Environmental Protection Department

Agreement No CE 57/2006 (EP) Review of Air Quality Objectives and Development of a Long Term Air Quality Strategy for Hong Kong - Feasibility Study

Appendix F

Health Impact and International Practices in setting AQO

ARUP

F1 Health Impact

F1.1 Health Impacts of Air Pollution

Based on the review of the international research studies on air pollution (European Commission, 2005; USEPA 2006; WHO, 2006; DEFRA, 2007), the potential health effects can be subdivided into short-term acute and long-term chronic impacts. Short-term effect usually refers to a dose of exposure within 1 hour to 1 day. Chronic effect generally refers to continuous exposure in a 70-year life-time period. Based on the study findings, **Table F1.1** summarises the health effects of different major air pollutants.

Table F1.1: Short-term and long-term health effect of key air pollutants

Pollutants	Short-term Impact	Long-term Impact
Particulate Matter	• Lung inflammatory reactions • Respiratory symptoms • Adverse effects on the cardiovascular system • Increase in medication usage • Increase in hospital admissions • Increases in short-term mortality	• Increases in cardio-pulmonary and lung cancer mortality • Increases in lower respiratory symptoms and reduced lung function in children • Increases in chronic obstructive pulmonary disease and reduced lung function in adults
Nitrogen Dioxide	• Changes in pulmonary function in asthmatics • Increases in bronchial responsiveness in asthmatics	• Decreases lung function • Increases in risk of respiratory symptoms
Sulphur Dioxide	• Changes in pulmonary function and respiratory symptoms in asthmatics	• Increases in mortality
Ozone	• Increases in lung inflammation • Increases in lung permeability • Increases in respiratory symptoms • Decreases in mucociliary clearance rates	• Reduction in lung function growth in children
Carbon Monoxide	• Fatigue in healthy people • Chest pain in those with heart disease • Impaired vision and coordination • Mortality at very high concentrations	• Effects in cardiovascular system • Effects in central nervous system
Lead	• Lead poisoning especially in children • Increases in blood pressure	• Effects in central nervous system • Effects in kidney system • Effects in reproductive system

In support of these, a thorough review of research on the potential health hazards of such pollutants to human has been carried out to draw on scientific evidence from a wide variety of toxicological and epidemiological studies linking exposure to air pollutants to their potential effects.

The US is one of the pioneers on health risk analysis and the investigation of air pollution related illnesses. The subject has also been studied by UK experts for a fairly long time. In addition, there are also several large-scale studies on air pollution related illness conducted in EU, by WHO and Australian experts. Some highlights of the key review findings are described below.

F1.1.1 Particular Matter (PM)

The level of PM in air would cause health impact. The evidence on airborne PM and its public health impact is consistent in showing adverse health effects at exposures that are currently experienced by urban populations in both developed and developing countries. The range of health effects is broad, but is predominantly to the respiratory and cardiovascular systems. All population is affected, but susceptibility to the pollution may vary with health or age. The risk for various outcomes has been shown to increase with exposure and there is little evidence to suggest a threshold below which no adverse health effects would be anticipated. In fact, the low end of the range of concentrations at which adverse health effects has been demonstrated is not greatly above the background concentration, which for particles smaller than 2.5μm ($PM_{2.5}$) has been estimated to be 3–5μg/m^3 in both the United States and western Europe. The epidemiological evidence shows adverse effects of PM following both short-term and long-term exposures, but no threshold values have been identified.

Short-term exposures

Whether the 24-hour or the annual average AQG, is the more restrictive tends to vary between countries, this being largely dependent on the specific characteristics of pollutant sources and their location. When evaluating their AQGs and interim targets, WHO generally recommended that the annual average take precedence over the 24-hour average since, at low levels, there is less concern about episodic excursions. Meeting the guideline values for the 24-hour mean will however protect against peaks of pollution that would otherwise lead to substantial excess morbidity or mortality.

Multi-city studies conducted in Europe (29 cities) and in the United States (20 cities) reported short-term mortality effects for PM_{10} of 0.62% and 0.46% per 10μg/m^3 (24-hour mean), respectively (Katsouyanni et al., 2001; Samet et al., 2000). A meta-analysis of data from 29 cities located outside western Europe and North America found a mortality effect of 0.5% per 10μg/m^3(Cohen et al., 2004), very similar in fact to that derived for Asian cities (0.49% per 10μg/m^3) (HEI International Oversight Committee, 2004). These findings suggest that the health risks associated with short-term exposures to PM_{10} are likely to be similar in cities in developed and developing countries, producing an increase in mortality of around 0.5% for each 10μg/m^3 increment in the daily concentration. Ultrafine particles (UF), i.e. particles smaller than 0.1μm in diameter, have recently attracted significant scientific and medical attention. These are usually measured as a number concentration. While there is considerable toxicological evidence of potential detrimental effects of UF particles on human health, the existing body of epidemiological evidence is insufficient to reach a conclusion on the exposure–response relationship of UF particles. Therefore no recommendations can be provided as to guideline concentrations of UF particles at this point in time.

Long-term exposures

Cohort studies reported there are robust associations between long-term exposure to $PM_{2.5}$ and mortality, including long-term exposure studies that use the ACS and the Harvard Six-Cities data (Dockery et al., 1993; Pope et al., 1995; HEI, 2000, Pope et al., 2002, Jerrett, 2005).

Prospective cohort studies of mortality associated with chronic exposures to air pollution of outdoor origins have yielded especially valuable insights into the adverse health effects of long-term PM exposures. Such cohort studies using subject-specific information about relevant covariates (such as cigarette smoking, occupation, etc.) typically are capable of providing more certain findings of long-term PM exposure effects than are purely “ecological studies” (Künzli and Tager, 1997). The new, better designed cohort studies, as discussed below, have largely confirmed the magnitude of PM effect estimates derived from past cross-sectional studies. The extensive Harvard Six-Cities Study (Dockery et al., 1993) and the initial American Cancer Society (ACS) Study (Pope et al., 1995) agreed in their findings of statistically significant positive associations between fine particles and excess mortality, although the ACS study did not evaluate the possible contributions of other air pollutants. Neither study considered multipollutant models, although the Six-City study did

examine various PM and gaseous pollutant indices (including total particles, $PM_{2.5}$, $SO4^{2-}$, H^+, SO_2, and O3) and found that sulphate and $PM_{2.5}$ fine particles were most strongly associated with mortality. The excess RR estimates originally reported for total mortality in the Six-Cities study (and 95 percent confidence intervals, CI) per increments in PM indicator levels were: Excess RR = 18% (CI: 6.8%, 32%) for 20μg/m^3 PM_{10}; excess RR = 13.0% (CI: 4.2%, 23%) for 10μg/m^3 $PM_{2.5}$; and excess RR = 13.4% (CI: 5.1%, 29%) for 5μg/m^3 $SO4^{2-}$. The estimates for total mortality derived from the ACS study were excess RR = 6.6% (CI: 3.5%, 9.8%) for 10μg/m^3 $PM_{2.5}$ and excess RR 3.5% (CI: 1.9%, 5.1%) for 5μg/m^3 $SO4^{2-}$. The ACS pollutant RR estimates were smaller than those from the Six-Cities study, although their 95% confidence intervals overlap. In some cases in these studies, the life-long cumulative exposure of the study cohorts included distinctly higher past PM exposures, especially in cities with historically higher PM levels (e.g., Steubenville, OH); but more current PM measurements were used to help estimate the chronic PM exposures. In the ACS study, the pollutant exposure estimates were based on 8-87 concentrations at the start of the study (during 1979-1983). In addition, the average age of the ACS cohort was 56, which could overestimate the pollutant RR estimates and perhaps underestimate the life-shortening associated with PM associated mortality. Still, although caution must be exercised regarding use of the reported quantitative risk estimates, the Six-Cities and ACS prospective cohort studies provided consistent evidence of significant mortality associations with long-term exposure to ambient PM. The Six-Cities cohort was preselected by the investigators to be a representative population for the U.S. midwest / eastern regions of the country heavily-impacted by both coal combustion and motor vehicle effluents. By contrast, the ACS study cohort was drawn from a large pool of volunteers who happened to live in communities where several years of fine particle and/or sulphate ambient air concentration data were available. It is important to note that the ACS had a relatively small proportion of people with less than high school education (12% versus 28% for Six-Cities) and, by inference, better diets and access to good health care than an average U.S. population. To the extent that the mortality impact is lower in the better educated portion of the population, the mortality experience of the ACS cohort likely provides an underestimate for the U.S. population as a whole.

In contrast to the Six-Cities and ACS studies, early results reported by Abbey et al. (1991) and Abbey et al. (1995a) from another prospective cohort study, the Adventist Health Study on Smog (AHSMOG), reported no significant mortality effects of previous PM exposure in a relatively young cohort of California nonsmokers. However, these analyses used TSP as the PM exposure metric, rather than more health-relevant PM metrics such as PM_{10} or $PM_{2.5}$, included fewer subjects than the ACS study, and considered a shorter follow-up time than the Six-Cities study (ten years versus 15 years for the Six-Cities study). Further, the AHSMOG study included only nonsmokers (indicated by the Six-Cities Study as having lower pollutant RR's than smokers), suggesting that a longer follow-up time than considered in the past (10 years) might be required to have sufficient power to detect significant pollution effects than would be needed in studies that include smokers (such as the Six-Cities and ACS studies). Thus, greater emphasis was placed in the 1996 PM Air Quality Criteria Document of US on the results of the Six-Cities and ACS studies.

Overall, the previously available chronic PM exposure studies collectively indicated that increases in mortality are associated with long-term exposure to ambient airborne particles; and effect size estimates for total mortality associated with chronic PM exposure indices appeared to be much larger than those reported from daily mortality PM studies. This suggested that a major fraction of the reported mortality relative risk estimates associated with chronic PM exposure likely reflects cumulative PM effects above and beyond those exerted by the sum of acute exposure events.

F1.1.2 Ozone (O_3)

The level of O_3 in air would cause health impact. Significant additions to the health effects evidence base have, however, come from epidemiological time-series studies. Collectively these studies have revealed positive, small, though convincing, associations between daily mortality and ozone levels, which are independent of the effects of particulate matter. Similar associations have been observed in both North America and Europe. These latest time-series studies have shown health

effects at ozone concentrations below the previous guideline of 120µg/m^3 but without clear evidence of a threshold. This finding, together with evidence from both chamber and field studies that indicates that there is considerable individual variation in response to ozone, provides a good case for reducing the WHO AQG for ozone from the existing level of 120µg/m^3 to 100µg/m^3 (daily maximum 8-hour mean). It is possible that health effects will occur below the new guideline level in some sensitive individuals. Based on time-series studies, the increase in the number of attributable deaths brought forward is estimated to be 1–2% on days when the 8-hour mean ozone concentration reaches 100µg/m^3 over that when ozone levels are at a baseline level of 70µg/m^3. Similar effects were observed in summer camp studies, involving exercising children. Although some would argue that these responses may not necessarily be adverse, and that they were seen only with vigorous exercise, these views are counterbalanced by the possibility that there are substantial numbers of persons in the general population that might be more susceptible to the effects of ozone than the relatively young and generally healthy individuals who participated in the chamber study. At 8-hour concentrations exceeding 240µg/m^3, significant health effects are considered likely. This conclusion is based on the findings of a large number of clinical inhalation and field studies. Both healthy adults and asthmatics would be expected to experience significant reductions in lung function, as well as airway inflammation that would cause symptoms and alter performance. There are additional concerns about increased respiratory morbidity in children. According to time-series evidence, exposure to concentrations of ozone of this magnitude, would result in a rise in the number of attributable deaths brought forward of 5–9%, relative to exposures at the estimated background level.

Höppe et al. (1995a,b) examined several potentially susceptible populations for changes in pulmonary function attributable to O_3 exposure in Munich, Germany. The forestry workers and athletes were discussed in the previous section(??). Senior citizens (n = 41) and juvenile asthmatics (n = 43) were also monitored on “low” O_3 (mean ½-hr max O_3 of 32 to 34 ppb) and “high” O_3 (mean ½-hr max O_3 of 64 to 74 ppb) days. Subjects were requested to stay outdoors for at least 2 hours just before the afternoon pulmonary function test. Clerks (n = 40) were considered the nonrisk control group. Although clerks spent the majority of their time indoors, their outdoor exposures on high O_3 days were similar to that of the four other risk groups. The results showed no significant O_3 effects on the senior citizens. Clinical studies also have consistently shown that seniors are less responsive to O_3 (Bedi et al., 1989; Drechsler-Parks, 1995). Asthmatics and clerks experienced slight reductions in FEV1 on high O_3 days. Among all risk groups, juvenile asthmatics experienced the largest O_3-related decline in FEV1, -84.0 mL (95% CI: -196.4, 28.4) per 40 ppb increase in mean ½-hr max O_3.

Several other panel studies performed spirometry in children, another potentially susceptible group (Avol et al., 1998; Chen et al., 1999; Cuijpers et al., 1994; Frischer et al., 1997; Linn et al., 1996; Romieu et al., 2002; Scarlett et al., 1996; Ulmer et al., 1997). All studies, with the exception of Avol et al. (1998) and Scarlett et al. (1996), observed a decrease in FEV1 associated with O_3 exposure. In a cohort of 154 children in Surrey, England, Scarlett et al. (1996) observed no association between ambient O_3 concentrations and FEV0.75 (0.2mL [95% CI: -3.6, 3.9] increase per 30 ppb increase in 8-hr max O_3 at a 1-day lag), but noted a small effect of PM_{10} on lung function. The mean 8-hr max O_3 concentration was 50.7ppb (range 6.8 to 128ppb). The study by Avol et al. (1998) examined three groups of children, asthmatic (n = 53), wheezy (n = 54), and healthy (n = 103). Ozone levels were reported as being very low (values not provided). The authors advised that noncompliance by the subjects might have been a problem, and further noted limitations in the analysis methods and other aspects of the study design.

One large study measured spirometric lung function in 895 school children in three towns in Taiwan (Chen et al., 1999). The 1-hr max O_3 concentrations ranged from 19.7 to 110.3ppb. Lung function was measured only once for each subject. The authors reported significant associations between diminished FEV1 and FVC with a 1-day lag of O_3 concentrations. Effect sizes were typical of those observed in past studies, i.e., 0.5 to 1.0mL decline in FEV1 per ppb 7-38 increase in O_3 concentration. Ozone was the only air pollutant associated with changes in lung function in

multipollutant models including SO_2, CO, PM_{10}, and NO_2. Linn et al. (1996) repeatedly measured spirometric lung function among 269 school children in three southern California communities (Rubidoux, Upland, and Torrance). Lung function was measured over 5 consecutive days, once in each of 3 seasons over 2 school years. Between-week variability was controlled in the analysis by seasonal terms in the model. Statistical power was limited by the relatively narrow range of exposures that were experienced within each week. In addition, the study was restricted to the school year, eliminating most of the "high" O_3 season from consideration. During the study period, 24-hr avg O_3 levels at the central monitoring site ranged up to 53 ppb (mean 23 ppb), whereas personal measurements ranged up to 16 ppb (mean 5 ppb). A mean change of -11.6mL (95% CI: -20.6, -2.6) (approximately a 1% decline) in FEV1 was observed from morning to afternoon per 20ppb increase in 24-hr average O_3. Other associations (involving individual morning or afternoon FVC and FEV1 measurements) went in the plausible direction, but the O_3 effect estimates were much smaller. Ulmer et al. (1997) examined 135 children aged 8 to 11 years in two towns in Germany from March to October 1994 for O_3 effects on pulmonary function at four time periods. The cross-sectional results at each of the four time points showed limited FVC and no FEV1 associations. However, the longitudinal analysis, which combined data from all four periods yielded a mean change of -87.5 mL (95% CI: -143.2, -31.7) (approximately a 5% decline) in FEV1 per 40 ppb increase in ½-hr max O_3 for the town with the higher O_3 levels (median ½-hr max of 50.6ppb versus 32.1ppb). In the cross-sectional analysis, only between-person variability was analyzed. The longitudinal analysis, in which the subjects provided multiple days of measurements, provided information on both between- and within-subject responses.

There are a limited number of new epidemiologic studies examining the effects of O_3 on FEV1; however, results from these studies indicate that acute exposure to O_3 is associated with declines in FEV1 in children. These results further support the negative effects of O_3 on lung function observed in the meta-analysis on children attending summer camp (Kinney et al., 1996a) and in the clinical literature.

The effects of acute O_3 exposure on PEF in asthmatics were examined in several panel studies. In Mexico City, two studies of asthmatic school children were carried out simultaneously in the northern (Romieu et al., 1996) and southwestern sections of the city (Romieu et al., 1997). In the northern study, 71 mildly asthmatic school children aged 5 to 13 years old, were followed over time for daily morning (before breakfast) and afternoon (bedtime) PEF. The mean 1-hr max O_3 was 190 ppb (SD 80). In single-pollutant models, O_3 concentrations at 0-, 1-, and 2-day lags were associated with diminished morning and afternoon PEF, but only the 0-day lag morning effect was significant. The O_3 effect became nonsignificant when $PM_{2.5}$ was added to the model. In the southwestern study, 65 mildly asthmatic children aged 5 to 13 years old were followed during the summer and winter for daily morning and afternoon PEF. The mean 1-hr max O_3 was 196ppb (SD 78). Ozone concentrations at a 0- and 1-day lag were associated with afternoon PEF, with larger effects at a 1-day lag. Associations involving O_3 were stronger than those involving PM_{10}. Several additional studies, both in the United States and in other countries, reported significant associations between O_3 exposure and decrements in PEF among asthmatics (Gielen et al., 1997; Jalaludin et al., 2000; Just et al., 2002; Ross et al., 2002; Thurston et al., 1997).

Mortimer et al. (2002) examined 846 asthmatic children from the National Cooperative Inner-City Asthma Study (NCICAS) for O_3-related changes in PEF. Children from eight U. S. urban areas (St. Louis, MO; Chicago, IL; Detroit, MI; Cleveland, OH; Washington, DC; Baltimore, MD; East Harlem, NY; and Bronx NY) were monitored from June through August 1993. Median 8-hr avg O_3 (10 a.m.-6 p.m.) concentrations ranged from 34ppb in Chicago to 58 ppb in Washington, DC. The mean 8-hr avg O_3 level across the eight cities was 48ppb. This study provides representative data for the United States, in so much as children from multiple cities throughout the East and Midwest were examined. Asthmatic children from urban areas are an important subgroup of potentially at-risk populations. Study children either had physician diagnosed asthma and symptoms in the past 12 months or respiratory symptoms consistent with asthma that lasted more than 6 weeks during the previous year.

Mortimer et al. (2002) examined O_3-related changes in PEF for single-day lags from 1 to 6 days and a multiday lag period of 5 days. Of all the pollutants examined, including O_3, PM_{10}, NO_2, and SO_2, none were associated with evening PEF. Only O_3 was found to be associated with morning PEF. Small morning effects were observed at 1- and 2-day lags. The effect of O_3 on morning outcomes increased over several days. A strong association between O_3 and PEF also was found with a multiday lag period (cumulative lag of 1 to 5 days). Unrestricted lag models suggested that the O_3 exposure from 3 to 5 days prior had a greater impact on morning % PEF than more immediate exposures. Mortimer et al. discussed biological mechanisms for delayed effects on pulmonary function, which included increased bronchial reactivity secondary to airway inflammation associated with irritant exposure.

Mortimer et al. (2002) further noted that small declines in morning PEF may be of uncertain clinical significance; thus they calculated the incidence of ≥10% declines in PEF. A 5 to 15% change in FEV1 has been expressed as having clinical importance to asthma morbidity (American Thoracic Society, 1991; Lebowitz et al., 1987; Lippmann, 1988). Although greater variability is expected in PEF measurements, a ≥10% change in PEF also may have clinical significance. In Mortimer et al. (2002), O_3 was associated with an increased incidence of ≥10% declines in morning PEF (odds ratio of 1.30 [95% CI: 1.04, 1.61] per 30 ppb increase in 8-hr avg O_3 for a 5-day cumulative lag). This finding suggests that exposure to O_3 might be related to clinically important changes in PEF in asthmatic children. This study also observed that excluding days when 8-hr avg O_3 levels were greater than 80 ppb provided effect estimates that were similar to those when all days were included in the analysis, indicating that the negative effect of O_3 on morning PEF persisted at levels below 80 ppb. There is some concern, however, regarding the lack of an association between O_3 and afternoon PEF.

Results from the multicities study by Mortimer et al. (2002), as well as those from several regional studies, provide evidence of a significant relationship between O_3 concentrations and PEF among asthmatics. Collectively, these studies indicate that O_3 may be associated with declines in lung function in this potentially susceptible population.

F1.1.3 Nitrogen Dioxide (NO_2)

The level of NO_2 in air would cause health impact. As an air pollutant, nitrogen dioxide (NO_2) has multiple roles, which are often difficult or sometimes impossible to separate from one another. Animal and human experimental studies indicate that NO_2 – at short-term concentrations exceeding 200μg/m^3 – is a toxic gas with significant health effects. Animal toxicological studies also suggest that long-term exposure to NO_2 at concentrations above current ambient concentrations has adverse effects.

Numerous epidemiological studies have used NO_2 as a marker for the cocktail of combustion-related pollutants, in particular, those emitted by road traffic or indoor combustion sources. In these studies, any observed health effects could also have been associated with other combustion products, such as ultrafine particles, nitrous oxide (NO), particulate matter or benzene. Although several studies – both outdoors and indoors – have attempted to focus on the health risks of NO_2, the contributing effects of these other, highly correlated co-pollutants were often difficult to rule out.

Most atmospheric NO_2 is emitted as NO, which is rapidly oxidized by ozone to NO_2. Nitrogen dioxide, in the presence of hydrocarbons and ultraviolet light, is the main source of tropospheric ozone and of nitrate aerosols, which form an important fraction of the ambient air $PM_{2.5}$ mass.

Short-term exposures

A number of short-term experimental human toxicology studies have reported acute health effects following exposure to 1-hour NO_2 concentrations in excess of 500μg/m^3. Although the lowest level of NO_2 exposure to show a direct effect on pulmonary function in asthmatics in more than one laboratory is 560μg/m^3, studies of bronchial responsiveness among asthmatics suggest an increase in responsiveness at levels upwards from 200μg/m^3.

Long-term exposures

Evidence has emerged, however, that increases the concern over health effects associated with outdoor air pollution mixtures that include NO_2. For instance, epidemiological studies have shown that bronchitic symptoms of asthmatic children increase in association with annual NO_2 concentration, and that reduced lung function growth in children is linked to elevated NO_2 concentrations within communities already at current North American and European urban ambient air levels. A number of recently published studies have demonstrated that NO_2 can have a higher spatial variation than other traffic-related air pollutants, for example, particle mass. These studies also found adverse effects on the health of children living in metropolitan areas characterized by higher levels of NO_2 even in cases where the overall city-wide NO_2 level was fairly low. Recent indoor studies have provided evidence of effects on respiratory symptoms among infants at NO_2 concentrations below 40μg/m^3. These associations cannot be completely explained by co-exposure to PM, but it has been suggested that other components in the mixture (such as organic carbon and nitrous acid vapour) might explain part of the observed association. Cohort studies such as the Six Cities study found a positive and statistically significant association of all-cause and cardio-pulmonary mortality with nitrogen dioxide and particles (78%) (Dockery et al., 1993). However, the re-analysis of the ACS studies showed that there is lack of convincing positive associations in the cause-specific mortality with nitrogen dioxide (HEI, 2000; Pope et al., 2002). There is currently insufficient evidence to quantify the possible but unproven effects of exposure to ambient concentrations of NO_2 on mortality.

F1.1.4 Sulphur Dioxide (SO_2)

The level of SO_2 in air would cause health impact. Studies indicate that reductions in mean lung function values among groups of normal subjects at rest have been seen in 10-minute exposures at 4000ppb (11440μg/m^3) and at 5000ppb (14300μg/m^3). No significant changes in group mean lung function have been seen below 1000 ppb (2860μg/m^3) even with exercise, although there are examples of airway resistance increasing in individuals at that value, with deep breathing. Several studies have shown fairly large changes in mean values of lung function indices with 600 ppb (1716μg/m^3) and heavy exercise and with 500 ppb (1430μg/m^3) and moderate or severe but not light exercises. From the information published to date, the overall conclusion is that the minimum concentration evoking changes in lung function in exercising asthmatics is of the order of 400ppb (1144μg/m^3), although there is the one example of small changes in airway resistance in two sensitive subjects at 100 ppb (286μg/m^3).

Short-term exposure (less than 24 hours)

The most direct information on the acute effects of sulphur dioxide comes from controlled chamber experiments on volunteers. Most of these studies have been for exposure periods ranging from a few minutes up to one hour. The exact duration is not critical, however, because responses occur very rapidly, within the first few minutes from commencement of inhalation; continuing the exposure further does not increase the effects. The effects observed include reductions in FEV_1 or other indices of ventilatory capacity, increases in specific airway resistance, and symptoms such as wheezing or shortness of breath. Such effects are enhanced by exercise, which increases the volume of air inspired, thereby allowing sulphur dioxide to penetrate further into the respiratory tract. An acute effect of short-term exposure at rest to 0.2ppm sulphur dioxide is a change in heart rate variability, in which normal young adults responded with small but statistically significant increases in both high and low frequency power, while asthmatic subjects responded with decreases in these parameters of comparable magnitude.

A wide range of sensitivity has been demonstrated, both among normal individuals and among those with asthma, who form the most sensitive group for pulmonary function changes. Continuous exposure–response relationships, without any clearly defined threshold, are evident. To develop a guideline value, the minimum concentrations associated with adverse effects in the most extreme circumstances (i.e. with asthmatic patients exercising in chambers) have been considered. An example of an exposure–response relationship for such subjects was given by Linn et al. (1987) and was expressed in terms of reductions in FEV_1 after a 15-minute exposure. Only small changes, not regarded as of clinical significance, were seen at 572μg/m^3 (0.2 ppm); reductions representing about 10% of baseline FEV_1 occurred at about 1144μg/m^3 (0.4 ppm); and reductions of about 15% occurred at about 1716μg/m^3 (0.6 ppm). The response was not greatly influenced by the severity of asthma. These findings are consistent with those reported from other exposure studies. WHO (2006) also reviewed one early series, which indicated that a small change in airway resistance was reported in two of the asthmatic patients at 286μg/m^3 (0.1 ppm).

Exposure over a 24-hour period

Observational time series studies have reported numerous mortality and morbidity risk estimates for sulphur dioxide in the past decade. The consistency of the association of sulphur dioxide with health outcomes appears to be less than that for PM. As mentioned above, however, there are exceptions and the magnitude of estimated risks were often comparable to that of PM. Interestingly, many of the researchers, in their discussions when sulphur dioxide associations were found, did not often interpret these sulphur dioxide associations with mortality or morbidity as causal but rather as “artefact”, and suggested that sulphur dioxide was acting as a “surrogate” for a source type. The reasoning offered for the sulphur dioxide levels being “too low” to be causal was based on prior knowledge. This situation highlights a limitation of the observational studies, the “surrogate” interpretation, i.e. that sulphur dioxide represents a source type (e.g. coal-fired power plant), or a mixture that can affect health through a co-pollutant (e.g. PM), or through pollutants that it is converted into (i.e. sulphuric acid and sulphates). Yet another interpretation is that PM becomes more toxic when sulphur dioxide coexists and gets adsorbed onto PM surfaces. Reanalysis of Philadelphia time series data some years ago found some indication that the proportional increment in daily mortality associated with TSP was greater at higher levels of sulphur dioxide. In any case, the current observational studies were not designed to resolve these issues. The Hong Kong intervention studies do suggest that reducing the use of sulphur-rich fuels leads to reductions in adverse health effects.

In the past, exacerbation of symptoms among panels of selected sensitive patients occurred consistently when the concentration of sulphur dioxide exceeded 250μg/m^3 (0.087 ppm) in the presence of PM. Such findings were related mainly to situations in which emissions from the inefficient burning of coal in domestic appliances was the main contributor to the pollution complex. More recent studies, involving the mixed stationary and vehicular sources that now dominate, have consistently demonstrated effects on daily mortality (total, cardiovascular and respiratory) and hospital emergency admissions for total respiratory causes and COPD at much lower levels of exposure (mean daily levels below 50μg/m^3). The Hong Kong “intervention” study (Hedley et al., 2002) indicated significant health benefits in reducing sulphur dioxide from a daily average of 44μg/m^3 to 21μg/m^3. As with ozone and PM, no obvious threshold levels have so far been identified in these population-based studies.

Long-term exposure

Earlier assessments examined findings on the prevalence of respiratory symptoms, respiratory illness frequencies or differences in lung function values in localities with contrasting concentrations of sulphur dioxide and PM, largely in the coal-burning era. The lowest observed adverse effect level (LOAEL) of sulphur dioxide was judged to be 100μg/m^3 (0.035ppm) annual average together with PM. The more recent studies related to the changed urban mixture have shown associations between ambient levels of sulphur dioxide and adverse effects at concentrations well below this level (18–27μg/m^3), but one major difficulty in interpretation is that

long-term effects are liable to be affected not only by current conditions. Another is the qualitatively and quantitatively different pollution of earlier years. Cohort studies of differences in annual mortality between areas with contrasting pollution levels indicate that there is a closer association with PM, sulphur dioxide and sulphate aerosol than with other measured air pollutants. The intervention study in Hong Kong (Hedley et al., 2002) showed a positive association of mortality with SO_2 in the absence of a change in particle concentrations.

F1.1.5 Carbon Monoxide (CO)

The level of CO in air would cause health impact. In general, CO diffuses rapidly across alveolar, capillary and placental membranes. Approximately 80-90% of the absorbed CO binds with hemoglobin to form carboxyhemoglobin (COHb), which is a specific biomarker of exposure in blood. The affinity of hemoglobin for CO is 200-250 times that for oxygen. During exposure to a fixed concentration of CO, the COHb concentration increase rapidly at the onset of exposure, starts to level off after 3 hours, and reaches a steady-state after 6-8 hours of exposure. It is noted that the elimination half-life in the fetus is much longer than in the pregnant mother.

The binding of CO with hemoglobin to form COHb reduces the oxygen-carrying capacity of the blood and impairs the release of oxygen from hemoglobin. These are the main causes of tissue hypoxia produced by CO at low exposure levels. At higher concentrations, the rest of the absorbed CO binds with other heme proteins such as myoglobin and with cytochrome oxidase and cytochrome P-450. The toxic effects of CO first become evident in organs and tissues with high oxygen consumption, such as the brain, heart, exercising skeletal muscle and the developing fetus. Severe hypoxia due to acute CO poisoning may cause both reversible, short-lasting, neurological deficits and severe, often delayed, neurological damage. The neurobehavioural effects include impaired coordination, tracking, driving ability, vigilance and cognitive performance at COHb levels as low as 5.1-8.2%.

In controlled studies (Anderson et al., 1973; Sheps et al., 1987; Adams et al., 1988; Allred et al 1989; Kleinman et al., 1989) involving patients with documented coronary artery disease, mean pre-exposure COHb levels of 2.9-5.9% (Corresponding to post-exercise COHb levels of 2.0-5.2%) have been associated with a significant shortening in the time to onset of angina, with increased elecrocardiographic changes and with impaired left ventricular function during exercise. In addition, ventricular arhythmias may be increased significantly at the higher range of mean post-exercise COHb levels. Epidemiological and clinical data indicate that CO from smoking and environmental or occupational exposures may contribute to cardiovascular mortality and to the early course of myocardial infarction. Current data from epidemiological studies and experimental animal studies indicate that common environmental exposures to CO in the developed world would not have atherogenic effects on humans (WHO 1999a).

During pregnancy, endogenous production of CO is increased so that maternal COHb levels are usually about 20% higher than the non-pregnant values. At steady-state, the fetal COHb levels are as much as 10-15% higher than the maternal COHb levels. There is a well-established and probably causal relationship between maternal smoking and low birth weight at fetal COHb levels of 2-10%. In addition, maternal smoking seems to be associated with a dose-dependent increase in perinatal deaths and with behavioural effects in infants and young children.

F1.1.6 Lead (Pb)

The level of Pb in air will have health impact. The level of lead in blood is the best available indicator of current and recent past environmental exposure and, with stable exposures, may also be a reasonably good indicator of lead bodyburden. The biological effects of lead can therefore be related to blood lead levels as an indicator of internal exposure. The relationship between blood lead concentrations and exposure to lead in air exhibits downward curvilinearity where the range of exposures is sufficiently large. At low levels of exposure the deviation from linearity is negligible and linear models of the relationship between intake and blood lead levels are satisfactory approximations.

The LOAEL for hematological and neurological effects in adults and children can be summarized as follows. Frank anemia is exhibited in adults at blood lead levels above 800 µg/l, and in children above about 700 µg/l. Hemoglobin production is reduced in adults at blood lead levels above 500 µg/l and in children above 250-300 µg/l. The presence of lead in the blood also inhibits delta-aminolaevulinic acid dehydrase (ALAD), an enzyme involved in heme biosynthesis, resulting in an accumulation of its substrate, ALA, in blood, plasma and urine (WHO 1987). Urinary ALA and coproporyphyrin are elevated in both adults and children above blood lead levels of about 400 µg/l. Erythrocyte protoporphyrin is found to increase in male adults at blood lead levels above 200-300µg/l, and in female adults and children above 150-200 µg/l. A reduction in vitamin D3 occurs in children at blood lead levels above 100-150 µg/l. Consequently, inhibition of ALAD in adults and children is likely to occur at blood lead levels of about 100 µg/l. However, because of its uncertain biological significance for the functional reserve capacity of the heme biosynthetic system, ALAD inhibition is not treated as an adverse effect here. Encephalopathic signs and symptoms appear not to occur in adults at lead concentrations in blood below 1000-1200 µg/l, and in children below 800-1000 µg/l.

Cognitive effects in lead workers have not been observed at blood lead levels below 500 µg/l, although reductions in nerve conduction velocity were found at concentrations as low as 300 µg/l. Elevation of free erythrocyte protoporphyrin has been observed at blood lead levels of 200- 300 µg/l. Central nervous system effects, as assessed by neurobehavioural endpoints, appear to occur in children at levels below 200 µg/l. Consistent effects have been reported for global measures of cognitive functioning, such as the psychometric intelligence quotient, at blood lead levels between 100-150 µg/l. Some epidemiological studies have indicated effects such as hearing impairment at blood lead levels below 100 µg/l. In addition, animal studies provide qualitative support for the claim that lead is a causative agent for hearing impairment.

F1.2 Rationales of Setting Air Quality Standards in Different Countries

F1.2.1 Terminology and Purposes of Air Quality Objectives

There are a wide range of terms and concepts adopted in reference to national and international air quality management, for examples, standards, objectives, target values and limit values. A review of the terminologies for ambient air quality requirements in different countries and their dependences have been conducted and summarised as follows.

HKAQO is a set policy objective for air quality management as to achieve and maintain an acceptable level of air quality to safeguard the health and well being of the community, and to promote the conservation and best use of air in the public interest. These AQOs are statutory objectives and established to promote the conservation and best use of air in the public interest. The protection of public health, even though not stated explicitly, is already a key consideration because to do otherwise will not be in “public interest”.

The Mainland China uses the terminology of Ambient Air Quality Standard (AQS). It stipulates the ambient air quality functional zone, standard class, pollutant item, maximum allowable concentration and the respective averaging time period, sampling and analytical methodology, and validity of statistical data without specification of the number of exceedences allowed. Taiwan also uses the terminology of AQS. It is defined as the concentration limit of the pollutants in the ambient air.

The United States also uses the terminology of AQS. There are 2 types of AQS established:

(i) National primary AQS: “Ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”

(ii) National secondary AQS: “A level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect public welfare

from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air."

The number of exceedences to be allowed has also been stipulated.

The European Union (EU) uses the terminology of Limit Values and Target Values. The former means a level fixed on the basis of scientific knowledge, with the aim of avoiding, preventing or reducing harmful effects on human health and/or the environment as a whole, to be attained within a given period and not to be exceeded once attained. The latter means a level fixed with the aim of avoiding, preventing or reducing harmful effects on human health and/or the environment as a whole, to be attained where possible over a given period. Allowances of exceedences and tolerance of allowance of the values have also been stipulated.

The United Kingdom uses AQO and differentiates it clearly from AQS. The AQO is defined as the maximum ambient concentration not to be exceeded, either without exception or with a permitted number of exceedences, within a specified timescale. AQS, on the other hand, is defined as the concentration of pollutants which can be taken to achieve a certain level of environmental quality and serves as the benchmark for setting AQO. The AQOs are regarded as a statement of policy intentions or policy targets and there is no legal requirement to meet these objectives except in as far as these mirror any equivalent legally binding limit values in EU legislation. In "The Air Quality Strategy for England, Scotland, Wales and Northern Ireland, Volume 1 – July 2007", the primary objective of UK Government and devolved administration is to ensure that all citizen should have access to outdoor air without significant risk to their health, where this is economically & technically feasible.

Australia refers to their AQO as "ambient air quality measure". The purpose of the ambient air quality measure, together with their numerical figures, is stipulated in their "National Environment Protection (Ambient Air Quality) Measure" made under the Australian National Environment Protection Council Act 1994 as follows:
"The desired environmental outcome of this Measure is ambient air quality that allows for the adequate protection of human health and well-being."
Similar to other countries/economies, the number of exceedences to be allowed has also been stipulated.

F1.3 Rationale of WHO AQG

F1.3.1 Sulphur Dioxide

Controlled studies involving exercising asthmatics indicate that a proportion experience changes in pulmonary function and respiratory symptoms after periods of exposure to SO_2 as short as 10 minutes (short-term exposures). Latest epidemiological studies in US and HK also show day-to-day changes in mortality, morbidity or lung function in relation to 24-hour average concentration of SO_2. Thus, short-term (10-minute) and 24-hour guidelines are derived for SO_2 pollution. Since compliance with the 24-hour level will assure low annual average levels, annual guideline is no longer needed. Some relevant scientific evidence and their rationale are summarized in **Table F1.2**.

Table F1.2: Key rationale of revising the WHO AQGs for sulphur dioxide

(A) Short-term exposures		**Reference**
Scientific evidence:	• Controlled studies involving exercising asthmatics show a proportion experience changes in pulmonary function and respiratory symptoms after periods of exposure to SO_2 as short as 10 minutes. Based on this evidence, it is recommended that a SO_2 concentration of 500 μg/m^3 should not be exceeded over averaging periods of 10-minutes duration. • Prior to 1987, guideline values for SO_2 were linked to corresponding values for PM and this approach led to the setting of an AQG value for SO_2 of 125 μg/m^3 as a 24-hour average.	Wong et al., 2002a
	• Recent time-series studies on hospital admissions for cardiac disease in Hong Kong and London, produced no evidence of a threshold for health effects of 24-hour SO_2 concentrations in the range of 5-40 μg/m^3. • In the American Cancer Society (ACS) study, significant associations between SO_2 and mortality were observed in the United States metropolitan areas, in which the mean SO_2 concentration recorded was 18 μg/m^3.	Pope et al., 2002
Selection of WHO AQG value:	• 24-hour mean SO_2 concentration of 20 μg/m^3 • 10-minute mean SO_2 concentration of 500 μg/m^3	
(B) Long-term exposures		
Scientific evidence:	• Since compliance with the 24-hour level will assure low annual average levels, annual guideline is no longer needed as compliance is natural with the 10-min level being the control factor.	WHO Global update 2005
Selection of WHO AQG value	• No guideline value proposed.	

F1.3.2 Nitrogen Dioxide

UK studies revealed that there is no adverse health effect for normal person with a concentration up to 4,688μg/m^3. In people with asthma, some studies have shown changes in these tests of lung function at exposures of around 560μg/m^3 (300ppb) when the subjects have been exercising. Studies of bronchial responsiveness among asthmatics suggest an increase in responsiveness at levels upwards from 200μg/m^3. This is an experimental threshold value to show response but it does not correspond to any adverse effect on pulmonary function.

Studies on indoor cooking have provided evidence of effects on respiratory symptoms among infants at NO_2 concentrations below 40μg/m^3, with the co-exposure to PM and other gaseous mixture (organic carbon and nitrous acid vapour). However, it is unclear as to what extent the health effects are attributable to NO_2 itself.

Since the previous WHO AQG value has not been challenged by more recent studies, as a precautionary approach to control complex mixtures of combustion-related pollution, the guideline values for NO_2 remain unchanged to protect the susceptible people, i.e. 40 μg/m^3 for annual mean

and 200 μg/m^3 for 1-hour mean. The rationale for the WHO AQG values is summarized in **Table F1.3**.

Based on the research findings and rationale for the AQG, the selected criteria are mainly focused on the susceptible group (infant or asthma) rather than normal person. In addition, owing to the fact that NO_2 is usually co-existed with other combustion-related pollution, NO_2 is usually used as a marker for complex combustion-generated pollutant mixtures, thus lower guideline values have been used. With respect to long-term exposure, there is low association in all-cause mortality due to inconclusive observations.

Table F1.3: Key rationale of the WHO AQGs for nitrogen dioxide

(A) Short-term exposures		**Reference**
Scientific evidence:	• A number of short-term experimental human toxicology studies have reported acute health effects following exposure to 1-hour NO_2 concentrations in excess of 500 μg/m^3. • The lowest level of NO_2 exposure showing a direct effect on pulmonary function in asthmatics in laboratory is 560 μg/m^3 when the subjects have been exercising. • Studies of bronchial responsiveness among asthmatics suggest an increase in responsiveness at levels upwards from 200 μg/m^3 (an experimental threshold value to show response).	WHO Global update 2005
Selection of WHO AQG value:	• 1-hour mean of 200 μg/m^3	
(B) Long-term exposures		**Reference**
Scientific evidence:	• Indoor studies (indoor cooking) have provided evidence of effects on respiratory symptoms among infants at NO_2 concentrations below 40 μg/m^3, with co-exposure to PM and other gaseous mixture (organic carbon and nitrous acid vapour). However, it is unclear as to what extent the health effects are attributable to NO_2 itself. • As NO_2 is adopted as a marker monitoring parameter for complex combustion-generated pollutant mixtures, a lower annual guideline value should be used as a means to control the complex mixtures of combustion-related pollution.	WHO Global update 2005
Selection of WHO AQG value:	• Annual mean NO_2 concentration of 40 μg/m^3.	

F1.3.3 Particulate Matter

The latest relevant key scientific evidence and the revised guideline values on PM are summarized in **Table F1.4**. Most of the long-term research data are conducted in US (majority) and Europe and further interpreted by experts in US and WHO. Based on known health effects, both short-term (24-hour) and long-term (annual mean) guidelines are included as indicators of PM pollution. The rationale for the WHO AQG values is also summarized. In the process of defining PM_{10} criteria, a ratio of 0.5 between (in the range of 0.5 to 0.8) $PM_{2.5}$ to PM_{10} has been adopted by WHO. This ratio is specific for areas of different nature and can be established by monitoring data. Data from EPD monitoring stations have indicated this ratio to be 0.7 for Hong Kong. This suggests the PM_{10}

guideline values applicable to Hong Kong would be lower than the WHO AQG in accordance with the principle of $PM_{2.5}$ to PM_{10} correlation.

Table F1.4: Key rationale of the WHO AQGs for PM

(A) Long-term exposures		**Reference**
Scientific evidence:	• Historical mean $PM_{2.5}$ concentration was 18 μg/m^3 (range, 11.0-29.6 μg/m^3) in the Six-Cities study, and 20 μg/m^3 (range, 9.0-33.5 μg/m^3) in the ACS study in the United States. • Increases in risk are apparent in the city with long-term $PM_{2.5}$ mean of 14.9 μg/m^3, indicating that health effect is still evident in the concentration range of 11-15 μg/m^3. • Daily exposure time-series studies showed that acute adverse health outcomes were observed when long-term $PM_{2.5}$ means are in the range of 13-18 μg/m^3.	Dockery et al., 1993; Pope et al., 1995; HEI, 2000; Pope et al., 2002; Jerrett, 2005
Selection of WHO AQG value:	• Annual mean $PM_{2.5}$ concentration of 10 μg/m^3. • Annual mean PM_{10} concentration of 20 μg/m^3 was based on $PM_{2.5}$ to PM_{10} ratio of WHO at 0.5. However, data from EPD monitoring stations indicate the Hong Kong $PM_{2.5}$ to PM_{10} ratio is 0.7. By substitution, this gives the applicable annual mean PM_{10} concentration at 14μg/m^3 for Hong Kong.	
(B) Short-term exposures		**References**
Scientific evidence:	• Multi-city studies conducted in Europe and in the United States reported short-term mortality effects for PM_{10} of 0.62% and 0.46% per 10 μg/m^3 (24-hour mean), respectively. • Mortality effect for PM_{10} was found to be 0.5% per 10 μg/m^3 outside western Europe and North America, and that derived for Asian cities was 0.49% per 10 μg/m^3. • For PM_{10}, the AQG for the 24-hour average is 50 μg/m^3, and reflects the relationship between the distributions of 24-hour means (and its 99th percentile) and annual average concentrations.	Katsouyanni et al., 2001; Samet et al., 2000 Cohen et al., 2004; HEI International Oversight Committee, 2004
Selection of WHO AQG value:	• 24-hour mean $PM_{2.5}$ concentration of 25 μg/m^3. • 24-hour mean PM_{10} concentration of 50 μg/m^3 was based on $PM_{2.5}$ to PM_{10} ratio of WHO at 0.5. However, data from EPD monitoring stations indicate the Hong Kong $PM_{2.5}$ to PM_{10} ratio is 0.7. By substitution, this gives the applicable 24-hour mean PM_{10} concentration at 35.7 μg/m^3 for Hong Kong.	

F1.3.4 Ozone

Since publication of the second edition of the WHO AQGs for Europe (WHO, 2000), large amount of evidences from epidemiological time-series studies have established relationship between daily mortality and ozone levels. With the latest epidemiological time-series studies findings, the short-term (8-hour) guidelines for ozone have been proposed. According to latest studies in US, there is little evidence to correlate between relationship of chronic O_3 exposure and increase risk of mortality. Annual average criterion has therefore not been proposed. Some relevant scientific evidence and their rationale are summarized in **Table F1.5**.

Table F1.5: Key rationale of WHO AQGs for ozone

(A) Short-term exposures		**Reference**
Scientific evidence:	• Time-series studies have shown health effects at ozone concentrations below the previous guideline of 120 μg/m^3 but without clear evidence of a threshold. • Evidence from both chamber and field studies indicate that there is considerable individual variation in response to ozone.	WHO Global update 2005
Selection of WHO AQG value:	• Daily maximum 8-hour mean O_3 concentration of 100 μg/m^3.	
(B) Long-term exposures		
Scientific evidence:	• No conclusive findings and low associations were found in the RR for all cause mortality.	WHO Global update 2005 & Air quality criteria for ozone and related photochemical oxidants (final) USEPA, 2006
Selection of WHO AQG value:	• No guideline value proposed.	

F1.3.5 Carbon Monoxide

The guideline values for CO are not included in WHO Global update 2005 (WHO, 2006). The previous guideline values from the second edition of the WHO AQGs for Europe (WHO, 2000) remain in effect. Epidemiological studies have shown that to protect nonsmoking, middle-aged and elderly population groups with documented or latent coronary artery disease from acute ischaemic heart attacks, and to protect the fetuses of nonsmoking pregnant women from untoward hypoxic effects, a COHb level of 2.5% should not be exceeded. Some relevant scientific evidence and their rationale are summarized in **Table F1.6**.

Table F1.6: Key rationale of WHO AQGs for carbon monoxide

(A) Short-term exposures		**Reference**
Scientific evidence:	• In healthy subjects, endogenous production of carbon monoxide results in COHb levels of 0.4–0.7%. • During pregnancy, elevated maternal COHb levels of 0.7–2.5%, mainly due to increased endogenous production, have been reported. • The COHb levels in non-smoking general populations are usually 0.5–1.5%, owing to endogenous production and environmental exposures. Nonsmokers in certain occupations (car drivers, policemen, traffic wardens, garage and tunnel workers, firemen, etc.) can have longterm COHb levels of up to 5%, and heavy cigarette smokers have COHb levels of up to 10%. • Well trained subjects engaging in heavy exercise in polluted indoor environments can increase their COHb levels quickly up to 10–20%. In indoor ice arenas, epidemic carbon monoxide poisonings have recently	Coburn et al., 1965; Longo, 1977; ACGIH Chemical Substances TLV Committee, 1991; USEPA, 1991

(A) Short-term exposures		Reference
	been reported. • The guidelines are based on the Coburn-Foster-Kane exponential equation, which takes into account all the known physiological variables affecting carbon monoxide uptake. The following guideline values (ppm values rounded) and periods of time-weighted average exposures have been determined in such a way that the COHb level of 2.5% is not exceeded, even when a normal subject engages in light or moderate exercise.	
Selection of WHO AQG value:	• The following guidelines were established: – 100,000 µg/m^3 for 15 minutes – 60,000 µg/m^3 for 30 minutes – 30,000 µg/m^3 for 1 hour – 10,000 µg/m^3 for 8 hours	

F1.3.6 Lead

The guideline values for Pb are not included in WHO Global update 2005 (WHO, 2006). The previous guideline values from the second edition of the WHO AQGs for Europe (WHO, 2000) remain in effect. Guidelines for lead in air will be based on the concentration of lead in blood. Critical effects to be considered in the adult organism include elevation of free erythrocyte protoporphyrin, whereas for children cognitive deficit, hearing impairment and disturbed vitamin D metabolism (Rosen et al., 1980; Mahaffey et al., 1982) are taken as the decisive effects. All of these effects are considered adverse. A critical level of lead in blood of 100 µg/l is proposed. It should be stressed that all of these values are based on population studies yielding group averages, which apply to the individual child only in a probabilistic manner. Although some lead salts have been found to be carcinogenic in animals, the evidence for a carcinogenic potential in humans is inadequate and will, therefore, not be considered here. Some relevant scientific evidence and their rationale are summarized in **Table F1.7**.

Table F1.7: Key rationale of WHO AQGs for lead

(A) Short-term exposures		Reference
Scientific evidence:	• Currently measured "baseline" blood lead levels of minimal anthropogenic origin are probably in the range10–30 µg/l. • Various international expert groups have determined that the earliest adverse effects of lead in populations of young children begin at 100–150 µg/l. Although it cannot be excluded that population effects may occur below this range, it is assumed to be prudent to derive a guideline value based on the lowest value in this range (100 µg/l). • It can be assumed that inhalation of airborne lead is a significant route of exposure for adults (including pregnant women) but is of less significance for young children, for whom other pathways of exposure such as ingested lead are generally more important. • It appears that 1 µg lead per m^3 air directly contributes approximately 19 µg lead per litre blood in children and about 16 µg per litre blood in adults, although it is	Schwartz et al., 1994; WHO, 1995

(A) Short-term exposures		Reference
	accepted that the relative contribution from air is less significant in children than in adults. These values are approximations, recognizing that the relationships are curvilinear in nature and will apply principally at lower blood lead levels. • It must be taken into account that, in typical situations, an increase of lead in air also contributes to increased lead uptake by indirect environmental pathways. To correct for uptake by other routes as well, it is assumed that 1 µg lead per m^3 air would contribute to 50 µg lead per litre blood. • It is recommended that efforts be made to ensure that at least 98% of an exposed population, including preschool children, have blood lead levels that do not exceed 100 µg/l. In this case, the median blood lead level would not exceed 54 µg/l. On this basis, the annual average lead level in air should not exceed 0.5µg/m^3. This proposal is based on the assumption that the upper limit of nonanthropogenic blood is 30 µg/l. These estimates are assumed to protect adults also. • To prevent further increases of lead in soils and consequent increases in the exposure of future generations, air lead levels should be kept as low as possible.	
Selection of WHO AQG value:	• Annual mean Pb concentration of 0.5 µg/m^3.	

F1.4 International Health Impact Studies

In recent years, there are many medical research studies conducted in HK to review the short-term health effect contributing from air pollutants (Wong et al., 2002a). These valuable studies findings can be adopted as the basis on short-term effect in local context. For recent studies in HK and Asia Pacific Regions (Aga et al., 2003), which had adopted the same assessment method as in EU using the 'Air Pollution and Health: A European Approach (APHEA) Phase II'. A relatively stronger association and a greater magnitude of short-term mortality effect for SO_2 (its correlations with others were the lowest when compared with other pollutants) and NO_2 than for PM_{10} is observed.

In a subtropical city such as HK, air pollution has stronger health effects during the cool rather than warm season and that oxidant pollutants are more important indicators of health effects than particulates. Recent research studies suggested that air pollution may show stronger short-term health effects in Hong Kong than those obtained in European cities (Wong et al., 2002a).

In terms of acute morbidity, a recent study has compared directly the effects of air pollution on hospital admissions in Mainland, HKSAR and London (Cohen et al., 2004). Similar associations were observed for PM_{10} and gaseous pollutants and hospital admissions for ischaemic heart disease in both locations, and the associations were strongest during seasons of low humidity in both cities, but no association with admissions for cardiac disease was observed in HK.

Most of the long-term scientific evidences were derived largely from studies in North America and Western Europe, where air pollution derived largely from combustion sources. USEPA identifies

the air quality standards based on these large samples health studies while WHO and Asian countries did not have these very long-term health studies and therefore relied on the US and Western Europe database.

Quantifying the magnitude of these long-term health impacts in other cities presents considerable challenges owing to the limited availability of information on both effects on health and on exposures to air pollution in many parts of the world. Without the local long-term data in hand, it is necessary to refer to the WHO/USEPA recommendations.

US, UK and WHO evaluations show that the effects of long-term PM exposure on mortality (life expectancy) seem to be attributable to $PM_{2.5}$ rather than to coarser particles (Department of Health, 2006; USEPA, 2006; WHO, 2006). The primary, carbon-centred, combustion derived particles have been found to have considerable inflammatory potency. Nitrates, sulphates and chlorides belong to components of PM showing lower toxic potency. It is important to state that there is no threshold level identified for PM in the studies (WHO, 2006). In terms of the long-term exposure effects, evidence from epidemiological studies of associations between long-term exposure to particulate air pollution ($PM_{2.5}$ and sulphate) and sulphur dioxide shows positive and statistically significant associations with a reduction in life expectancy due to increased deaths from cardiovascular rather than respiratory disease, a most important finding (Department of Health, 2006).

The US have recently conducted a systematic human health review from the ozone impact. It is found that the ozone uptake in humans is increased by exposure to NO_2 and SO_2 and decreased during the O_3 exposure. Ozone was generally found to be associated with respiratory hospitalizations and asthma emergency department visits during warm season but not during the cool season. After the adjustment for the effect of co-pollutants, such as PM, there is evidence to support independent O_3 effects on respiratory hospital admissions. Robust associations have been identified between various measures of daily O_3 concentrations and increase risk of mortality. Nonetheless, there is little evidence for a relationship between chronic O_3 exposure and increase risk of lung cancer of mortality. The limited evidence suggests that if a population threshold exists in O_3 health effect, it is likely near the lower limit of ambient O_3 concentration in US. Along with the pathophysiologic understanding of asthma as a chronic inflammatory disease, asthmatics are likely a susceptible population that requires protection from O_3 exposure. The elderly population (above 65 years) appears to be at great risk of O_3-related mortality and hospitalizations compared to all age population (USEPA, 2006).

In latest UK review studies, the associations between both daily and long-term average concentrations of air pollutants and effects on the cardiovascular system are causal, reflected by a variety of outcome measures including risk of death and of hospital admissions (Department of Health, 2006). The public health implication is not as large as those arising from factors such as family history, active smoking and hypertension, but should be taken as precautionary approach in future planning. Recent research findings show no convincing associations between all-cause mortality and nitrogen dioxide, ozone or carbon monoxide (WHO, 2006). The RR for all-cause mortality, cardiopulmonary mortality, lung cancer mortality and all other cause mortality are low.

There is heterogeneity between epidemiological results obtained in differing geographical locations and this should be further reviewed. Design of studies is recommended to separate the effects of different components of traffic-generated pollution, and significant contributions from sources other than vehicles. It is also suggested that further work should use oxides of nitrogen (NOx), a known vasoactive compound, as a better marker for traffic-generated pollutants than NO_2.

In the UK Panel review, studies indicated that people with healthy lungs, whether at rest or exercising, show little response to experimental inhalation of nitrogen dioxide at concentrations well above those occurring in the ambient air, even during extreme pollution episodes. Very small changes in sensitive tests of lung function have been recorded at exposures between 4,688 µg/m3 (2,500 ppb) to 14,063 µg/m3 (7,500 ppb). In people with asthma, some studies have shown changes in these tests of lung function at exposures of around 563 µg/m3 (300 ppb) when the

subjects have been exercising, though other studies have shown no changes at higher concentration (Department of the Environment, 1996).

F1.5 Risk-Concentration Relationships of Air Pollution

Epidemiological studies have been widely adopted to assess the health effects of air pollution. In Hong Kong, many time-series studies have been conducted to establish the relationships between health outcomes, including daily mortality, hospital admissions and general practitioner consultations (Wong et al., 1997; Wong et al., 1998; Wong et al., 1999; Wong et al., 2000; Wong et al., 2001; Wong et al., 2002a, b, c; Wong et al., 2003; Yu et al., 2004; Wong et al., 2006). Time-series approach is the most commonly adopted study design to investigate the short-term effects of air pollution on health. This design specifically caters for matched daily series of exposure and outcome data which aims (Schwartz et al., 1996) to quantify adverse short-term effects of the current levels of air pollutants on health. In the time-series approach, statistical modelling is performed, taking into consideration the characteristic (approximately Poisson) distribution, overdispersion and positive autocorrelation of the outcome data. Daily meteorological variables (temperature and humidity) and others (seasonal changes, holidays, day of the week, time trends) may be included as confounding variables in the statistical model. The health effects of individual pollutants expressed as the relative risks (the ratio of the risk of disease or death among the exposed to the risk among the unexposed) or excess risks (derived from the corresponding relative risk minus one) are then evaluated.

Based on the findings from the recent time-series studies showed that an increase of 10 g/m^3 concentration of pollutants was associated with a 0.6% to 2.1% increase across all disease categories (non-accidental, respiratory, COPD, Cardiovascular, Cardiac, IHD) for NO_2; 1.4% to 3.9% increase across all disease categories for SO_2; 0.2% to 0.9% increase across all disease categories for PM_{10} and with 0.6% to 0.8 increase across all disease categories for O_3 (Wong et al., 2002a). The results for hospital admissions show that, except for asthma, all the criteria pollutants (NO_2, SO_2, PM_{10} and O_3) were associated with increased admissions across all the disease categories. For an increase of 10 g/m^3 concentration, there was a 0.5% to 1.9% increase for NO_2; 0.5% to 2.4% increase for SO_2; 0.4% to 1.0% increase for PM_{10}; and 0.2% to 0.6% increase for O_3. The number of cardiorespiratory deaths (the summation of cardiovascular and respiratory deaths) estimated based on the relative risks per 10 g/m^3 change in concentration of the pollutant which produced greatest effect would be 243 deaths a year (based on SO_2). The number of cardiorespiratory deaths attributed to per 10 g/m^3 change in NO_2, PM_{10} and O_3 concentrations would be 134, 57 and 34, respectively. Other studies on the effects of air pollution on mortality and hospital admissions in Hong Kong also concluded that there is significant association between air pollutant concentrations with mortality and hospital admissions showing the strong short-term effects of air pollutants on human health (Wong et al., 1997; Wong et al., 1998; Wong et al., 2002a).

A time-series study on the effects of air pollution on general practitioner (GP) consultations in Hong Kong by Wong et al. (2003) showed that the excess risks of GP consultations for all respiratory diseases and upper respiratory tract infections (URTI) were highest for NO_2 (at 3.8% and 3.2%, respectively), and lower for O_3 (at 2.9% for all respiratory diseases and 2.5% for URTI) and PM_{10} (at 2.3% for all respiratory diseases and 2.2% for URTI). The results of the studies were compared with similar studies in UK. Both studies showed that there is a significant association between GP consultations for respiratory diseases and air pollutant concentrations (PM_{10}, $PM_{2.5}$, NO_2 and O_3 in Hong Kong, and PM_{10} and SO_2 in UK). Findings in the study and from other earlier Hong Kong studies (Wong et al., 2001; Wong et al., 2002a) provide evidence that air pollution contributes to a significant amount of morbidity for respiratory diseases in the community.

Studies (Wong et al., 2002a; Wong et al., 2003; Hedley et al., 2006) considered that the epidemiological studies in other countries can be applied to Hong Kong. It is recommended that local data would be preferred for assessment of health effects and hence data from studies (Wong et al., 2002a) is adopted. Relative Risk has been used to describe the risk of an event (of

developing a disease) relative to exposure and it is a ratio of the probability of the event occurring in the exposed group versus the non-exposed group. There are many research studies conducted locally on short-term air pollutants exposure, and thus the short-term health risk findings and recommendation are directly applicable to HK condition. The most relevant and recent study conducted by Wong et al. (2002a) will be adopted in the socio-economic analysis to determine the affected population and the respective cost of illness under the various assessment scenarios of the different WHO, AQGs and Interim Target levels in the next Formulation of Recommendation Stage. The relevant RR data for short-term mortality and hospital admission for various pollutants are listed in **Tables F1.8** and **F1.9**.

On the long-term exposure to pollutants, there is a lack of long historical health data and systematic analysis on local data is not feasible. Nonetheless, it is quite clear that industrial (e.g. power plant) and vehicular activities are the dominant sources in HK, PRD and the regions beyond, which are similar to the conditions encountered in the North America and Western Europe of which the WHO studies health database are originated. Cohort studies are well adopted methods to study the long-term health effects of pollution. Several cohort studies have been conducted, mainly in U.S: the Harvard Six Cities study (Dockery et al., 1993), the Seventh Day Adventists Study (Abbey et al., 1999), the American Cancer Society cohort study (Pope et al., 1995) which has been extended in a recent paper (Pope et al., 2002). Recent report on the effect of long-term exposure to air pollution on mortality has studied the scientific evidence on the long-term health effect of pollution (COMEAP, 2007). Findings from the study showed that the current scientific evidence indicated there is strong association between PM, especially $PM_{2.5}$, and mortality. Positive associations between SO_2 and mortality have also been found in many but not all other studies. Moreover, the associations of all-cause mortality with nitrogen dioxide and with carbon monoxide are unconvincing. It has been also showed that the overall evidence is weak for the effect of long-term exposure to ozone on mortality. A paper on Report of a study of cancer risks of diesel particulates in Hong Kong critically reviewed US research findings in this area to determine their applicability for the calculation of cancer cases in Hong Kong (Wong 2000). The paper calculated these cancer cases as a percentage of total related cancer cases and compared the results with those of major US cities. In Hong Kong, the paper estimated that the cumulative number of lung cancer due to diesel exhaust over a life time of 70 years is around 8000 cases.

The RR for long-term mortality conducted by Pope et al. (2002) are commonly adopted by other countries and are therefore proposed for use as the best available substitution in the socio-economic analysis. The relevant data is given in **Table F1.8**.

Table F1.8: Relative risks (RR) and 95% confidence intervals for short-term mortality for 10 μg/m^3 increase in levels of air pollutants [Source: Wong et al. (2002a)]

Pollutants	Non- accidental mortality, all ages	Respiratory mortality, all ages	COPD mortality, all ages	Cardiovascular mortality, all ages	Cardiac mortality, all ages	IHD mortality, all ages
NO_2	1.0064 (1.0036-1.0091)	1.0081 (1.0024-1.0138)	1.0107 (1.0000-1.0215)	1.0094 (1.0044-1.0144)	1.0134 (1.0065-1.0204)	1.0209 (1.0131-1.0288)
SO_2	1.0136 (1.0093-1.0178)	1.0162 (1.0077-1.0248)	1.0247 (1.0074-1.0424)	1.0161 (1.0078-1.0244)	1.0312 (1.0203-1.0423)	1.0389 (1.0261-1.0519)
PM_{10}	1.0024 (1.0001-1.0046)	1.0040 (0.9995-1.0085)	1.0087 (1.0000-1.0174)	1.0037 (0.9997-1.0077)	1.0017 (0.9961-1.0072)	1.0033 (0.9969-1.0097)
O_3	0.9989 (0.9963-1.0016)	1.0062 (1.0009-1.0116)	1.0081 (0.9974-0.0189)	0.9984 (0.9935-1.0033)	0.9981 (0.9912-1.0050)	0.9952 (0.9872-1.0031)

Table F1.9: Relative risks (RR) and 95% confidence intervals for hospital admissions for 10 μg/m^3 increase in levels of air pollutants [Source: Wong et al. (2002a)]

Pollutants	Respiratory, all ages	Respiratory, 65+	Asthma, 15-64 years	Cardiovascular, all ages	Cardiac, all ages	IHD, all ages
NO_2	1.0054 (1.0027-1.0080)	1.0191 (1.0159-1.0223)	1.0077 (0.9990-1.0165)	1.0073 (1.0048-1.0098)	1.0112 (1.0084-1.0140)	1.0078 (1.0035-1.0121)
SO_2	1.0076 (1.0034-1.0118)	1.0242 (1.0192-1.0293)	1.0052 (0.9912-1.0194)	1.0108 (1.0072-1.0144)	1.0154 (1.0111-1.0197)	1.0060 (0.9992-1.0129)
PM_{10}	1.0050 (1.0028-1.0071)	1.0104 (1.0078-1.0130)	0.9949 (0.9877-1.0022)	1.0037 (1.0018-1.0057)	1.0049 (1.0027-1.0072)	1.0057 (1.0022-1.0093)
O_3	1.0055 (1.0031-1.0079)	1.0049 (1.0018-1.0080)	1.0054 (0.9967-1.0143)	1.0024 (1.0001-1.0047)	1.0034 (1.0007-1.0061)	1.0057 (1.0015-1.0100)

Table F1.10: Relative Risks and 95% confidence intervals for long-term mortality for 10μg/m^3 increase in levels of air pollutants [Source: Pope et al. (2002)]

Pollutants	All-cause mortality	Cardio-pulmonary mortality	Lung cancer mortality
$PM_{2.5}$	1.06 (1.02-1.11)	1.09 (1.03-1.16)	1.08 (1.01-1.16)
SO_2	1.03 (1.01-1.04)	-	-

F2 International Approach to Air Quality Management

F2.1 United States

Air quality management in US is stipulated under the Clean Air Act Amendment of 1990 (CAA) which specified provisions to control pollutants that causes acid rain; introduced new operating permit program; and specified program to control pollutants that cause stratospheric ozone depletion. The purposes of the CAA are:

- to protect and enhance the quality of the Nation's air resources so as to promote the public health and welfare and the productive capacity of its population;
- to initiate and accelerate a national research and development program to achieve the prevention and control of air pollution;
- to provide technical and financial assistance to State and local governments in connection with the development and execution of their air pollution prevention and control programs; and
- to encourage and assist the development and operation of regional air pollution prevention and control programs.

In accordance with the CAA, both primary and secondary NAAQS for short-term and long-term exposures are set. The rationales are listed as follows:

- Primary NAAQS shall be ambient air quality standards for the attainment and maintenance, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.
- Secondary NAAQS shall specify a level of air quality for the attainment and maintenance, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.

In order to identify the latest health effect, the US Environmental Protection Agency (USEPA) will complete a thorough review of the criteria and the national ambient air quality standards at five-year intervals. A committee will be established to advise the USEPA of any additional knowledge as required before national ambient air quality standards is revised and on any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such standards.

On top of the NAAQS which are identified based on the health risk studies, the US has also formulated an Air Quality Strategy which stipulates the time frame for implementation. In view of the US practical situation, there were many urban areas that could not attain the NAAQS. Non-attainment areas will be established. The CAA extended the time for states to achieve compliance. Nonetheless, the State required constant progress review on reducing emissions and established provisions for sanctions on the counties that did not meet the conditions. Degrees of severity for non-attainment areas were established for ozone, carbon monoxide, PM_{10} and $PM_{2.5}$. The State Implementation Program requirements for air quality plan will vary in accordance with the degrees of severity.

The USEPA has conducted various health impact studies in the review of NAAQS recently for particulate matter and ozone, and has revised the NAAQS for particulate matter in September 2006, while the revision of NAAQS for ozone is still in progress (USEPA, 2005, 2006, 2007a, 2007b).

Moreover, as part of the NAAQS review process, Criteria Document and Staff Paper are drafted for the pollutant under review. The Criteria Document forms primarily from the scientific assessments on air quality analyses, human health exposure and risk, while the Staff Paper includes the peer-reviewed publications in relation to the pollutant in question in the scientific literatures, the description and evaluation of these studies' methods and findings, and the synthesis of the entire

body of evidence to develop an integrated, generally qualitative assessment designed to inform the central policy-relevant issues.

F2.2 United Kingdom

Air quality management in UK is stipulated under the Environment Act 1995 which requires the UK Government and the devolved administrations for Scotland, Wales and Northern Ireland to produce a national air quality strategy containing standards, objectives and measures for improving ambient air quality and to keep these policies under review. The primary objective is to ensure that all citizens should have access to outdoor air without significant risk to their health, where this is economically and technically feasible. This strategy is based on the AQS recommended by the expert panel at which no significant health effects would be expected in the population as a whole. The AQS, set as the benchmarks for setting objectives, are set purely with regard to scientific and medical evidence on the effects of the particular pollutant on health, or on the wider environment, as minimum or zero risk levels with regard to the short-term and long-term exposures. The AQO in the strategy then aims to move air quality as close to these AQS as possible. This is the same rationale proposed by the WHO AQG global update 2005.

In establishing the AQS, UK has formulated a team of Expert Panel with the aspiration of reviewing the latest health risk finding every 5 years, with particular reference to the levels of airborne pollutants at which no or minimum effects on human health are likely to occur; taking account of the best available evidence of the effects of air pollution on human health; but without reference to the practicality of abatement or mitigation measures, the economic costs and economic benefits of pollution control measures or other factors pertinent to the management rather than the assessment of risk. Where appropriate, for example for pollutants where no threshold for adverse effects can be determined, the Panel shall recommend exposure-response relationships or other information Government might use to set policy objectives. However, in practice only two pollutants, 1,3,-butadiene and particulate matter have been revisited.

The UK Environment Act 1995 requires the air pollution control strategy to include statements on “standards relating to the quality of air”, and “objectives for the restriction of the levels at which particular substances are present in the air”. Standards have been used as benchmarks or reference points for the setting of objectives. Specifically, standards are the concentrations of pollutants in the atmosphere which can broadly be taken to achieve a certain level of environmental quality. The standards are based on assessment of the effects of each pollutant on human health including the effects on sensitive subgroups or on ecosystems. Whereas, objectives are policy targets often expressed as a maximum ambient concentration not to be exceeded, either without exception or with a permitted number of exceedences, within a specified timescale.

The AQS, as the basis for setting objectives, are set purely with regard to scientific and medical evidence on the effects of the particular pollutant on health, or, in the appropriate context, on the wider environment, as minimum or zero risk levels. In the area of the effects on human health this is the approach adopted by the WHO, and by Expert Panel on Air Quality Standards (EPAQS) in the UK who last reported on pollutants of national importance.

The health impact assessments of pollutants are included in the Air Quality Strategy prepared by the Department for Environment Food and Rural Affairs (DEFRA). The marginal change in mortality and hospital admissions relative to the baseline scenario is presented in the assessment (DEFRA, 2007).

F2.3 European Union

The European Union policy on air quality aims to develop and implement appropriate instruments to improve air quality. In 1996 the Air Quality Framework Directive was adopted which established a Community framework for the assessment and management of ambient air quality in the EU. The Framework Directive also provided a list of priority pollutants for which air quality objectives would be established in daughter legislation. There were subsequently four daughter directives in respect

of particular pollutants and a Council Decision to bring about the reciprocal exchange of air quality monitoring information:

- Council Directive 96/62/EC on ambient air quality assessment and management (“Framework Directive"), OJ L 296, 21.11.1996, p.55.
- Council Directive 1999/30/EC relating to limit values for sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter and lead in ambient air, OJ L 163, 29.6.1999, p.41 ("First Daughter Directive").
- Values for benzene and carbon monoxide in ambient air, O.J. L 313, 13.12.2000, p. 12 ("Second Daughter Directive").
- Directive 2002/3/EC of the European Parliament and of the Council relating to ozone in ambient air, OJ L 67, 9.3.2002, p.14 ("Third Daughter Directive").
- Council Decision 97/101/EC establishing a reciprocal exchange of information and data from networks and individual stations measuring ambient air pollution within the member States, OJ L 35, 5.2.1997, p.14 ("Exchange of Information Decision").

There is no legislative requirement to review the EU air quality limits on a regular basis. In May 2008, EU however, has merged and updated the limit values and target values in its Directive 2008/50/EC on ambient air quality and cleaner air for Europe by referencing to the WHO AQGs.

On top of the legislative framework, EU has also organised periodic air pollution control review programme. The Sixth Environment Action Programme (EAP), "Environment 2010: Our future, Our choice", includes Environment and Health as one of the four main target areas where new effort is needed. Air pollution is one of the issues included under Environment and Health. Whilst overall air quality trends in the Community are encouraging, continued efforts and vigilance are still needed. The objective considered in the Sixth Environment Action Programme is to achieve levels of air quality that do not give rise to unacceptable impacts on, and risks to, human health and the environment. The Community is acting at many levels to reduce exposure to air pollution: through EC legislation, through work at the wider international level in order to reduce cross-border pollution, through working with sectors responsible for air pollution and with national, regional authorities and NGOs, and through research. The focus for the next ten years will be implementation of air quality limits and coherency of all air legislation and related policy initiatives.

The EU is also implementing the Clean Air for Europe (CAFE) which is a programme of technical analysis and policy development that underpinned the development of the Thematic Strategy on Air Pollution under the Sixth Environmental Action Programme for the following objectives:

- to develop, collect and validate scientific information relating to the effects of outdoor air pollution, emission inventories, air quality assessment, emission and air quality projections, cost-effectiveness studies and integrated assessment modeling, leading to the development and updating of air quality and deposition objectives and indicators and identification of the measures required to reduce emissions;
- to support the implementation and review the effectiveness of existing legislation, in particular the air quality daughter directives, the decision on exchange of information, and national emission ceilings as set out in recent legislation, to contribute to the review of international protocols, and to develop new proposals as and when necessary;
- to ensure that the sectoral measures that will be needed to achieve air quality and deposition objectives cost-effectively are taken at the relevant level through the development of effective structural links with sectoral policies;
- to determine an overall, integrated strategy at regular intervals which defines appropriate air quality objectives for the future and cost-effective measures for meeting those objectives;

- to disseminate widely the technical and policy information arising from implementation of the programme.

In collaboration with WHO, EU has formulated air quality limit based on the health risk findings. The Thematic Strategy and the CAFE Directive (SEC(2005) 1133) aims to develop a long-term, strategic and integrated policy advice to protect against significant negative effects of air pollution on human health and the environment. In addition, priorities for future action will be set; existing ambient air quality legislation and the National Emission Ceilings Directive will be reviewed to reach the long-term environmental objectives. Apart from the significant negative impacts on and risks to human health and the environment, the strategy includes no exceedence of critical loads and levels for natural ecosystems.

There are local components and transboundary contributions to observed effects. Several pollutants contribute to the same or multiple effects and pollutants interact. Moreover, there are prominent synergies and tensions between air pollution and other environmental problems such as climate change. These issues are addressed in a systematic and cross-cutting way so that benefits can be maximised. The Strategy on air pollution is built upon an integrated assessment of different environmental and health effects and aims to provide the most cost-effective solution for the chosen level of objectives. It is considered that economic, social and environmental dimensions should be considered in an integrated and balanced manner.

F2.4 Mainland

Air quality management in Mainland is stipulated under the Law of the People's Republic of China on the Prevention and Control of Atmospheric Pollution. This law was formulated for the purpose of preventing and controlling atmospheric pollution, protecting and improving people's environment and the ecological environment, safeguarding human health, and promoting the sustainable development of economy and society. The State Council and the local people's governments at various levels must incorporate the protection of the atmospheric environment into their national economic and social development plans, make rational plans for the distribution of industrial layout, strengthen the scientific research on the prevention and control of atmospheric pollution, adopt preventive and curative measures against atmospheric pollution, and protect and improve the atmospheric environment. The State takes measures to control or gradually reduce, in a planned way, the total amount of the main atmospheric pollutants discharged in local areas. The local people's governments at various levels shall be responsible for the quality of the atmospheric environment under their own jurisdictions, making plans and taking measures to make the quality of the atmospheric environment under their own jurisdictions meet the prescribed standard. State Environmental Protection Agency (SEPA) will review the air pollution status and prepares control plan every 5 years on the overall environmental control strategy and time frame. Environmental goals in the 5-year plans provided impetus for forced shutdown or relocation of urban factories, funding for installation of more advanced treatment, and justification for a raft of environmental laws and regulations.

The latest ambient AQS were issued by environmental authorities in 1996 under GB 3095-1996 “Ambient Air Quality Standard”. The rationale to derive these AQS is to improve the air quality, maintain clean and sustainable environment, prevention of damage of ecological system and protection of public health on human, and encourage on business development. Regarding Level 2 of the AQS, the criteria are specifically aiming to protect human health, and plant and animal in both villages and cities. These take into consideration on the potential impacts from the long-term and short-term exposure.

There are some systematic health risk studies conducted in collaboration with various local and overseas universities, World Bank, National Institute of Health, National Institute of Environmental Health Sciences and USEPA. Relevant documents can be found in World Bank documentations, international research papers and local medical journals. Nonetheless, there is no official documentation on findings of the various health risk analysis approach.

F2.5 Australia

On 26 June 1998, the Australia's National Environment Protection Council (NEPC) made Australia's first set of National Environment Protection Measure for Ambient Air Quality (the 'Air NEPM'). The NEPC is its statutory body with law making powers established under the National Environment Protection Council Act 1994 (Commonwealth) and corresponding legislation in the other jurisdictions.

The desired environmental outcome of Air NEPM is ambient air quality that allows for the adequate protection of human health and well-being. As stated in the "National Environment Protection (Ambient Air Quality) Measure – Revised Impact Statement, 1998", the NEPC will, in setting air quality standards the NEPC has examined the latest health related air pollution research from around the world, examined the information available on the current state of the major airsheds and, taking into account the technology that is readily available, assessed what level of air quality we could achieve within ten years, without significant social and economic disturbance.

On top of the NAQS which aim for the protection of human health and well-being, the State Environment Protection Policy (Ambient Air Quality) legislation (SEPP) sets out the attainment program for achieving the Air NEPM in accordance with their local condition. Additional monitoring and reporting protocols for six common pollutants plus visibility are also specified.

F2.6 Hong Kong

The Air Pollution Control Ordinance (APCO) (Cap 311) is the principal legislation for air quality management. The Ordinance covers specific areas related to air pollution emissions from power plant, motor vehicle, and major industrial (Specified Process). Air pollution emissions from vessels, motor vehicles, railways locomotive and aircraft are excluded from the APCO.

The Environmental Protection Department of HKSARG has defined its overall policy objective for air quality management in Hong Kong as to achieve and maintain an acceptable level of air quality to safeguard the health and well being of the community, and to promote the conservation and best use of air in the public interest. Air Quality Objectives (AQO) for seven widespread air pollutants, i.e. SO_2, NO_2, CO, O_3, lead, TSP and PM_{10} were established in 1987. These standards were derived from scientific analyses of the relationship between pollutant concentrations in air and the associated adverse effects of the polluted air on the health of the public, mainly from reference to US researches. There is no requirement for regular review of the air quality objectives.

There are many systematic health risk studies initiated by EPD of HKSAR and various universities in HK, some of the relevant large scale studies [Ref 53-63]. These studies have been promoting a revision of the AQO in light of the latest research findings on health impact. They also established the relative risks associated with mortality and hospital admissions for the different air pollutants.

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