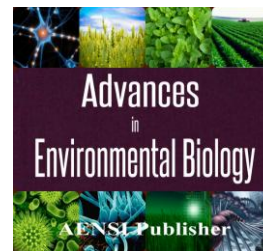




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### A Global Challenge of Air Pollution and Public Health: A Mini Review

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#### ABSTRACT

Economic development, urbanization, transportation and rapid population growth are major driving forces of air pollution all over the globe. In contrast to developed countries, air pollution levels in developing countries and in countries in transition, are still at relatively high levels, due to economic development. "Air pollution and population health" is one of the most important environmental and public health issues. In recent years, several hundred epidemiological studies have emerged showing adverse health effects associated with short-term and long-term exposure to air pollutants. Time-series studies conducted in Asian cities also showed similar health effects on mortality associated with exposure to particulate matter (PM), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>) and ozone (O<sub>3</sub>) to those explored in Europe and North America. The World Health Organization (WHO) published the "WHO Air Quality Guidelines (AQGs), Global Update" in 2006. These updated AQGs provide much stricter guidelines for gaseous and particulate pollutants. Considering that current air pollution levels are much higher than the WHO-recommended AQGs, interim targets for these four air pollutants are also recommended for member states, especially for developing countries in setting their country-specific air quality standards. In conclusion, ambient air pollution is a health hazard. It is more important in Asian developing countries within the context of pollution level and population density. Improving air quality has substantial, measurable and important public health benefits.

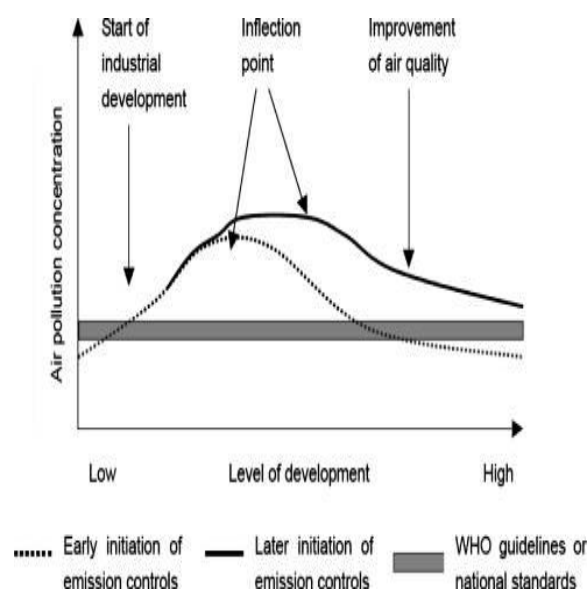
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#### INTRODUCTION

Clean air is one of the basic requirements of human health and well-being. However, during the process of economic development, air pollution has been and continues to be a significant health hazard worldwide. The driving forces of air pollution include economic development, urbanization, energy consumption, transportation and motorization, as well as increase of urban population. Exposure to air pollutants is a problem of increasing concern due to the diversity of the pollutants in question, adverse effects observed in a broad range of air pollution levels, and the vast number of people at risk. The effects of air pollution can sometimes be observed even when the pollution level is below the level indicated by air quality guidelines. Individuals differ widely in genetic predisposition and physiological response to pollutants. Young children, the elderly, persons with predisposed diseases, such as cardiovascular and pulmonary diseases, and workers in certain industries may be at a higher risk owing to their increased biological sensitivities and different exposure patterns. The most common ambient air pollutants encountered in our daily life are particulate matter (PM), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), ozone (O<sub>3</sub>), carbon monoxide (CO), and carbon dioxide (CO<sub>2</sub>). The environmental Kuznets curve (EKC) can be used to study the relationship between economic development and air pollution levels. During the early stage of economic development, air pollution level is generally low. However, when economic development reaches an intermediate stage, air pollution concentration levels tend to increase appreciably or even rise sharply if no effective ameliorating measures are taken. It might then reach an inflexion point later at a higher development stage due to better environmental awareness and relevant control measures taken in protecting the environment (Fig. 1). As zero risk is neither practical nor necessary, it is crucial to set appropriate air pollutant guidelines for air pollution management to meet.

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**Fig. 1:** Environmental Kuznets curve (EKC).

*Global trend in air pollution level:*

Particulate matter (PM), SO<sub>2</sub>, NO<sub>2</sub> and O<sub>3</sub> are considered classical/traditional air pollutants, and commonly used as indicator pollutants for fuel combustion and traffic-related air pollution. In the middle of the twentieth century, total suspended particulate (TSP) levels were very high in some large cities. For instance, during the London episode in 1952, ambient TSP and SO<sub>2</sub> levels reached several thousands of micrograms per cubic meter (µg/m<sup>3</sup>); [2].

In Shenyang, a heavy-industry city in north-eastern China, TSP and SO<sub>2</sub> levels were in the range of hundreds to thousands of µg/m<sup>3</sup> [3].

After decades of effort in air pollution management control, air pollutant levels in most developed countries have been decreasing dramatically. However, in many developing countries, as well as countries in transition, though air pollution levels have been slightly decreasing or has remained stable, they are still higher than those in developed countries. The World Health Organization has summarized the annual average concentrations of particulate matter less than 10 µm in aerodynamic diameter (PM<sub>10</sub>), NO<sub>2</sub> and SO<sub>2</sub>, as well as the 1 h average maximum concentrations of O<sub>3</sub> for different regions in the world (Table 1). In most European and North American cities, the PM<sub>10</sub> annual average concentration levels are generally lower than 50 µg/m<sup>3</sup>. The highest levels of PM<sub>10</sub> are found in Asia, Africa and Latin America. In Asia, PM is still the major and most important air pollutant, though in some large cities, such as certain cities in China, a slight decrease in PM<sub>10</sub> levels has been noted during the economic development over the last few decades [4].

**Table 1:** Ranges of annual average concentrations of PM<sub>10</sub>, NO<sub>2</sub>, SO<sub>2</sub> and 1 h average concentrations of ozone, based on selection of urban data (µg/m<sup>3</sup>).

Region	Annual average concentration			Ozone (1 h max. concentration)
	PM <sub>10</sub>	NO <sub>2</sub>	SO <sub>2</sub>	
Africa	40–150	35–65	10–100	120–300
Asia	35–220	20–75	6–65	100–250
Australia/New Zealand	28–127	11–28	3–17	120–310
Canada/United States	20–60	35–70	9–35	150–380
Europe	20–70	18–57	8–36	150–350
Latin America	30–129	30–82	40–70	200–600

SO<sub>2</sub> levels have been decreasing in most parts of the world. They have declined substantially in the United States and Europe in particular. In some Asian cities, (e.g., Bangkok, New Delhi, and Jakarta), the ambient SO<sub>2</sub> levels are low due to the low sulfur content of the fuel used there. On the other hand, in Chinese cities, although the SO<sub>2</sub> level has declined quite substantially, it is still relatively high. In larger cities in Latin America and Africa, there has also been a moderate decline in SO<sub>2</sub> levels [3]. On the other hand, such a tendency has not been observed for traffic-related air pollutants, i.e., NO<sub>2</sub> and O<sub>3</sub>. To the contrary, in countries in transition, levels of NO<sub>2</sub> and O<sub>3</sub> tend to increase due to the increased number of motor vehicles. Megacities where annual average NO<sub>2</sub> exceeded the WHO air quality criteria of 40 µg/m<sup>3</sup> were Beijing, Shanghai, Tokyo, Osaka, New York, Los Angeles, San Paulo and Mexico, while in New Delhi, Mumbai and Calcutta the annual average NO<sub>2</sub> concentrations were lower than 40 µg/m<sup>3</sup> [5].

Ozone and some of its precursors are transported long distances and across borders (transboundary movements) in the atmosphere, and so they can be considered to be a regional and even a global problem. With regard to its adverse health effects, atmospheric ozone concentrations are usually assessed as 1 h maximum or maximum 8 h average concentrations because they are closely associated with sunlight. The highest levels of ozone and NO<sub>2</sub> are found in Latin America and in some large cities of other developed countries.

*Adverse health effects associated with exposure to air pollution:*

Exposure to ambient air pollution has been associated with a series of adverse health effects, ranging from subclinical effects, physiological changes in pulmonary functions and the cardiovascular system, to clinical symptoms, outpatient and emergency-room visits, hospital admissions, and finally to premature death. Most of the recent evidence focuses on respiratory and cardiovascular effects associated with exposure to short-term and long-term exposure to air pollution. High-risk subgroups include young children, the elderly, persons with predisposed diseases, and persons with low socioeconomic status (SES). The increased risk of air pollution-related health effects seems to be relatively low, generally in the range of only a few percent. However, the total number of people affected worldwide is quite significant. More importantly, most state-of-the-art epidemiological studies using sophisticated statistical tools have shown that the pollutant concentration associated with increased risk is quite low. In other words, even when the WHO 2000 Air Quality Guidelines and/or individual country-specific air quality standards are met, the increased health risk still exists.

*Health effects of short-term exposure to air pollutants:*

Epidemiological studies are generally not available for most environmental chemicals. However, in recent years many epidemiological studies, time-series studies in particular, have emerged that show statistically significant associations between exposure to classical air pollutants and adverse health effects including mortality and morbidity. Nevertheless, more evidence is needed to establish a causal relationship, and the underlying mechanism needs to be further explored.

The epidemiological studies used to explore the relationship between changes in air pollution levels over the short term and changes in various health endpoints are time-series studies, panel studies and case-crossover studies. In time-series studies, repeated observations of exposure and health outcomes (daily mortality or morbidity) are made over time within the same study population in a geographically defined area. The analysis centers on comparing variations in exposure status over time with changes in health outcomes over time. A time-series study based on aggregate data is essentially a temporal comparison study that examines an association between a variable exposure and a variable health outcome. Since observations are made within the same population, the influence of many confounding factors can thus be avoided. Access to air pollution data and death records and other health data has been increasing in many cities worldwide, so hundreds of time-series studies exploring short-term exposure effects of air pollution have emerged.

*PM:*

Many time-series studies have explored the acute health effects associated with short-term exposure to airborne particulates [6]

PM<sub>10</sub> is used as an indicator for airborne particulates as there are extensive monitoring data for PM<sub>10</sub> throughout the world. Substantial evidence shows that PM exposure is linked to a variety of adverse effects on mortality (nonaccidental all-cause mortality, cardiovascular and respiratory mortality) and morbidity (hospital admissions, outpatient and emergency-room visits, asthma attacks, acute respiratory infection of young children, etc.). Risk for acute events, including myocardial infarction and stroke, has been assessed [7 – 9]

The evidence of airborne PM and adverse health effects is consistent in various cities, both in developed and developing countries. Most time-series studies show a positive association between PM concentration and an increased risk for total and cause-specific mortality. A WHO task group summarized the relative risk estimates (and 95% confidence interval) for a 10 µg/m<sup>3</sup> PM<sub>10</sub> increase for all-cause mortality of all ages, for respiratory mortality of all ages, and for cardiovascular mortality of all ages. They are 1.006 (1.004–1.008), 1.013 (1.005–1.020), and 1.009 (1.005–1.013), respectively. It can be seen that the risk estimates for respiratory and cardiovascular mortality are larger than that for all-cause mortality. These summarized data were based on the results for 33, 18 and 17 European cities [10] In another meta-analysis, an increase of 10 µg/m<sup>3</sup> PM<sub>10</sub> is associated with an increase in all-cause mortality of 0.46, 0.62, and 0.49% in the United States (30 city studies), Europe (21 city studies), and Asia (4 city studies), respectively [ 11].

In some studies positive associations between fine particles (PM<sub>2.5</sub>) and daily mortality were not observed. A study conducted in a district of Chongqing in China showed no positive association between daily ambient PM<sub>2.5</sub> concentration and daily mortality. However, a positive association was found between daily ambient SO<sub>2</sub> concentration and daily mortality, especially respiratory and cardiovascular mortality. When PM<sub>2.5</sub> was controlled in the model, the association found between daily ambient SO<sub>2</sub> and mortality remained unchanged [12].

Most of the time-series studies have shown an increased risk with exposure to air pollution. However, associations observed in time-series studies could only reflect the situation that occurs just a short period prior to the time of death, and mostly among elderly people already suffering from respiratory or cardiovascular diseases. Therefore the association observed in time-series studies may not necessarily imply a significant health effect for the total population. Nevertheless, there is little evidence to suggest a threshold below which no adverse health effects would be anticipated.

In comparison with studies on mortality, there are fewer epidemiological studies on the association between morbidity and ambient air pollution levels. Hwang *et al.* studied the effects of air pollution on daily clinic visits for lower respiratory tract illness in Taiwan during 1998. The study included 50 townships and city districts in Taiwan where ambient air monitoring stations of the Taiwan Air Quality Monitoring Network are located. It was found that PM<sub>10</sub>, NO<sub>2</sub>, CO, and SO<sub>2</sub> showed significant effects on daily clinic visits due to lower respiratory tract illness including acute bronchitis, acute bronchiolitis and pneumonia. In contrast, daily clinic visit rates were found not associated with maximum hourly ozone levels. People aged 65 years and over are more susceptible to the effects of PM<sub>10</sub> than other age groups [13]. In another paper, Hwang *et al.* reported that NO<sub>2</sub> exposure was related to increased schoolchildren absence due to respiratory illness in the subsequent three days [14].

In order to better understand the underlying mechanism of adverse effects associated with exposure to air pollutants found from many epidemiological studies, several plausible mechanistic pathways have been described [15].

Toxicological studies have shown that airborne particulates exert their effects on health mainly through inflammatory and oxidative stress-related processes. PM may have direct effects on the respiratory tract, including production of an inflammatory response, exacerbation of existing airway disease or impairment of pulmonary defense mechanisms. Inflammation is an important mechanism for producing many of the health effects of PM. Inhaled PM may increase production of antigen-specific immunoglobulins, alter airway reactivity to antigen, or enhance susceptibility of the lungs to microbial infection. There are also extrapulmonary effects of PM. One potential pathway is via systemic transport of cytokines produced in the lungs by an inflammatory response. Another potential pathway is through unfavorable effects on coagulation properties that lead to increased risk of stroke or myocardial infarction. There is also the possibility that PM may have a direct effect on the heart, resulting in changes in blood pressure, heart rate, and heart rate variability.

#### SO<sub>2</sub> :

As mentioned above, many time-series studies have been conducted to explore the association between PM and daily mortality and morbidity; about 60% of these studies also examined the health impacts of SO<sub>2</sub>. Associations between SO<sub>2</sub> exposure and daily mortality (including all-cause, cardiovascular and pulmonary mortality) were found in most of these studies, but the consistency of association between SO<sub>2</sub> exposure with daily mortality appeared to be less than that for PM. On the other hand, some studies found that the association of SO<sub>2</sub> and mortality was stronger than that of PM [16]. Some researchers argued that SO<sub>2</sub> might serve as a “surrogate” for urban air pollution from fossil fuel combustion [17].

Associations between emergency hospital admissions for asthma and SO<sub>2</sub> have been reported in some studies, but not in others [18]. Likewise, associations between hospital admissions for chronic obstructive pulmonary disease (COPD) and SO<sub>2</sub> were found to be significantly positive in some studies, but not in others [19].

Association between SO<sub>2</sub> and cardiac disease hospital admissions was found both in London and Hong Kong despite their differences in climate and ethnicity<sup>20</sup>. A meta-analysis analyzed the time-series studies performed in Asia on SO<sub>2</sub> and respiratory and cardiovascular hospital admissions, in which positive associations were also found<sup>11</sup>.

However, it could not be concluded whether SO<sub>2</sub> per se is positively correlated with hospital admissions or acts as a surrogate for a mixture of urban air pollutants.

A time-series analysis comparing daily rates of SIDS (sudden infant death syndrome) and daily concentrations of air pollution during a 16-year period was conducted in 12 Canadian cities. The results showed that ambient SO<sub>2</sub> and NO<sub>2</sub> might be important risk factors for SIDS<sup>21</sup>.

#### NO<sub>2</sub> :

Many time-series studies have been conducted to explore the association between NO<sub>2</sub> exposure and daily mortality. NO<sub>2</sub> daily concentrations are found to be significantly associated with increased all-cause, cardiovascular and respiratory mortality. A meta-analysis on daily mortality and 24 h NO<sub>2</sub> levels (20.4–103.3 µg/m<sup>3</sup>) indicated that the overall effect estimate from the single pollutant model for all-cause mortality was 2.8% per 45 µg/m<sup>3</sup> increase of NO<sub>2</sub>, which fell to 0.9% in multipollutant models, including particles [22]. The European APHEA-1 (Air Pollution and Health, a European Approach) study found a 1.3% increase in daily deaths (95% CI 0.9–1.8) per 50 µg/m<sup>3</sup> increase of NO<sub>2</sub> (1 h maximum) [23].

The effect remained statistically significant after adjusting for black smoke. The APHEA-2 study found that PM effects on daily mortality were stronger in areas with high levels of NO<sub>2</sub> [24].

The US National Morbidity and Mortality Air Pollution Study (NMMAPS) showed that daily mortality increased from 0.3 to about 0.4 % per 10 ppb (18.8 µg/m<sup>3</sup>) increase of NO<sub>2</sub>. Although a causal association cannot be indicated, short-term variations of NO<sub>2</sub> clearly predict an increase in daily mortality [25 – 26]

The results of most of the time-series studies on NO<sub>2</sub> and hospital admissions/emergency room visits for respiratory and cardiovascular diseases as well as doctor visits for asthma in children show an independent NO<sub>2</sub> effect. Controlling for other pollutants lowers the effect estimates at times, and at other times makes them statistically insignificant<sup>27</sup>.

In some studies, NO<sub>2</sub>, rather than PM, was found to be associated with asthma hospital admissions [28]. An effect of NO<sub>2</sub> has been noted in most panel studies evaluating aggravation of asthma in children, showing a clear effect of NO<sub>2</sub> on incidence of viral infections among asthmatics<sup>29</sup>. Health risks from nitrogen oxides may result from NO<sub>2</sub> per se or its products, including O<sub>3</sub> and secondary fine particles. It is difficult to determine whether the independent effects observed for NO<sub>2</sub> and PM are really effects of the gaseous pollutant NO<sub>2</sub>, or independent effects of regionally transported particles and locally produced fine and ultrafine particles. However, NO<sub>2</sub> levels are generally considered a reasonable indicator of exposure to traffic-related emissions.

### O<sub>3</sub> :

Combined evidence from time-series studies show positive associations between daily mortality and ozone levels, independent of the effects of particulate matter. Risk estimates on ozone related mortality are higher in the warmer season. Temperature plays a significant role in the magnitude of the coefficients<sup>30–32</sup>.

A meta-analysis on 95 US urban communities studies showed that a 20 µg/m<sup>3</sup> increase in ozone was associated with a 0.52% increase in total mortality and a 0.64% increase in cardiovascular and respiratory mortality [33]

In another meta-analysis of 144 effect estimates from 39 time-series studies, a 10 µg/m<sup>3</sup> increase in ozone was associated with a 0.87% (95% CI 0.55–1.18) increase in all-cause mortality for all seasons and a 1.34% increase (95% CI 0.45–3.17) in the warmer season. For cardiovascular disease, the same increment of ozone was associated with a 1.11% (95% CI 0.68–1.53) increase during all seasons and a 2.45% increase (95% CI 0.88–4.1) in the warmer season [

Ito et al. [32] conducted a meta-analysis of 43 studies; each 20 µg/m<sup>3</sup> increase in 1 h maximum ozone was associated with a 0.39% increase (95% CI 0.26–0.51) in all-cause mortality. There was no appreciable modification by including PM as co pollutant in the models. Effects were larger for the warmer season.

In Asia, ozone associated with mortality due to stroke was reported in Seoul; an increase of 34 µg/m<sup>3</sup> ozone had a RR 1.06 (95% CI 1.02–1.10) on stroke mortality [34]

In a Chinese study, a significant effect of ozone on daily mortality was found in Shanghai; furthermore, O<sub>3</sub> pollution was found to have stronger health effects in the cold than in the warm season in the city [35]

There is no clear evidence of a threshold for ozone. Time-series studies have shown effects at ozone concentrations as low as 75 µg/m<sup>3</sup>(1 h mean) [31]

Combined evidence shows that ozone exposure is significantly associated with increase in morbidity. The most common health end-points are school absenteeism, hospital or emergency room admissions for asthma, respiratory infections, and exacerbation of chronic airway diseases. Children, elderly people, asthmatics and those with chronic obstructive airway diseases are more sensitive to ozone exposure. Effects of ozone on respiratory hospital admissions seem stronger during the warmer season.

### *Health effects of long-term exposure to air pollutants:*

There was evidence showing chronic adverse health effects associated with long-term exposure to air pollution, especially PM. Two long-term exposure studies, i.e., the US American Cancer Society (ACS) study and Harvard Six-City cohort study, reported associations between long-term exposure to PM<sub>2.5</sub> and mortality. In the ACS Cancer Prevention Study II (CPS-II), a prospective cohort study, 500,000 adults linked with air pollution data from 1982 to 1998 were followed. Confounders including cigarette smoking, BMI, diet, occupational exposure, age, sex, race, education, and alcohol were controlled. A 10 µg/m<sup>3</sup> increase of PM<sub>2.5</sub> was associated with approximately 4, 6 and 8% increases in the risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Coarse particle fraction (PM<sub>2.5–15</sub>) and TSP (total suspended particulates) were not consistently associated with mortality. Thresholds were not apparent in these studies. However, a PM<sub>2.5</sub> annual mean level of 10 µg/m<sup>3</sup> is found to be the lowest level at which all-cause, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to PM<sub>2.5</sub>. Although adverse health effects cannot be entirely ruled out, even at such low level, these levels are expected to effectively reduce the health risks [36]

The US Harvard six-city prospective cohort study showed that mortality rates were most strongly associated with cigarette smoking. After adjusting for smoking and other risk factors, statistically significant associations

between air pollution and mortality were observed. The adjusted mortality rate ratio for the most polluted cities as compared with the least polluted cities was 1.26 (95% CI 1.08–1.47). Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not with death from other causes. Mortality was most strongly associated with fine particulates, including sulfates. It was thus concluded that long-term exposure to fine particles (PM<sub>2.5</sub>) and sulfur oxide-related air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

Long-term exposure to fine particles (PM<sub>2.5</sub>) has been linked to incidence of cardiovascular disease and death among postmenopausal women. The authors studied 65,893 postmenopausal women without previous cardiovascular disease in 36 US metropolitan areas from 1994 to 1998, with a median follow-up of six years. Hazard ratios were estimated for the first cardiovascular event, adjusting for age, race or ethnic group, smoking status, educational level, household income, body-mass index, diabetes, and hypercholesterolemia. A total of 1,816 women had one or more fatal or nonfatal cardiovascular events, including death from coronary heart disease or cerebrovascular disease, coronary revascularization, myocardial infarction, and stroke. Each increase of 10 µg/m<sup>3</sup> of PM<sub>2.5</sub> was associated with a 24% increase in the risk of a cardiovascular event (hazard ratio 1.24; 95% CI 1.09–1.41) and a 76% increase in the risk of death from cardiovascular disease (hazard ratio 1.76; 95% CI 1.25–2.47). The risk of cerebrovascular events was also associated with increased levels of PM<sub>2.5</sub> (hazard ratio 1.35; 95% CI 1.08–1.68).

#### *Causal evidence from intervention study:*

Numerous studies have shown evidence for the association of air pollution with increased daily death. However, there is little direct evidence that diminished particulate or gaseous air pollution levels could lead to reductions in death rates or increase in life-expectancy. In other words, it shows an association rather than a causal relation in the context of air pollution level and death rates. Two citywide air pollution regulations were put into practice during a short period in Hong Kong and Dublin. The direct and immediate health benefit of decreased air pollution level provides convincing evidence supporting a causal relationship between air pollution level and death rates.

The Irish Government banned the sale of bituminous coal within the city of Dublin on 1 September 1990, as Dublin's air quality deteriorated in the 1980s after using cheaper bituminous coal for domestic heating. After the ban on coal sales, average black smoke concentrations in Dublin declined by 35.6 µg/m<sup>3</sup> (70%). Non-trauma, respiratory, and cardiovascular death rates were compared for 72 months before and after the ban of coal sales. Adjusted non-trauma death rates decreased by 5.7% (95% CI 4–7,  $P < 0.0001$ ), respiratory deaths by 15.5% (12–19,  $P < 0.0001$ ), and cardiovascular deaths by 10.3% (8–13,  $P < 0.0001$ ). About 116 fewer respiratory deaths and 243 fewer cardiovascular deaths were seen per year in Dublin after the ban [39]

In Hong Kong, the sulfur content of fuels used by all power plants and vehicles was reduced to 0.5% over a weekend in July 1990. The ambient air SO<sub>2</sub> levels declined from 44 to 21 µg/m<sup>3</sup>, about 50% decrease, while PM<sub>10</sub>, NO<sub>2</sub> and ozone levels did not change. The decreased SO<sub>2</sub> level in ambient air is associated with a reduction in number of deaths among people residing there. All-cause mortality decreased by 2.1% (about 600 deaths per year associated with 10,268 person-years of life per year), respiratory disease mortality decreased by 3.9%, and cardiovascular disease mortality decreased by 2.0%. Average gain in life expectancy for females per year of exposure to the decreased SO<sub>2</sub> level was 20 days, for males 41 days. In this intervention study it was found that SO<sub>2</sub> exposure was consistently associated with mortality, while PM<sub>10</sub> exposure was only marginally associated with mortality [39]

In these two intervention studies the reduced black smoke and SO<sub>2</sub> levels leading to reductions in daily mortality supported a causal relationship between air pollutant exposure and daily mortality.

#### *WHO Air Quality Guidelines:*

The WHO Air Quality Guidelines are an international reference on the adverse effects of exposure to air pollutants on human health. WHO summarized the scientific knowledge on health hazards related to air pollutants, providing risk estimates for exposure to air pollutants and recommending air quality guidelines for member states to develop their own national air quality standards. The first edition of the WHO Air Quality Guidelines for Europe (WHO AQG) was published in 1987, and was updated in 2000. As numerous new data have emerged in recent years, it was decided to revise and update the second version. The latest global updated version of the WHO AQG was published in 2006 (Table 2) [40]

This updated AQG focused on four classical air pollutants, namely particulate matter, ozone, nitrogen dioxide and sulfur dioxide. As numerous data have emerged recently that show that even at low concentration levels adverse health effects still exist and that no clear threshold value can be established, in order to protect human health from air pollution the updated air quality guidelines are much stricter than those recommended in the second version, published in 2000. Considering the actual high air pollution levels in most developing countries, it is not feasible, indeed virtually impossible, to meet the strict criteria. Thus the WHO further provides interim targets for these four pollutants for decision makers of member states so that they can set their

own goals for controlling air pollution in order to protect human health to some extent at various development stages. Meantime, it was emphasized that the updated AQG suits every person in every country

#### Conclusion:

In conclusion, ambient air pollution is a health hazard. It is a global challenge, as evidence shows that adverse effects still exist even at relatively low air pollutant concentrations, and so no threshold values for classical air pollutants can be established based on the available data. It is more important in Asian developing countries due to the severe pollution levels and high population densities associated with them. Improving air quality has substantial, measurable and important public health benefits. Efforts should be made and goals set in order to control air pollution in every country.

**Table 2:** Global updated WHO Air Quality Guidelines.

Pollutants	Averaging time	AQG values ( $\mu\text{g}/\text{m}^3$ )
Particulate matter		
PM <sub>2.5</sub>	1 year	10
	24 h (99th percentile)	25
PM <sub>10</sub>	1 year	20
	24 h (99th percentile)	50
Ozone O <sub>3</sub>	8 h daily maximum	100
Nitrogen dioxide NO <sub>2</sub>	1 year	40
	1 h	100
Sulfur dioxide SO <sub>2</sub>	24 h	20
	10 min	500

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