hui DSC. Effects of air pollution on asthma hospitalization rates in different age groups in Kong Hong. *Clinical and Experimental Allergy* 2007; 37:1312-1319.

Ko FWS, Tam W, Chan DPS, Wong TW, Tung AH, Lai CKW, Hui DSC. Temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong. *Thorax* 2007; 62:779-784.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA III, Thurston G, Calle EE, Thun MJ. 2009. Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality. HEI Research Report 140. Health Effects Institute, Boston, MA.

Künzli N, Kaiser R, Medina S, Studnicka M, Oberfeld G, Horak F. *Health costs due to road traffic-related air pollution – an assessment project of Austria, France and Switzerland*. Prepared for the Third Ministerial Conference for Environment and Health, London, 1999.

Lee JH, Röbbel N and Dora C. Cross-country analysis of the institutionalization of Health Impact Assessment. Social Determinants of Health. Discussion Paper Series 8 (Policy & Practice). Geneva, World Health Organization, 2013.

Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, Amann M et al (2012) A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the global burden of disease study 2010. Lancet 380(9859): 2224–2260.

National Research Council (Committee on Health Impact Assessment) (2011). Improving Health in the United States: The Role of Health Impact Assessment. Washington DC: National Academies Press.

Ong SG, Liu J, Wong CM, Lam TH, Tam AYC, Daniel L, Hedley AJ. Studies on the respiratory health of primary school children in urban communities of Hong Kong. *Science of the Total Environment*

1991; 106:121-35.
Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 2002; 287(9):1132-41.
Quigley, R; den Broeder, L; Furu, P; Bond, A; Cave, B; Bos, R (2006). Health Impact Assessment: International Best Practice Principles. Special Publication Series No. 5. Fargo, USA: International Association for Impact Assessment (IAIA).
Tam AYC, Wong CM, Lam TH, Ong SG, Peters J, Hedley AJ. Bronchial responsiveness in children exposed to atmospheric pollution in Hong Kong. *Chest* 1994; 106:1056-60.
Tam WWS, Wong TW, Ng L, Wong SYS, Wong AHS. Association between air pollution and general outpatient clinic consultations for upper respiratory tract infection in Hong Kong. *PLOS ONE* 2014; 9(1) e86913:1-6. Published 23 Jan 2014. (Accepted: 17 Dec 2013). Accessible at: http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0086913
Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015 (p23). Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKingsReport14072015final.pdf
Wong CM, Ma S, Hedley AJ, Lam TH. Does ozone have any effect on daily hospital admissions for circulatory diseases? *Journal of Epidemiology and Community Health* 1999; 53:580-81.
Wong CM, Ma S, Hedley AJ, Lam TH. Effect of air pollution on daily mortality in Hong Kong. *Environmental Health Perspectives* 2001; 109(4):335-340.
Wong CM, Thach TQ, Chau PYK, Chan EKP, Chung RYN, Ou C-Q, Yang L, Peiris JSM, Thomas GN, Lam TH, Wong TW, Hedley AJ 2010. Part 4. Interaction between Air Pollution and Respiratory Viruses: Time Series Study of Daily Mortality and Hospital Admissions in Hong Kong. In: *Public Health and Air Pollution in Asia (PAPA): Coordinated Studies of Short-Term Exposure to Air Pollution and Daily Mortality in Four Cities.* HEI Research Report 154, Health Effects Institute, Boston, MA.
Wong TW, Tam WS, Yu TS, Wong AHS. Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. *Occupational and Environmental Medicine* 2002; 59:30-35.
Wong TW, Tam W, Yu ITS, Wun YT, Wong AHS, Wong CM. Association between Air Pollution and General Practitioner Visits for Respiratory Diseases in Hong Kong. *Thorax* 2006; 61:585-591.
World Health Organization (2002). Technical Briefing – Health Impact Assessment: A tool to include health on the agenda of other sectors. Copenhagen: WHO Regional Office for Europe.
World Health Organization (2005). Health Impact Assessment Toolkit for Cities, Document 1: Vision to Action (Background document: concepts, processes, methods). Copenhagen: WHO Regional Office for Europe.
World Health Organization (2006). *Air Quality Guidelines Global Update 2005*. World Health

Organization Regional Office for Europe, Copenhagen.
World Health Organization (2013a). Review of evidence on health aspects of air pollution – REVIHAAP Project. Technical Report. World Health Organization Regional Office for Europe, Copenhagen.
World Health Organization (2013b). Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration-response functions for cost-benefit analysis of particulate matter, ozone and nitrogen dioxide. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf
Yu TS, Wong TW, Wang XR, Song H, Wong SL, Tang JL. Adverse effects of low-level air pollution on respiratory health of school children in Hong Kong. *Journal of Occupational and Environmental Medicine* 2001; 43:310-16.

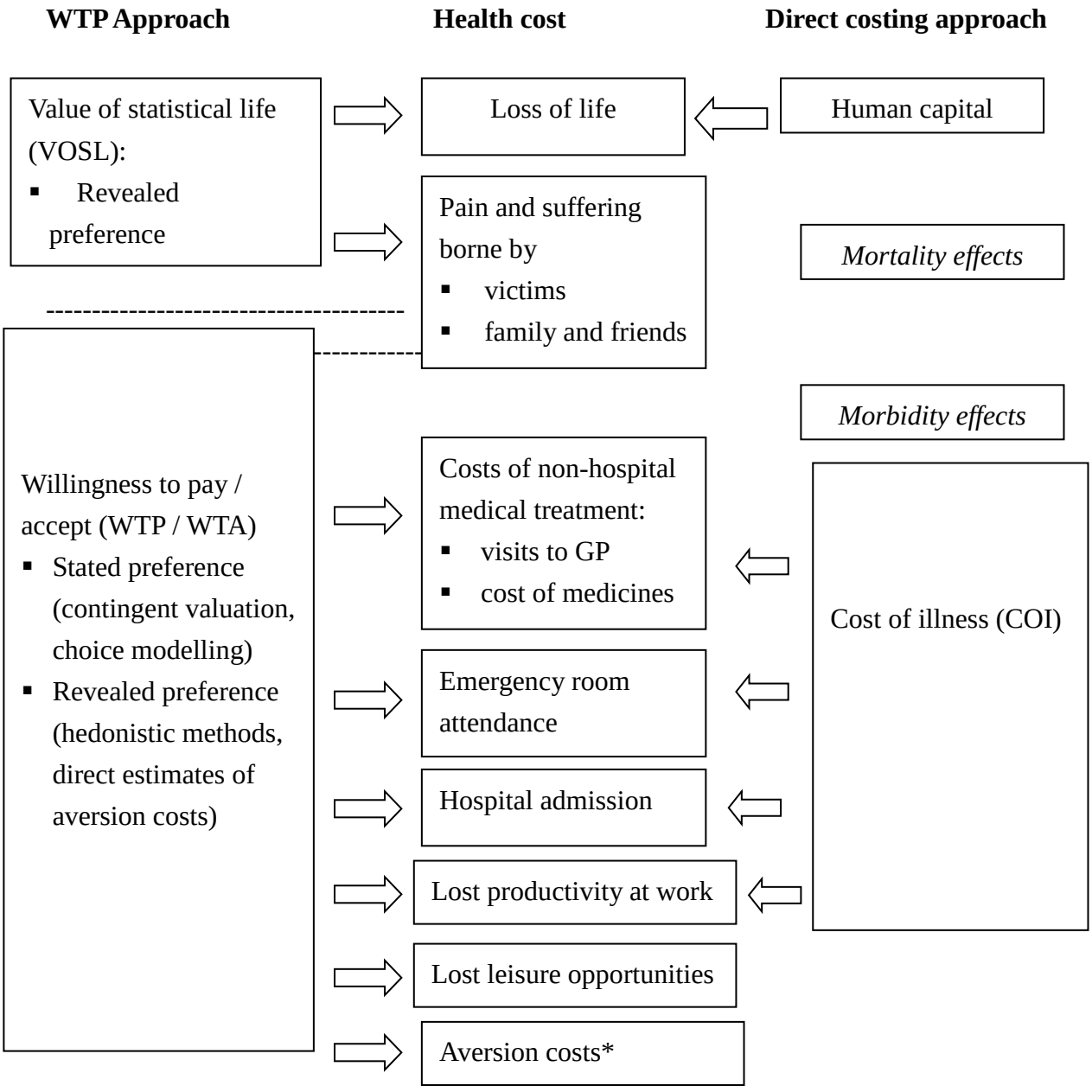
Chapter 3: Economic Impact Assessment (EIA)

Economic impact assessment (EIA) refers to the valuation of health impacts caused by air pollution. EIA is important in air pollution as the latter produces 'external costs' that are not borne by the polluter, but by those whose health and life are being affected. Although it is not a legal requirement to conduct EIA in most countries, an economic evaluation of policy options has been required under the U.S. Environmental Protection Agency regulations for rules and regulatory actions that have a "significant impact – those with an annual effect on the economy of US$100 million or more (USEPA 2016), or adversely affect in a material way the economy, a sector of the economy, productivity, competition, jobs, the environment, public health or safety, or State, local or tribal governments or communities. In many European countries, it is customary to perform EIA studies for major developmental projects. There are two approaches (i) cost of illness (COI) that assesses the direct medical costs of illness, such as diagnosis and treatment for these illnesses, and indirect costs such as the loss of productivity, and (ii) the '*willingness to pay*' (WTP) approach, that makes use of surveys to assess an individual's willingness to pay to avoid specified illnesses or loss of life. The WTP approach has been adopted by the European and Australian (Sydney) HIA studies, as it includes a comprehensive list of values that include the direct and indirect costs of illness, and in addition, other components such as pain and suffering from illnesses, perceived quality of life. Premature death has also been valued using the 'statistical value' of a life lost as a unit of measurement. In general, the valuation using the WTP approach is typically several times higher than that using the COI approach. Figure 1 illustrates the two approaches and the components they measure.

Explanatory note:

Willingness to pay (WTP): In health economics, 'willingness to pay' refers to the maximum sum an individual (or a government) is willing to pay to acquire some good or service, or the maximum sum an individual (or government) is willing to pay to avoid a prospective loss. It is usually elicited from stated or revealed preference experiments. In normative analyses based on user preferences, it should be remembered that willingness to pay is highly conditioned by the subjects' ability to pay. On the other hand, 'willingness to accept' (WTA) is the minimum amount of money that a person is willing to accept to abandon a good or to put up with something negative, such as an illness, or air pollution. It is equivalent to the minimum monetary amount required for sale of a good or acquisition of something undesirable to be accepted by an individual. Measures of willingness to accept typically reveal higher values than measures of willingness to pay. Unlike WTP, WTA is not constrained by an individual's wealth. (Source: Wikipedia: https://en.wikipedia.org/wiki/Willingness_to_accept;Dictionary.comhttp://www.dictionarycentral.com/definition/willingness-to-pay.html)

Figure 1: Valuation methodologies and coverage of health costs (Source: Centre for International Economics, 2001)



* Aversion costs can be associated with both mortality and morbidity effects.

Reference: Centre for International Economics 2001. Health costs of transport emissions in Sydney – Consultancy 2 – Economic Valuation Methodologies. Prepared for the NSW Environmental Protection Authority, Sydney.

3.1 Techniques in Valuation of Morbidity

3.1.1 Willingness to pay: stated preference method

There are three approaches to the WTP to valuate air pollution-related illnesses: (i) Contingent valuation (CV); (ii) Choice modelling, and (iii) Conjoint analysis. For CV, the values estimated are contingent to a hypothetical situation presented to the subjects in the survey. The drawback is that it is often difficult to determine in monetary terms the amount that one is "willing to pay to avoid" in a hypothetical situation. The choice modelling method presents respondents with different sets of choices and asks them to choose one option from several alternatives, one of which must be in monetary terms. This enables the respondents to "trade off" one choice with another, to determine the most preferred options. In conjoint analysis, respondents are asked to rate their choices instead of choosing an option. Respondents are not forced to make a decision in a market environment. Hence, conjoint analysis is not considered as a valid measure of welfare.

3.1.2 Willingness to pay: revealed preference method

There are two common approaches: the hedonistic pricing method and 'averting behaviour'. Hedonistic pricing relies on the availability of market data to identify implicit prices, e.g., of clean air. It assumes that the total price of a good or service can be considered as an aggregate of different attributes. For example, house prices are affected by several variables, including a district of good air quality. On the contrary, averting behaviour describes actions by individuals to mitigate or avoid health impacts, e.g., purchase of air purifiers, or behavioural changes, e.g., staying indoors, or moving to a cleaner location. This provides an indication of the costs a person is willing to pay to avoid certain health outcomes.

3.2 Valuation of mortality

3.2.1 Willingness to pay: value of a statistical life (VOSL)

The value of a statistical life (VOSL) is an aggregate measure of a community's willingness to pay to reduce the risk of premature mortality. It is a WTP-based measure, so to the extent that people take into account the pain and suffering of family and relatives, these costs will be reflected in the WTP estimate that they report and therefore in the overall VOSL measure. (Centre for International Economics, 2001).

‘VOSL is the valuation of small changes in the risk of death. This is an appropriate measure because any change in public policy will generally result only in small changes in the health of an individual or in the risk of a major change in that health status. VOSL is calculated as follows:

VOSL = average individual WTP for risk reduction / observed change in risk
= δWTP / δr (where r – risk of dying)

WTP for risk reduction can be estimated by either revealed preference or state preference methods. Revealed preference methods include the compensating wage differential approach (hedonistic method that assumes that higher wages are paid for more risky occupations to compensate for the higher risk of injury or death) and averting behaviour studies. A large number of studies have attempted to estimate VOSL, and these estimates are commonly applied to the valuation of a fatal risk change.’ (Department of Environment and Conservation (NSW) 2005).

3.2.2 Direct costing method – the human capital approach

This approach only assumes life as a productive capital and estimates its value in terms of discounted future earnings lost. The human capital approach uses the labour market as a surrogate for a non-existent market for human life (Commonwealth Department of Finance, 1991). It does not aim to capture the full costs associated with the death of a person. The pain, suffering and fear felt by the individual or those left behind are not considered. This approach does not take account the value that individuals place on their own lives, or the value of non-work activity and other non-tangible elements of human life. This approach therefore tends to underestimate what people are willing to pay for reducing the risk of premature death (Centre for International Economics, 2001).

3.2.3 Value of life years (VOLY) and quality adjusted life years (QALY)

The value of life years (VOLY) approach attempts to adjust the VOSL for the age at death (i.e., to reflect the change in life expectancy). This method assesses the value that an individual place on a change in life expectancy – a gain or loss in life-years lived. The quality adjusted life years (QALY) approach attempts to weight the life expectancy of an individual with a perceived health status (ranked from zero to one, with zero equal to death and one

equal to perfect health). Essentially, the QALY estimates the effects on health status before death. Both methods depend on a VOSL estimate.

Both methods have appeal by valuating the life according to age of death and change in health status among those affected. The major criticism of these approaches is that the assumption that WTP decreases with increasing age has not been substantiated in research. On the contrary, literature has shown that WTP shows an inverted U-shape, with elderly people's WTP to reduce the risk of premature death significantly higher than that suggested by the VOSL approach (Centre for International Economics, 2001).

3.3 Justification for the WTP approach in valuation of life

In the WHO Report (2015), justifications for using WTP as a standard method in the derivation of VOSL is described below: 'In the language of present-day economics, which has developed far beyond but nonetheless remains descended from the tradition of Quesnay and Smith, *value* is a measure of the things that individuals in their millions value in the ordinary sense of the word, and *cost* is a measure of their loss, whether absolutely or as a means of securing other valuable things…. These things include *consumption, leisure*, *health* and *life*. The cost of mortality is by far the *largest* component of the cost of air pollution. The impact of premature deaths of working-age people from air pollution on the national accounts will be through the loss of labour inputs to production and the outputs of it. Those responsible for measuring, analyzing and forecasting changes in gross domestic product (GDP) will have an interest in measuring this impact. Clearly, however, a calculation that stops counting at retirement age and implicitly places a zero value on the death of a person of 65 years or over will yield a very different estimate from the economist's estimate of the value to the individual. Even before the point of death, there are different calculations at work addressing different features of the same reality: *counting the lost output as a result of the patient's absence from work is not the same as counting the patient's own loss* … None of this is to deny the validity, importance or policy relevance of accounting, including national accounting. But the information yielded needs to be considered separately from the information on economic costs. Thus, there is a case for bringing to the attention *of decision-makers simultaneously, side-by-side, both the economic cost estimates of air pollution and the estimates of its direct impact on GDP*…. Present-day economics possesses a standard method by which to measure the cost of mortality at the level of society as a whole: VOSL, as derived from aggregating individuals' willingness to pay (WTP) to secure a marginal reduction in the risk of premature death. Despite its unfortunate name, suggesting a monetary judgement on the worth of an individual life, this method is safely

grounded in economic first principles, seeking to aggregate the valuations by individuals of the value to individuals.' (WHO 2015).

As WTP depends on surveys of a hypothetical scenario, the validity of its estimates has often been criticized by conventional economists. However, the valuation of life using the WTP approach is comprehensive compared to the obvious deficiencies of the human capital methodology. VOSL estimated by WTP (as well valuations of morbidities) has been used by health economists in their research for decades. The advantage of assigning a monetary value to life and health is that it serves as a common denominator to assess the health impact of air pollution (as well as other environmental pollutants), by comparing the costs and benefits of different policy options for air pollution control. It should be noted that there is no standardized methodology by WHO to valuate morbidity. This depends on the health outcomes to be determined, the medical expenditure to treat these outcomes, which often vary widely across countries, and the lack of data on WTP to avoid these health outcomes. COI of selected health outcomes is often the only method used. However, it is important to note that *mortality is the dominant component of the total cost*. In the WHO Report, the chosen indicative estimate for the additional cost of morbidity – as shown by the OECD (OECD, 2014) – about 10%. This implies that morbidity constitutes approximately 9% of the estimated total cost of health impacts from air pollution, with mortality accounting for about 91% of the total.

3.4 Economic data relevant to HIA and EIA study in Hong Kong

3.4.1 Cost of morbidity

For the direct 'cost of illness' approach, we use cost data on hospital beds and general outpatient consultations published by the Hospital Authority. For private outpatient attendances, we conducted a survey of private general practitioners (GPs) in Hong Kong to obtain the average consultation fees that they charged for upper respiratory infection consultations in 2015 (See 3.4.2 below). To estimate the indirect cost due to loss of productivity from air pollution-related illnesses, the median income published by the Census and Statistics Department will be used. There are no local data on WTP for hospital illnesses. Direct costs of hospital illnesses and outpatient services have been published by the Hospital Authority, the largest public provider of hospital services in Hong Kong.[1] As costs of air

[1] The Hospital Authority provides about 90% of all hospital beds in Hong Kong. The rest is provided by the private sector.

pollution-related illnesses – cardiopulmonary diseases requiring hospital care, and upper respiratory infections that are managed in general outpatient services – will be used in HIA, the costs that are relevant to our HIA study are listed in Table 3.1 below:

Table 3.1: Charges for 'non-eligible persons'[2] for services provided by the Hospital Authority and charges by private general practitioners for upper respiratory infections consultations (derived from our survey, described in 3.4.2 below)

Service	**Fees**
*Provided by Hospital Authority**	
Accident & Emergency	$990 per attendance
In-patient (general hospitals)	$4,680 per day **
In-patient (psychiatric hospitals)	$1,940 per day
Intensive care ward/unit	$23,000 per day
High dependency ward/unit	$12,000 per day [N1]
Specialist out-patient (including allied health services)	$1,110 per attendance
General out-patient	$385 per attendance
Derived from survey of doctors (2015)	
Median charge by private general practitioner per consultation	$250 (Range: $120 - $800)

* Source: Hospital Authority, Fees and Charges (based on data published in Hong Kong Gazette 2013) Accessed at: https://www.ha.org.hk/visitor/ha_visitor_text_index.asp?Content_ID=10045
** Revised to $4,910 per day in 2015-16.

3.4.2 Survey on the consultation fees charged by private medical practitioners

We collected data on fees charged for outpatient medical consultations by private general practitioners from the most recent survey by the Hong Kong Medical Association (HKMA). This survey covered 620 private doctors, including 370 specialists (including family physicians) and 233 non-specialist medical practitioners. We have also conducted a survey of private medical doctors. Details are presented below:

3.4.2.1 Background of survey

[2] Charges to non-eligible persons are not subsidized by the Government and generally reflect the full costs of medical services. Eligible local residents pay much lower (subsidized) fees, e.g., hospital charges amount to $50 for admission and $100 for daily maintenance, inclusive of all medicines, investigations and meals. Attendance for general outpatients is $45 per consultation.

Under section 4.7(b) of the Tender, we are required to perform a questionnaire survey of consultation charges by private doctors (with a sample size not less than 200) to ascertain the direct health care costs to the treatment of air pollution-associated respiratory illnesses. In our study of air pollution and GP consultations in 2000-02, (a study commissioned by the Environmental Protection Department), we demonstrated a statistically significant, positive association between air pollution and GP consultations for upper respiratory tract infections (URTI). Although the association between air pollution and asthmatic attacks are well-known, we did not detect a significant association in our study owing to the relatively small number of consultations for asthma, in contrast to the much larger numbers of URTI consultations. Accordingly, we sought the consultation charges for an episode of URTI instead of asthma in our survey.

Our initial approach was a web-based questionnaire survey of a network of private family physicians who are honorary tutors of the School of Public Health and Primary Care. The sampling frame was, however, with 176 eligible doctors on our list. The response rate was low, at 16.5%, with 29 private doctors completing our questionnaire. Hence, we used the results only as a guide, to be compared with that from the Hong Kong Medical Association (HKMA), which conducts surveys of GP charges among its members once every 4 years. The most up-to-date survey was carried out in 2014 (Hong Kong Medical Association Newsletter, 2014), and our telephone survey that aims to cover the requisite sample size of at least 200.

3.4.2.2 Methods

We used the Primary Care Directory, a list of primary health care providers in the private sector published by the Department of Health (available at: https://apps.pcdirectory.gov.hk/PUBLIC/MAIN/MAIN.ASPX) as a sampling frame, and systematically sampled over 300 doctors (allowing for replacements for invalid samples / no answers[3]) from each of 18 districts on the list, with a sampling fraction of 1 in 5 and proportionate sampling reflecting the numbers of doctors listed in the districts. Our target sample size was at least 200 doctors, as stipulated in the Study. As the Directory makes no distinction between generalists from specialists, we eliminated specialists[4] from our sample

[3] No answers were different from non-response. A call with no one answering was regarded as an invalid sample and a replacement sample would be used. An unsuccessful response was one which the respondent did not provide the information we sought.

[4] Most GPs who are not specialists in other specialties than family medicine describe themselves as family physicians, whether they possess the specialist qualification (Fellow of the Hong Kong College of Family Physicians) or not. Some

as far as possible except for paediatricians (who sees URTI among children) and family physicians with specialist qualifications. From 16 to 21 April 2015, telephone calls were made by a trained interviewer to the clinics and the following information were sought: charge per consultation for 'a flu', whether the charge includes medicine prescribed, and if so, the number of days of medicine given, and the specialty of the doctor. Calls with no replies / no dialling tone were replaced by another sample on the reserve list. Unsuccessful interviews after contact was made were recorded as such.

3.4.2.3 Results

Our findings are presented in Table 3.2. The mean consultation charge (all districts) was $265 with a median of $250 and a range from $120 to $800. Most doctors' consultation charges were inclusive of medicine, with an average of 2.7 days, a median of 3 days and a range from 2 to 7 days. 5 of 265 (1.9%) charged consultation fees and medicines separately. Most were family physicians, but we did not enquire whether the doctor was a registered specialist in family medicine or general practitioner. 13 were paediatricians, and there was a respirologist, a geriatrician, an endocrinologist and a nephrologist. All of them saw patients with respiratory illnesses. There are variations in charges between districts. In general, highest charges were observed in Central and Western, Wanchai, Yau Tsim Mong and Islands[5] at an average of over $300 (ranging from $301 to $342). The median charges in these districts, except Islands, were likewise higher than in other districts (ranging from$270 to $295). The non-response rate was very low (with 4 out of 263 or 1.5%).

In our telephone survey, we did not seek the number of days of sick leave given for an upper respiratory illness.

3.4.2.4 Discussion

Our response rate was much higher than in other surveys of medical consultation charges. An important reason was that our interviewer posed as a potential customer. Hence, she was able to elicit a very high response rate. By contrast, our web-based survey could only elicit a response rate of 16.5%, similar to the HKMA survey of 15.5%. The latter survey has the advantage of a much larger sample size, with 620 respondents. However, a low response rate implies there might be considerable selection bias in the results. A major concern about formal surveys is the under-reporting of the fees charged. Compared with the HKMA survey

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paediatricians have qualifications in both paediatrics and family medicine.

[5] The average charge in Islands was affected by one high value. The median differed little from other districts.

(median $240 for 2 days medicine and $325 for 3 day's medicine), our findings were similar, even though our sample consists of fewer specialists – 60% of the HKMA respondents were specialists, whereas only 13 (6%) of our respondents were specialists outside family medicine. Our web-based survey of 29 family physicians (many of whom were specialists in family medicine) yielded a mean consultation charge of $384, and a median charge of $280. The mean value was affected by extreme values, while the median was somewhat higher than our median of $250. (See Table 3.3.) However, our web-based sample was too small for any scientific interpretation, and only served as a reference to assess the approximate amount of the consultation charges by private doctors. We consider that our approach using direct questioning of the charges was more reliable in finding out the true fees charged, as the enquiry was made by a potential client without the clinic's awareness that we were conducting a survey.

Table 3.2: Consultation fees (in HK$) charged by private doctors (n=263) by district, using telephone interview

	No. of clinics	Consultation fees (HK$)				No. of refusals	Mean days of medicine	Median days of medicine
		Mean	Min	Max	Median			
HK East	22	262.73	185	360	260	0	2.8	3
HK South	8	273.75	180	400	255	0	2.6	2.5
Island	5	342	200	800	210	0	2.8	3
Kwai Tsing	15	224	180	280	220	0	2.5	2.5
Kwun Tong	21	246.19	150	320	250	0	2.8	3
Central & Western	13	300.772	160	800	270	0	3	3
Kowloon City	10	246.5	150	320	247.5	0	2.7	3
Shatin	18	274.72	220	320	280	0	2.8	3
Taipo	12	273.33	120	700	240	0	2.5	3
Yuen Long	22	270	190	650	240	0	2.8	3
North	8	238.75	200	290	240	0	2.8	3
Sai Kung	12	259.17	200	320	260	0	2.9	2.5
Shamshuipoo	15	234	190	300	240	0	2.6	3
Tsuen Wan	16	243.75	190	590	220	1	2.5625	3
Tuen Mun	16	235.625	160	360	227.5	1	2.5625	3
Wanchai	10	328	220	500	287.5	1	3.4	3
Wong Tai Sin	12	227.5	190	280	225	0	2.5833	2.75
Yau Tsim Mong	28	311.6071	170	600	295	1	2.6667	3
All districts	263	264.962	120	800	250	4	2.736641	3

Table 3.3: Consultation fees (HK$) charged by private doctors using web-based survey (n=29)

	Acute respiratory illness	Asthma or COPD
Mean	$383.8	$450.7
Median	$280	$360
Min	$200	$220
Max	1000	1000

3.4.3 Cost of mortality

Like morbidity, few local WTP data on air pollution are available to estimate the economic costs of premature deaths. Since VOSL is the single most important item (>90%) in the cost estimate, we have reviewed the literature on VOSL to identify a value appropriate for Hong Kong. Viscusi and Aldy (2003) published a paper that reviewed market estimates of VOSL in both developed and developing countries, based on earlier published studies. In developed countries, studies by different researchers with different sources of data (e.g., labour data, accident statistics, insurance and injury compensation data), in the same country reported a wide range of VOSL that are much higher than that from studies conducted in developing countries. The U.S. reported a range from US$3.8 – $9.0 million, median = US$7.0 milllion (converted to year 2000 dollar value). The range for Canada was from US$2.2 – $21.7 million (also 2000 value). The corresponding values form Japan, U.K. and Australia were: US$9.7 million, $4.2 - $74.1 million, and $4.2 – 11.5 million respectively. VOSLs in developing countries are: US$0.8 million for South Korea, US$1.0 – 4.1 million for India, US$0.2 – 0.9 million for Taiwan, and US$1.7 million for Hong Kong. VOSL estimates from selected countries published in the critical review are summarized in Table 3.4. The Hong Kong VOSL value was based on a local study (Siebert and Wei,1998) that used the 'hedonic wage methodology', and adjusted to year 2000 dollar value. The original VOSL reported by Siebert and Wei was HK$10.8 million (about US$1.4 million) and was based on an estimate of wage compensation for fatal jobs in Hong Kong, using job fatality data. This estimate was much higher than workers' compensation for permanent total incapacity (with a maximum of HK$1.728 million in 1996). We also made reference to economic data used by the World Health Organization Regional Office for Europe (WHO, 2015). The baseline VOSL is US$3 million. WHO made adjustments in countries of the European Region, based on their per capita gross domestic product (GDP). VOSL ranges from the lowest estimate of US$0.44 (Tajikistan and Uzbekistan) to US$6.28 (Luxembourg) in 2010. Data from other countries, such as the U.S. ($10 million), the United Kingdom (US$3.55 million) will also be used as references. The Australian EIA study (Department of Environment and Conservation (NSW) 2005) was based on a lower estimate of A$1,004,000 (US$722,880) and an upper estimate ofA$2,500,000 (US$1,800,000).

VOSL estimates in Asian countries are much lower except Japan and one study result in India (Shanmugan 2001). The average VOSL in China has been estimated as RMB795,000 or US$119,250 (Wang & He, 2010). In another study by Chen et al (2010), an average value of RMB$1 million or US$150,000 (valued in 2006) for premature death, published by World Bank in 2007 [6], was used. The VOSL estimates can be considered with reference to the per

[6] The World Bank 2007 report made reference to several studies, and used the value reported by Krupnick et al

capita GDP of these countries (Table 3.5). For example in 2014, the per capital GDP in the U.S. is US$46,405.26; in the U.K., US$40,967.70; Singapore, US$38087.89; Hong Kong's per capita GDP, at US$34,222.29, is 8.89 times that of China, at US$3,865.88. If we use the more conservative VOSL estimate of RMB795,000 in China (Wang & He, 2010, World Bank Paper), and adjust this amount by the ratio of per capita GDP of Hong Kong to China (8.89 times), the estimated VOSL for Hong Kong would amount to: 795,000 x 1.19 / 7.75 x 34222.29 / 3,865.88 = US$1,080,620.21 (in 2010 value), assuming an exchange rate (in 2016) of HK$1.19 per RMB, and HK$7.75 per US$. If we adjust the 2006 value of RMB 1 million (Chen et al, 2010) with Hong Kong's per capita GDP, the corresponding value of VOSL is US$1,359,271 (26% higher than the adjusted VOSL from Wang's data). However, this estimate is still much lower than the WHO estimate of US$3 million for European countries in 2015. An adjustment of the European VOSL to Hong Kong's per capital GDP (relative to EU, at US$30,240.87) would yield an estimate of US$2,650,980 (about 2.5 times the adjusted value using Chen's VOSL and about 2 times that of Wang's VOSL). If we use the Taiwan VOSL of US$700,000, based on the latest Taiwan figure (Liu and Hammitt, 1999), the adjusted VOSL for Hong Kong is US$1,071,599. Using South Korea's VOSL of US$800,000 (Kim and Fishback, 1993), the adjusted VOSL for Hong Kong is US$1,114,480. Hence, an estimate of VOSL at US$1.08 million for Hong Kong, adjusted for per capital GDP, is in line with similar estimates in neighbouring Asian countries. We used the adjusted VOSL from the European value of US$2,650,980 as the upper limit, and the adjusted VOSL from Wang's data of US$1,080,620.21 as the lower limit in our EIA. Both are expressed in 2010 dollar value. These are adjusted to 2012 values at US$2,872,817 and US$1,171,048 respectively, using an annual mean inflation rate of 4.1 % (2010 – 14)[7], for the economic impact assessment of 2012 mortality. We have chosen to take the average of these two (upper and lower) VOSL estimates, which amounts to US$2,021,933 as the VOSL for our economic impact assessment. The lower estimate from China (which is quite similar to the VOSL from Taiwan, adjusted to Hong Kong's per capita GDP) represents the lower limit from an Asian country that is ethnically and culturally similar to Hong Kong, except for differences in the political system and level of economic development. The upper estimate from WHO European Region (also adjusted to Hong Kong's per capital GDP) represents a standard promulgated by a highly acclaimed authority on health. This estimate is recommended for European countries, with obvious cultural differences from the Hong Kong people. The decision of taking the mean of these two (lower and upper) estimates is arbitrary. Yet, this

(2006) of RMB1.4 million, based on pooled data from Chongqing and Shanghai, and then adjusted to RMB 1 million to reflect income differences between these cities and the rest of China.

[7] Source: Census and Statistics Department Annual Reports Accessed at: http://www.gov.hk/en/about/abouthk/factsheets/docs/statistics.pdf

mean value (US$2.02 million) is fairly close to the VOSL published in Hong Kong (Siebert and Wei, 1998) when adjusted to 2012 value. The Hong Kong VOSL was adjusted to US$1.7 million in year 2000 by Viscusi and Aldy (2003). This, when further adjusted to 2012 dollar value (assuming an annual inflation rate of 4.1%, as described before), amounts to US$1,842,258 – about 90% of US$2.02 million – the mean value of lower and upper limits of VOSL estimate that we proposed to use. Hence, we consider our use of the mean VOSL derived from the lower and upper estimates as a reasonable estimate of the Hong Kong VOSL. Viscusi and Aldy (2003) remarked that 'VOSL should not be regarded as a universal constant or some "right number" that researchers aim to infer from market evidence. Rather, the VOSL reflects the wage-risk tradeoffs that reflect the preferences of workers in a given sample." They cautioned that "transferring the estimates of VOSL to non-labour market contexts, as the case in benefit-cost analyses of environmental health policies for example, should recognize that different populations have different preferences over risks and different values on life-saving."

3.4.4 The application of VOSL to public policy decisions

Viscusi and Aldy (2003) remarked that 'Regulatory agencies in the United States, the United Kingdom, and Canada, have been most prominent in their use of VOSL estimates to value the benefits of proposed environmental, health, and safety rules. The USEPA guidelines recommends a value of VOSL of $6.2 million in its 2002 economic analyses of regulations.' The UK Cabinet Office also developed guidelines for economic analyses for the government's regulatory and policy-making agencies (U.K. Cabinet Office, 2000; HMS Treasury 1997). The UK Department of the Environment, Transport and Regions (DETR) has used a willingness-to-pay base value of preventing a fatality since 1988. A value equivalent to US$1.2 million was selected in 1998, and was still being used in 2003. Whereas US agencies rely on market-based VOSL estimates, the UK emphasizes on contingent valuation estimates. In Canada, guidelines for benefit-cost analysis were published in 1995. The VOSL for transport-related projects used ranged from US$0.4 to US$3.2 million. The European Commission (EC) since 2000 started to prepare guidance for benefits analysis to improve the benefit-cost analysis procedures within the EC. Australia has also used VOSL in its study of the economic impact of air pollution in Sydney (Department of Environment and Conservation (NSW), 2005).

Table 3.4: Summary of labour market studies of the value of a statistical life (Viscusi and Aldy, 2003) by author / country / income level

Country	Author (year)	Sample	Risk variable	Mean risk	Non-fatal risk included?	Workers' comp included?	Average income level (2000 US$)	Implicit VOSL (million, 2000 US$)
U.S.	26 studies by various authors, 1974 – 2000	Population survey, Panel Study of Income Dynamics, and others	Bureau of Labour Statistics, Society of Actuaries, NIOSH survey, and others	0.00008 – 0.001	Variable	Variable	$21,636 – 49,019	Median: $7.0
U.K.	5 studies, various authors, 1982 – 2001	General Household Survey, Social Change and Economic Life Initiative Survey	OPCS Occupational Mortality Decennial Survey 1970-72 , 80 – 82, 79 – 83, 86 – 88	0.000038 – 0.0001	Variable	No	$12,810 – $20,163	$4.2 - $74.1
Canada	6 studies, various authors, 1989 – 1999	National Survey of Class Structure and Labour Process, National Election Study, Labour Survey and others	Labour Canada and Quebec Occupational Health and Safety Board, Labour Canada and Statistics Canada, Quebec Workers' Compensation Board	0.0001 – 0.00025	Variable	Variable	$19,962 – 43,840	$2.2 – $10.3
Australia	2 studies (Kniesner and Leeth 1991; Miller, Mulvey and Norris 1997)	Manufacturing data, Australian Census of Population and Housing 1991	Industrial Accidents, Australian Bureau of Statistics, Worksafe Australia, National Occupational Health and Safety Commission	0.000068 –0.0001	Variable	Variable	$23,307 - $27,177	$4.2 (1991 study); $11.3 – 19.1(1997 study)
Japan	Kniesner and Leeth 1991	Two-digit manufacturing data	Yearbook of Labour Statistics Japan	0.00003	Yes	No	$44,863	$9.7
Austria	Weiss, Maier and Gerking 1986	Austrian Microcensus File of Central Bureau of Statistics	Austrian Social Insurance Data on job-related accidents	NA	Yes	No	$12,011	$3.9, $6.5
Switzerland	Baranzini and Ferro Luzzi 2001	Swiss Labour Force Survey	Swiss National Accident Insurance Company	0.000059, 0.000064	No	No	$47,400	$6.3, $8.6
India	3 studies (Shan-mugam	Author's survey of blue collar	Administrative Report of Factories Act	0.000104	Variable	No	$778	$1.0 - $4.1

	1996/7, 2000 & 01)	manufacturing workers, Madras, India						
(Table 3.4 continued)								
South Korea	Kim and Fishback 1993	Ministry of Labour's Report on Monthly Labour Survey and Survey on Basic Statistics for the Wage Structures	Ministry of Labour's Analysis for Industrial Accidents	0.000485	Yes	Yes	$8,125	$0.8
Taiwan	2 studies (Liu, Hammitt and Liu 1997; Liu and Hammitt 1999)	Taiwan Labour Force Survey; Authors' survey of petrochemical workers	Taiwan Labour Insurance Agency; Workers' assessed fatality risk at work;	0.000225 – 0.000382 (1997 study); 0.000513 (1999 study)	Variable	No	$5,007 - $6,088 (1997 study); $18,483 (1997 study)	$0.2 - $0.9 (1997 study); $0.7 (1999 study)
Hong Kong	Siebert and Wei 1998	Hong Kong Census 1991	Labour Department	0.000139	No	No	$11,668	$1.7

Table 3.5: Per capita GDP * by country / city / region by year

Country / city	Per capita GDP (US$)	Year
Luxembourg	79,511.21	Dec 2013
Macau	52,476.87	Dec 2014
United States	46,405.26	Dec 2014
United Kingdom	40,967.70	Dec 2014
Singapore	38,087.89	Dec 2014
Australia	37,828.25	Dec 2014
Japan	37,595.18	Dec 2014
Hong Kong	34,222.29	Dec 2014
European Union	30,240.87	Dec 2014
New Zealand	29,201.12	Dec 2013
South Korea	24,565.56	Dec 2014
Taiwan*	22,355	Dec 2014
Malaysia	7,304.14	Dec 2014
China	3,865.88	Dec 2014
Thailand	3,451.33	Dec 2014
Indonesia	1,865.85	Dec 2014
Philippines	1,649.35	Dec 2014

*Reference: http://www.tradingeconomics.com/hong-kong/gdp-per-capita
**Reference for Taiwan: http://twbusiness.nat.gov.tw/old/pdf/sec9.pdf

3.5 References

Centre for International Economics 2001. *Health costs of transport emissions in Sydney – Consultancy 2 – Economic Valuation Methodologies*, Prepared for the NSW Environment Protection Authority, Sydney.

陈仁杰,陈秉衡,阚海东.我国 113 个城市大气颗粒物污染的健康经济学评价. 中国环境科学 (China Environmental Science) 2010,30(3)：410~415.

Commonwealth Department of Finance (1991). Handbook of Cost-Benefit Analysis, Australian Government Publishing Service, Canberra.

Department of Environment and Conservation (NSW). *Air Pollution Economics: Health costs of air pollution in the Greater Sydney Metropolitan Region*, Sydney 2005.

Hong Kong Medical Association News. Hong Kong Medical Association, November 2014.

OECD (2014). The cost of air pollution: health impacts of road transport. Paris: Organisation for Economic Co-operation and Development. Doi: 10.1787/9789264210448-en (Accessed at: http://dx.doi.org/10.1787/9789264210448-en)
Primary Care Directory, Department of Health. Source: https://apps.pcdirectory.gov.hk/PUBLIC/MAIN/MAIN.ASPX

Ki, SW and Fishback PV. Institutional change, compensating differentials, and accident risk in American railroading, 1892-1945. *Journal of Economic History* 1993; 53(4):796-823.

Siebert WS, Wei X. Wage compensation for job risks: the case of Hong Kong. *Asian Economic Journal* 1998; 12(2):171-181.

USEPA 2016. Economic Resource Analysis Document, Analytical Guidance Document, 2.2 Statutory and Administrative Requirements for Economic Analysis of Regulations. Environmental Protection Agency, U.S. Source: http://www3.epa.gov/ttn/ecas/econdata/Rmanual2/2.2.html (Accessed 27/02/2016)

Viscusi WK and Aldy JE. The value of a statistical life: a critical review of market estimates throughout the world. Working Paper 9487, National Bureau of Economic Research, Cambridge MA, 2003. (Also published in *The Journal of Risk and Uncertainty* 2003; 27(1):5-76.

Wang H, He J. The value of statistical life – a contingent investigation in China. Policy Research Working Paper 5421, Washington D C: World Bank, 2010.

World Bank. Cost of pollution in China. Washington D C: World Bank, 2007.
WHO Regional Office for Europe, OECD 2015. Economic cost of the health impact of air pollution in Europe: Clean air, health and wealth, Copenhagen: WHO Regional Office for Europe.

Chapter 4: Choice of health endpoints for health and economic impact assessment

4.1 Health endpoints

Health endpoints refer to the effects on health, either self-limiting or permanent, that are causally related to air pollution. As described in Section 2, the list of health outcomes causally associated with air pollution is long. A review of the literature shows that premature mortality due to long-term air pollution exposure is the predominant health endpoint used, due to both the severity of the outcome and its associated economic loss. As premature death is the predominant health outcome, some HIA studies, notably in the U.K., used *only* premature death as a measure of the burden of air pollution. The other important health risks associated with short-term changes in air pollutant levels are hospital admissions for cardiovascular and respiratory diseases. For premature death (from both short- and long-term exposure to air pollution) and hospital admissions for cardiopulmonary diseases, the evidence is unequivocal, and they have been used in most HIA and EIA studies. The health impact of hospital admissions for cardiovascular and respiratory diseases and their associated hospital costs will be estimated, as unit health risks and community burden of these diseases are available locally. These hospital illnesses will include those diseases that have been well-documented to be associated with air pollution, such as bronchial asthma, acute bronchitis and chronic obstructive airway diseases. HIA of these individual diseases is not feasible because of the lack of local data on their incidence and prevalence (that are required to establish the baseline disease burden).[8] Moreover, the costs associated with specific treatment of these diseases in an outpatient setting are not available for the assessment of 'cost of illness'. GP consultations for upper respiratory infections have been shown both in U.K. (Hajat et al, 2002) and local studies (Wong TW, et al, 2002 and 2006; Tam et al, 2014) to be associated with short-term exposure to air pollutants. This will be included in this HIA and EIA study.

It is important to ensure that health endpoints do not overlap with one another. For example, when mortality risk resulting from long-term exposure to $PM_{2.5}$ is used as a major health endpoint, mortality due to short-term changes in $PM_{2.5}$ concentrations should have already been included in that estimate. Separate assessments of premature mortality from short- and

[8] The study by Künzli et al (1999) also included the incidence of chronic bronchitis in adults, acute bronchitis in children, asthma attacks in children and adults, and restricted activity days in adults as health endpoints. There are many studies published overseas on the associations of these diseases and conditions with air pollution. However, using these as health outcomes in our study is hampered by the lack of local data. For HIA, it is not advisable to use health data from other countries, owing to differences in the incidence and prevalence of these diseases between Hong Kong and many western countries.

long-term exposures and summing them up will result in over-estimation of the mortality risk. Hospital admissions as a health endpoint do not overlap with mortalities, although they may involve the same individuals.

4.2 Strength of evidence

From the strength of the evidence, the associations between premature mortality, a higher risk of morbidity from cardiovascular and respiratory illnesses, and air pollution are regarded to be causal by the scientific community. Table 4.1 summarises the strength of evidence of a cause-effect relationship between air pollution and diseases / mortalities (WHO 2005; WHO 2013). All the health endpoints chosen for our HIA and EIA, as described in 4.1, are based on sufficient scientific evidence of causality from exposure to air pollution.

Table 4.1: Health outcomes associated with air pollution, source and strength of evidence and their RRs

Health outcomes		Exposure to air pollution	Studies used in HIA	Level of Evidence *§
Mortality, all cause		Long-term	US (Pope et al, 2002, ACS study)*; RR=1.06; Hoek's meta-analysis RR of 1.062 (HRAPIE Report, WHO 2013)	Sufficient to infer causality §
			Hong Kong: (Wong CM et al, 2015) RR all-cause mortality = 1.14 (higher than Pope's 1.06)	
Cause-specific mortality	Cardiovascular	Short-term	Hong Kong: Wong CM, et al (PAPA Study, 2010).	Sufficient to infer causality *§
	Respiratory			Sufficient to infer causality *§
Hospital admissions	Cardiovascular	Short-term	Hong Kong: Wong CM, et al (PAPA Study, 2010)	Sufficient to infer causality *§
	Respiratory			Sufficient to infer causality *§
Specific diseases				
Chronic bronchitis	Incidence	Long-term	Abbey et al, 1993 (US);	Suggestive
	Prevalence	Long-term	Cai et al, 2013 (Europe)	Insufficient
Bronchitis in children	Incidence	Long-term	US: Dockery et al, 1996, 1998; Braun-Fahrländer et al, 1997;	Sufficient to infer causality *§
Asthma attacks in children	Prevalence	Short-term	Europe: Roemer et al, 1993 (Wageningem); Gielen et al, 1997 (Amsterdam); Segala et al, 1998 (Paris).	Sufficient to infer causality *§
Asthma attacks in adults	Prevalence	Short-term	Europe: Dusseldorp et al, 1995 (Wijk an Zee, NL); Hiltermann et al, 1998 (Leiden, NL); Neukirch et al, 1998 (Paris)	Sufficient to infer causality *§
Hospitalization for asthma	Attacks	Short-term	Ko et al, 2007 (Hong Kong)	Sufficient to infer causality
Upper respiratory illnesses (URI)	Incidence	Short-term	Hong Kong: Wong TW, et al, 2006 (EPD)	Sufficient to infer causality
Lung function in children		Long-term	Gao et al, 2013; He et al, 2010; Yu et	Sufficient to infer causality

		al, 2001	
Lung growth in children	Long-term	Gaudermann et al, 2007, 2015	Sufficient to infer causality
Cardiopulmonary fitness (VO_{2max})	Long-term	Gao et al, 2013; Yu et al, 2004	Highly suggestive
Restricted activity days in adults	Short-term	Ostro, 1990;	Sufficient
Sickness absence in adults	Short-term	Hong Kong: Wong TW, et al, 2002 (EPD Report)	Sufficient
Low birth weight (LBW), preterm births, intrauterine growth retardation (IUGR) and birth defects	Long-term	One positive Hong Kong study (Chung et al, 2013)	Suggestive but inconsistent for LBW, preterm births and IUGR; Limited evidence for birth defects *
Neurodevelopmental deficit in children		Freire et al, 2010;	Mainly through ingestion, but air pollution also contributes to *indirect* exposure *
Cognitive defects in adults		Ranft, et al, 2009;	Inconclusive §
Childhood leukaemia		Different authors *	Insufficient *
Atherosclerosis	Long-term	Brook et al, 2010;	Strong supportive evidence from experimental studies §
Diabetes mellitus	Long-term	Brook et al, 2008;	Suggestive §

Note: Health outcomes with sufficient evidence are highlighted in this table.

* *Effects of air pollution on children's health and development: A review of the evidence. WHO Special Programme on Health and Development, European Centre for Environment and Health, Bonn Office, 2005.*

§ Review of Evidence on health aspects for air pollution – REVIHAAP Project. Technical Report, World Health Organization Regional Office for Europe, Denmark, 2013.

4.3 References

Brook RD et al. (2008). The relationship between diabetes mellitus and traffic-related air pollution. *Journal of Occupational and Environmental Medicine*, 50(1):32–38.

Brook RD, et al. (2010). Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*, 121(21):2331–2378.

Cai Y, Schikowski T, Adam, M, et al. Cross-sectional associations between air pollution and chronic bronchitis: an ESCAPE meta-analysis across five cohorts. *Thorax* 2014;**69**:1005-1014.

Chung MK, Lao TT, Ting YH, Suen SH, Leung TY, Lau TK, Wong TW. Environmental Factors in the First Trimester and Risk of Oral-Facial Clefts in the Offspring. *Reproductive Sciences 2013. Published online*
http://rsx.sagepub.com/content/early/2012/12/07/1933719112466311.full

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at:
https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. *New England Journal of Medicine* 1993; 329:1753-59.

Dusseldorp A, Kruize H, Brunekreef B, et al. Associations of PM10 and airborne iron with respiratory health of adults living near a steel factory. *American Journal of Respiratory and Critical Care Medicine* 1995; 152:1032-39.

Freire C et al. (2010). Association of traffic-related air pollution with cognitive development in children. *Journal of Epidemiology and Community Health*, 64(3):223–228.

Gao Y, Chan EYY, Zhu Y, Wong TW. Adverse effect of outdoor air pollution on cardiorespiratory fitness in Chinese children. *Atmospheric Environment 2013;* 64:10-17.

Gauderman WJ, Vora H, McConnel R, Berhane K, Gililand F, Thomas D, Lurmann F, Avol E, Künzli N, Jerrett M, Peters J. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *Lancet* 2007; 369(9561):571-577.

Gaudermann WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, Chang R, Lurmann F, Gilliland F. Association of improved air quality with lung development in children. *The New England Journal of Medicine* 2015; 372(10):905-913.

Gielen MH, van der Zee SC, van Wijnen JH, et al. Acute effects of summer air pollution on respiratory health of asthmatic children. *American Journal of Respiratory and Critical Care Medicine* 1997; 155:2105-08.

Hajat S, Anderson HR, Atkinson RW, et al. Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. Occupational and Environmental Medicine 2002;59:294–9.

He QQ, Wong TW, Du L, Jiang ZQ, Gao Y, Qiu H, Liu WJ, Wu JG, Wong A, Yu TSI. Effects of ambient air pollution on lung function growth in Chinese schoolchildren. *Respiratory Medicine* 2010*;* 104:1512-1520.

Hiltermann TJN, Stolk J, van der Zee SC, et al. Asthma severity and susceptibility to air pollution. Eur Respir J 1998; 11:686-93.

Ko FWS, Tam WS, Wong TW, Lai CKW, Wong GWK, Leung TF, Ng S, Hui DSC. Effects of air pollution on asthma hospitalization rates in different age groups in Kong Hong. *Clinical and Experimental Allergy* 2007; 37:1312-1319.

Ko FWS, Tam W, Chan DPS, Wong TW, Tung AH, Lai CKW, Hui DSC. Temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong. *Thorax* 2007; 62:779-784.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA III, Thurston G, Calle EE, Thun MJ. 2009. Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality. HEI Research Report 140. Health Effects Institute, Boston, MA.

Ong SG, Liu J, Wong CM, Lam TH, Tam AYC, Daniel L, Hedley AJ. Studies on the respiratory health of primary school children in urban communities of Hong Kong. *Science of the Total Environment* 1991; 106:121-35.

Ostro B. Associations between morbidity and alternative measures of particulate matter. *Risk Analysis* 1990; 10(3):421-27.

Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 2002; 287(9):1132-41.

Ranft U et al. (2009). Long-term exposure to traffic-related particulate matter impairs cognitive function in the elderly. *Environmental Research*, 109(8):1004–1011.

Roemer W, Hoek G, Brunekreef B. Effect of wintertime air pollution on respiratory health of children with chronic respiratory symptoms. *American Review of Respiratory Diseases* 1993; 147:118-24.

Segala C, Fauroux B, Just J, et al. Short-term effects of winter air pollution on respiratory health of asthmatic children in Paris. *European Respiratory Journal* 1998; 11:677-685.

Tam AYC, Wong CM, Lam TH, Ong SG, Peters J, Hedley AJ. Bronchial responsiveness in children exposed to atmospheric pollution in Hong Kong. *Chest* 1994; 106:1056-60.

Tam WWS, Wong TW, Ng L, Wong SYS, Wong AHS. Association between air pollution and general outpatient clinic consultations for upper respiratory tract infection in Hong Kong. PLOS ONE 2014; 9(1) e86913:1-6. Accessible at: http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0086913

Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015. Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKings

Report14072015final.pdf
Wong CM, Ma S, Hedley AJ, Lam TH. Does ozone have any effect on daily hospital admissions for circulatory diseases? *Journal of Epidemiology and Community Health* 1999; 53:580-81.
Wong CM, Ma S, Hedley AJ, Lam TH. Effect of air pollution on daily mortality in Hong Kong. *Environmental Health Perspectives* 2001; 109:355-40.
Wong CM, Vichit-Vadakan N, Kan H, Qian Z, and the PAPA Project Teams. Public Health and Air Pollution in Asia (PAPA): A Multi-city Study of Short-term Effects of Air Pollution on Mortality. *Environmental Health Perspectives* 2008; 116(9):1195-1202.
Wong CM, Thach TQ, Chau PYK, Chan EKP, Chung RYN, Ou C-Q, Yang L, Peiris JSM, Thomas GN, Lam TH, Wong TW, Hedley AJ. Public Health and Air Pollution in Asia (PAPA): Coordinated studies of short-term exposure to air pollution and daily mortality in four cities. Part 4: Interaction between air pollution and respiratory viruses: Time series study of daily mortality and hospital admissions in Hong Kong. Research Report No. 154, Health Effects Institute, Boston, MA, U.S.A. November 2010.
Wong CM, Lai HK, Tsang H, Thack TQ, Thomas GN, Lam KBH, Chan KP, Yang L, Lau AKH, Ayres JG, Lee SY, Chan WM, Hedley AJ, Lam TH. Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents. *Environmental Health Perspectives* 2015; 123:1167-1172. DOI:10.1289/ehp.1408264. Accessed at: http://dx.doi.org/10.1289/ehp.1408264
Wong TW, Lau TS, Yu TS, Neller A, Wong SL, Tam W, Pang SW. Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong. *Occupational and Environmental Medicine* 1999; 56(10):679-683.
Wong TW, Tam WS, Yu TS, Wong AHS. Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. *Occupational and Environmental Medicine* 2002; 59:30-35.
Wong TW, Wun YT, Yu TS, Tam WS, Wong CM, Wong AHS. Air pollution and GP consultations for respiratory illnesses. *Journal of Epidemiology and Community Health* 2002; 56:949-50.
Wong TW, Wun YT, Yu TS, Tam W, Wong CM, Hedley AJ, Lam TH, Thach TQ. Short-term effects of air pollution on morbidity of the general population: a continuation study. Research Report to Environmental Protection Department, Hong Kong Government, Department of Community & Family Medicine, The Chinese University of Hong Kong, February 2002.
Wong TW, Tam W, Yu ITS, Wun YT, Wong AHS, Wong CM. Association between Air Pollution and General Practitioner Visits for Respiratory Diseases in Hong Kong. *Thorax* 2006; 61:585-591.
World Health Organization (2005). Effects of air pollution on children's health and development: A review of the evidence. WHO Special Programme on Health and Development, European Centre for Environment and Health, Bonn Office, 2005.
World Health Organization (2013). Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration-response functions for cost-benefit analysis of particulate matter,

ozone and nitrogen dioxide. Accessed at:
http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf

Yu TS, Wong TW, Wang XR, Song H, Wong SL, Tang JL. Adverse effects of low-level air pollution on respiratory health of school children in Hong Kong. *Journal of Occupational and Environmental Medicine* 2001; 43:310-16.

Yu TS, Wong TW, Liu HJ. Impact of air pollution on cardiopulmonary fitness in schoolchildren. *Journal of Occupational and Environmental Medicine* 2004; 46:946-52.

Chapter 5 Exposure to air pollution

5.1 Exposure assessment, personal exposure and vertical profile of pollutant concentrations

It is well-known that exposure to air pollution varies from one person to another, and depends on the time-activity analysis of an individual – where the person stays, how high are the concentrations of the air pollutants relevant to HIA and EIA, and for how long in the day. Such time activity patterns differ from weekdays to weekends and holidays, and by age group, occupation, and socioeconomic status. It is not practical to estimate personal exposure based on exposure studies of small groups of individuals (usually students or young adults) and extrapolate these findings to the general population. Likewise, concentrations of air pollutants in different micro-environments such as homes, place of work, the roadside, inside vehicles and other locations differ substantially. It is not possible to assess the exposure by a "representative population group" in the community.

The concentrations of air pollutants recorded from roadside monitoring stations are much higher than in ambient stations, except O_3. This is explained by the proximity of the stations from the major source of pollution – traffic. There is a vertical gradient – the concentrations of these pollutants fall with distance above roadside level, owing to the effect of dilution. However, it is not possible to assess exposure to air pollution according to the vertical distance of households from the roadside. First, 'vertical' distribution of population data in Hong Kong does not exist in the public domain.[9] Second, household air quality is dependent on several factors, such as the presence of indoor sources of air pollution, the ventilation pattern, the width of the streets / roads, the volume of traffic, the proximity to main roads and the topography of the neighbourhood. Hence, it is not feasible to 'tailor-make' an HIA according to detailed exposure assessment of population sub-groups. Instead, we used the exposure assessment method conventionally used in HIA, as reported in studies by developed countries (and China).

For the practical purpose of HIA and EIA, mean long-term (annual) ambient concentrations of air pollutants have been used as a proxy for exposure in all studies that we have reviewed. Hence, we have decided to follow the accepted methodology of HIA and EIA in the developed countries (U.K., Australia, Europe, U.S.) that we have reviewed.

[9] That demographic data by floor of the household were not collected in the census was confirmed through personal communication between the author and the former Director of Census and Statistics Department and his colleagues.

5.2 Choice of data on air pollutants for Health and Economic Impact Assessment

A fundamental methodological issue in developing the HIA tool is whether a single pollutant or multiple pollutants should be used in the model. The major criteria air pollutants in Hong Kong are: particulate matter (PM) of aerodynamic diameter less than 10 μm (PM_{10}) and 2.5 μm ($PM_{2.5}$); nitrogen dioxide (NO_2); sulphur dioxide (SO_2); and ozone (O_3). These pollutants have similar short-term effects on health, as shown in many local and overseas epidemiological studies. However, both PM_{10} and $PM_{2.5}$ are strongly correlated with NO_2, and to a lesser extent, with SO_2. The high correlations between these air pollutants make it difficult for the user to interpret the results if all three were included in the HIA, as there will inevitably be double-counting of health effects. In terms of the magnitude of health risk from short-term exposure, the gaseous air pollutants such as NO_2 and O_3 have been shown to be dominant among the criteria air pollutants in Hong Kong, with higher relative risks (RR) than PM and SO_2. However, many scientists argue that NO_2 most likely acts as a proxy for PM or other traffic-related combustion products such as volatile organic chemicals (VOCs). SO_2 is not included in our HIA. No long-term AQG is recommended in the WHO Air Quality Guidelines (2015), and no RR is recommended in the HRAPIE Report (WHO 2013). Another reason for excluding SO_2 in our HIA is that the ambient concentration of SO_2 has been controlled to a very low level in Hong Kong (annual level below the WHO AQG). Hence, its associated health outcomes could not be assessed if we use the WHO AQG as the target (counter-factual) concentration.

5.2.1 Particulate matter

Particulate matter of respirable size is the main air pollutant that is causally linked to mortality. Künzli et al (1999) used PM_{10} as the single indicator pollutant in their study on the health costs of air pollution, owing to incompleteness of data on $PM_{2.5}$ in those countries being studied. This approach has been followed in the HIA study in Sydney Metropolitan Region in Australia (Department of Environment and Conservation, NSW, 2005). In the past decade, $PM_{2.5}$ has been widely accepted by the scientific community to be more relevant than PM_{10} in its effects on health, owing to its smaller size and higher penetration into the lung parenchyma. Moreover, the relative risks of the different types of mortality (such as total mortality, mortality from cardiovascular and respiratory diseases, and mortality due to lung cancer) from exposure to $PM_{2.5}$ are well documented (Pope et al, 2002) and have been used in HIA locally (Airport Authority Hong Kong, 2014) and abroad (COMEAP, 2010; Walton et al, 2015). $PM_{2.5}$ is therefore chosen as the preferred indicator for PM effects in our study.

5.2.2 Nitrogen dioxide

In a recent review by the WHO (REVIHAAP, WHO 2013a; HRAPIE, WHO 2013b), the authors recommended using NO_2 in HIA in addition to $PM_{2.5}$. The reason for the inclusion of NO_2 is that more and more epidemiological evidence has accumulated that NO_2 has an independent effect on mortality. However, as the concentrations of NO_2 and $PM_{2.5}$ are highly correlated, there is a risk of "double-count" of their impacts on health. The extent of overlap has been estimated to range from 0 – 33%. The HIA study by Walton et al (2015), King's College, London, included the NO_2 effect on mortality, using a conservative estimate of a 30% overlap in its effect on mortality with $PM_{2.5}$. Even if NO_2 acts as a proxy for some other unmeasured emissions (e.g., VOCs) from traffic sources, a reduction of NO_2 through regulatory means will also result in a corresponding decrease of the "underlying cause" of health impact. In our HIA study, we used the annual mean concentrations of 28.6 $\mu g/m^3$ for $PM_{2.5}$ and 52.7 $\mu g/m^3$ for NO_2. These values are averaged from all ambient air monitoring stations except Tap Mun in 2014 (EPD 2014) and are the most up-to-date data available on air pollutants. (It should be noted that for the mortality and hospital statistics, the most up-to-date data are in 2012. Hence, we have used the most up-to-date data on health as well as air pollution in our HIA study, even though they are not from the same year. The actual differences in the concentrations of the two air pollutants between 2012 (27.9 $\mu g/m^3$ for $PM_{2.5}$ and 54.8 $\mu g/m^3$ for NO_2) and 2014 (28.6 g/m^3 for $PM_{2.5}$ and 52.7 $\mu g/m^3$ for NO_2) are small. The differences in hospital and mortality statistics between 2012 and 2014 should likewise be small and will not materially affect the HIA results.

5.2.3 Ozone

Ozone is poorly correlated with $PM_{2.5}$ and has been considered to exert an independent effect on respiratory morbidity. Epidemiological evidence of chronic effects on health is less conclusive, compared to effects from short-term exposure. The American Cancer Society study (Pope et al, 1995) and the Harvard Six City study (Dockery et al, 1993) reported no association of O_3 with mortality, although a re-analysis (Health Effects Institute 2000) showed a significant association between warm-weather O_3 levels and cardiopulmonary mortality. In the World Health Organization Air Quality Guidelines 2005 Global Update (WHO, 2006), a long-term AQG is not recommended for O_3. In the Health Risks of Air Pollution in Europe – HRAPIE project (WHO 2013), long-term exposure to O_3 in summer months and respiratory mortality is classified as a "Group B" or "extended set" of health effects (i.e., with more uncertainty than Group A effects), with a recommended RR of 1.014 (95% CI: 1.005 – 1.024). For short-term effects of O_3 on all-cause mortality (classified as Group A*, that is, where effects can be added to effects attributable to other air pollutants),

the HRAPIE experts recommend an RR of 1.0029 (95% CI: 1.0014 – 1.0043). The exposure metric is the daily maximum of the 8-hour mean concentration of O_3. Ozone effects have not been included in HIAs and cost-benefit analyses in continental Europe, Australia or U.K. (Künzli et al, 1999; DEC 2005; COMEAP 2010; Walton et al, 2015). Reasons to exclude O_3 are: (i) There is some degree of double-counting of morbidities from short-term exposure; (ii) There is higher uncertainty of mortality effects from long-term exposure to O_3; and (iii) There are considerable difficulties in modelling changes in O_3 concentrations that result from air pollution control strategies, because of the complexity of O_3 formation from its components and its dependence on sunshine. In accordance with international practice in HIA, we have excluded O_3 in this study.

5.2.4 Toxic air pollutants

Similarly, toxic air pollutants (TAPs) have also been excluded from the above-named studies, owing to lack of data on their concentrations and the absence of RRs of these chemicals on mortality and morbidity. TAPs comprise a wide variety of organic chemicals and heavy metals, commonly generated from traffic and industrial sources. Common examples of TAPs include: formaldehyde, acetaldehyde, propionaldehyde, acrolein, 1-3-butadiene, benzene, toluene, xylene, polycyclic aromatic hydrocarbons, nickel, chromium, and cadmium. The omission of TAPs from the HIA will result in an under-estimation of the true impact of air pollution. However, most carcinogenic TAPs are associated with lung cancer risk, and overlaps with the effects of $PM_{2.5}$, which is now classified as a Group 1 carcinogen by the WHO. As emissions from traffic vehicles are a common source of TAPs as well as the criteria air pollutants – PM, NO_2 and SO_2, the inclusion of TAPS in HIA would result in a certain degree of double-counting of lung cancer cases.

5.3 References

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

Department of Environment and Conservation (NSW). *Air Pollution Economics: Health costs of air pollution in the Greater Sydney Metropolitan Region*, Sydney 2005.

Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. An association between air pollution and mortality in Six U.S. cities. *The New England Journal of Medicine* 1993;

329:1753-1759.
Health Effects Institute. 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality: A Special Report of the Institute's Particle Epidemiology Reanalysis Project*. Health Effects Institute, Cambridge MA.
Künzli N, Kaiser R, Medina S, Studnicka M, Oberfeld G, Horak F. *Health costs due to road traffic-related air pollution – an assessment project of Austria, France and Switzerland*. Prepared for the Third Ministerial Conference for Environment and Health, London, 1999.
Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine* 1995; 151(3):669-674.
Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015. Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKingsReport14072015final.pdf
World Health Organization (2006). *Air Quality Guidelines Global Update 2005*. World Health Organization Regional Office for Europe, Copenhagen.
WHO 2013a. Review of Evidence on health aspects for air pollution – REVIHAAP Project. Technical Report, World Health Organization Regional Office for Europe, Denmark, 2013. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf
WHO 2013b. Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration-response functions for cost-benefit analysis of particulate matter, ozone and nitrogen dioxide. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf

Chapter 6: Methodology of the Instrument for Health and Economic Impact Assessment

6.1 Methods used in Health Impact Assessment

The health impact assessment (HIA) follows the conventional methodology used in the literature (COMEAP, 2010; Department of Environment and Conservation (NSW), 2005; Kunzli et al, 1999; WHO, 2014;Walton et al, 2015; WHO, 2016). An important step in HIA is the derivation of the proportions of health outcomes (mortalities and morbidities) that are estimated to be the results of exposure to air pollution. This proportion is known as the 'attributable fraction' (AF) in epidemiology. The AF is related to the relative risk (RR) of the health outcome that results from exposure to air pollution (i.e., the risk of premature death or the risk of being ill resulting from air pollution) by the formula: AF = (RR-1) / RR

Hence, identifying the appropriate RRs for the health outcomes under study is an important step in HIA (See Chapter 2). RRs are quoted as the risk per unit change in the concentrations of the air pollutants concerned, commonly in 10 $\mu g/m^3$. To assess the impact of a change in the air pollution level from the current level (x $\mu g/m^3$) to a target level (y $\mu g/m^3$), often termed the 'counterfactual', it is necessary to determine the RR of this change (i.e., instead of a unit change in concentration, the RR is now expressed as that from a change from x to y). To estimate this change, we first determine the RR per 1 $\mu g/m^3$. This is done by taking the natural logarithm of the RR per 10 $\mu g/m^3$ and divide it by 10 to obtain the regression coefficient. We then take the exponential of this new regression coefficient to obtain the RR per 1 $\mu g/m^3$. For any change from the current air pollutant concentration to the desired level (counterfactual), the corresponding RR is calculated by multiplying the unit regression coefficient (per 1 $\mu g/m^3$) by the change in concentration (current level - counterfactual) and then taking the exponential. This process will yield the AF for any specified change in concentration of any air pollutant under study. The health impact of this change is given by the formula:

Attributable health outcomes = Baseline health outcome data x Attributable fraction

The baseline health outcome data refer to the annual number of non-accidental deaths, emergency hospital admissions for cardiovascular and respiratory diseases, and GP and GOPC visits for upper respiratory infections. The AF refers to the fraction of a specified health outcome (deaths / hospitalizations / GP visits / GOPC visits) for a specified change in the air pollutant concentrations.

6.2 Methods used in Economic Impact Assessment

In economic impact assessment (EIA), the economic impact attributable to a specified change in air pollutant concentration is given by:

Economic impact = Attributable health outcome x unit cost of that health outcome

The unit cost of premature death is expressed as the value of statistical life (VOSL), as described in Chapter 3. The unit cost of hospital illnesses is expressed as the unit cost of hospitalization (cost per bed-day, derived from published data by the Hospital Authority) multiplied by the mean duration of hospital stay for cardiovascular and respiratory diseases. The indirect economic impact due to loss of earnings is estimated from the number of days off work, multiplied by the daily wage (derived from the median monthly income, published by the Census and Statistics Department). The unit cost of a GP visit is derived from a survey conducted as part of this Study (Chapter 3, Section 3.4.2). The unit cost of GOPC visit is derived from data published by the Hospital Authority.

6.3 Need for updating health and economic data in the Instrument

The instrument for HIA and EIA is developed as an Excel spreadsheet. Inputs to this instrument include the following variables:

1. Baseline health outcome data, including (i) number of non-accidental deaths; (ii) number of emergency hospital admissions for cardiovascular and respiratory diseases respectively; (iii) number of general practitioners visits for URTI; and (iv) number of general outpatients clinics visits for URTI
2. RR per 10 $\mu g/m^3$
3. Current air pollutant concentration
4. Target air pollutant concentration
5. Unit VOSL
6. Unit cost of hospitalization
7. Unit cost of GP visit
8. Unit cost of GOPC visit
9. Unit daily wage
10. Mean duration of hospitalization

In the instrument that we have developed, the RRs are chosen by literature review, and the current and target air pollutant concentrations have been specified using ambient air pollution data from the Environmental Protection Department (EPD) and the World Health Organization Air Quality Guidelines Glorabl Update 2005 (WHO 2006). Although RRs and mean duration of hospital stay are unlikely to change much over time, updating of the relevant inputs to the instrument, such as RR (from local epidemiological studies) on RR of mortality from long-term effects of air pollution, new local studies on hospital activities) would be required on a regular basis. Available new data can be readily updated by inputing them in the instrument. In addition, baseline health data, data on unit costs and median daily wage, which changes from year to year, will need to be updated when HIA and EIA is performed in future. A common way to smooth out the year-to-year fluctuations in health outcome data is to use the mean value for a longer period (e.g., five years) instead of relying on an annual figure alone.

6.4 References

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

Department of Environment and Conservation (NSW). *Air Pollution Economics: Health costs of air pollution in the Greater Sydney Metropolitan Region*, Sydney 2005.

Künzli N, Kaiser R, Medina S, Studnicka M, Oberfeld G, Horak F. *Health costs due to road traffic-related air pollution – an assessment project of Austria, France and Switzerland*. Prepared for the Third Ministerial Conference for Environment and Health, London, 1999.

Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015. Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKingsReport14072015final.pdf

World Health Organization (2006). *Air Quality Guidelines Global Update 2005*. World Health Organization Regional Office for Europe, Copenhagen.

World Health Organization. WHO Expert Meeting: Methods and tools for assessing the health risks of air pollution at local, national and international level. Meeting Report, Bonn, Germany, 12-13 May 2014. WHO Regional Office for Europe, 2014.

World Health Organization. Health Risk Assessment of Air Pollution: General Principles. WHO Regional Office for Europe, 2016. Assessed at:

http://www.euro.who.int/__data/assets/pdf_file/0006/298482/Health-risk-assessment-air-pollution-General-principles-en.pdf?ua=1

Chapter 7: Results of Health Impact Assessment

7.1 Effect of different assumptions on HIA

The choice of different factors that contribute to the health impacts may substantially affects the estimates. Data including population and health statistics – total and cause-specific mortality, the total number of hospital admissions for cardiovascular and respiratory diseases, and attendances at general outpatient clinics are relatively stable, but do change from year to year (rising slowly with time). The estimated total number of consultations to private GP for upper respiratory infections may vary more, depending on the assumptions used for the estimations. We have chosen the latest available data. For hospital and mortality data, we have chosen the annual statistics in 2012. The RRs used, whether from local studies or internationally well-accepted values, are relatively stable, and generally do not vary with time. The long-term mean concentrations of the relevant air pollutants in Hong Kong obviously determine the burden of air pollution. For air pollutant concentrations, we have used the mean annual concentrations of $PM_{2.5}$ and NO_2 from all ambient air monitoring stations except Tap Mun in 2014 as our latest available data. By far, the value of the "counter-factual", i.e., the concentrations of the air pollutants that may be taken as the "change" (thealternative scenario) from the current concentrations, is the single most important variable that affects the outcomes of the HIA. Different researchers have different choices of counter-factual used in HIA and the subsequent EIA. In the COMEAP (2010), the authors considered the use of "non-anthropogenic" $PM_{2.5}$ concentration as their counter-factual, by subtracting the sea salt concentrations from the total $PM_{2.5}$ concentration. Thus, all the other components of $PM_{2.5}$ are regarded as man-made (therefore, preventable), and the concentration of "anthropogenic $PM_{2.5}$" were used to assess the burden of air pollution on health. Another counter-factual used was 7 $\mu g/m^3$, the lowest concentration of $PM_{2.5}$ observed in Pope's study, whose RR has been widely used internationally (including Chinese HIA studies). The rationale is that 7 $\mu g/m^3$ is the lower limit of scientific validity in the use of Pope's RR (all-cause mortality) of 1.06 for $PM_{2.5}$. Hence, extrapolating this RR to $PM_{2.5}$ levels below 7 $\mu g/m^3$ would be invalid. In this study, we have instead chosen two counter-factuals: a 1 $\mu g/m^3$ change, as a unit of change in HIA, and 10 $\mu g/m^3$, the 2005 Air Quality Guideline proposed by WHO (2006). The use of the former is to facilitate the calculation of a unit RR (RR for a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration) for the calculation of the attributable fraction (AF), while the WHO AQG – the annual mean $PM_{2.5}$ concentration of 10 $\mu g/m^3$ is used as a counter-factual that is widely considered as an acceptable level from the public health perspective. The choice of the WHO AQGs as counter-factuals is appropriate as they are the long-term goals of the Environmental Protection Department in its review of Air Quality Objectives (AQOs) in Hong Kong. The WHO AQG of 10 $\mu g/m^3$ for $PM_{2.5}$ is not meant to be a level where there is

no health impact – in fact, there is no evidence for a safe threshold for $PM_{2.5}$. Instead, 10 $\mu g/m^3$ is considered by WHO as an achievable level of $PM_{2.5}$. Below this level, statistical uncertainties in the exposure-response function are much higher. Nevertheless, WHO advises that a country should not just stop at 10 $\mu g/m^3$ if it is able to achieve an even lower level, because a lower level of $PM_{2.5}$ would have additional public health benefit.

Most HIA studies that we have reviewed used the RR estimated by Pope's study (2002): 1.06 for all-cause mortality, despite the publication of a higher RR (1.078) by Krewski (2009) using an extension analysis of Pope's original data. Using this higher RR would increase the health outcomes by about 17%.

The summed effects of $PM_{2.5}$ and NO_2 on premature mortality, obtained by conducting a separate assessment using NO_2 data is conducted, will necessarily be larger than $PM_{2.5}$ effects alone, even after allowing for a 30% overlap with $PM_{2.5}$ effects. This decision will affect the final results of mortality assessment in HIA.[10]

[10] There is more certainty in the HIA obtained using only the mortality burden of $PM_{2.5}$. This approach is the WHO recommendation for the 'limited set' of more certain concentration-response functions. Adding together the effects of $PM_{2.5}$ and the effects of NO_2 assuming no overlap between NO_2 and $PM_{2.5}$ is the WHO recommendation for the 'extended set' of concentration-response functions (using RR for NO_2=1.055). In this study, we used the approach by Walton et al (2015) to add together the effects of $PM_{2.5}$ and the effects of NO_2, but reducing the effects of NO_2 by 30%, taking into the account of the possible maximum size of the overlap between NO_2 and $PM_{2.5}$. The RR for NO_2 is thus adjusted downwards to 1.039 to reflect a 30% overlap in effect.

7.2 Premature mortalities attributable to $PM_{2.5}$ and NO_2

Estimates of premature mortalities attributable to $PM_{2.5}$ and NO_2 in our HIA are presented in Tables 6.1 and 6.2.

Table 7.1: Estimates of premature mortalities attributed to $PM_{2.5}$ using air quality data in 2014 and mortality data in 2012

Health Outcomes	Annual no. of deaths	Unit RR per 10 $\mu g/m^3$	Annual $PM_{2.5}$ conc.§	Target $PM_{2.5}$ conc.	Change in exp. conc.	RR for 1$\mu g/m^3$ **	RR for change in exp. conc.	Attributable fractionΨ	Annual attributable deaths	Remark
All-cause mortality (Hypothetical no.)	100,000	1.06 *	28.6	27.6	1	1.005844	1.005844	0.00581	581	Pope's RR; Assuming a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration
All non-external ϕ cause mortality	42,017	1.06 *	28.6	27.6	1	1.005844	1.005844	0.00581	244	Pope's RR; Assuming a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration
All non-external cause mortality	42,017	1.02	28.6	27.6	1	1.001982	1.001982	0.001978	83	Lower 95% CL of RR; Assumes a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration
All non-external cause mortality	42,017	1.11	28.6	27.6	1	1.010491	1.010491	0.010382	436	Upper 95% CL of RR; Assumes a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration
All non-external cause mortality	42,017	1.06 *	28.6	10 φ	18.6	1.005844	1.114471	0.102714	4,316	Pope's RR; Assumes $PM_{2.5}$ changes to 10 $\mu g/m^3$
All non-external cause mortality	42,017	1.02	28.6	10 φ	18.6	1.001982	1.03752	0.036163	1,519	Lower 95% CL of RR; Assumes $PM_{2.5}$ changes to 10 $\mu g/m^3$
All non-external cause mortality	42,017	1.11	28.6	10 φ	18.6	1.010491	1.214229	0.176432	7,413	Upper 95% CL of RR; Assumes $PM_{2.5}$ changes to 10 $\mu g/m^3$
All non-external cause mortality	42,017	1.078 #	28.6	27.6	1	1.007539	1.007539	0.007483	314	Krewski's RR;
All non-external cause mortality	42,017	1.046	28.6	27.6	1	1.004507	1.004507	0.004487	189	Krewski's L95% CL of RR
All non-external cause mortality	42,017	1.115	28.6	27.6	1	1.010945	1.010945	0.010826	455	Krewski's U95% CL of RR
All non-external cause mortality	42,017	1.078 #	28.6	10	18.6	1.007539	1.149929	0.130381	5,478	Krewski's RR;
All non-external cause mortality	42,017	1.046	28.6	10	18.6	1.004507	1.087249	0.080247	3,372	Krewski's L95% CL of RR
All non-external cause mortality	42,017	1.115	28.6	10	18.6	1.010945	1.224422	0.183288	7,701	Krewski's U95% CL of RR

* RR according to Pope et al (2002).

** 1 μg/m^3 is used as the smallest unit change in concentration of air pollutant to *facilitate* the calculation of the unit change in RR in relation to the assumed changes in air pollution concentrations from the current level to a counter-factual level. The unit RR is approximately one-tenth of the RR per 10μg/m^3. Mathematically, this unit RR (per 1 μg/m^3) is derived by dividing the regression coefficient (anti-log of RR per 10 μg/m^3) by 10, and then taking the logarithm of this new regression coefficient.

RR according to Krewski (2009)

§ $PM_{2.5}$ concentration based on the mean annual concentration of $PM_{2.5}$ in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Ψ Attributable fraction (AF) = (RR-1)/RR

ϕ 2012 mortality data from Census and Statistics Department: All non-external causes of death (42,017) = Total deaths (43,672) - deaths from external cause (1,655)

φ Assuming a counter-factual of 10 μg/m^3, the WHO AQG annual concentration for $PM_{2.5}$. This implies that the prevailing concentration of $PM_{2.5}$ in Hong Kong can be reduced hypothetically to an annual mean concentration recommended by WHO, a level that is considered to be *acceptable* from the public health viewpoint.

Table 7.2: Estimates of premature mortalities attributed to NO_2 (assuming a 30% overlap with $PM_{2.5}$ effect using air quality data in 2014 and mortality data in 2012

Health Outcomes	Annual no. of deaths	Unit RR* per 10 μg/m^3	Annual NO_2 conc. §	Target NO_2 conc.	Change in exp. conc.	RR for 1μg/m^3	RR for change in exp. conc.	Attributable fractionΨ	Annual attributable deaths	Remark
All non-external ϕ cause mortality	42,017	1.039	52.7	51.7	1	1.003833	1.003833	0.003819	160	RR using HRAPIE recommendation of 30% overlap with $PM_{2.5}$ mortality
All non-external ϕ cause mortality	42,017	1.039	52.7	40^{φ}	12.7	1.003833	1.049788	0.047427	1,993	(Ditto); Central RR
All non-external cause mortality	42,017	1.022	52.7	40	12.7	1.002179	1.028023	0.027259	1,145	Lower 95% CL of RR
All non-external cause mortality	42,017	1.056	52.7	40	12.7	1.005464	1.071651	0.06686	2,809	Upper 95% CL of RR

* RR using HRAPIE (WHO 2013) recommendation of 30% overlap with $PM_{2.5}$ mortality

Ψ Attributable fraction (AF) = (RR-1)/RR

ϕ 2012 mortality data from Census and Statistics Department: All non-external causes of death (42,017) = Total deaths (43,672) - deaths from external cause (1,655)

$^{\varphi}$ Assuming a counter-factual of 40 μg/m^3, the WHO annual AQG.

§ NO_2 concentration based on the mean annual concentration of NO_2 in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Table 7.3: Estimates of premature mortalities (all non-external cause) attributed to the combined effects of $PM_{2.5}$ and NO_2

RR used / change in conc.	Annual deaths ($PM_{2.5}$) 1 μg/m^3	Annual deaths (NO_2)	Annual deaths (combined)
Central RR /	244	160	404
Lower 95% CI	83	91	174
Upper 95% CI	436	228	664
RR used / change in conc.	Annual deaths* ($PM_{2.5}$) 18.6 μg/m^3	Annual deaths* (NO_2) 12.7 μg/m^3	Annual deaths* (combined)
Central RR	4,316**	1,993**	6,308**
Lower 95% CI	1,519**	1,145**	2,665**
Upper 95% CI	7,413**	2,809**	10,222**

* Assume counter-factual for $PM_{2.5}$= 10 μg/m3; NO_2 = 40 μg/m3;

** In this table, the combined no. of deaths are slightly different from the sums of deaths from $PM_{2.5}$ and NO_2. This is because the numbers from $PM_{2.5}$ and NO_2 have been rounded up to whole numbers. The actual figures is 4,315.72 for $PM_{2.5}$, and 1992.74 for NO_2, Hence, the total is 6,308.46, rounded down to 6,308. This approach is also used for the number of deaths for the lower and upper 95% CI of the respective RRs.

Using the RR of 1.06 (Pope et al, 2002), and a counter-factual of 10 μg/m^3 for $PM_{2.5}$, we estimate that the annual premature mortality of attributable to $PM_{2.5}$ to be 4,316 (95% CI: 1,519 – 7,413). The figure obtained by using Krewski's slightly higher RR was slightly higher, at 5,478 deaths per annum (95% CI: 3,372 – 7,701). These estimates are lower than figures reported in other countries, due to the conservative counter-factual of 10 μg/m^3 that we used for $PM_{2.5}$. As mentioned in 6.1, some HIA studies have used "all anthropogenic" $PM_{2.5}$ (total $PM_{2.5}$ – sea salt $PM_{2.5}$), or a counter-factual of 7 μg/m^3, the lowest concentration in Pope's study, with much higher estimates of attributable health outcomes.

We have also estimated the additional effect on premature mortality by NO_2, according to REVIHAAP and HRAPIE Reports (WHO 2013a; WHO 2013b). Using a counter-factual of 40 μg/m^3, the WHO AQG for annual NO_2 concentrations, we have derived an additional 1,993 deaths (95% CL: 1,145 – 2,809), making a total of 6,309 deaths per annum (95% CL: 2,664 – 10,222). In the London study by King's College (Walton et al, 2015), a counter-factual of 0 μg/m^3 for NO_2 (and 20 μg/m^3 for sensitivity analysis) was used.

We have also estimated the premature mortality attributed to short-term changes in air pollution. The annual number of premature deaths is 318 for all-cause mortality (using the same concentrations of $PM_{2.5}$ and counter-factual, and Pope's RR for $PM_{2.5}$). Using cause-specific mortalities, we have derived a slightly higher number of 247 cardiovascular deaths and 171 respiratory deaths per year. This appears to be counter-intuitive, as the sum of the estimated cardiovascular and respiratory deaths is higher than the estimated all-cause

deaths attributable to air pollution. However, these estimates are derived from different RRs – the RR for all-cause mortality is lower than that for cardiovascular and pulmonary mortalities. We must note, however, that estimates of premature deaths from short-term exposure to air pollution have already been included in the estimates using the RR for long-term exposure.

7.3 Morbidities (hospital admissions) attributable to $PM_{2.5}$ and NO_2

Tables 7.4 and 7.5 show the estimated annual increase in hospital admissions for cardiovascular and respiratory diseases using data on hospital admissions from Hospital Authority. Note that these estimates do not include admissions into private hospitals (that contribute to about 10% of the total number of hospital beds in Hong Kong). The numbers of cardiovascular and respiratory hospital admissions attributable to 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration are 47 and 101 respectively, with a total of 148 cardio-respiratory admissions. Using the counter-factual of 10 $\mu g/m^3$ in $PM_{2.5}$ concentration (a 18.6 $\mu g/m^3$ change), the corresponding numbers (rounded-up to whole numbers) are 863 and 1,868 respectively, with a total of 2,730[11] (95% CI: 1,730 – 3,735) cardio-respiratory admissions. The corresponding numbers attributable to1 $\mu g/m^3$ change in NO_2 concentration are 71 and 78 for cardiovascular and respiratory hospital admissions, with a total of 149 cardio-respiratory admissions. The numbers using a counter-factual of 40 $\mu g/m^3$ in NO_2 concentration (a 12.7 $\mu g/m^3$ change) are 891 for cardiovascular diseases and 991 for respiratory diseases, with a total of 1,882 cardio-respiratory admissions. These figures overlap, as RRs derived from single pollutant models have been used in the estimates. Hence, it is not appropriate to add these numbers up. Instead, we shall take the higher figure of the two, to reflect the burden of diseases from air pollution in our HIA and EIA. The major reason for a lower figure derived from NO_2, despite its higher unit RR is the choice of a counter-factual of 40 $\mu g/m^3$, the WHO 2005 AQG that has been presently used as AQO in Hong Kong. By contrast, the corresponding WHO AQG that we used for $PM_{2.5}$ as counter-factual is 10 $\mu g/m^3$, much lower than the mean annual concentration of 28.6 $\mu g/m^3$ in Hong Kong (2012 data).

7.4 Morbidities (clinic attendances) attributed to air pollution

7.4.1 Private GP consultations for upper respiratory infections

[11] The slight discrepancies in the sum total (2,730) with the numbers attributable to $PM_{2.5}$ and NO_2 arises because the sum total is derived from adding the individual numbers in decimals, not by adding the rounded-off whole numbers attributable to these 2 pollutants (863 and 1,868), which gives a total of 2,731.

We have also estimated the annual number of consultations for upper respiratory infections (URI) to private general practitioners (GPs) and general outpatient clinics (GOPC) run by the Hospital Authority. Despite the availability of a list of registered medical practitioners, there is no register of private GPs' clinics in Hong Kong. The list of clinics from the Primary Care Directory published by the Department of Health (used in our survey of GP consultation fees) is not comprehensive (listing is on a voluntary basis), and do not distinguish between generalist, family medicine, or specialist (e.g., paediatrics practice that is relevant for asthma consultations by children, or cardiologists and respirologists who may provide service to patients with heart and lung diseases). Hence, we used the estimate of 26,000,000 outpatient consultations[12] (2014 estimate) to private doctors from the Hong Kong Government report – My Health My Choice, Health Reform Second Stage Public Consultation Report (Food and Health Bureau, 2010). We then used the proportion of consultations for primary medical care as 86% of all consultations in Hong Kong (Thematic Household Survey, quoted in Food and Health Bureau, 2010) to estimate the number of visits to private GPs, the proportion of URI among all cases seen (46.9%) and the percentage of new URI cases from all cases (78%) [13], and estimated the total number of consultations for new cases of URI to be 8,179,735 per annum. A substantially higher figure of 21,575,032 per annum was estimated, based on an updated estimate of 3,949 [14] private GPs (2013 data) and the same proportion of new cases and URI obtained from the author's 2002 Report (Wong TW et al, 2002). This estimate can be regarded as the upper bound of the estimate of the annual total consultations for new cases of URI, while the former estimate of 8,179,735 as the lower bound. We consider that the mean estimate, at 14,877,384 annual GP visits, as a representative figure for our HIA.

7.4.2 General Outpatients Clinics consultations for upper respiratory infections

By contrast, data from attendances in GOPCs are available from the Hospital Authority. The total number of consultations in the year 2012 – 13 was 5,633,407 (Hospital Authority, 2013). Using the same proportion of new cases to all cases (78%), and URI cases to all cases (46.9%), we estimated the annual total number of new URI cases seen at Hospital Authority

[12] My Health, My Choice, Healthcare Reform Second Stage Public Consultation Report, Appendix C, p87.

[13] Wun et al, 2002. Wun YT, Wong TW, Tam W, Yu TS. Patient characteristics of encounters in general practice. *The Hong Kong Practitioner* 2002;24:59-65.

[14] Total no. of resident medical doctors = 12,401 (end 2013 data), less no. of doctors in DH (495) and HA (5107) = 6,799 that is the total no. of medical doctors in the private sector. As the no. of specialists (5,700) are roughly equally divided in the private and public sector, we assume that there are 2,850 specialists in the private sector. Therefore, the no. of private GPs in the private sector = 6,799 – 2,850 = 3,949.

GOPCs to be 2,060,813. Taking the lower bound estimate for cases seen by private GPs (8,179,735) this amounts to about ¼ of cases seen by private GPs, or 20.1% of all new cases of URI seen in both private and public sector. If we take the upper bound of our estimate for GP cases, the number seen at GOPCs of the Hospital Authority represents 9.5% of cases seen by private GPs, or 8.7% of all new URI cases seen by both sectors.

7.4.3 Private GP and GOPC consultations for URI attributable to air pollution

Tables 7.6 to 7.9 show the annual number of URI cases attributable to $PM_{2.5}$ and NO_2 seen by private GPs and Hospital Authority GOPCs respectively. From the lower estimate of 8,179,735 and upper estimates of 21,575,032 annual consultations to private GPs for new cases of URI, the numbers of cases attributed to $PM_{2.5}$ (counter-factual = 10 $\mu g/m^3$) were 310,159 and 818,081 (mean = 564,120) respectively. The corresponding numbers attributed to NO_2 (counter-factual = 40 $\mu g/m^3$) were 301,372 (low), 794,906 (high), and 548,140 (mean) respectively. As these estimates are based on RRs derived from single pollutant models (i.e., the effects of other pollutants have not been adjusted), we shall take the larger number, derived from $PM_{2.5}$. As described before, the number of cases attributed to NO_2 is smaller than that to $PM_{2.5}$, despite a higher unit RR of 1.03 for NO_2 (versus 1.021 for $PM_{2.5}$). This is influenced by the values of the counter-factual chosen for the assessment (40 $\mu g/m^3$ for NO_2, and 10 $\mu g/m^3$ for $PM_{2.5}$).

For GOPC consultations, we used the RR for PM_{10} and NO_2 derived from a recent GOPC time series study (Tam et al, 2014). The RR for $PM_{2.5}$ was not estimated, and we have used the RR for PM_{10} as a proxy, bearing in mind that this RR is likely to be under-estimated, as RR for $PM_{2.5}$ is generally higher than that for PM_{10}. The number of new URI new cases attributable to $PM_{2.5}$ is 1,028 per 1$\mu g/m^3$ change in $PM_{2.5}$ concentration, and 19,029 per year, using the same counter-factuals for $PM_{2.5}$ as before. The number of new URI cases attributed to NO_2 is 2,050 (per 1$\mu g/m^3$ change in NO_2 concentration), and 25,878 using the same counter-factual for NO_2 concentrations as before. We shall use the NO_2 estimate, which is higher than the $PM_{2.5}$ estimates (from NO2), as both are estimated using single-pollutant RRs.

The annual number of estimated GOPC consultations for new URI cases is 8.3% of the lower estimate of cases seen by private GPs, and 3.1% of the higher estimate.

Table 7.4: Estimates of increased hospital admissions attributed to $PM_{2.5}$ using hospital admission statistics

Hospital admissions	Annual no. of patients	Unit RR per 10 $\mu g/m^3$ §	Annual $PM_{2.5}$ conc.φ	Target $PM_{2.5}$ conc.	Change in conc.	RR for 1 $\mu g/m^3$	RR for change in exp. conc.	Attributable fractionΨ	Annual attributable cases	Remark
CVS diseases (Hypothetical)	100,000	1.0066	28.6	27.6	1	1.000658	1.000658	0.000658	66	RR from Qiu et al (2013) §
CVS diseases*	70,934	1.0066	28.6	27.6	1	1.000658	1.000658	0.000658	47	hospital admissions in 2012
CVS diseases*	70,934	1.0036	28.6	27.6	1	1.000359	1.000359	0.000359	25	Lower 95%C L
CVS diseases*	70,934	1.0097	28.6	27.6	1	1.000966	1.000966	0.000965	68	Upper 95%C L
CVS diseases*	70,934	1.0066	28.6	10	18.6	1.000658	1.012311	0.012161	863	Central RR
CVS diseases*	70,934	1.0036	28.6	10	18.6	1.000359	1.006706	0.006662	473	Lower 95%C L
CVS diseases*	70,934	1.0097	28.6	10	18.6	1.000966	1.018117	0.017795	1,262	Upper 95%C L
Respiratory diseases hypothetical	100,000	1.0097	28.6	27.6	1	1.000966	1.000966	0.000965	96	R from Dr. Qiu Hong § (personal communication)
Respiratory diseases*	104,953	1.0097	28.6	27.6	1	1.000966	1.000966	0.000965	101	
Respiratory diseases*	104,953	1.0065	28.6	27.6	1	1.000648	1.000648	0.000648	68	Lower 95%C L
Respiratory diseases*	104,953	1.0129	28.6	27.6	1	1.001283	1.001283	0.001281	134	Upper 95%C L
Respiratory diseases*	104,953	1.0097	28.6	10	18.6	1.000966	1.018117	0.017795	1,868	Central RR
Respiratory diseases*	104,953	1.0065	28.6	10	18.6	1.000648	1.012124	0.011979	1,257	Lower 95% C L
Respiratory diseases*	104,953	1.0129	28.6	10	18.6	1.001283	1.024127	0.023559	2,473	Upper 95% C L
Annual cardiovascular and respiratory admissions attributable to $PM_{2.5}$ (= 863 + 1868)									2,731	Assuming 10 $\mu g/m^3$ as counter-factual

* Based on data on hospital admissions in 2012, Hospital Authority

Ψ Attributable fraction (AF) = (RR-1)/RR

§ RR from Dr. Qiu Hong, School of Public Health, the University of Hong Kong (personal communication)

φ $PM_{2.5}$ concentration based on the mean annual concentration of $PM_{2.5}$ in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Table 7.5: Estimates of increased hospital admissions attributed to NO_2 using hospital admission statistics

Hospital admissions	Annual no. of patients	Unit RR per 10 $\mu g/m^3$	Annual NO_2 conc.§	Target NO_2 conc.	Change in exp. conc.	RR for 1 $\mu g/m^3$	RR for change in exp. conc.	Attributable fractionΨ	Annual attributable cases	Remark
CVS diseases (Hypothetical)	100,000	1.01	52.7	51.7	1	1.000996	1.000996	0.000995	99	2012 hospital data; RR from PAPA study; Annual NO_2 based on EPD 2014 Air Quality Report
CVS diseases*	70934	1.01	52.7	51.7	1	1.000996	1.000996	0.000995	71	(ditto)
CVS diseases*	70934	1.0073	52.7	51.7	1	1.000728	1.000728	0.000727	51	L95%C L
CVS diseases*	70934	1.0126	52.7	51.7	1	1.001253	1.001253	0.001251	89	U95%C L
CVS diseases*	70934	1.01	52.7	40	12.7	1.000996	1.012717	0.012557	891	Central RR
CVS diseases*	70934	1.0073	52.7	40	12.7	1.000728	1.00928	0.009195	652	L95%C L
CVS diseases*	70934	1.0126	52.7	40	12.7	1.001253	1.016029	0.015776	1,119	U95%C L
Respiratory diseases (Hypothetical)	100000	1.0075	52.7	51.7	1	1.000747	1.000747	0.000747	75	CM Wong: PAPA study 2011
Respiratory diseases*	104953	1.0075	52.7	51.7	1	1.000747	1.000747	0.000747	78	(ditto)
Respiratory diseases*	104953	1.005	52.7	51.7	1	1.000499	1.000499	0.000499	52	L95%C L
Respiratory diseases*	104953	1.01	52.7	51.7	1	1.000996	1.000996	0.000995	104	U95%C L
Respiratory diseases	104953	1.0075	52.7	40	12.7	1.000747	1.009535	0.009445	991	Central RR
Respiratory diseases	104953	1.005	52.7	40	12.7	1.000499	1.006354	0.006314	663	L95%C L
Respiratory diseases	104953	1.01	52.7	40	12.7	1.000996	1.012717	0.012557	1,318	U95%C L
Annual cardiovascular and respiratory admissions attributable to NO_2 (= 891 + 991)									1,882	Assuming 40 $\mu g/m^3$ as counter-factual

* Based on data on hospital admissions in 2012, Hospital Authority

Ψ Attributable fraction (AF) = (RR-1)/RR

§ NO_2 concentration based on the mean annual concentration of NO_2 in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Table 7.6: Estimates of URI consultations to private GPs, attributed to $PM_{2.5}$ using low and high estimates of total GP numbers

Upper respiratory infections (new cases)	Annual no. of patients	Unit RR per 10 $\mu g/m^3$	Annual $PM_{2.5}$ conc.φ	Target $PM_{2.5}$ conc.	Change in exp. conc.	RR for 1$\mu g/m^3$	RR for change in exp. conc.	Attributable fraction$^{\Psi}$	Annual attributable cases	Remark
GP consultations (hypothetical)	1,000,000	1.021	28.6	27.6	1	1.00208	1.00208	0.002076	2,076	Central RR
GP consultations, low estimate*	8,179,735	1.021	28.6	27.6	1	1.00208	1.00208	0.002076	16,982	Central RR
GP consultations, low estimate*	8,179,735	1.01	28.6	27.6	1	1.000996	1.000996	0.000995	8,135	L95%C L
GP consultations, low estimate*	8,179,735	1.032	28.6	27.6	1	1.003155	1.003155	0.003145	25,724	U95%C L
GP consultations, low estimate*	8,179,735	1.021	28.6	10	18.6	1.00208	1.039412	0.037918	310,159	Central RR
GP consultations, low estimate*	8,179,735	1.01	28.6	10	18.6	1.000996	1.01868	0.018337	149,995	L95%C L
GP consultations, low estimate*	8,179,735	1.032	28.6	10	18.6	1.003155	1.060338	0.056904	465,462	U95%C L
GP consultations, high estimate§	21,575,032	1.021	28.6	27.6	1	1.00208	1.00208	0.002076	44,792	central RR
GP consultations, high estimate§	21,575,032	1.01	28.6	27.6	1	1.000996	1.000996	0.000995	21,457	L95% C L
GP consultations, high estimate§	21,575,032	1.032	28.6	27.6	1	1.003155	1.003155	0.003145	67,852	U95% C L
GP consultations, high estimate§	21,575,032	1.021	28.6	10	18.6	1.00208	1.039412	0.037918	818,081	Central RR
GP consultations, high estimate§	21,575,032	1.01	28.6	10	18.6	1.000996	1.01868	0.018337	395,630	L95% C L
GP consultations, high estimate§	21,575,032	1.032	28.6	10	18.6	1.003155	1.060338	0.056904	1,227,712	U95% C L
Mean annual GP consultations for new URI attributable to $PM_{2.5}$ (= 310,159 + 818,081)/2									564,120	Mean of low and high estimates of GP numbers

* Based on annual private GP consultation of 26,000,000, 86% of all private doctor consultations are for primary care; 78% are new cases, and 46.9% URTI).

$^{\Psi}$ Attributable fraction (AF) = (RR-1)/RR

§ Based on an estimate of 3949 private GPs in HK, and applying the ratio of URTI new cases from the 2002 Study of 65,556 URTI new cases among 12 GPs.

φ $PM_{2.5}$ concentration based on the mean annual concentration of $PM_{2.5}$ in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Table 7.7: Estimates of URI consultations to private GPs, attributed to NO_2 using low* and high§ estimates of total GP numbers

Upper respiratory infections (new cases)	Annual no. of patients	Unit RR per 10 $\mu g/m^3$	Annual NO_2 conc.φ	Target NO_2 conc.	Change in exp. conc.	RR for 1$\mu g/m^3$	RR for change in exp. conc.	Attributable fraction$^{\Psi}$	Annual attributable cases	Remark
GP consultations (hypothetical)	1,000,000	1.03	52.7	51.7	1	1.00296	1.00296	0.002952	2,952	Central RR
GP consultations, low estimate*	8,179,735	1.03	52.7	51.7	1	1.00296	1.00296	0.002952	24,143	Central RR
GP consultations, low estimate	8,179,735	1.02	52.7	51.7	1	1.001982	1.001982	0.001978	16,182	L95%C L
GP consultations, low estimate	8,179,735	1.04	52.7	51.7	1	1.00393	1.00393	0.003914	32,019	U95%C L
GP consultations, low estimate	8,179,735	1.03	52.7	40	12.7	1.00296	1.038253	0.036844	301,373	Central RR
GP consultations, low estimate	8,179,735	1.02	52.7	40	12.7	1.001982	1.025468	0.024836	203,150	L95%C L
GP consultations, low estimate	8,179,735	1.04	52.7	40	12.7	1.00393	1.051072	0.04859	397,454	U95%C L
GP consultations, high estimate§	21,575,032	1.03	52.7	51.7	1	1.00296	1.00296	0.002952	63,679	central RR
GP consultations, high estimate	21,575,032	1.02	52.7	51.7	1	1.001982	1.001982	0.001978	42,682	L95% C L
GP consultations, high estimate	21,575,032	1.04	52.7	51.7	1	1.00393	1.00393	0.003914	84,453	U95% C L
GP consultations, high estimate	21,575,032	1.03	52.7	40	12.7	1.00296	1.038253	0.036844	794,906	Central RR
GP consultations, high estimate	21,575,032	1.02	52.7	40	12.7	1.001982	1.025468	0.024836	535,832	L95% C L
GP consultations, high estimate	21,575,032	1.04	52.7	40	12.7	1.00393	1.051072	0.04859	1,048,333	U95% C L
Annual GP consultations for new URI attributable to PM2.5 (= 301,373 + 794,906)/2									548,140	Mean of low and high estimates of GP numbers

* Based on annual private GP consultation of 26,000,000, 86% of all private doctor consultations are for primary care; 78% are new cases, and 46.9% URTI).

$^{\Psi}$ Attributable fraction (AF) = (RR-1)/RR

§ Based on an estimate of 3949 private GPs in HK, and applying the ratio of URTI new cases from the 2002 Study of 65,556 URTI new cases among 12 GPs.

φ NO_2 concentration based on the mean annual concentration of NO_2 in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

Table 7.8: Estimates of the annual number of new URI consultations to GOPCs, attributed to $PM_{2.5}$

Upper respiratory infections (new cases)	Annual no. of patients	Unit RR per 10 $\mu g/m^3$	Annual $PM_{2.5}$ conc.φ	Target $PM_{2.5}$ conc.	Change in exp. conc.	RR for 1$\mu g/m^3$	RR for change in exp. conc.	Attributable fraction$^{\Psi}$	Annual attributable cases	Remark
GOPC consultations (hypothetical)	1,000,000	1.005	28.6	27.6	1	1.000499	1.000499	0.000499	499	Central RR for PM_{10} based on Tam et al, 2014[15]
GOPC new URI consultations *	2,060,813[16]	1.005	28.6	27.6	1	1.000499	1.000499	0.000499	1,028	Central RR for PM_{10}
GOPC new URI consultations *	2,060,813	1.002	28.6	27.6	1	1.0002	1.0002	0.0002	412	L95%C L
GOPC new URI consultations *	2,060,813	1.009	28.6	27.6	1	1.000896	1.000896	0.000896	1,846	U95%C L
GOPC new URI consultations *	2,060,813	1.005	28.6	10	18.6	1.000499	1.00932	0.009234	19,029	Central RR for PM_{10}
GOPC new URI consultations *	2,060,813	1.002	28.6	10	18.6	1.0002	1.003723	0.003709	7,644	L95%C L
GOPC new URI consultations *	2,060,813	1.009	28.6	10	18.6	1.000896	1.016805	0.016527	34,059	U95%C L

* Based on annual GOC consultation of 5,633,407 in 2012-13, assuming 78% are new cases, and 46.9% are URI.

$^{\Psi}$ Attributable fraction (AF) = (RR-1)/RR

φ $PM_{2.5}$ concentration based on the mean annual concentration of $PM_{2.5}$ in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

[15] In Tam's study, only RR for PM_{10} was available. This is used as a proxy of RR for $PM_{2.5}$ in our HIA study. In general, RR for PM_{10} is slightly lower in magnitude than that for $PM_{2.5}$.

[16] No. of new URI consultations based on the same assumption as for private GP consultations, with 78% of consultations being new cases, and 46.9% of all GOPC cases are URI.

Table 7.9: Estimates of the annual number of new URI consultations to GOPCs, attributed to NO_2

Upper respiratory infections (new cases)	Annual no. of patients	Unit RR per 10 $\mu g/m^3$	Annual NO_2 conc.φ	Target NO_2 conc.	Change in conc.	RR for 1$\mu g/m^3$	RR for change in exp. conc.	Attributable fraction$^{\Psi}$	Annual attributable cases	Remark
GOPC consultations (hypothetical)	1,000,000	1.01	52.7	51.7	1	1.000996	1.000996	0.000995	995	Central RR based on Tam et al, 2014
GOPC new URI consultations *	2,060,813	1.01	52.7	51.7	1	1.000996	1.000996	0.000995	2,050	Central RR based on Tam et al, 2014
GOPC new URI consultations *	2,060,813	1.006	52.7	51.7	1	1.000598	1.000598	0.000598	1,232	Lower 95% C L
GOPC new URI consultations *	2,060,813	1.013	52.7	51.7	1	1.001292	1.001292	0.001291	2,660	Upper 95% C L
GOPC new URI consultations *	2,060,813	1.01	52.7	40	12.7	1.000996	1.012717	0.012557	25,878	Central RR based on Tam et al, 2014
GOPC new URI consultations *	2,060,813	1.006	52.7	40	12.7	1.000598	1.007626	0.007568	15,597	Lower 95% C L
GOPC new URI consultations *	2,060,813	1.013	52.7	40	12.7	1.001292	1.016539	0.01627	33,529	Upper 95% C L

* Based on annual GOC consultation of 5,633,407 in 2012-13, assuming 78% are new cases, and 46.9% are URI.

$^{\Psi}$ Attributable fraction (AF) = (RR-1)/RR

φ NO_2 concentration based on the mean annual concentration of NO_2 in ambient stations (except Tap Mun) in 2014, from EPD's Air Quality Report (2014)

7.5 Years of life lost

Table 7.10a shows the expected years of life lost (EYLL) based on Pope's RR of 1.06 (i.e., this table shows the estimated numbers of life years lost per 10 $\mu g/m^3$ change in $PM_{2.5}$ concentration). The life table was developed by the COMEAP (2010) group and adapted to Hong Kong mortality statistics (2012). Table 7.10b shows the corresponding EYLL based on the adjusted RR of mortality for NO_2 (=1.039 per 10 $\mu g/m^3$ change in NO_2).

Table 7.10a: Estimated life of years lost per 10 $\mu g/m^3$ change in $PM_{2.5}$ concentration

Age	No. of deaths	Attributable fraction	No. of attributable deaths	Life Expectancy	Expected Years of life lost
0	137	0.056604	7.754717	83.6	648.2943
1-4	33	0.056604	1.867925	78.9	147.3792
5-9	30	0.056604	1.698113	74	125.6604
10-14	24	0.056604	1.358491	69	93.73585
15-19	63	0.056604	3.566038	64	228.2264
20-24	135	0.056604	7.641509	59.1	451.6132
25-29	148	0.056604	8.377358	54.2	454.0528
30-34	235	0.056604	13.30189	49.3	655.783
35-39	367	0.056604	20.77358	44.5	924.4245
40-44	612	0.056604	34.64151	39.7	1375.268
45-49	986	0.056604	55.81132	35	1953.396
50-54	1,573	0.056604	89.03774	30.4	2706.747
55-59	2,185	0.056604	123.6792	26	3215.66
60-64	2,620	0.056604	148.3019	21.7	3218.151
65-69	2,626	0.056604	148.6415	17.9	2660.683
70-74	3,743	0.056604	211.8679	14.2	3008.525
75-79	6,172	0.056604	349.3585	11	3842.943
80-84	7,713	0.056604	436.5849	8.7	3798.289
85+	14,489	0.056604	820.1321	8.7	7135.149
Total	43,891		2,484.396		36,643.98
				Average per person	14.75 (years)

On average, 36,643 (95% CI: 12,694 – 64,154) years of life are lost from 2,484 (95% CI: 861 – 4,350) attributed deaths with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration. This amounts to a mean loss of 14.75 years of life for an individual who dies from air pollution. The corresponding annual number of attributable deaths and total years of life lost for a 1 $\mu g/m^3$ change in $PM_{2.5}$ concentration are respectively 255 (95% CL: 87 – 456) and 3,761 (95% CL: 1,281 – 6,721). For a 18.6 $\mu g/m^3$ reduction in $PM_{2.5}$ concentration (i.e., a change

from the annual mean concentration from 28.6 μg/m^3 to the WHO AQG of 10 μg/m^3), the corresponding annual number of attributable deaths and total years of life lost are 4,508 (95% CL: 1,587 – 7,744) and 66,494 (95% CL: 23,411 – 114,218) respectively.

Table 7.10b: Estimated life of years lost per 10 μg/m^3 change in NO_2 concentration

Age	No. of deaths	Attributable fraction	No. of attributable deaths	Life Expectancy	Expected Years of life lost
0	137	0.037536	5.142445	83.6	429.9084
1-4	33	0.037536	1.238691	78.9	97.73272
5-9	30	0.037536	1.126083	74	83.33013
10-14	24	0.037536	0.900866	69	62.15977
15-19	63	0.037536	2.364774	64	151.3455
20-24	135	0.037536	5.067372	59.1	299.4817
25-29	148	0.037536	5.555342	54.2	301.0995
30-34	235	0.037536	8.820982	49.3	434.8744
35-39	367	0.037536	13.77575	44.5	613.0207
40-44	612	0.037536	22.97209	39.7	911.9919
45-49	986	0.037536	37.01059	35	1295.371
50-54	1,573	0.037536	59.04427	30.4	1794.946
55-59	2,185	0.037536	82.01636	26	2132.425
60-64	2,620	0.037536	98.34456	21.7	2134.077
65-69	2,626	0.037536	98.56978	17.9	1764.399
70-74	3,743	0.037536	140.4976	14.2	1995.066
75-79	6,172	0.037536	231.6728	11	2548.4
80-84	7,713	0.037536	289.5159	8.7	2518.788
85+	14,489	0.037536	543.8604	8.7	4731.586
Total	43,891		1647.497		24300.00
				Average per person	14.75 (years)

The annual number of years of life lost attributable to NO_2 is smaller, at 24,300 years (95% CI: 13,936 – 34,331) from 1,647 (95% CI: 945 – 2,328) deaths for a 10 μg/m^3 increase in NO_2 concentration. The annual number of years of life lost for a 1 μg/m^3 increase is 2,472 (95% CI: 1,407 – 3,518) from 168 (95% CI: 95 - 239) deaths. For a 12.7 μg/m^3 change in NO_2 concentration, the annual number of years of life lost is 30,703 years (95% CI: 17,647 – 43,284) from 2,082 deaths (1,196 – 2,935).

As the overlapping effect of NO_2 (with that of $PM_{2.5}$) has already been adjusted, the total number of life years lost from NO_2 can be added to that of $PM_{2.5}$. The annual total number of

life years attributable to these two pollutants amounts to 60,943 years (from 4,131 deaths) per 10 μg/m^3 increase in each pollutant. The annual total number of life years attributable to both pollutants at their respective counter-factuals is 97,197 years (from 6,590 deaths).

If the loss of life is borne only by those who suffered from premature deaths, the average loss of life expectancy is 14.75 years.[17]

7.6 Sensitivity Analysis

To test the variations in the health impact using different RRs, we have used an RR of 1.062 (95% CI: 1.041, 1.083) as recommended by HRAPIE Report (WHO 2013b) in our Instrument as a sensitivity analysis. In addition, we have tested the RR reported by Krewski et al (2009) in their extended study based on Pope's original study. The results are shown in Table 7.11.

Table 7.11: Sensitivity analysis of health impact assessment with different RRs

RR per 10 μg/m^3	Annual $PM_{2.5}$ conc.	Target $PM_{2.5}$ conc.	Change in exp. conc.	RR per 1ug/m^3	RR per change in exp. conc.	Attributable fraction:	Annual attributable deaths	Remark
1.06	28.6	10	18.6	1.005844	1.114471	0.102714	4315.72	Pope's ACS Study
1.02	28.6	10	18.6	1.001982	1.03752	0.036163	1519.45	Pope's ACS Study L95% CL
1.11	28.6	10	18.6	1.010491	1.214229	0.176432	7413.16	Pope's ACS Study U95% CL
1.062	28.6	10	18.6	1.006034	1.118386	0.105854	4447.67	Hoek's meta-analysis
1.04	28.6	10	18.6	1.00393	1.075677	0.070353	2956.03	Hoek's meta-analysis L95% CL
1.083	28.6	10	18.6	1.008005	1.159869	0.137834	5791.36	Hoek's meta-analysis U95% CL
1.078	28.6	10	18.6	1.007539	1.149929	0.130381	5478.21	Krewski's extended study
1.046	28.6	10	18.6	1.004507	1.087249	0.080247	3371.75	Krewski's extended study L95% CL
1.115	28.6	10	18.6	1.010945	1.224422	0.183288	7701.23	Krewski's extended study U95% CL

Using the WHO (Hoek's meta-analysis) RR of all-cause mortality (at 1.062 per 10 μg/m^3) and

[17] If we assume the loss of life years is to be equally borne by the Hong Kong population (7,070,388), the average "shortening of life expectancy" for an individual with a life expectancy of 82 years (averaged for both genders) would be quite small, at about 3.5 days. The reality is probably in between – some people die much earlier, and suffer from a significant shortening of life expectancy, while others are less affected, with much smaller shortening of life expectancy.

Krewski's extended analysis (at 1.078 per 10 μg/m^3), the total numbers of attributable deaths are 4,448 and 5,478 (3.06% and 26.9% higher than that using Pope's RR), respectively. While the number of attributable deaths obtained from the WHO RR are fairly close to that using Pope's RR, the 95% confidence interval is much smaller (i.e., more precise) than that using the latter RRs. By contrast, the attributable deaths using Krewski's RR is substantially higher. We caution against using this higher RR because of the supralinear relationship of the exposure response function, in which the RR flattens at higher $PM_{2.5}$ concentrations. As the annual mean $PM_{2.5}$ concentration in Hong Kong is substantially higher (about 3 times) than in most US cities, using Pope's original RR would be more conservative than using Krewski's RR, which would have over-estimated the risk of $PM_{2.5}$ in the local context. It should be noted, however, that regardless of the RRs used, the 95% confidence intervals (CI) in all 3 scenarios are fairly wide, and the estimated attributable deaths using the central RRs are well within the 95% CI – 1.04 to 1.083 (derived from the WHO meta-analysis), the narrowest 95% CI of the 3 sets of figures.

7.7 References

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

Food and Health Bureau, 2010. My Health, My Choice. Healthcare Reform Second Stage Public Consultation Report, Appendix C, p87. Food and Health Bureau, The Government of the Hong Kong Special Administrative Region, 2010.

Environmental Protection Department, 2014. Air Quality Report. Hong Kong Government.

Hoek G, Krishnan R, Beelen R, Peters A, Ostro B, Brunekreef B, et al. Long-term air pollution exposure and cardio-respiratory mortality: a review. Environmental Health 2013; 12(1):43.

Hospital Authority Annual Report, 2012 – 2013. Hospital Authority, Hong Kong, Appendix 9, p211.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA III, Thurston G, Calle EE, Thun MJ. 2009. *Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality*. HEI Research Report 140. Health Effects Institute, Boston, MA.

Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 2002; 287(9):1132-41.

Qiu H, Yu ITS, Wang XR, Tian LW, Tse LA, Wong TW. Differential Effects of Fine and Coarse Particles on Daily Emergency Cardiac Hospitalizations in Hong Kong. *Atmospheric Environment* 2013; 64:296-302.

Tam WWS, Wong TW, Ng L, Wong SYS, Wong AHS. Association between air pollution and general outpatient clinic consultations for upper respiratory tract infection in Hong Kong. *PLOS ONE* 2014; 9(1) e86913:1-6. Published 23 Jan 2014. (Accepted: 17 Dec 2013). Accessible at: http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0086913

Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015. Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKingsReport14072015final.pdf

World Health Organization (2006). *Air Quality Guidelines Global Update 2005*. World Health Organization Regional Office for Europe, Copenhagen.

WHO 2013a. Review of Evidence on health aspects for air pollution – REVIHAAP Project. Technical Report, World Health Organization Regional Office for Europe, Denmark, 2013. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf

WHO 2013b. Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration-response functions for cost-benefit analysis of particulate matter, ozone and nitrogen dioxide. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf

Wun YT, Wong TW, Tam W, Yu TS. Patient characteristics of encounters in general practice. *The Hong Kong Practitioner* 2002;24:59-65.

Chapter 8: Economic impact assessment

8.1 Premature mortality

8.1.1 Valuation using the value of statistical life approach

The economic impacts of premature mortality attributable to air pollution, using different assumptions of the value of a statistical life (VOSL) are presented in Table 8.1. Our estimates of VOSL are based on the average of two values, described in Section 3.4.3. The higher value is derived from a VOSL of US$ 3 million (2010 value), from WHO Regional Office for Europe (WHO, 2015), and adjusted to US$2,872,817 (2012 value) using the per capita GDP for Hong Kong in 2010, and a mean annual inflation rate of 4.1% (2 years, from 2010 to 2012), while the lower value is based on the VOSL in China of RMB795,000 in China (Wang & He, 2010, World Bank Paper), and adjusted by the Hong Kong per capital GDP and inflation rate to US$1,171,048 (2012 value). The average VOSL from these two values is US$2,021,933 (HK$15,710,419, or about HK$15.7 million).

The economic impact for 244 estimated deaths (based on 2012 mortality statistics) that may be attributed to a 1 $\mu g/m^3$ change in $PM_{2.5}$ ranges from US$285,872,209.7 (95% CI: US$97,340,285.01 – US$510,822,118.01) , using the lower estimate, to US$701,302,204.39 (95% CI: US$238,795,357.28 – US$1,253,149,712.56), using the higher VOSL estimate. Using the average VOSL of US$2,021,933, the economic impact is US$493,587,329.10 (95% CI: US$168,067,862.70 – US$881,986,133.39. This amounts to HK$3,835,173,547.13 (95% CI: HK$1,305,887,293.19 – HK$6,853,032,256.42), using an exchange rate of HK$7.77 per US$. When a counter-factual for $PM_{2.5}$ of 10 $\mu g/m^3$ against the annual mean concentration of 28.6 $\mu g/m^3$ is used in the estimate, the corresponding values are: US$5,053,911,607.84, or HK$39,268,893,192.95, or HK$39.27 billion, (95% CI: US$1,779,352,057.83 – US$8,681,165,736.75) using the lower VOSL estimate, and US$12,398,264,788.05, or HK$96,334,517,403.13, or HK$96.33 billion (95% CI: US$4,365,109,577.69 – US$21,296,650,955.69) for the higher VOSL. Using the average VOSL of US$2,021,933, the economic impact is US$8,726,090,355.80 (95% CI: US$3,072,231,577.49 – US$14,988,912,052.80). This is equivalent to HK$67,801,722,064.60 (95% CI: HK$23,871,239,357.08 – HK$116,463,846,650.24). The average estimate, therefore, is HK$67.80 billion, with a 95% CI ranging from HK$23.87 billion to HK$116.46 billion. It should be noted that all the above figures reflect an economic impact on an annual basis.

Table 7.10 (Section 7.5) shows that are 505 estimated deaths attributable to $PM_{2.5}$ (per 10 $\mu g/m^3$) among those aged 15 – 64 (20.4% of total attributable deaths) in Hong Kong, and 1,967 deaths among those aged 65 and above (79.6% of total). If we assume the productivity loss of those aged 65 and above (i.e., "officially retired" and economically unproductive) as nil, the overall productivity loss of deaths attributed to air pollution is very small, at 0.13%, 0.32%, and 0.19% of VOSL, using European, Chinese and their average estimates respectively. This clearly indicates the values given by individuals of their own lives are not limited to productivity alone. It should be noted, however, that the economic impact based on VOSL has already included all losses, including loss of productivity. Hence it is inappropriate to sum up the two values.

The independent effects of NO_2 on premature mortality have also been evaluated (Table 8.2). The economic impact for a 1 $\mu g/m^3$ change in NO_2 is US$324,408,059.49 (95% CI: US$184,674,956.59 – US$461,648,561.42) for the average VOSL estimate. The range varies from the lower estimate of US$187,888,228.37 to the higher VOSL estimate of US$460,927,730.17. In HK dollar values, the estimate of economic impact averages at HK$2,520,650,622.23 (HK$2.52 billion) and ranges from HK$1,459,891,534.42 (1.46 billion) to HK$3,581,408,463.38 (HK$3.58 billion). When a counter-factual for NO_2 of 40 $\mu g/m^3$ against the annual mean concentration of 52.7 $\mu g/m^3$ is used in the estimate, the corresponding value is US$4,029,189,709.04 (95% CI: US$2,315,776,744.37 - US$5,680,125,455.55). The range varies from the lower VOSL of US$2,333,595,895.81 to US$5,724,781,529.54 for the higher VOSL estimate. The equivalent value in HK dollar (using the average VOSL) is HK$31,306,804,039.26 (HK$31.31 billion), with a range from HK$18,132,040,110.41 (HK$18.13 billion) to HK$44,481,552,484.50 (HK$44.48 billion).

The combined effects of $PM_{2.5}$ and NO_2, assuming a 30% overlap (WHO 2013a ,b) are presented in Table 7.3 (See also Section 5.2). The economic impact on premature mortality for a 1 $\mu g/m^3$ change in NO_2 and $PM_{2.5}$ is US$817,995,388.59 (95% CI: $352,742,819.29 - $1,343,634,694.80), using the average VOSL. It ranges from a lower VOSL estimate of US$473,760,438.07 to the higher estimate of US$1,162,229,934.56. In HK dollar, the average VOSL is HK$6,355,824,169.36 (HK$6.36 billion), with a range from HK$3,681,118,603.77 (HK$3.68 billion) to HK$9,030,526,591.51 (HK$9.03 billion). The economic impact on premature mortality for a 18.6 $\mu g/m^3$ change in $PM_{2.5}$ and a 12.7 $\mu g/m^3$ change NO_2 is much higher than the unit change, at US$12,755,280,064.85 (95% CI: $5,388,008,321.86 - $20,669,037,508.35) for the average VOSL estimate, US$7,387,507,503.65 for the lower VOSL estimate, and US$18,123,046,317.58 for the higher VOSL estimate. In HK dollars, the values are: average VOSL: HK$99,108,526,103.86 (HK$99.11 billion); lower VOSL:

HK$57,400,933,303.36 (HK$57.40 billion), and higher VOSL: HK$140,816,069,887.63 (HK$140.82 billion). The productivity loss amounts to US$23,877,817.97 (= HK$185,530,645.60) [95% CI: US$10,086,323.57 (=HK$78,370,734.11) to US$38,692,330.76 (=HK$300,639,409.99)]. This amount is 0.32%, 0.19% and 0.13% of the economic impact using low, average and high VOSL estimates respectively.

When evaluating the long-term economic impact of an air pollution control strategy, a commonly adopted practice is to discount the values of future reduction in the number of premature deaths by an arbitrary percentage. This is because people and society value goods and services that are available at present more than when they are available at a later date. This is also known as 'time preference'. The benefits on health that occurs in future years therefore have to be discounted to the present value. The choice of a proper discount rate is relevant in the assessment of the economic impact of a policy or strategy that brings long-term health benefits to the population, where later benefits should be properly discounted according to the time when the expected improvement in air quality will occur. Discount rates of 0%, 1.5%, 3% and 6% have been used in the COMEAP (2000) study as a sensitivity analysis of the effect of discounting on the value of the health impact. The UK Intergovernmental Group on Costs and Benefits (IGCB) recommends a discounting rate of 1.5%. In the estimate of long-term benefits resulting from, say number of lives saved, one also needs to take into account the assumption that the 'willingness to pay', or the value of a statistical life will rise with economic growth (known as 'uplifting'). In the UK, the HMS Treasury has recommended an annual rate of uplifting of 2%, close to the discounting rate of 1.5% recommended by IGCB. In our EIA, we have presented the values without discount or uplifting. We have no local data on either the local discounting rate or the annual percentage for uplifting. Using an undiscounted approach is also justified as our results are expressed as economic impact in one year, instead of the cumulative impact over decades. When we make long-term projections, we need to take into account of the effects of these opposing rates.

8.1.2 Productivity loss from premature mortality

By contrast, the estimated losses of productivity from the loss of lives are much smaller, at US$923,992.91 (=HK$7,179,424.94, using the average VOSL), with a 95% CI ranging from US$314,622.17 (= HK$2,444,614.22) to US$1,651,073.46 (=HK$12,828,840.76), per 1 $\mu g/m^3$ change in $PM_{2.5}$. On the basis of counter-factual (10 $\mu g/m^3$) used, the central estimate (per 18.6 $\mu g/m^3$) is US$16,335,195.78 (=HK$126,924,471.20, or HK$126.92 million), with a 95% CI ranging from US$5,751,201.54 (= HK$44,686,835.97) to US$28,059,165.44 (=HK$218,019,715.44). These values are 0.19% of the estimated value using the average

VOSL. The impact per 1 μg/m^3 change in NO_2 is US$607,290.20 (=HK$4,718,644.86), with a 95% CI ranging from US$345,710.56 (=HK$2,686,171.04) to US$864,203.71 (=HK$6,714,862.80). The impact per 12.7 μg/m^3 is US$7,542,622.19 (=HK$58,606,174.40), with a 95% CI ranging from US$4,335,122.03 (=HK$33,683,898.14) to US$10,633,165.32 (=HK$82,619,694.55). The productivity loss from the combined effects of both pollutants is at US$1,531,283.11 (=HK$11,898,069.80), with a 95% CI ranging from US$660,332.72 (=HK$5,130,785.26) to US$2,515,277.16 (=HK$19,543,703.56) per 1 μg/m^3 change in each pollutant, and at US$23,877,817.97 (=HK$185,530,645.60), 95% CI at US$10,086,323.57 (=HK$78,370,734.11) to US$38,692,330.76 (=HK$300,639,409.99) for the respective counter-factuals used (10 μg/m^3 of $PM_{2.5}$ and 40 μg/m^3 of NO_2).

Table 8.1: Economic impact of premature mortality attributable to $PM_{2.5}$, using VOSL and the loss of productivity approaches

RR (per 10 μg/m^3)	Change in $PM_{2.5}$ conc. (μg/m^3)	No. of attributable deaths	VOSL-Europe* (US$)	VOSL-China§ (US$)	VOSL – averageΨ (US$)	Unit productivityφ loss (US$)
1.06	1	244	$701,302,204.39	$285,872,209.70	$493,587,329.10	923,992.91
1.02 (L95%)	1	83	$283,795,357.28	$97,340,285.01	$168,067,862.70	314,622.17
1.11 (U95%)	1	436	$1,253,149,712.56	$510,822,118.01	$881,986,133.39	$1,651,073.46
1.06	18.6	4,316	$12,398,264,788.05	$5,053,911,607.84	$8,726,090,355.80	$16,335,195.78
1.02 (L95%)	18.6	1,519	$4,365,109,577.69	$1,779,352,057.83	$3,072,231,577.49	$5,751,201.54
1.11 (U95%)	18.6	7,413	$21,296,650,955.69	$8,681,165,736.75	$14,988,912,052.80	$28,059,165.44

* Unit VOSL-Europe = US$2,872,817, adjusted to Hong Kong per capita GDP, 2010 value.

§ Unit VOSL-China = US$1,171,048, adjusted to Hong Kong per capita GDP, 2010 value.

Ψ Unit VOSL= US$2,021,933, average of Europe and China values.

φ Unit productivity loss = HK$12,000 per month per person (gender-adjusted) [Source: Census and Statistics Department. http://www.censtatd.gov.hk/hkstat/sub/gender/employment_earnings/

Note: economic impact is calculated without rounding off the no. of attributable deaths. Results are slightly different from that calculated by rounding off the no. of attributable deaths.

Table 8.2: Economic impact of premature mortality attributable to NO_2, using VOSL and the loss of productivity approaches

RR (per 10 $\mu g/m^3$)	Change in NO_2 conc. ($\mu g/m^3$)	No. of attributable deaths	VOSL-Europe* (US$)	VOSL-China[§] (US$)	VOSL – average[Ψ] (US$)	Unit productivity[φ] loss (US$)
1.039	1	160	$460,927,730	$187,888,228	$324,408,059	$607,290
1.022 (L95%)	1	91	$262,391,165	$106,958,657	$184,674,957	$345,711
1.056 (U95%)	1	228	$655,922,741	$267,374,154	$461,648,561	$864,204
1.039	12.7	1993	$5,724,781,530	$2,333,595,896	$4,029,189,709	$7,542,622
1.022 (L95%)	12.7	1145	$3,290,318,126	$1,341,234,217	$2,315,776,744	$4,335,122
1.056 (U95%)	12.7	2809	$8,070,475,615	$3,289,772,487	$5,680,125,456	$10,633,165

* Unit VOSL-Europe = US$2,872,817, adjusted to Hong Kong per capita GDP, 2010 value.

[§] Unit VOSL-China = US$1,171,048, adjusted to Hong Kong per capita GDP, 2010 value.

[Ψ] Unit VOSL= US$2,021,933, average of Europe and China values.

[φ] Unit productivity loss = HK$12,000 per person per month (gender-adjusted)

Note: economic impact is calculated without rounding off the no. of attributable deaths. Results are slightly different from that calculated by rounding off the no. of attributable deaths.

Table 8.3: Economic impact of premature mortality attributable to the combined effects of $PM_{2.5}$ and NO_2, using VOSL and the loss of productivity approaches

RR (per 10 $\mu g/m^3$)	Change in conc. ($\mu g/m^3$)		No. of attributable deaths	VOSL-Europe* (US$)	VOSL-China§ (US$)	VOSL -averageΨ (US$)	Unit productivity loss φ (US$)
	$PM_{2.5}$	NO_2					
Central	1	1	404	$1,162,229,935	$473,760,438	$817,995,389	$1,531,283
Lower 95%	1	1	174	$501,186,522	$204,298,942	$352,742,819	$660,333
Upper 95%	1	1	665	$1,909,072,453	$778,196,271	$1,343,634,695	$2,515,277
Central	18.6	12.7	6308	$18,123,046,318	$7,387,507,504	$12,755,280,065	$23,877,818
Lower 95%	18.6	12.7	2,665	$7,655,427,704	$3,120,586,275	$5,388,008,322	$10,086,324
Upper 95%	18.6	12.7	10,222	$29,367,126,570	$11,970,938,224	$20,669,037,508	$38,692,331

* Unit VOSL-Europe = US$2,872,817, adjusted to Hong Kong per capita GDP, 2010 value.

§ Unit VOSL-China = US$1,171,048, adjusted to Hong Kong per capita GDP, 2010 value.

Ψ Unit VOSL= US$2,021,933, average of Europe and China values.

φ Unit productivity loss = HK$12,000 per person per month (gender-adjusted)

Note: economic impact is calculated without rounding off the no. of attributable deaths. Results are slightly different from that calculated by rounding off the no. of attributable deaths.

8.2 Cost of illness (COI) estimate

8.2.1 Hospital illnesses

The annual number of hospital admissions for cardiovascular and respiratory illnesses attributable to air pollution, based on statistics on emergency hospital admissions to Hospital Authority (HA) hospitals, are shown in Table 7.4. As the estimates of hospital admissions are based on single-pollutant models, we shall take the higher of two estimates from the attributable deaths due to $PM_{2.5}$ and NO_2. For hospital admissions from cardiovascular illnesses, the costs estimates using NO_2 are higher than that from $PM_{2.5}$, while for admissions from respiratory illnesses, cost estimates using $PM_{2.5}$ are higher. The direct costs of illness are about $16.67 million and $34.96 million for hospital admissions from cardiovascular and respiratory illnesses respectively (based on the counter-factuals used), or a total sum of $51.63 million per year. Like cost estimates for premature deaths, the values used for the counter-factuals (the level of air pollutants estimated to be achieved) have significant influence on the cost estimates, besides the mean concentrations of the air pollutants concerned. The corresponding values (taking the maximum impact from either of the two pollutants) are: HK$1.90 million per unit change in $PM_{2.5}$ (for respiratory diseases) and HK$1.32 million per unit change in NO_2 (for cardiovascular diseases), or a total of HK$3.22 million per year.

The unit cost of intensive care unit (ICU) is $23,000 per bed-day and is about 5 times higher than that for a general hospital bed-day. A certain percentage of patients will require a period of stay in the ICU. However, we could not estimate the costs associated with patients staying in the intensive care unit (ICU), as we have no hospital data on the proportion of patients suffering from cardiovascular and respiratory illnesses using ICU and their respective period of stay. Hence, our estimate of the direct cost of illness (COI) estimate is lower than the true cost.

In addition to hospital bed costs, all the patients with cardiovascular and respiratory diseases estimated to be attributable to air pollution were admitted to HA hospitals through the Accident and Emergency Department, The unit cost of each attendance is HK$990. The annual direct cost of A&E attendance (for both diseases combined) amounts to HK$146,433 per $\mu g/m^3$ change in $PM_{2.5}$, and HK$2,702,954 per 18.6 $\mu g/m^3$ change in $PM_{2.5}$ (Table 7.4b). The corresponding cost is HK$147,449 per $\mu g/m^3$ change in NO_2, and HK$1,863,164 per 12.7 $\mu g/m^3$ change in NO_2. The total direct hospital cost of cardiovascular and respiratory illnesses including A & E visits (Table 7.4c), amounts to HK$53,813,364 (53.81 million) for $PM_{2.5}$ (per 18.6 $\mu g/m^3$ change), and $37,093,902 (37.10 million) for NO_2 (per 12.7 $\mu g/m^3$ change). These costs are estimated based on single-pollutant models (which do not take into account the overlap in effects of other air pollutants). Hence, we shall take the higher of the two estimates, i.e., that attributable to $PM_{2.5}$.

8.2.2 Productivity loss associated with hospital illnesses

The associated productivity loss was estimated based on the median length of hospital stay (4 days, for both cardiovascular and respiratory illnesses), instead of this period plus the sick leave granted by the attending doctor, owing to the lack of information on the latter figures. The loss of earnings associated with one episode of hospital illness for either cardiovascular or respiratory illnesses is $1,600 (based on a median monthly wage of $12,000). Hence the total loss of productivity is estimated to be 2,730 episodes [18] x $1,600 = $4,368,320, rounded to $4.37 million per year (using the higher number of attributable hospital admissions from estimates using $PM_{2.5}$). This figure is small (8.2%) compared to the direct medical cost of illness.

[18] This number is the sum of 1,867.6 attributable no. of respiratory disease admissions, and 862.6, attributable no. of cardiovascular admissions.

Table 8.4a: Estimated costs of annual hospital admissions for cardiovascular and respiratory illnesses attributable to $PM_{2.5}$ and NO_2

Pollutant	Annual attributable hospital admissions [19]						Daily hospital cost: (@HK$4,680 per bed-day)						Total cost per illness[20] (median length of stay: 4 days)					
	Cardio-vascular		Respiratory		Total		Cardiovascular		Respiratory		Total		Cardiovascular		Respiratory		Total	
$PM_{2.5}$ ($\mu g/m^3$)	1	18.6	1	18.6	1	18.6	1	18.6	1 $\mu g/m^3$	18.6 $\mu g/m^3$	1	18.6	1	18.6	1	18.6	1	18.6
Central RR	47	863	101	1,868	148	2,731	$218,309	$4,037,139	$473,920	$8,740,464	$692,229	$12,777,603	$873,237	$16,148,556	$1,895,680	$34,961,854	$2,768,917	$51,110,410
Lower 95% CL	25	473	68	1,257	93	1,730	$119,274	$2,211,488	$318,131	$5,883,628	$437,405	$80,951,116	$477,094	$8,845,952	$1,272,523	$23,534,510	$1,749,617	$32,380,462
Upper 95% CL	68	1,262	134	2,473	202	3,735	$320,306	$5,907,368	$629,167	$11,571,522	$949,473	$17,455,150	$1,281,223	$23,629,474	$2,516,668	$46,286,087	$3,797,891	$69,915,561
NO_2 ($\mu g/m^3$)	1	12.7	1	12.7	1	12.7	1	12.7	1	12.7	1	12.7	1	12.7	1	12.7	1	12.7
Central RR	71	891	78	991	149	1,882	$330,158	$4,168,697	$366,873	$4,638,987	$697,031	$8,807,684	$1,320,632	$16,674,789	$1,467,493	$18,555,949	$2,788,125	$35,230,738
Lower 95% CL	52	652	52	663	104	1,315	$241,371	$3,052,405	$244,919	$3,101,389	$486,290	$6,153,794	$965,483	$12,209,621	$979,677	$12,405,556	$1,945,160	$24,615,177
Upper 95% CL	89	1,119	104	1,318	193	2,437	$415,410	$5,237,262	$488,507	$6,167,949	$903,917	$11,405,211	$1,661,641	$20,949,046	$1,954,026	$24,671,796	$3,615,667	$45,620,842

Note: 1. The costs are calculated from attributable hospital admissions without rounding off the decimals. Hence, the results are slightly different from that using round numbers of hospital admissions.

2. To avoid over-estimation of the cost, we adopt the higher estimate (i.e., that attributed to $PM_{2.5}$), instead of summing up the costs attributed to each pollutant, because attributable cases are estimated from RRs based on single-pollutant models.

[19] The annual total of cardiovascular hospital admissions in 2012 was 70,934, while that for respiratory diseases was 140,953.

[20] For both cardiovascular and respiratory diseases, the median length of stay was 4 days. (2012 HA data).

Table 8.4b: Estimated cost of annual Accident and Emergency Department visits of hospital admissions for cardiovascular and respiratory illnesses

Pollutant	Annual attributable A & E visits prior to hospital admissions [21]						Cost of A & E visit (@HK$990 per visit)					
	Cardio-vascular		Respiratory		Total		Cardio-vascular		Respiratory		Total	
$PM_{2.5}$	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3
Central RR	47	863	101	1,868	148	2,731	$46,181	$854,010	$100,252	$1,848,944	$146,433	$2,702,954
Lower 95% CL	25	473	68	1,257	93	1,730	$25,231	$467,815	$67,297	$1,244,614	$92,528	$1,712,429
Upper 95% CL	68	1,262	134	2,473	202	3,735	$67,757	$1,249,636	$133,093	$2,447,822	$200,850	$3,697,458
NO_2	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3
Central RR	71	891	78	991	149	1,882	$69,841	$881,840	$77,608	$981,324	$147,449	$1,863,164
Lower 95% CL	52	652	52	663	104	1,315	$51,059	$645,701	$51,810	$656,063	$102,869	$1,301,764
Upper 95% CL	89	1,119	104	1,318	193	2,437	$87,875	$1,107,882	$103,338	$1,304,758	$191,213	$2,412,640

Note: 1. The costs are calculated from attributable hospital admissions without rounding off the decimals. Hence, the results are slightly different from that using round numbers of hospital admissions.

2. To avoid over-estimation of the cost, we adopt the higher estimate (i.e., that attributed to $PM_{2.5}$), instead of summing up the costs attributed to each pollutant, because attributable cases are estimated from RRs based on single-pollutant models.

[21] The annual total of cardiovascular hospital admissions in 2012 was 70,934, while that for respiratory diseases was 140,953.

Table 8.4c: Total hospital cost (A & E visit and hospitalization) for cardiovascular and respiratory illnesses

Pollutant	Annual attributable A & E visits prior to hospital admissions [22]						Total cost (A & E visit (@HK$990 per visit and 4 days of hospital stay)					
	Cardio-vascular		Respiratory		Total		Cardio-vascular		Respiratory		Total	
$PM_{2.5}$	1μg/m^3	18.6μg/m^3	1μg/m^3	18.6μg/m^3	1μg/m^3	18.6μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3	1 μg/m^3	18.6 μg/m^3
Central RR	47	863	101	1,868	148	2,731	$919,418	$17,002,566	$1,995,932	$36,810,798	$2,915,350	$53,813,364
Lower 95% CL	25	473	68	1,257	93	1,730	$502,325	$9,313,767	$1,339,820	$24,779,124	$1,842,145	$61,589,922
Upper 95% CL	68	1,262	134	2,473	202	3,735	$1,348,979	$24,879,109	$2,649,761	$48,733,909	$3,998,740	$73,613,018
NO_2	1μg/m^3	12.7μg/m^3	1μg/m^3	12.7μg/m^3	1μg/m^3	12.7μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3	1 μg/m^3	12.7 μg/m^3
Central RR	71	891	78	991	149	1,882	$1,390,473	$17,556,629	$1,545,101	$19,537,273	$2,935,574	$37,093,902
Lower 95% CL	52	652	52	663	104	1,315	$1,016,543	$12,855,322	$1,031,487	$13,061,619	$2,048,030	$25,916,941
Upper 95% CL	89	1,119	104	1,318	193	2,437	$1,749,516	$22,056,929	$2,057,364	$25,976,554	$3,806,880	$48,033,483

[22] The annual total of cardiovascular hospital admissions in 2012 was 70,934, while that for respiratory diseases was 140,953.

8.2.3 General Practitioners (GP) consultations

8.2.3.1 Direct cost of illness for private GP visits and associated productivity loss

Tables 8.5 – 8.6 summarize the economic impacts of private GP consultations for new cases of URI and their associated productivity loss attributable to $PM_{2.5}$ and NO_2.

Table 8.5: Cost of illness for private GP visits for URI (new cases) attributable to $PM_{2.5}$ and associated productivity loss

Annual attributable cases of URI[§]	Total cost of GP visits ($250 per session)	Productivity loss (=median daily wage = $12,000/30 = $400)	COI + productivity loss	Remarks*
16,982	$4,245,478.56	$6,792,765.70	$11,038,244.26	Lower estimate of GP no.; RR=Central estimate ; cases per 1μg/m^3
8,135	$2,033,764.79	$3,254,023.66	$5,287,788.45	Lower estimate of GP no.; RR=L95% CL; cases per 1μg/m^3
25,725	$6,431,134.97	$10,289,815.95	$16,720,950.91	Lower estimate of GP no.; RR=U95% CL; cases per 1μg/m^3
310,159	$77,539,662.14	$124,063,459.43	$201,603,121.57	Lower estimate of GP no.; RR=Central estimate; cases per 18.6 μg/m^3
149,995	$37,498,771.47	$59,998,034.35	$97,496,805.83	Lower estimate of GP visits; RR=L95% CL; cases per 18.6 μg/m^3
465,462	$116,365,533.87	$186,184,854.20	$302,550,388.07	Lower estimate of GP visits; RR=U95% CL; cases per 18.6 μg/m^3
44,792	$11,197,958.57	$17,916,733.71	$29,114,692.27	Higher estimate of GP visits,; RR=Central estimate; cases per 1μg/m^3
21,457	$5,364,298.39	$8,582,877.43	$13,947,175.83	Higher estimate of GP visits,; RR=L95% CL; cases per 1μg/m^3
67,852	$16,962,889.30	$27,140,622.88	$44,103,512.18	Higher estimate of GP visits,; RR=U95% CL; cases per 1μg/m^3
818,081	$204,520,152.68	$327,232,244.29	$531,752,396.96	Higher estimate of GP visits,; RR=Central estimate; cases per 18.6 μg/m^3
395,630	$98,907,504.30	$158,252,006.88	$257,159,511.18	Higher estimate of GP visits; RR=L95% CL; cases per 18.6 μg/m^3
1,227,712	$306,928,042.97	$491,084,868.76	$798,012,911.73	Higher estimate of GP visits; RR=U95% CL; cases per 18.6 μg/m^3
30,887	$7,721,718.56	$12,354,749.70	$20,076,468.27	**Mean estimate of GP visits; RR=Central estimate; cases per 1μg/m^3**
564,120	$141,029,907.41	$225,647,851.86	$366,677,759.27	**Mean estimate of GP visits; RR=Central estimate; cases per 18.6μg/m^3**

* Lower estimate of GP visits for new URI illness = 8,179,735 per annum; higher estimate = 21,575,032

§ Attributable cases of URI are estimated using a change of $PM_{2.5}$ concentration of 1 μg/m^{3}and 18.6 μg/m^3 (i.e., annual mean concentration of 28.6 μg/m^3 and a counter-factual of 10 μg/m^{3}μg/m^3) respectively.

Table 8.6: Cost of illness for private GP visits for URI (new cases) attributable to NO_2 and associated productivity loss

Annual attributable cases of URI[§]	Total cost of GP consultation ($250 per session)	Productivity loss (=median daily wage = $12,000/30 = $400)	COI + productivity loss	Remarks*
24,143	$6,035,654.65	$9,657,047.44	$15,692,702.09	Lower estimate of GP no.; RR=Central estimate ; cases per 1μg/m^3
16,182	$4,045,499.29	$6,472,798.87	$10,518,298.16	Lower estimate of GP no.; RR=L95% CL; cases per 1μg/m^3
32,019	$8,004,668.50	$12,807,469.60	$20,812,138.09	Lower estimate of GP no.; RR=U95% CL; cases per 1μg/m^3
301,373	$75,343,131.87	$120,549,010.99	$195,892,142.86	Lower estimate of GP no.; RR=Central estimate; cases per 12.7 μg/m^3
203,150	$50,787,416.83	$81,259,866.93	$132,047,283.73	Lower estimate of GP visits; RR=L95% CL; cases per 12.7μg/m^3
397,454	$99,363,569.71	$158,981,711.54	$258,345,281.25	Lower estimate of GP visits; RR=U95% CL; cases per 12.7μg/m^3
63,679	$15,919,762.56	$25,471,620.09	$41,391,382.56	Higher estimate of GP visits,; RR=Central estimate; cases per 1μg/m^3
42,682	$10,670,489.27	$17,072,782.83	$27,743,272.09	Higher estimate of GP visits,; RR=L95% CL; cases per 1μg/m^3
84,453	$21,113,272.593	$33,781,236.14	$54,894,508.73	Higher estimate of GP visits,; RR=U95% CL; cases per 1μg/m^3
794,906	$198,726,540.81	$317,962,465.30	$516,689,006.11	Higher estimate of GP no.; RR=Central estimate; cases per 12.7 μg/m^3
535,832	$133,957,899.18	$214,332,638.69	$348,290,537.88	Higher estimate of GP visits; RR=L95% CL; cases per 12.7μg/m^3
1,048,333	$262,083,324.68	$419,333,319.48	$681,416,644.16	Higher estimate of GP visits; RR=U95% CL; cases per 12.7μg/m^3
43,911	$10,977,708.60	$17,564,333.77	$28,542,042.37	**Mean estimate of GP visits; RR=Central estimate; cases per 1μg/m^3**
548,139	$137,034,836.34	$219,255,738.14	$356,290,574.48	**Mean estimate of GP visits; RR=Central estimate; cases per 12.7μg/m^3**

* Lower estimate of GP visits for new URI illness = 8,179,735 per annum; higher estimate = 21,575,032

§ Attributable cases of URI are estimated using a change of $PM_{2.5}$ concentration of 1 μg/m^{3}and 18.6 μg/m^3 (i.e., annual mean concentration of 28.6 μg/m^3 and a counter-factual of 10 μg/m^{3}μg/m^3) respectively.

8.2.3.2 Direct cost of illness for General Outpatients Clinics (GOPC) visits and associated productivity loss

Tables 8.7 – 8.8 summarize the economic impacts of GOPC consultations for new cases of URI and their associated productivity losses attributable to $PM_{2.5}$ and NO_2.

Table 8.7: Cost of illness for GOPC visits for URI (new cases) attributable to $PM_{2.5}$ and associated productivity loss

Annual attributable cases of URI[§]	Total cost of GOPC consultation ($385 per session)	Productivity loss (median daily wage: $12,000/30 = $400)	COI + productivity loss	Remarks*
1,028	$395,619.35	$411,033.09	$806,652.45	RR=Central estimate ; cases per 1μg/m^3
412	$158,508.29	$164,683.94	$323,192.23	RR=L95% CL; cases per 1μg/m^3
1,846	$710,559.15	$738,243.27	$1,448,802.41	RR=U95% CL; cases per 1μg/m^3
19,029	$7,326,320.13	$7,611,761.17	$14,938,081.30	RR=Central estimate; cases per 18.6 μg/m^3
7,644	$2,943,076.70	$3,057,742.02	$6,000,818.72	RR=L95% CL; cases per 18.6 μg/m^3
34,059	$13,112,755.48	$13,623,642.06	$26,736,397.54	RR=U95% CL; cases per 18.6 μg/m^3

* Total annual consultations to HA GOPCs=5,633,407 (2012-13)

§ Attributable cases of URI are estimated using a change of $PM_{2.5}$ concentration of 1 μg/m^{3}and 18.6 μg/m^3 (i.e., annual mean concentration of 28.6 μg/m^3 and a counter-factual of 10 μg/m^{3}μg/m^3) respectively. These numbers are rounded up. The $ values in columns 2 and 3 are calculated from the attributable cases expressed in decimal places and do not correspond with the sum obtained by multiplying the whole number of attributable cases.

Table 8.8: Cost of illness for GOPC visits for URI (new cases) attributable to NO_2 and associated productivity loss

Annual attributable cases of URI[§]	Total cost of GOPC consultation ($385 per session)	Productivity loss (median daily wage: $12,000/30 = $400)	COI + productivity loss	Remarks*
2,050	$789,079.53	$819,822.88	$1,608,902.41	RR=Central estimate ; cases per 1μg/m^3
1,232	$474,483.40	$492,969.77	$967,453.17	RR=L95% CL; cases per 1μg/m^3
2,660	$1,024,128.55	$1,064,029.66	$2,088,158.21	RR=U95% CL; cases per 1μg/m^3
25,878	$9,963,211.82	$10,351,388.91	$20,314,600.73	RR=Central estimate; cases per 12.7 μg/m^3
15,597	$6,004,902.55	$6,238,859.80	$12,243,762.35	RR=L95% CL; cases per 12.7 μg/m^3
33,529	$12,908,670.25	$13,411,605.46	$26,320,275.71	RR=U95% CL; cases per 12.7 μg/m^3

* Total annual consultations to HA GOPCs=5,633,407 (2012-13)

§ Attributable cases of URI are estimated using a change of NO_2 concentration of 1 μg/m^{3}and 12.7 μg/m^3 (i.e., annual mean concentration of 52.7 μg/m^3 and a counter-factual of 40 μg/m^{3}μg/m^3) respectively. These numbers are rounded up. The $ values

in columns 2 and 3 are calculated from the attributable cases expressed in decimal places and do not correspond with the sum obtained by multiplying the whole number of attributable cases.

We use the results of our 2015 survey on GP charges for URI, at $250 per consultation in our estimate of the direct cost of illness (COI). Using the lower estimate of 8,179,735 annual GP consultations for new URI cases, we estimate that the annual direct COI is $4,245,479 for a change in $PM_{2.5}$ concentration of 1 $\mu g/m^3$, and $77,539,662 for the counter-factual used (18.6$\mu g/m^3$). Using the higher estimate of 21,575,032 annual GP consultations, we estimate that the annual COI to be $11,197,959 for a 1 $\mu g/m^3$ change and $204,520,153 for a 18.6 $\mu g/m^3$ change (counter-factual = 10 $\mu g/m^3$). For a mean estimate of GP visits, the annual COI for a 1 $\mu g/m^3$ and a 18.6$\mu g/m^3$ change in $PM_{2.5}$ concentration are $7,721,719 and $141,029,907 respectively (Table 7.5). The corresponding figure for a 1 $\mu g/m^3$ change in NO_2 concentration is $6,035,655 and that for a 12.7$\mu g/m^3$ change (counter-factual = 40 $\mu g/m^3$) is $75,343,132 for the lower estimate of GP numbers, $15,919,763 for a 1 $\mu g/m^3$ change in NO_2 concentration and $198,726,541 for a 12.7 $\mu g/m^3$ change (counter-factual = 40 $\mu g/m^3$) for the higher estimate of GP numbers. For a mean estimate of GP visits, the annual COI for a 1 $\mu g/m^3$ and a 12.7$\mu g/m^3$ change in NO_2 concentration are $10,977,709 and $137,034,836 respectively (Table 7.6). Although the unit economic impact for NO_2 is higher than that for $PM_{2.5}$ (owing to a higher RR), the overall economic impact is less, because the change in concentration of NO_2 is lower (at a higher counter-factual).

The COI for GOPC visits is lower than that for GP visits, at HK$395,619 per 1 $\mu g/m^3$ change in $PM_{2.5}$, and HK$789,080 per 1 $\mu g/m^3$ change in NO_2 (Tables 7.7 and 7.8). At their respective counter-factuals, the COI are HK$7, 326,320 for $PM_{2.5}$ and HK$9,963,212 for NO_2. The productivity loss for 1 $\mu g/m^3$ change in $PM_{2.5}$ is HK$411,033 and that for 1 $\mu g/m^3$ change in NO_2 is HK$819,823, while that for the respective counter-factuals are HK$7,611,761 and HK$10,351,389 (Tables 7.7 and 7.8). The total costs (COI + productivity loss) are: HK$806,652 per 1 $\mu g/m^3$ change in $PM_{2.5}$ and HK$1,608,902 per 1 $\mu g/m^3$ change in NO_2, and HK$14,938,081 and HK$20,314,601 for changes to their respective counter-factuals (Tables 7.7 and 7.8).

The total economic impact of air pollution-related visits for URI by air pollutant is summarized in Table 7.9. For $PM_{2.5}$, the total direct COI from GP and GOPC visits, based on a mean estimate of the former, is 8.12 million per 1 $\mu g/m^3$ and 148.36 million per 18.6 $\mu g/m^3$. For NO_2, the COI per 1 $\mu g/m^3$ is higher, at 11.77 million, but the total COI per 12.7 $\mu g/m^3$ is lower, at $146.99 million. The total productivity loss from 1 $\mu g/m^3$ change of $PM_{2.5}$ is 12.76 million and that from 18.6 $\mu g/m^3$ is $233.26 million. Likewise, the productivity loss from 1 $\mu g/m^3$ change of NO_2 is higher, at $18.38 million, but the loss from a change of 12.7 $\mu g/m^3$ is slightly lower than that for $PM_{2.5}$, at $229.61 million. The total economic impact (combined value of COI and productivity loss) amounts to $20.89 million per 1 $\mu g/m^3$ change in $PM_{2.5}$, $30.15 million per 1 $\mu g/m^3$ change in NO_2, $381.62 million per 18.6 $\mu g/m^3$ change in $PM_{2.5}$, and $376.60 million per 12.7 $\mu g/m^3$ change in

NO_2.

Table 8.9: Total economic impact of air pollution-related GP and GOPC visits for URI by air pollutant, based on central RR and different estimates of GP visits

	Cost of GP visits (million HK$)				Productivity loss from GP visits (million HK$)				Total economic impact of GP visits (million HK$)			
Pollutant	$PM_{2.5}$		NO_2		$PM_{2.5}$		NO_2		$PM_{2.5}$		NO_2	
Change in conc. ($\mu g/m^3$)	1	18.6	1	12.7	1	18.6	1	12.7	1	18.6	1	12.7
GP visits lower estimate	4.24	77.54	6.04	75.34	6.79	124.06	9.66	120.55	11.04	201.60	15.69	195.89
GP visits upper estimate	11.20	204.52	15.92	198.73	17.92	327.23	25.47	317.96	29.11	531.75	41.39	516.69
GP visits mean estimate	7.72	141.03	10.98	137.03	12.35	225.65	17.56	219.26	20.08	366.68	28.54	356.29
GOPC visits	0.40	7.33	0.79	9.96	0.41	7.61	0.82	10.35	0.81	14.94	1.61	20.31
	Total cost (million HK$)				Total productivity loss (million HK$)				Total economic impact			
GP visits lower estimate + GOPC visits	4.64	84.87	6.83	85.30	7.20	131.61	10.48	131.19	11.85	216.54	17.30	216.49
GP visits upper estimate + GOPC visits	11.6	211.85	16.71	208.69	18.33	334.84	26.29	328.60	29.92	546.69	43.00	537.29
GP visits mean estimate + GOPC visits	8.12	148.36	11.77	146.99	12.76	233.26	18.38	229.61	20.89	381.62	30.15	376.60

Lower estimate of annual GP visits for new cases of URTI = 8,179,735

Upper estimate of annual GP visits for new cases of URTI = 21,575,032

Mean estimate of annual GP visits for new cases of URTI = 14,877,384

Annual GOPC visits for new cases of URTI = 2,060,813

8.3 References

Census and Statistics Department, the Government of Hong Kong Special Administrative Region. Source: http://www.censtatd.gov.hk/hkstat/sub/gender/employment_earnings/

Hospital Authority Annual Report (2013 – 2013), Hospital Authority, Hong Kong. Accessed at: http://www.ha.org.hk/upload/publication_13/477.pdf

Wang H, He J. The value of statistical life – a contingent investigation in China. Policy Research Working Paper 5421, Washington D C: World Bank, 2010.

World Bank. Cost of pollution in China. Washington D C: World Bank, 2007.

WHO 2013a. Review of Evidence on health aspects for air pollution – REVIHAAP Project. Technical Report, World Health Organization Regional Office for Europe, Denmark, 2013. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf

WHO 2013b. Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration-response functions for cost-benefit analysis of particulate matter, ozone and nitrogen dioxide. Accessed at: http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf

WHO Regional Office for Europe, OECD 2015. Economic cost of the health impact of air pollution in Europe: Clean air, health and wealth, Copenhagen: WHO Regional Office for Europe.

Chapter 9: Discussion of study findings

9.1 Summary of health impact assessment (HIA)

After our literature review, we performed a health impact assessment (HIA) based on local health statistics, local unit health risks (if available) and unit health risks derived from reputable and widely used studies. The results are in line with HIAs performed in several developed countries. A key message is: air pollution exerts a significant health impact on Hong Kong's residents. A unit change in $PM_{2.5}$ concentration (1μg/m^3) is causally associated with 244 premature deaths in Hong Kong, and a reduction from the annual mean $PM_{2.5}$ concentration (28.6 μg/m^3) to the WHO air quality guideline (AQG) level (10 μg/m^3) is estimated to prevent 4,316 premature deaths (95% confidence limit: 1,519 – 7,413).[23] Estimates based on NO_2, which is now recognized to exert an independent effect (assuming 30% overlap with $PM_{2.5}$ effects) on mortality gives 1,993 (95% CI: 1,145 – 2,809) additional deaths , based on a reduction of NO_2 level from 52.7 μg/m^3 to the WHO AQG of 40 μg/m^3. The combined effects of these two air pollutants therefore are responsible for an annual death toll of 6,308 (95% CI: 2,665 – 10,222) using the same counter-factuals ($PM_{2.5}$ = 10 μg/m^3; NO_2 = 40 μg/m^3).

Likewise, a unit change (per 1 μg/m^3) in $PM_{2.5}$ concentration is causally associated with 148 cardio-respiratory hospital admissions, while a change using the stated counter-factual ($PM_{2.5}$ = 10 μg/m^3) is associated with 2,730 hospital admissions for cardio-respiratory diseases. The corresponding values are similar for NO_2, with 149 cardio-respiratory hospital admissions for a unit change in concentration. Using the counter-factual for NO_2 (at 40 μg/m^3), however, results in a lower number of 1,882 cardio-respiratory admissions. The total annual admissions for cardiovascular and respiratory diseases into HA hospitals, according to the most up-to-date data available (2012), are 70,934 and 104,953 respectively (total =175,887).

The annual number of GP visits for new cases of upper respiratory tract infections (URI) is much higher than that for hospital admissions, estimated at a range from 8.18 million to 21.58 million annually. The annual number of new URI cases seen by private GPs attributed to 1μg/m^3 increase in $PM_{2.5}$ concentration ranges from 16,982 to 44,792 (mean = 30,887), and from 310,159 to 818,081 (mean = 564,120) using 10 μg/m^3 as the counter-factual for $PM_{2.5}$ concentrations. The corresponding URI new cases attributed to 1μg/m^3 increase in NO_2 concentrations are slightly higher, ranging from 24,143 to 63,679 (mean = 43,911). Using the

[23] A slightly higher number (5,478; 95% CI: 3,372 – 7,701) is derived when using a more recently reported RR for a 10 μg/m^3 increase in $PM_{2.5}$ of 1.078 (Krewski et al, 2009) obtained from an extended analysis of the original study (Pope et al, 2002).

counter-factual of 40 μg/m^3, the attributable number of URI new cases range from 301,373 to 794,906 (mean = 548,139), slightly lower than the corresponding figures for $PM_{2.5}$. The annual number of new URI cases seen at General Outpatient Clinics attributed to NO_2 is 2,050 (per 1μg/m^3 change in NO_2 concentration), and 25,878 using the same counter-factual for NO_2 concentrations as before. The number of new URI new cases attributable to $PM_{2.5}$ is lower, at 1,028 per 1μg/m^3 change in $PM_{2.5}$ concentration, and 19,029 per year, using the same counter-factuals for $PM_{2.5}$ as before.

Using a life-table approach, a 10 μg/m^3 change in $PM_{2.5}$ concentration is associated with a loss of 2,484 lives and a total of 36,643 life years (about 15 years lost per premature death), while a 10 μg/m^3 change in NO_2 concentration is associated with a loss of 1,647 lives and a total of 24,300 life years. The combined effects are a loss of 4,131 lives with a total of 60,943 life years annually, for every 10 μg/m^3 increase in both pollutants. The combined number of life-years lost at the respective counter-factuals is 97,197 from 6,590 deaths.

9.2 Implications of HIA findings

We have shown that using internationally accepted unit risk estimates, the burden of air pollution on health in Hong Kong is substantial. The number of premature deaths attributable to air pollution ($PM_{2.5}$ and NO_2 combined) is 6,308 deaths. This is not as high as 'death tolls' published in other HIA studies that use anthropogenic $PM_{2.5}$ as a measure of health burden. In those scenarios, the counter-factuals are non-anthropogenic $PM_{2.5}$, which is substantially lower than 10 μg/m^3, the counter-factual that we use in this study.

Compared to premature mortality, the assessment of life years lost has been considered as a more appropriate indicator of the adverse effect of air pollution on survival (COMEAP 2010). The reason is that the assessment of life years represents not only the loss of life, but the extent of shortening of life expectancy in different ages affected by air pollution. However, the drawback of expressing health impact in terms of the numbers of life years lost is that this is not a easy-to-comprehend concept, compared to the number of premature deaths. For example, it is difficult for a policy maker to decide whether a certain number of life years lost is too high or not.

The health impact on hospital admissions for cardio-respiratory diseases is also substantial. Compared to premature mortality, hospital admissions attributable to air pollution is 1.55%[24]

[24] Derived from 2,730 hospital admissions for cardiovascular and respiratory diseases out of a total of 175,887 hospital admissions for these illnesses.

of all hospital admissions (about one-tenth the percentage attributed to premature mortality). This can be explained by the fact that the RR used for short-term exposure to air pollution is one order of magnitude lower than the RR used for assessing mortality from long-term exposure to air pollution. No unit risk of hospital admissions for *long-term* exposure to air pollution is available in the literature. It should be noted that the hospital admissions for cardio-respiratory diseases attributable to the air pollutants that we have estimated do not reflect the total health burden of air pollution. These numbers are derived from the counter-factuals of air pollutant concentrations as stated in the study. If we had assessed the health burden of, for example, all anthropogenic $PM_{2.5}$, as in European, Australian, U.S. and U.K. studies, the burden to health (like in the case of premature mortality) will become much higher, (because the counter-factual, the concentrations of non-anthropogenic concentrations of $PM_{2.5}$ is very low).

9.3 Limitations of HIA findings

Our HIA results are based on health outcomes for which relevant data are available. For specific health outcomes that have been assessed in other HIA studies, such as the prevalence of asthma, the incidence and prevalence of chronic obstructive pulmonary disease, acute bronchitis, and lost days from work, we could not find the corresponding local incidence and prevalence data (or data related to the costs of medical treatment of these conditions for our EIA study). Hence, our HIA is a conservative assessment that has omitted the above health conditions that are generally recognized to be causally associated with air pollution. Our estimates of hospitalizations, as mentioned earlier, do not include private hospital data, although private hospitals only constitute about 10% of hospital beds in Hong Kong. We have not included visits to Accident and Emergency Departments (without hospitalization) for cardiovascular and respiratory illnesses, as the relevant statistics are not available in the HA.

Another limitation is that we have not assessed the health impact of all air pollutants. Instead, we have chosen the most relevant ones among the “criteria air pollutants” – $PM_{2.5}$ and NO_2. There are points for and against using $PM_{2.5}$ alone for HIA, versus using more than one air pollutant, e.g., adding NO_2 effects or O_3 effects. Different tools may be required for different policy options. For example, the use of a multi-pollutant model that incorporates the effects of $PM_{2.5}$ and NO_2 would be more suitable for policy options that involve roadside NO_2 reduction, to estimate the role of long-term effects of NO_2 on health. The U.K. King’s College (Walton et al, 2015) study uses the WHO REVIHAAP (WHO 2013) viewpoint that there is evidence for an independent effect of long-term exposure NO_2 on mortality risk, and that because of the correlation between NO_2 and $PM_{2.5}$, there is an overlap of effects ranging

from 0 to 33%.[25] In the UK study (Walton et al, 2015), a conservative estimate of a 30% overlap was used. We have adopted the same approach in our study. A thorough discussion of uncertainties in HIA can be found in Chapter 4 of a recent WHO report 'Health risk assessment of air pollution: General Principles' (WHO 2016).

We have not studied the effects of SO_2 mainly because the prevailing average concentration of SO_2 is low (annual mean excluding roadside and Tap Mun station data = 11.6 μg/m^3; EPD 2014), with limited scope for further reduction. O_3 has not been included in most HIA. Its formation depends on many factors, including other air pollutants and weather conditions. Furthermore, O_3 can be transported over long distances, making it difficult to model changes in local concentrations using local control strategies. The effect of long-term exposure to O_3 on survival has not been well-documented. Hence, no AQG for long-term exposure to O_3 has been recommended. The health impact of toxic air pollutants (TAPs) has not been estimated. Their effects are small compared to criteria air pollutants, in particular $PM_{2.5}$. Some TAPs are carcinogenic, and their health impacts have been expressed as an increase in the risk of lung cancer. Mortality from lung cancer has already been assessed as part of the all-cause mortality. Non-cancer health impact, resulting from short-term and long-term exposure is considered to be insignificant, as the ambient concentrations to which people are exposed are in general well-below the threshold levels for most TAPs. The fact that air pollutants exist as a mixture contributes to the uncertainty in the outcomes of the HIA based on individual air pollutants (WHO 2016).

All our health impact estimates have been done using the central RR as the most likely scenario, with the lower and upper 95% confidence intervals (CI) as the possible range of our estimates. This practice is in line with other HIA studies. Unlike other potential sources of error in our estimate, uncertainties from 95% CI can be assessed quantitatively.

A major source of uncertainty is the baseline data on private GP consultations. For private GP data, where no official figures are available, we have attempted to produce two estimates – a higher and a lower one, based on the best available data on GP consultations from various sources. The range of estimates is wide (310,159 – 818,081 URI cases attributable to $PM_{2.5}$), and it is difficult to determine which estimate is more accurate than the other. However, even the lower estimate of the annual attributable GP consultations for URI represents a substantial health burden. The number of GOPC cases is much smaller (19,029 cases attributable to $PM_{2.5}$; 25,878 cases attributable to NO_2), but they add to the overall health impact on

[25] 0% overlap means both NO_2 effects and PM effects can be added up, whereas the 30% overlap assumption means in their calculation, the RR for NO_2 is reduced by 30%, and the health impact of this reduced amount of health impact will be added to that attributed to PM.

non-hospital illnesses.

We have extrapolated the RR by Pope et al (which applies to adults aged 30 and above) to cover the 'below 30' age group. The RR 'below 30 years' age group is probably smaller than Pope's RR of 1.06, as the latter RR comprises the elderly age group who are most vulnerable to air pollution. The total no. of deaths among the 'below 30 years' age group in Hong Kong is very small (529 in 2014). Hence, the attributable deaths from air pollution – which comprise mostly heart and lung diseases, should be even smaller.[26] We consider that if the HIA is conducted for the '30 and above' age groups only, the results would be marginally smaller than the total number of attributable deaths for all age groups if we assume the RR in the 'below 30' age groups to be 1.06 as well.

9.4 Summary of economic impact assessment (EIA)

We estimated that the annual economic impact of premature mortality attributable to a 1 $\mu g/m^3$ change in $PM_{2.5}$ is HK$3,835,173,546 (HK$3.84 billion), using the average VOSL. This value ranges from HK$2,221,227,069 (HK$2.22 billion) using the low VOSL estimate, to HK$5,449,118,128 (HK$5.45 billion), using the high VOSL estimate. The economic impact attributable to a counter-factual of 10 $\mu g/m^3$ in $PM_{2.5}$ concentration (from 28.6 $\mu g/m^3$) is HK$67,801,722,064 (HK$67.8 billion), using the average VOSL estimate, and ranges from HK$39,268,893,193 (HK$39.2 billion) using the low VOSL estimate, to HK$96,334,517,403 (HK$96.3 billion), using the high VOSL estimate. The economic impact is substantial, even without taking into account the independent effect of NO_2 on mortality. The economic impact attributed to a 1 $\mu g/m^3$ change in NO_2 averages at HK$2,520,650,622 (HK$2.52 billion), using the average VOSL estimate, and ranges from HK$1,459,891,534 (HK$1.46 billion) using the low VOSL estimate, to HK$3,581,408,463 ($3.58 billion), using the high VOSL estimate. The corresponding values attributable to a 12.7 $\mu g/m^3$ change in NO_2 are:

HK$31,306,804,039 (HK$31.3 billion), using the average VOSL estimate,
HK$18,132,040,110 (HK$18.1 billion), using the low VOSL estimate, and
HK$44,481,552,485 (HK$44.8 billion), using the high VOSL estimate.

When these figures are combined, the total economic impact is huge, at HK$6,355,824,169 (HK$6.36 billion), using the average VOSL estimate, and ranges from HK$3,681,118,604 (HK$3.68 billion) to HK$9,030,526,592 (HK$9.03 billion), using respectively the low and

[26] Assuming an AF of 10% for $PM_{2.5}$ and 5% for NO_2, the total no. of attributable deaths for those aged below 30 years would be about 80.

high VOSL estimates. For a 18.6 μg/m^3 change in $PM_{2.5}$ and a 12.7 μg/m^3 change NO_2, the combined economic impact is HK$99,108,526,105 (HK$99.1 billion), average VOSL; HK$57,400,933,306 (HK$57.4 billion), low VOSL; and HK$140,816,069,891 (HK$140.8 billion), high VOSL. The range of these estimates is wide because of different assumptions of the VOSL.

By contrast, the estimated losses of productivity from the loss of lives are much smaller, ranging from HK$2,444,614 (using lower 95% CI of RR) to HK$12,828,841 (using upper 95% CI of RR), per 1 μg/m^3 change in $PM_{2.5}$. Expressed as the counter-factuals used, the values are from HK$44.7 million to HK$218 million, less than one percent of the lower VOSL estimate at the lower 95% CI of RR. Productivity loss from hospital illnesses is likewise small, at about HK$3 million per year.

The direct COI from hospital illnesses at HK$53.81 million (for a 18.6 μg/m^3 change in $PM_{2.5}$) is 0.08% of the VOSL using the average VOSL estimate of HK$67.63 billion (for the same change in $PM_{2.5}$), and the estimated productivity loss from hospital admissions (both cardiovascular and respiratory illnesses, for the same counter-factual) is $4.4 million (about 8.1% of the direct COI). The direct COI for NO_2 (12.7 μg/m^3 change in concentrations) is HK$37.09 million (0.055%), and an estimated productivity loss of HK$3.01 million, slightly lower than the corresponding values for $PM_{2.5}$.

Based on a counterfactual of 40 μg/m^3 for NO_2, the annual COI for private GP consultations for URI ranges from a low estimate (low GP estimate and lower 95% CI of RR) of HK$50.79 million to a high of HK$262.08 million (high GP estimate and upper 95% CI of RR). The central estimate (central RR) is: HK$75.34 million (low GP estimate), HK$198.73 million (high GP estimate), and HK$137.03 million (mean GP estimate). At a counterfactual of 10 μg/m^3 for $PM_{2.5}$, the range is HK$37.50 million (low GP estimate and lower 95% CI of RR) to HK$306.93 million (high GP estimate and upper 95% CI of RR). The central estimate (central RR) is: HK$77.54 million (low GP estimate), HK$204.52 million (high GP estimate), and HK$141.03 million (mean GP estimate). The estimated COI for GP visits based on the central RR of either air pollutant is higher than the hospital COI. Also, the productivity losses from the URI are about 50 – 60% higher than the COI (Tables 7.5 and 7.6). The COI of GOPC visits for URI is much lower, at HK$7.33 million for a 18.6 μg/m^3 change in $PM_{2.5}$ and HK$9.96 million for a 12.7 μg/m^3 change in NO_2. In contrast to GP visits, the productivity losses from GOPC visits are only slightly higher than their corresponding COI, because the unit cost for GOPC is much higher than the GP charges.

9.5 Implications of EIA findings

As mentioned in Section 3, the economic impact assessed by direct cost of illness (COI) is but a small sub-set of the total economic impact, which is generally regarded by health economists to be best assessed by the 'willingness-to-pay' (WTP) methodology. Economic impact assessment (EIA) reports in Europe, Australia and New Zealand have shown clearly that direct COI is but a fraction of the economic impact using the WTP approach. The COMEAP (2010) suggests that COI results are likely to mislead policy makers, the public and the polluters by grossly under-estimating the true cost of air pollution. The major drawback using the WTP approach, however, is the lack of local data, which are essential in producing valid results, as WTP varies widely between communities, owing to the diversity of their culture, and socioeconomic conditions.

9.6 Limitations of EIA findings

The most critical comments about economic valuation results from non-health economists are on the validity of WTP estimates. Their arguments are that since WTP values are based on hypothetical situations, it is impossible to valuate one's life or one's health. Yet, after decades of research into the methodology, health economists have found that the WTP values in general reflect the COI plus other attributes not otherwise assessed, such as opportunity costs of being healthy. WTP has been used extensively in the valuation of life and health. We have described the major limitations of the human capital approach to life. Clearly, life cannot be worthless after a person supposedly stops his / her economic productivity. This is even less so if the subject (and not a disconnected economist) is asked to value his / her own life. In our study, the problem is not with the VOSL approach, but with the actual valuation placed on a life. This is partly circumvented by the use of the WHO statistics (WHO 2015) and World Bank data (Wang et al, 2010), for which we adjust the VOSL by the per-capita GDP of Hong Kong, against the VOSL estimates in China and WHO European Region. The limitation of COI is its incompleteness, while the limitation of WTP is the absence of local WTP data on morbidity and VOSL. We consider that the adjusted VOSL is a reasonable approach, but the lack of WTP data on hospital illnesses and minor URI is a major limitation of our EIA. We believe the presence of local WTP data would give a more realistic estimate of health and morbidity than the COI approach alone.

In some European and U.K. EIA studies (COMEAP 2010; European Commission 2013) that use the loss of life years approach, researchers have placed a value on a unit life year – value of life-year (VOLY) in the economic impact assessment, although without abandoning the

use of the value of statistical life (VOSL). However, in the U.S., the USEPA Science Advisory Board advised the continued use of VOSL (USEPA 2001:26).[27] Studies have shown that the value of a life year is not uniform throughout life, but follows a U-shape, with a higher valuation at both ends of the lifespan. If the same value is given to a life year lost, this implies that the elderly has a lower valuation of their lives compared to younger people. The converse has been found to be true. Hence, we have refrained from attaching a value to a life year lost, but instead perform the more conventional assessment of valuating premature mortality by assigning a VOSL.

9.7 Future uses of findings in this study

The HIA and EIA studies that we have conducted have illustrated that we have a great need for better and more comprehensive data for a more accurate assessment. An HIA and EIA that grossly under-estimate the "true" situation will do more harm than good. Our findings agree with those in international HIAs and EIAs in that premature mortality is the predominant health and economic impact, and the magnitude of the HIA and EIA clearly shows air pollution exerts a heavy toll on health and the economy. This is in agreement with findings by the WHO Global burden of Disease Project (Lim et al, 2012) that ambient air pollution is one of the major, yet preventable risk factor to ill-health. The present study has two major contributions. First, it shows the burden of air pollution is high, and second, it enables the policy maker to assess the health impact and cost-benefit of an air quality improvement policy or strategy. This evidence-based environmental policy is the direction that most developed countries are taking in recent years, and Hong Kong must not lag behind.

Regular updating of diseases to be included in HIA is necessary in the light of up-to-date scientific evidence. Some possible effects of air pollution that are under research, such as the impairment of cognitive functions of adults and neurodevelopmental deficit of children , would radically change the HIA results if evidence for a causal-relationship is strengthened. Likewise, updating of exposure response functions, exposure data, local health data (hospital admissions and mortality, disease prevalence data and economic data) are necessary to ensure the validity of our study over time. A five-year period is a reasonable time frame for updating the HIA and EIA results.

[27] Further discussions on this issue can be found in the WHO report 'Economic cost of the health impact of air pollution in Europe' (WHO 2015).

9.8 **References**

Committee on the Medical Effects of Air Pollution (COMEAP). The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom. A Report by the Committee on Medical Effects of Air Pollution. Health Protection Agency, United Kingdom 2010. Accessed at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304641/COMEAP_mortality_effects_of_long_term_exposure.pdf

Environmental Protection Department (2014). Air Quality in Hong Kong 2014, Environmental Protection Department, The Government of the Hong Kong Special Administrative Region.

European Commission 2013. Commission staff working document. Impact assessment. Brussels: European Commission (SWD (2013) 531 final; http://ec.europa.eu/governance/impact/ia_carried_out/docs/ia_2013_0531_en.pdf

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA III, Thurston G, Calle EE, Thun MJ. 2009. *Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality*. HEI Research Report 140. Health Effects Institute, Boston, MA.

Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, Amann M et al (2012) A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the global burden of disease study 2010. Lancet 380(9859): 2224–2260.

Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 2002; 287(9):1132-41.

USEPA 2001. Review of the draft analytical plan for EPA's second prospective analysis – benefits and costs of the Clean Air Act 1990 – 2020. An advisory by a special panel of the Advisory Council on Clean Air Compliance Analysis. Washington, DC: United States Environmental Protection Agency (EPA-SAB-COUNCIL-ADV-01-004; http://yosemite.epa.gov/sab/sabproduct.nsf/7CCBBFE15CD4C8B185256F17005E3079/$File/council_avd_04004.pdf.

Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impact of Air Pollution in London. King College London, 2015. Accessed at: http://www.kcl.ac.uk/lsm/research/divisions/aes/research/ERG/research-projects/HIAinLondonKingsReport14072015final.pdf

World Health Organization (2013). Review of evidence on health aspects of air pollution – REVIHAAP Project. Technical Report. World Health Organization Regional Office for Europe, Copenhagen.

WHO Regional Office for Europe, OECD 2015. Economic cost of the health impact of air pollution in Europe: Clean air, health and wealth, Copenhagen: WHO Regional Office for Europe.

World Health Organization (2016). Health Risk Assessment of Air Pollution: General Principles. Copenhagen: WHO Regional Office for Europe, 2016.

Chapter 10: Non-health effects of air pollution

We have conducted a review of non-human health effects of air pollution. Some of these effects indirectly affect human health, through other pathways. The literature on these effects is large, and we shall highlight below some of the more important findings that affect our economy and well-being.

10.1 Effects on wildlife and domestic animals

Animals are exposed to air pollutants via three pathways: 1) inhalation of gases or small particles; 2) ingestion of particles suspended in food or water; or 3) absorption of gases through the skin. In general, only soft-bodied invertebrates (e.g. earthworms), or animals with thin, moist skin (e.g. amphibians) are affected by the absorption of pollutants. An individual's response to a pollutant varies greatly and depends on the type of pollutant involved, the duration and time of exposure, and the amount taken up by the animal. The individual's age, sex, health, and reproductive condition also play a role in its response. There is a great deal of variability between animal classes, species, and even genotypes, in terms of the level of tolerance to a particular pollutant.

Losses of wildlife resulting from air pollution have been reported since the 1880s (Newman, 1979). Exposure to environmental chemicals can have negative consequences for wildlife and even cause localized population extinctions (Brown et al, 2009). There are few toxicological studies on the effects of air pollutants on wild life and domestic animals. Most are observational studies from incidents, with few quantitative data. Before 1970, (the year the Clean Air Act was enacted in the U.S.), 11 reports of massive die-offs of wildlife have been reported, involving air pollution by arsenic, fluoride, hydrogen sulphide or asbestos. After 1970, more than 50 incidents have been reported in the US, Canada, Japan, India and several European countries (England, Poland, Sweden, the former Czekoslovakia and Yugoslavia). Besides the above pollutants, cadmium, lead, zinc, sulphur dioxide, nitrogen oxides, oxidants, fly ash and cement dust have been incriminated. Effects ranged from deaths and injuries to behavioral changes. The low number of incidents is believed to be due to lack of awareness of the problem. From controlled experiments, more is known about the effects of air pollution on domestic and laboratory animals than wildlife. Mice, rabbits and birds are found to be sensitive to levels of sulphur dioxide below the US national ambient air quality standard. Behavioral changes in deer mice, physiological changes in rabbits, and changes in the nesting dynamics of insectivorous birds have also been observed. Hydrogen sulphide, a toxic gas associated with oil and gas fields, has been associated with wildlife deaths in the US. Its toxicity to farm animals is well known, but the toxicity to and tolerance levels in wildlife

species have not been documented. Photochemical oxidants are associated with genetic changes, respiratory problems and blindness in birds and mammals. Arsenic has long been known to be toxic to wildlife and domestic animals. Cadmium and fluoride are toxic to wildlife. Fluorosis (fluoride poisoning) causes gross malformations of bones and teeth. Plants take up gaseous fluoride and store it in their tissues, and fluoride in particulate form is deposited on leaf surfaces and stays there until washed off. Herbivores are best known for exhibiting symptoms of fluoride poisoning. However, earthworms and other soil invertebrates also accumulate fluoride, which is, in turn, passed on to the animals that eat them. Lead causes acute toxicity from lead shot studies, but the effects of chronic exposure are not known. Most studies on trace metal contamination focus on their concentrations in tissues, but little is known about the biological or population effects on wildlife.

Sulphur dioxide and nitrogen oxides emitted as a result of fossil fuel combustion undergo chemical transformation in the atmosphere, and occur as sulphate, nitrate, and hydrogen ions when dissolved in precipitation known as "acid rain". Many studies have demonstrated that surface water acidification can lead to a decline in, and loss of, fish populations. Acid deposition is a possible cause of declines in amphibian populations.

Organic and synthetic chemicals, such as dioxins and organochlorines, affect wildlife. Dioxins bioaccumulate, or build up in the body by concentrating in body fat, and they are resistant to biological breakdown. A study of earthworms showed they accumulated dioxin up to five times the concentration found in the soil, although this dose was not lethal to the worms. Nevertheless, this non-lethal accumulation could have strong ecological implications, since earthworms are a major source of food for a number of bird and small mammal species, many of which have exhibited carcinogenic, reproductive, and immunotoxic effects after exposure to low levels of dioxins.

Oxidative stress is the unifying feature underlying the toxicity of anthropogenic pollution (e.g., heavy metals, polycyclic aromatic hydrocarbons, and nitrogen-oxides) and the ultimate culprit in the development of many diseases. In a meta-analysis, it was concluded that there was an overall increase in oxidative stress when wild animals are exposed to pollution (Issaksson 2010).

Tables 10.1 and 10.2 show the toxicological data of selected gaseous and particulate air pollutants respectively and their effects on wildlife.

In the U.S., secondary standards of air quality are established to protect public welfare, which includes wildlife. A review of the effects of these air pollutants on animals shows that

biological effects can occur at levels below these standards. Standards for fluoride levels in forage are established to protect the economic value of animal husbandry rather than fluorosis in animals including wildlife. Some areas are designated “non-attainment areas” for some secondary standards. In the U.S. and Canada, occasional incidents from oil and gas fields and factories have led to high levels of sulphur dioxide and other gases, with deaths and injuries to birds and water fowls, wild mammals and domestic animals. Wildlife in Third World countries are considered to be at high risk, owing to deficient legislation and poor enforcement, and unfavorable economic factors that hinder air pollution control.

Table 10.1: Toxicological data of selected gaseous air pollutants and their effects on wildlife

	Sulphur dioxide	Hydrogen sulphide	Photochemical oxidants
Sensitive wildlife	Mice, hare, insectivorous birds	Rabbits, owls, songbirds, raccoons, mice, bats, deer, antelopes	Mice, bighorn sheep, sparrows
Toxicity levels	0.018 – 0.059 ppm continuously for 7 months causes changes in behavior of deer mice	Unknown	Unknown
Symptoms in wildlife	Reduced blood enzyme levels in hares; behavioral avoidance in mice and birds	Die-offs, lung haemorrhages	Blindness in sheep, genetic changes in mice, respiratory lesions in birds
Sources of contamination	Fossil fuel combustion, smelting	Petrochemical plants, kraft mills, oil and gas fields	Urban sources
Route of exposure	Inhalation	Inhalation	Adsorption, inhalation
Tolerance levels / standards	Unknown	Unknown	Unknown
Diagnostic methods	None established	None formally established, but tissue assay has been sued	None established
References (See footnote)	1, 2, 3, 4	5, 6, 7, 8, 9,10	11,12,13

Footnote:

1. **Chilgren, J.D.** 1979. Small mammal investigation at ZAPS: Demographic studies and responses to gradient levels of SO_2. In E.M. Preston and T.L. Gullet, eds., *The Bioenvironmental Impacts of a Coal-Fired Power Plant.* Fourth Interim Report, December 1978, Colstrip, MT. EPA-600/3-79-044. U.S. Environmental Protection Agency, Corvallis, OR, p764-791.
2. **Newman, J.R., E. Novakova** and **J.T. McClave.** 1985. The influence of industrial air emissions on the nesting ecology of the house martin, *Delichon urbica* ,in Czechoslovakia. *Biol. Conser.* **31**:229-248.
3. **Novakova, E., A. Finkova** and 2. **Sova.** 1973. Etude preliminaire des proteines sanguines chez le lievre commun expose aux pollutions industrielles [Preliminary study of blood proteins of the common hare exposed to industrial pollution]. In Nemzetkozi Vadaszati Tudomanyos Konferencia Eloadasai, Budapest, September 16-18, 1971, pp. 69.44.
4. **Mikova, M.** and **E. Novakova.** 1979. Variation of corneal glycosaminoglycan values of hares in relation to environmental pollution by industrial emissions. *J. Toxicol. Environ. Health* 5:891-896.
5. **Bicknell, W.B.** 1984. A cooperative hydrogen-sulfide monitoring study-The Lone Butte oil field, McKenzie County, ND. Habitat Resources Field Office, U.S. Fish and Wildlife Service, Bismarck, ND.
6. **Harris, R.D.** 1971. Birds collected (die off) at Prince Rupert, British Columbia, September 1971. Unpublished Final Report. Canadian Wildlife Service, Vancouver, BC.
7. **Lillie, L.E.** 1981. Biomedical effects of sulfur containing emissions. In V. Geist and A.H. Legge, eds., *Proceedings*, The Effects of Sour Gas on Wildlife, Calgary, Alberta, The University of Calgary, Calgary, Alberta, June 3, 1981. Occasional Papers **No. 5**, p9-36.
8. **O'Gara, G.** 1982. Riley Ridge: Gas sours wildlife in Wyoming. *High County News* 14:10-11.
9. **Mikova, M.** and **E. Novakova.** 1979. Variation of corneal glycosaminoglycan values of hares in relation to environmental pollution by industrial emissions. *J. Toxicol. Environ. Health* **5**:891-896.
10. **Yant, W.P.** and **R.R. Sayers.** 1927. Hydrogen sulfide as a laboratory and industrial poison. *J. Chem.Educ.* **4**:613-619.
11. **Light, J.T.** 1973. The effects of oxidant air pollution on forest ecosystems of the San Bernadino Mountains, Section B. In O.C. Taylor, ed., *Oxidant Air Pollution Effects on a Western Coniferous Forest Ecosystem.* Task B Report. Air Pollution Research Center, University of California-Riverside, Riverside, CA, pp. B1-B14.
12. **Richkind, K.E.** 1979. Genetic responses to air pollution in mammalian populations. Ph.D. thesis, University of California, Los Angeles, CA.
13. **Wellings, S.R.** 1970. Respiratory damage due to atmospheric pollutants in the English sparrow, *Passer domesticus*, in Project Clean Air. Research Project S-25. Department of Pathology, University of California, Davis, CA.

Table 10.2: Toxicological data of selected particulate air pollutants on their effects in wildlife

	Arsenic	Cadmium	Fluoride	Lead	Selenium
Sensitive wildlife	Deer, rabbits	Songbirds, owls, badgers, shrews	Deer, mice, owls, songbirds, foxes	Mice, rats, pigeons, peregrine falcons, swallows, bats	Wild herbivores
Toxicity levels	Unknown	Unknown	Unknown	Unknown	Unknown
Symptoms in wildlife	Death; emaciation; loss of hair; defective antler formation; cirrhosis of liver; accumulation in bones, keratinized tissues, liver and kidney	Death, accumulation in kidney and liver; reduced blood enzyme levels; joint lesions	Dental and skeletal changes; jaws fractures; lameness, accumulation in bones, nesting and growth reduced in birds	Tissue accumulation in bone and kidney; reduced ALAD levels in blood of birds, secondary poisoning	None reported but concern raised
Sources of airborne contamination	Fossil fuel combustion, smelting	Fossil fuel combustion, smelting	Aluminium smelting, fertilizer plants, fossil fuel combustion	Automobiles, smelting, refining	Problem hypothesized from deposition of SO_2 and SO_4^{2+} in Se deficient soil
Pathways of contamination	Ingestion of contaminated forage (moderate bioaccumulation)	Ingestion, inhalation (high bioaccumulation)	Ingestion of contaminated food by deer, of contaminated mice and insects by owls; inhalation of contaminated air by songbirds (high bioaccumulation)	Ingestion of contaminated food, inhalation (high bioaccumulation)	Ingestion of contaminated forage (moderate bioaccumulation)
Tolerance levels / standards	Unknown	>10mg/kg* in kidney or liver indicates contamination; 13-15 ppm* in tissue is significant hazard; 200 ppm* in kidney or 5 ppm* in whole body is life threatening	None; fluorosis induced at daily intake of 25 ppm*		Unknown but is estimated that diet containing >5 ppm* or levels of 4 $\mu g/m^3$ in air are harmful
Diagnostic methods	None established; in domestic animals, violent behavior and urine and other tissue levels are indicators	None established	Dental classification scheme suggested for cattle; fluoride levels in bones	Suggested combination of symptoms; Pb levels in blood, faeces and kidneys; reduced ALAD levels in blood	None established
References (See footnote)	1,2,3,4,5,6	2, 5,7,8, 10,11,12,13	14,15, 16,17, 18,19, 20, 21, 22, 23	3,24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34,	35, 36

* Wet weigh

Footnote:

1. **Clarke, E.G.C.** and **M.L. Clarke.** 1967. *Garner's Veterinary Toxicology*, 3rd ed. Bailliere Tindall & Cassell, London, UK.
2. **Jenkins, D.W.** 1980. *Biological Monitoring of ToxicTrace Metals,* Vol. I-Biological Monitoring andSurveillance. EPA-60013-80-089. US. Environmental Protection Agency, Las Vegas, NV.
3. **Wojcik, W.** 1980. Estimation of contamination of small game by heavy metals in the region of copper works. *Ekologia Polska* **28**:601-614.
4. **Hais, K.** and **J. Masek.** 1969. Vcinky nekterych exhalaci na hospodarska zvirata [Effects of some emissions on agricultural animals]. *Ochr. Ovtduzi* **3**:122-125.
5. **Mankovska, B.** 1982. Influence of industrial fumes around an aluminum plant on roe game. *Folia Venatoria* **12**:283-287.
6. **Prell, H.** 1936. Die Schadigung der Tierwelt durch die Fernwirkungen von Industrieal Gasen [Injury to the animal world through the distant effects of industrial waste gases]. *Arch. Gewerbepath. Gewerbehyg.***7**:656-670.
7. **Sileo, L.** and **W.N. Beyer.** 1985. Heavy metals inwhite-tailed deer living near a zinc smelter in Pennsylvania. *J. Wildl. Dis.* **21**:289-296.
8. **Eisler, R.** 1985. Cadmium hazards to fish, wildlife,and invertebrates: A synoptic review. Contaminant Hazard Reviews Report No. 2. Biological Report 85(1.2). US. Fish and Wildlife Service, Laurel, MD.
9. **Mankovska, B.** 1982. Influence of industrial fumes around an aluminum plant on roe game. *Folia Venatoria* **12**:283-287.
10. **Nishino, U., M. Arari, I. Senda** and **K. Kuboto.** 1973. Kankyo osen no suzume ni oyobosu eikyo [Influence of environmental pollution on the sparrow]. *Jpn. J. Publ. Health* **20**:1.
11. **Martin, M.H.** and **P.J. Coughtrey.** 1976. Comparison between levels of lead, zinc, and cadmium within a contaminated environment. *Chemosphere* **5**:15-20.
12. **Hutton, M.** and **G.T. Goodman.** 1980. Metal contamination of feral pigeons, *Columbia livia,* from the London area: Part 1 -Tissue accumulation of lead, cadmium and zinc. *Environ. Pollut. (Ser. A)* **22**:207-217.
13. **Tjell, J.C., T.H. Christensen** and **R. Bro-Rasmussen.** 1983. Cadmium in soil and terrestrial biota, with emphasis on the Danish situation. *Ecotoxicol. Environ. Safety* **7**:122-140.
14. **Hoffman, D.J., O.H. Pattee** and **S.N. Wiemeyev.** 1985. Effects of fluoride on screech owl reproduction: Teratological evaluation, growth, and blood density in hatchlings. *Toxicol. Lett.* **26**: 19-24.
15. **Suttie, J.W., R.J. Hamilton, A.C. Clay, M.L. Tobin** and **W.G. Moore.** 1985. Effects of fluoride ingestion on white-tailed deer *(Odocoileus virginianus). J. Wildl. Dis.* **21**:283-288.
16. **Newman, J.R.** 1984. Fluoride standards and predicting wildlife effects. *Fluoride* **17**:41-47.
17. **Walton, K.C.** 1984. Flouroide in fox bone near an aluminum reduction plant in Anglesey, Wales, and elsewhere in the United Kingdom. *Environ. Pollut. (Ser. **B) 1:273-280.***
18. **Robinette, W.L., D.A. Jones, *G.* Rogers** and **J.S. Gashwiler. 1957.** Notes on tooth development and wear for Rocky Mountain mule deer. *J. Wildl. Munage.* **21:**135-152.
19. **Karstad, L. 1967.** Fluorosis in deer *(Odocoileus virginianus). Bull. Wildl. Dis. Assoc.* **3**:42-46.
20. **Kay, C.E. 1975.** Fluoride distribution in different segments of the femur, metacarpus and mandible of mule deer. *Fluoride* **8**:92-97.
21. **Kay, C.E., P.C. Tourangeau** and **C.C. Gordon. 1975.** Industrial fluorosis in wild mule and whitetail deer from western Montana. *Fluoride* **8: 182-191.**

22. **Newman, J.R.** and **M. Yu. 1976.** Fluorosis in blacktailed deer. *J. Wildl. Dis.* **12**:39-41.

23. **Ohi, *G.*, H. Seki, K. Akiyama** and **H. Yagyu. 1974.** The pigeon, a sensor of lead pollution. *Bull. Environ.Contam. Toxicol.* **12**:92-98.

24. **Clarke, E.G.C.** and **M.L. Clarke.** 1967. *Garner's Veterinary Toxicology*, 3rd ed. Bailliere Tindall & Cassell, London, UK.

25. **Demayo, A., M.C. Taylor, K.W. Taylor** and **P.V. Hodson.** 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife, plants and livestock. *Crit. Rev. in Environ. Control* **12**:257-305.

26. **Grue. C.E. T.J. O'Shea** and **D.J. Hoffman.** 1984. Lead'concentrations and reproduction in highway nesting barn swallows. *Condor* **86**:383-389.

27. **Tansey, M.F.** and **R.R. Roth.** 1970. Pigeons: A new role in air pollution. *J. Air Pollut. Control Assoc.* **20**: 307-309.

28. **DeMent, D.H., J.J. Chisolm, J.C. Barber** and **J.D. Strandberg.** 1986. Lead exposure in an "urban" peregrine falcon and its avian prey. *J Wildl. Dis.* **22**: 238-244.

29. **Hutton, M.** 1982. The role of wildlife species in the assessment of the biological impact of chronic exposure to persistent chemicals. *Ecotoxicol. Environ. Safety* **6**:471-478.

30. **Jenkins, D.W.** 1980. *Biological Monitoring of Toxic Trace Metals*, Vol. I-Biological Monitoring and Surveillance. EPA-60013-80-089. US. Environmental Protection Agency, Las Vegas, NV.

31. **Wren, C.D.** 1986. Mammals as biological monitors of environmental metal levels. *Environ. Monit. Assess.* **6:** 127- 144.

32. **Hutton, M.** and **G.T. Goodman.** 1980. Metal contamination of feral pigeons, *Columbia livia,* from the London area: Part 1 -Tissue accumulation of lead, cadmium and zinc. *Environ. Pollut. (Ser. A)* 22x207-2 17.

33. **Way, C.A.** and **G.D. Schroder. 1982.** Accumulation of lead and cadmium in wild populations of the commensal rat, *Ruttus norvegicus. Arch. Environ. Contam. Toxicol.* **11:**407-417.

34. **Hirao, Y.** and **C.C. Patterson. 1974.** Lead aerosol pollution in the High Sierra overrides natural mechanisms which exclude lead from a food chain. *Science* **184**: 989-992.

35. **Eisler, R.** 1985. Selenium hazards to fish, wildlife, and invertebrates: A synoptic review. Contaminant Hazard Review Report No. ***5 ,*** Biological Report 85(1.5). US. Fish and Wildlife Service, Laurel, MD.

36. **Shaw, *G.G.*** and **L. Cocks.** 1982. The effect of SO_2 on food quality for wild herbivores. *Proceedings,* Acid Forming Emissions in Alberta and Their Ecological Effects. Alberta Department of Environment, Canadian Petroleum Association, Oil Sands Environmental Study. Edmonton. Alberta. March 9-12, 1982, pp. 571-587.

In addition to affecting individual animals or populations directly, air pollutants also affect wildlife indirectly by causing changes in the ecosystem. Vegetation affords cover for protection from predators and weather, provides breeding and nesting habitat, and also serves as a food source. Therefore, any change in vegetation could indirectly affect animal populations. Many studies have found that invertebrates show a preference for, or are better able to establish themselves in, air pollution-injured vegetation.

10.2 Air pollution and damage to crop yield and vegetation

Sulphur dioxide and nitrogen oxides both combine with water in the atmosphere to create acid rain. Acid rain acidifies the soils and waters where it falls, killing off plants. Many industrial processes produce large quantities of pollutants including sulphur dioxide and nitrous oxide. These are also produced by car engines and are emitted in the exhaust. When sulphur dioxide and nitrous oxide react with water vapour in the atmosphere, acids are produced. The result is what is termed acid rain, which causes serious damage to plants. In addition, other gaseous pollutants, such as ozone, can also harm vegetation directly. Whilst acid rain is a major cause of damage to vegetation, air pollutants can also be harmful directly. These include sulphur dioxide and ozone.

Extensive plant damage due to tropospheric ozone was first observed during the Los Angeles smog episodes. In the early 1950s, Haagen-Smit reported that such plant damage could be reproduced in the laboratory by the reaction of organic trace gases or car exhaust with nitrogen oxides (NO_x) in the presence of sunlight (Haagen-Smit et al, 1952, 1954). The influence of ozone on vegetation is dependent on the ozone dose and plant phenotype (Pleijel et al, 1991; Heath et al, 2008; Iriti et al, 2009). Ozone enters leaves through plant stomata during normal gas exchange in the daylight hours and impairs plant metabolism, leading to yield reduction in agricultural crops (Wilkinson et al, 2012; Ainsworth et al, 2012; Leisner & Ainsworth, 2012). In certain phenotypes, ozone exposure interferes with the hormone levels in plants and has been shown to lead to the accumulation of ethylene in the leaves. The presence of ethylene in the leaves interferes with the functioning of abscisic acid, a hormone which normally controls stomata closure and reduces water loss under drought conditions (Wilkinson et al, 2012). Consequently, such plant phenotypes, when exposed to both drought and O_3 stress, will continue to lose water despite the potential for dehydration. Tropospheric ozone causes damage to crops at elevated levels (Sinha et al, 2015). The ozone-induced physiological damage such as lower yields and inferior crop quality lead to large economic losses (Avnery et al, 2011a, b; Van Dingenen et al, 2009; Giles, 2005; Wilkinson et al, 2012). A study in India estimated that the total economic cost losses from reduced crop yield (rice, maize, cotton and wheat) in Punjab and Haryana amounted to USD 6.5 to 2.2 billion in the

fiscal year of 2012–2013 and USD 3.7 to 1.2 billion in the fiscal year of 2013– 2014. This economic loss estimate represents a very conservative lower limit based on the minimum support price of the crop, which is lower than the actual production costs. The upper limit for ozone-related crop yield losses in all of India currently amounts to 3.5–20% of India's GDP. The authors further observed that mitigation of high surface ozone would require relatively little investment in comparison to the economic losses incurred presently. Therefore, ozone mitigation can yield massive benefits in terms of ensuring food security and boosting the economy. The co-benefits of ozone mitigation also include a decrease in the ozone-related mortality and morbidity and a reduction of the ozone-induced warming in the lower troposphere (Avnery et al, 2011a, b) .

Field surveys that studied the effect of ozone on refuge vegetation have been conducted within the Moosehorn National Wildlife Refuge in northeastern Maine (Davis, 2007). A low incidence of foliar symptoms was reported among some species – Fraxinus spp. (ash), Populus spp. (aspen), Corylus cornuta (beaked hazelnut), Prunus serotina (black cherry), Prunus pensylvanica (pin cherry), Apocynum androsaemifolium (spreading dogbane),and a viburnum tentatively identified as Viburnum nudum var. cassinoides (withe-rod). Ozone levels are expressed as SUM60, the accumulation of ozone concentrations of 60 ppb or greater during the growing season. Therefore , the threshold level of SUM60 ozone capable of inducing symptoms on sensitive vegetation within this refuge and Class-I Wilderness area is less than1 8,000p ppb-hrs and maybe as low as 10,000p ppb-hrs. The authors suggested that the findings can be used in making air quality management decisions, including those related to review of 'Prevention of Significant Deterioration' permits, and the data might serve as input into formulating more stringent National Ambient Air Quality Standards for ozone.

10.3 Air pollution and freshwater sources [28]

Acidification of freshwaters was a problem that was first identified in Scandinavia during the early 1970s, at which time many scientific studies were initiated. Since then the concerns that were voiced have been justified, and now thousands of lakes and rivers are known to be acidified. Ample evidence from chemical and biological studies of typical lakes proves that increased acidification has taken place. Evidence suggests that rapid acidification has been taking place at some sites for at least 100 years and is still occurring today. The effects of freshwater acidification are as follows.

[28] Source: http://www.air-quality.org.uk/17.php (Accessed on 10 Dec 2015)

- Carbon source changes from carbonate (HCO_3) to carbon dioxide (CO_2).
- Release of toxic metals.
- Phosphorus is retained.
- Freshwater fauna and flora gradually changes.
- Short-term pH depressions have direct toxic effects on susceptible organisms.

Freshwater acidification occurs in areas of high sulphur deposition and where soils are derived from granite or other rocks resistant to weathering. The magnitude of acidification in the last 150 years is a lot greater that in the last 100,000 years. It seems that atmospheric pollution is the major cause of acidification. There is evidence that in the past decade there has been a significant decrease in the acidity of rain and this is reflected by a small decline in acidity of some lakes.

10.4 Air pollution and soil (See footnote 22, p87)

The damage that occurs to ecosystems from acidic deposition is dependent on the buffering ability of that ecosystem. This buffering ability is dependent on a number of factors, the two major ones being soil chemistry and the inherent ecosystem sensitivity to acidification. Indirect damage to ecosystems is largely caused by changes in the soil chemistry. Increasing soil acidity can affect micro-organisms which break down organic matter into nutrient form for plants to take up. Increasing soil acidity also allows aluminium (a common constituent of soil minerals) to come into solution. In its free organic form, aluminium is toxic to plant roots and can lock up phosphate, thereby reducing the concentrations of this important plant nutrient.

When acid rain falls, it can affect forests as well as lakes and rivers. To grow, trees need healthy soil to develop in. Acid rain is absorbed into the soil making it virtually impossible for these trees to survive. As a result of this, trees are more susceptible to viruses, fungi and insect pests. Long-term changes in the chemistry of some sensitive soils may have already occurred as a result of acid rain. As acid rain moves through the soils, it can strip away vital plant nutrients through chemical reactions, thus posing a potential threat to future forest productivity. Poisonous metals such as aluminium, cadmium and mercury, are leached from soils through reacting with acids. Plant life in areas where acid rain is common may grow more slowly or die as a result of soil acidification. In the Green Mountains of Vermont and the White Mountains of New Hampshire in the United States 50% of the red spruce have died in the past 25 years. There has also been noted a reduced amount of growth in existing trees as measured by the size of growth rings of the trees in these areas. These effects occur because acid rain leaches many of the existing soil nutrients from the soil. The number of micro-organisms present in the soil

also decreases as the soil becomes more acidic. This further depletes the amount of nutrients available to plant life because the micro-organisms play an important role in releasing nutrients from decaying organic material. In addition, the roots of plants trying to survive in acidic soil may be damaged directly by the acids present. Finally, if the plant life does not die from these effects, then it may be weakened enough so that it will be more susceptible to disease or other harsh environmental influences like cold winters or high winds.

10.5 Effects of air pollution on tourism

Tourism and travel is a fundamental contributor to the global economy and particularly imperative for many developing countries. Tourism has a potential driver for growth of the world economy. The tourism economy represents 5 % of the world GDP, while it contributes to 6–7 % of total employment. International tourism ranks fourth (after fuels, chemicals, and automotive products) in global exports, with an industry value of US $1 trillion a year, accounting for 30% of the world's exports of commercial services or 6 % of total exports; 935 million international tourists were recorded in 2010 and 4 billion domestic arrivals in 2008. In over 150 countries, tourism is one of the five top export earners, and in 60, it is the number one export. It is the main resource of foreign exchange for one third of developing countries and one half of less-developed countries (UNEP 2011). Of concern is the effect of climate change on tourism. A major objective of tourism studies has thus been to quantify the environmental impacts of leisure-related activities and to evaluate these with acceptable levels of pollution (Gosling et al, 2005). Saenz-de-Miera and Rosselló (2013) discovered tourists' involvement to air pollution through an analysis of tropospheric ozone levels in Mallorca (Spain). The results show that the rising tourism activity in Mallorca is connected with rising daily concentrations of tropospheric ozone, which is created by transport, air conditioning, and other activities. In another study, Saenz-de-Miera and Rosselló (2014) conclude that, "Tourism has begun to be acknowledged as being a significant contributor to the increase in environmental externalities, especially to climate change" (Saenz-de-Miera & Rossello, 2014). Tourism destinations are gradually more concerned about global climate change and considering becoming involved in the adoption of mitigation policies that reduce global emissions (Arana et al, 2013). To explore the relationship between climate change, air pollution, and tourism development, Sajjad et al (2014) studied the effects of climate change and air pollution on tourism indicators in South Asia, the Middle East and North Africa, sub-Saharan Africa, and East Asia and the Pacific regions over a period from 1975–2012. On average, climatic factors and air pollution deplete the forest area; however, the intensity of their effects varied region to region. The more pronounced factors to affect forest depletion include hydrofluorocarbons (HFC) in East Asia and the Pacific region, perfluorocarbon (PFC) in the Middle East North Africa (MENA) region, NO_X in the

sub-Saharan Africa and East Asia and the Pacific, and carbon emissions in South Asia. The authors concluded that climatic factors and air pollution severely affect the tourism industry. Furthermore, there are multiple channels to affect the climatic factors, air pollution, and tourism indicators; however, the direction of cause-effect relationship remains diverse.

The impact of the 1997 and 1998 haze-related air pollution episodes on the tourism industry in Brunei Darussalam has been analyzed using multiple regression analysis and monthly arrival data of tourists from January 1995 to September 1999 (a sample of 57 monthly observations). Conservative estimates of the impact of the haze-related air pollution using ordinary least square regression are a reduction of 3.75% in the number of tourists and direct economic loss to the tourism industry of about one million Brunei dollars (B$). Using the Poisson regression analysis, monthly arrivals are estimated to be reduced by about 28.7% resulting in total direct loss of about B$8 million (Anaman & Looi, 2000). Singapore and Malaysia suffered long periods of haze from forest fires in Indonesia since October 2015. However, no studies on the effects of air pollution on tourism have been published in these countries.

Clean air, clear skies and spectacular views of natural landscapes have been the primary attractions of national parks and wilderness areas throughout the world and across generations. In the U.S., research has documented that human-made pollutants in the atmosphere have dramatically obscured visibility in national parks and wilderness areas. Mace et al (2004) highlighted that regional haze has reduced visibility from a natural visual range of 225 km to 145 km in wilderness areas in western USA. Visibility in parks located in the eastern USA has also been reduced from 145 km to as low as 22 km (Newman, 1979). Cone (1996) posits that visitors in Grand Canyon National Park today see only half the distance of those who visited this natural wonder a century ago. Hence, impaired visibility, which is largely attributable to human-made air pollution, both negatively impacts and possibly serves as a barrier to recreational opportunities for visitors.

In a study using time-series econometric models of monthly visitation data of the Great Smoky Mountain National Park (in North Carolina and Tennessee), the most visited national park in the USA, Poudyl et al (2013) examine the effect of impaired visibility on visitation demand. The results indicate monthly visitation is significantly affected by the cumulative effect of the visibility condition in both the current and preceding months. The authors estimated that a programme aiming to improve the average visibility by 10% (5.5 km) from the current level could result in an increase of roughly one million recreational visits annually.

There is increasing concerns about Hong Kong's poor air quality and its impact on Hong Kong's competitiveness as a travel destination. Little research attention has been given to investigate whether air pollution will affect tourists' experience. Cheung and Law surveyed over 1,000 in-bound tourists at the Hong Kong International Airport in 2000 to elucidate the relationship between air quality and tourism (Cheung & Law, 2000). The results reveal that when choosing Hong Kong as a travel destination, air quality is a minor concern to both Asian and Western travelers. However, Asian visitors seem to be more conscious of air quality when selecting a destination than Western visitors. In addition, the Asian tourists' standard of what constitutes acceptable air quality is lower than their Western counterparts. This finding is in contrast to the common belief that the majority of the Westerners who have an environmental friendly mind are more likely to be influenced by the air quality than Asians. The study also reveals that the air quality in Hong Kong is still acceptable to tourists. Nevertheless, there is an urgent need to improve the air quality in Hong Kong to enhance tourists' satisfaction.

In December 2015, $PM_{2.5}$ rose to unprecedented concentrations Beijing. This episode has been attributed largely to domestic coal burning, vehicle exhaust and discharges from industry. In a questionnaire survey on the impact of haze on travel in Beijing, haze pollution was found to have considerable impact on travel. Tourist arrivals could decrease by a small margin, but the most significant impact could be on the temporal distribution of tourist arrivals, due to tourists' avoidance psychology (Zhang et al, 2015).

10.6 Air pollution and building damage (See footnote 22, p87)

Since the beginning of the Industrial Revolution soiling and degradation of buildings in urban areas has been noticeable. The cause of this has often been attributed to the effects of air pollution. The pollutants that form acid rain are principally sulphur dioxide and nitrogen oxides; both of these are released from the combustion of fossil fuels like coal and oil. The list of materials affected by acid deposition is very long as most materials are liable to some degree of damage. Those most vulnerable are: limestone; marble; carbon-steel; zinc; nickel; paint and some plastics. Stone decay can take several forms, including the removal of detail from carved stone, and the build-up of black gypsum crusts in sheltered areas. Metal corrosion is caused primarily by oxygen and moisture, although SO_2 does accelerate the process. Most structures and buildings are affected by acid deposition to some degree because few materials are safe from these effects. In addition to atmospheric attack structures that are submerged in acidified waters such as foundations and pipes can also be corroded. Wet and dry deposition both contribute to the corrosion of materials. Dry deposition consists of gaseous and particulate matter that falls to Earth close to the source of emissions causing direct damage. Sulphur

dioxide often falls as dry deposition within 30 km of its source. Wet deposition occurs when the pollutants are spread high into the atmosphere, where they react with water vapour in clouds to form dilute acids. The effects are felt much further afield and therefore wet deposition can affect areas that are many tens of kilometres away from any sources of pollution.

Calcium carbonate in certain stones dissolves in dilute sulphuric acid to form calcium sulphate:

$CaCO_3 + H_2SO_4 + H_2O \rightarrow CaSO_4.2H_2O + CO_2$

This has two effects. Firstly it causes the surface of the stone to break up; secondly, a black skin of gypsum (calcium sulphate) forms which blisters off exposing more stone. When the gypsum crystals form they can grow into the stone, and the process may continue for up to 50 years. This is known as the Memory Effect. The interactions between materials and pollutants are very complex and many variables are involved. Deposition of pollutants onto surfaces depends on atmospheric concentrations of the pollutants and the climate and micro-climate around the surface. The effects of acid deposition on modern buildings are considerably less damaging than the effects on ancient monuments. Limestone and calcareous stones which are used in most heritage buildings are the most vulnerable to corrosion and need continued renovation. Evidence of the damaging effect of acid deposition can be seen throughout the world. For example, world famous structures as the Taj Mahal, Cologne Cathedral, Notre Dame, the Colosseum and Westminster Abbey have all been affected.

The main pollutants affecting materials are sulphur dioxide and sulphates, nitrogen oxides and nitrates, chlorides, carbon dioxide and ozone. The materials most sensitive to pollutants are calcareous building stones and ferrous metals. Manifestations of damage include losses of mass, changes in porosity, discoloration and embrittlement. Table 9.3 summarises the effect of air pollution on materials (Rao et al, 2014).

Table 10.3: The effect of air pollution on materials

MATERIAL EFFECTED	RANGE OF SENSITIVITY
Brick	very low
Concrete	low
Mortar	moderate to high
sandstone, limestone, marble	high
Unalloyed steel	high
Stainless steel	very low
Nickel and nickel-plated steel	high
Zinc and galvanised steel	high
Aluminium	very low
Copper	low

To obtain estimates of the economic cost of damage from acid deposition it is necessary to know how decay rates are related quantitatively to pollutants and meteorological parameters (damage functions), and the distribution of materials exposed in buildings and in geographical areas. However, there are few damage functions available and those in existence lack general applicability (Butlin, 1990).

10.7 References

Ainsworth, E. A., Yendrek, C. R., Sitch, S., Collins, W. J., and Emberson, L. D. The effects of tropospheric ozone on net primary productivity and implications for climate change, *Ann Rev Plant* 2012; **63**, 637–661.

Anaman KA, Looi CN. Economic impact of haze-related air pollution on the tourism industry in Brunei Darussalam. *Economic Analysis and Policy* 2000; **3**(2):133-134.

Araña JE, León CJ,Moreno-Gil S, Zubiaurre AR. A comparison of tourists' valuation of climate change policy using different pricing frames. *J Travel Res* 2013; **52**(1):82–92.

Avnery, S., Mauzerall, D. L., Liu, J., and Horowitz, L. W. Global crop yield reductions due to surface ozone exposure 1: Year 2000 crop production losses and economic damage, *Atmos Environ* 2011; **45**:2284–2296.

Avnery, S., Mauzerall, D. L., Liu, J., and Horowitz, L. W. Global crop yield reductions due to surface ozone exposure 2: year 2030 potential crop production losses and economic damage under two scenarios of O3 pollution, *Atmos Environ* 2011; **45**:2297–2309.

Brown AR, Hosken DJ, Balloux F, Bickley LK, LePage G, Owen SF, et al.,. Genetic variation, inbreeding and chemical exposure — combined effects in wildlife and critical considerations for ecotoxicology. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences* 2009; **364**:3377–3390.

Butlin RN. Effects of air pollutants on buildings and materials. *Proceedings of the Royal Society of Edinburgh. Section B. Biological Sciences* 1990; **97**:255-272.

Cheung C, Law R. The Impact of air quality on tourism: the case of Hong Kong. *Pacific Tourism Review* 2000; **5**(1):69-74.

Cone, M. (1996), 'Strategy set for treating Grand Canyon smog problem', *Las Vegas Review Journal*, pp A1–A3.

Davis DD. Ozone-Induced Symptoms on Vegetation within the Moosehorn National Wildlife Refuge in Maine. Northeastern Naturalist 2007; **14**(3):403-414.

Giles, J. Hikes in surface ozone could suffocate crops. *Nature* 2005; **435**:7, doi:10.1038/435007a,.

Gössling S, Peeters P, Ceron JP, Dubois G, Patterson T, Richardson RB. The eco-efficiency of tourism. *Ecol Econ* 2005; **54**(4):417–434.

Haagen-Smit, A. J. Chemistry and Physiology of Los Angeles smog. *Ind Eng Chem* 1952; **44:** 1342–1346.

Haagen-Smit, A. J. and Fox, M. M. Photochemical ozone formation with hydrocarbons and automobile exhaust, *JAPCA J Air Waste Ma* 1954; **4**:105–109.

Heath, R. L. Modification of the biochemical pathways of plants induced by ozone: What are the varied routes to change? *Environ Pollut* 2008; **155**:453–463.

Iriti, M. and Faoro, F. Chemical diversity and defence metabolism: How plants cope with pathogens and ozone pollution. *Int J Molec Sci* 2009; **10**:3371–3399.

Isaksson C. Pollution and Its Impact on Wild Animals: A Meta-Analysis on Oxidative Stress. *EcoHealth* 2010; **7**:342–350.

Leisner, C. P. and Ainsworth, E. A. Quantifying the effects of ozone on plant reproductive growth and development. *Global Change Biol* 2012; **18**:606–616.

Mace, B.L., Bell, P.A., and Loomis, R.J. (2004), 'Visibility and natural quiet in national parks and wilderness areas', *Environment and Behavior*, Vol 36, pp 5–31.

Newman, J.R. Effects of industrial air pollution on wildlife. *Biological Conservation* 1979; **15**:181-190.

Pleijel, H., Skärby, L., Wallin, G., and Sellden, G. Yield and grain quality of spring wheat (triticum aestivum l., cv. drabant) exposed to different concentrations of ozone in open–top chambers. *Agr Environ Pollut* 1991; **69**:151–168.

Poudyal NC, Paudel B, Green GT. Estimating the impact of impaired visibility on the demand for visits to national parks. *Tourism Economics* 2013; **19**(2), 433–452. doi: 10.5367/te.2013.0204.

Rao NV, Rajasekhar, M, Rao GC. Detrimental effect of air pollution, corrosion on building materials and historical structures. American Journal of Engineering Research 2014; **3**(3):359-364.

Saenz-de-Miera O, Rosselló J. Tropospheric ozone, air pollution and tourism: a case study of Mallorca. *J Sustain Tour* 2013; **21**(8):1232–1243.
Saenz-de-Miera O, Rosselló J. Modeling tourism impacts on air pollution: the case study of PM_{10} in Mallorca. *Tour Manag* 2014; **40**:273–281.
Sajjad F, Noreen U, Zaman K. Climate change and air pollution jointly creating nightmare for tourism industry. *Environ Sci Pollut Res* 2014; **21**:12403–12418. DOI 10.1007/s11356-014-3146-7.
Sinha B, Singh Sangwan K, Maurya Y, Kumar V, Sarkar C, Chandra BP, Sinha V. Assessment of crop yield losses in Punjab and Haryana using 2 years of continuous in situ ozone measurements. *Atmos Chem Phys* 2015; **15**:9555–9576.
UNEP (2011) Tourism: Investing in energy and resource efficiency. United Nations Environment Programme, 2011. Online available at: http://www.unep.org/resourceefficiency/Portals/24147/scp/business/tourism/greeneconomy_tourism.pdf (accessed on 09 Dec 2015).
Van Dingenen, R., Dentener, F. J., Raes, F., Krol, M. C., Emberson, L., and Cofala, J. The global impact of ozone on agricultural crop yields under current and future air quality legislation. *Atmos Environ* 2009; **43**:604–618.

Wilkinson, S., Mills, G., Illidge, R., and Davies,W. J. How is ozone pollution reducing our food supply? J Exp Bot 2012; **63**:527–536. doi:10.1093/jxb/err317.
Zhang A, Zhong L, Xu Y, Wang H, Dang L. Tourists' Perception of Haze Pollution and the Potential Impacts on Travel: Reshaping the Features of Tourism Seasonality in Beijing, China. *Sustainability* 2015; **7**: 2397-2414; doi:10.3390/su7032397.

Chapter 11: Conclusion

After an extensive literature review of health and non-health effects of air pollution, we have performed a health and economic impact assessment (HIA, EIA) of air pollution in Hong Kong, using the most up-to-date data available (air quality data in 2014 and health data in 2012) as an example. Our approach in HIA is based on studies in Europe, Australia and the U.K., with necessary modifications owing to the lack of local health and economic data. Our findings are in agreement with that from other studies – that air pollution exerts a heavy toll on health and the economy. We have developed a worksheet to quantify the health and economic impacts. This worksheet can be used to estimate the burden of air pollution and the benefits of air pollution control. By using different air quality data that may be derived from modelling assumptions, the output from this worksheet will enable us to predict the health impact (within 95% confidence limit), and economic impact (within a wider range) the benefits of air pollution control. This tool will enable the use of cost-benefit analysis of different policy options, and represent an evidence-based approach in policy decisions that have hitherto been based on empirical data. We acknowledge the shortcomings and limitations of our findings and assumptions in our worksheet. Further revisions of this worksheet, with the accumulation and availability of more local health and economic data, will enable a more precise and robust assessment.