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WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide

Global update 2005

Summary of risk assessment

World Health Organization

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Contents

Preface

Clean air is considered to be a basic requirement of human health and well-being. However, air pollution continues to pose a significant threat to health worldwide. According to a WHO assessment of the burden of disease due to air pollution, more than 2 million premature deaths each year can be attributed to the effects of urban outdoor air pollution and indoor air pollution (caused by the burning of solid fuels). More than half of this disease burden is borne by the populations of developing countries[1].

The WHO air quality guidelines are designed to offer guidance in reducing the health impacts of air pollution. First produced in 1987[2] and updated in 1997,[3] these guidelines are based on expert evaluation of current scientific evidence. Given the wealth of new studies on the health effects of air pollution that have been published in the scientific literature since the completion of the second edition of the *Air quality Guidelines for Europe*, including important new research from low-and middle-income countries where air pollution levels are at their highest, WHO has undertaken to review the accumulated scientific evidence and to consider its implications for its air quality guidelines. The result of this work is presented in this document in the form of revised guideline values for selected air pollutants, which are applicable across all WHO regions. These guidelines are intended to inform policy-makers and to provide appropriate targets for a broad range of policy options for air quality management in different parts of the world.

The new information included in this latest update of the *Air quality guidelines* relate to four common air pollutants: particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2). The scope of this review reflects the availability of new evidence on the health effects of these pollutants and their relative importance with regard to current and future health effects of air pollution in each of the WHO regions. For air pollutants not considered in the present document the conclusions presented in the WHO *Air quality guidelines for Europe*[3] remain in effect.

The process leading to the present revision of the air quality guidelines is summarized in the report of the WHO Working Group Meeting, which convened in Bonn, 18–20 October 2005[4]. This report lists the members of the Working Group who reviewed the available evidence and who recommended the guideline values presented here. A full report, to include a detailed assessment of the available scientific evidence, as well as the revised introductory chapters of the WHO *Air quality guidelines* will be published later in 2006.

[1] World health report 2002. Reducing risks, promoting healthy life. Geneva, World Health Organization, 2002.

[2] Air quality guidelines for Europe. Copenhagen, World Health Organization Regional Office for Europe, 1987 (WHO Regional Publications, European Series, No. 23).

[3] Air quality guidelines for Europe, 2nd ed. Copenhagen, World Health Organization Regional Office for Europe, 2000 (WHO Regional Publications, European Series, No. 91).

[4] Available at http://www.euro.who.int/Document/E87950.pdf.

Role of the guidelines in protecting public health

The WHO air quality guidelines (AQGs) are intended for worldwide use but have been developed to support actions to achieve air quality that protects public health in different contexts. Air quality standards, on the other hand, are set by each country to protect the public health of their citizens and as such are an important component of national risk management and environmental policies. National standards will vary according to the approach adopted for balancing health risks, technological feasibility, economic considerations and various other political and social factors, which in turn will depend on, among other things, the level of development and national capability in air quality management. The guideline values recommended by WHO acknowledge this heterogeneity and, in particular, recognize that when formulating policy targets, governments should consider their own local circumstances carefully before adopting the guidelines directly as legally based standards.

The WHO AQGs are based on the now extensive body of scientific evidence relating to air pollution and its health consequences. Although this information base has gaps and uncertainties, it offers a strong foundation for the recommended guidelines. Several key findings that have emerged in recent years merit special mention. Firstly, the evidence for ozone (O_3) and particulate matter (PM) indicates that there are risks to health at concentrations currently found in many cities in developed countries. Moreover, as research has not identified thresholds below which adverse effects do not occur, it must be stressed that the guideline values provided here cannot fully protect human health.

Secondly, an increasing range of adverse health effects has been linked to air pollution, and at ever-lower concentrations. This is especially true of airborne particulate matter. New studies use more refined methods and more subtle but sensitive indicators of effects, such as physiological measures (e.g. changes in lung function, inflammation markers). Therefore the updated guidelines could be based both on these sensitive indicators, in addition to the most critical population health indicators, such as mortality and unscheduled hospitalizations.

Thirdly, as our understanding of the complexity of the air pollution mixture has improved, the limitations of controlling air pollution through guidelines for single pollutants have become increasingly apparent. Nitrogen dioxide (NO_2), for example, is a product of combustion processes and is generally found in the atmosphere in close association with other primary pollutants, including ultrafine (UF) particles. It is itself toxic and is also a precursor of ozone, with which it coexists along with a number of other photochemically generated oxidants. Concentrations of NO_2 are often strongly correlated with those of other toxic pollutants, and being the easier to measure, is often used as a surrogate for the pollutant mixture as a whole. Achieving guideline concentrations for individual pollutants such as NO_2 may therefore bring public health benefits that exceed those anticipated on the basis of estimates of a single pollutant's toxicity.

The present revision of the WHO *Air quality guidelines for Europe* provides new guideline values for three of the four pollutants examined. For two of them (particulate matter and ozone), it is possible to derive a quantitative relationship between the concentration of the pollutant as monitored in ambient air and specific health outcomes (usually mortality). These relationships are invaluable for health impact assessments and allow insights into the mortality and morbidity burdens from current levels of air pollution, as well as what health improvements could be expected under different air pollution reduction scenarios. The burden-of-disease estimates can also be used for the purpose of estimating the costs and benefits of interventions that reduce air pollution. Approaches to, and the

limitations of, health impact assessments are summarized in the full report supporting the updated guidelines.

Air pollutant concentrations should be measured at monitoring sites that are representative of population exposures. Air pollution levels may be higher in the vicinity of specific sources of air pollution, such as roads, power plants and large stationary sources, and so protection of populations living in such situations may require special measures to bring the pollution levels to below the guideline values.

The following sections of this document present the WHO AQGs for PM, ozone, NO_2 and SO_2, and in each case give the rationale for the decision to revise the guideline value or to retain the existing value. As noted above, the epidemiological evidence indicates that the possibility of adverse health effects remains even if the guideline value is achieved, and for this reason some countries might decide to adopt lower concentrations than the WHO guideline values as their national air quality standards.

In addition to guideline values, **interim targets** are given for each pollutant. These are proposed as incremental steps in a progressive reduction of air pollution and are intended for use in areas where pollution is high. These targets aim to promote a shift from high air pollutant concentrations, which have acute and serious health consequences, to lower air pollutant concentrations. If these targets were to be achieved, one could expect significant reductions in risks for acute and chronic health effects from air pollution. Progress towards the guideline values should, however, be the ultimate objective of air quality management and health risk reduction in all areas.

Air quality guidelines and their rationale

Particulate matter

Guidelines

$PM_{2.5}$:	**10 μg/m³ annual mean**
	25 μg/m³ 24-hour mean
PM_{10}:	**20 μg/m³ annual mean**
	50 μg/m³ 24-hour mean

Rationale

The evidence on airborne particulate matter (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 are 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 2.5 μm ($PM_{2.5}$) has been estimated to be 3–5 μg/m³ in both the United States and western Europe. The epidemiological evidence shows adverse effects of PM following both short-term and long-term exposures.

As thresholds have not been identified, and given that there is substantial inter-individual variability in exposure and in the response in a given exposure, it is unlikely that any standard or guideline value will lead to complete protection for every individual against all possible adverse health effects of particulate matter. Rather, the standard-setting process needs to aim at achieving the lowest concentrations possible in the context of local constraints, capabilities and public health priorities. Quantitative risk assessment offers one way of comparing alternative control scenarios and of estimating the residual risk associated with a particular guideline value. Both the United States Environmental Protection Agency and the European Commission have recently used this approach to revise their air quality standards for PM. Countries are encouraged to consider adopting an increasingly stringent set of standards, tracking progress through the monitoring of emission reductions and declining concentrations of PM. To assist this process, the numerical guideline and interim target values given here reflect the concentrations at which increased mortality responses due to PM air pollution are expected based on current scientific findings.

The choice of indicator for particulate matter also requires consideration. At present, most routine air quality monitoring systems generate data based on the measurement of PM_{10} as opposed to other particulate matter sizes. Consequently, the majority of epidemiological studies use PM_{10} as the exposure indicator. PM_{10} represents the particle mass that enters the respiratory tract and, moreover, it includes both the coarse (particle size between 2.5 and 10 μm) and fine particles (measuring less than 2.5 μm, $PM_{2.5}$) that are considered to contribute to

the health effects observed in urban environments. The former is primarily produced by mechanical processes such as construction activities, road dust re-suspension and wind, whereas the latter originates primarily from combustion sources. In most urban environments, both coarse and fine mode particles are present, but the proportion of particles in these two size ranges is likely to vary substantially between cities around the world, depending on local geography, meteorology and specific PM sources. In some areas, the combustion of wood and other biomass fuels can be an important source of particulate air pollution, the resulting combustion particles being largely in the fine ($PM_{2.5}$) mode. Although few epidemiological studies have compared the relative toxicity of the products of fossil fuel and biomass combustion, similar effect estimates are found for a wide range of cities in both developed and developing countries. It is, therefore, reasonable to assume that the health effects of $PM_{2.5}$ from both of these sources are broadly the same. By the same token, the WHO AQG for PM can also be applied to the indoor environment, specifically in the developing world, where large populations are exposed to high levels of combustion particles derived from indoor stoves and fires.

Although PM_{10} is the more widely reported measure, and also the indicator of relevance to the majority of the epidemiological data, for reasons that are discussed below, the WHO AQGs for PM are based on studies that use $PM_{2.5}$ as an indicator. The $PM_{2.5}$ guideline values are converted to the corresponding PM_{10} guideline values by application of a $PM_{2.5}/PM_{10}$ ratio of 0.5. A $PM_{2.5}/PM_{10}$ ratio of 0.5 is typical of developing country urban areas and is at the bottom of the range found in developed country urban areas (0.5–0.8). When setting local standards, and assuming the relevant data are available, a different value for this ratio, i.e. one that better reflects local conditions, may be employed.

Based on known health effects, both short-term (24-hour) and long-term (annual mean) guidelines are needed for both indicators of PM pollution.

Long-term exposures

An annual average concentration of 10 $\mu g/m^3$ was chosen as the long-term guideline value for $PM_{2.5}$. This represents the lower end of the range over which significant effects on survival were observed in the American Cancer Society's (ACS) study (Pope et al., 2002). Adoption of a guideline at this level places significant weight on the 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). In all of these studies, robust associations were reported between long-term exposure to $PM_{2.5}$ and mortality. The historical mean $PM_{2.5}$ concentration was 18 $\mu g/m^3$ (range, 11.0–29.6 $\mu g/m^3$) in the Six-Cities study and 20 $\mu g/m^3$ (range, 9.0–33.5 $\mu g/m^3$) in the ACS study. Thresholds were not apparent in any of these studies, although the precise period(s) and pattern(s) of relevant exposure could not be ascertained. In the ACS study, statistical uncertainty in the risk estimates becomes apparent at concentrations of about 13 $\mu g/m^3$, below which the confidence bounds significantly widen since the concentrations are relatively far from the mean. According to the results of the Dockery et al. (1993) study, the risks are similar in the cities with the lowest long-term $PM_{2.5}$ concentrations (i.e. 11 and 12.5 $\mu g/m^3$). Increases in risk are apparent in the city with the next-lowest long-term $PM_{2.5}$ mean (i.e. 14.9 $\mu g/m^3$), indicating that health effects can be expected when annual mean concentrations are in the range of 11–15 $\mu g/m^3$. Therefore, an annual mean concentration of 10 $\mu g/m^3$ can be considered, according to the available scientific literature, to be below the mean for most likely effects. Selecting a long-term mean $PM_{2.5}$ concentration of 10 $\mu g/m^3$ also places some weight on the results of daily exposure time-series studies that examine the relationships between exposure to $PM_{2.5}$ and acute adverse health outcomes. In these studies, long-term (i.e. three- to four-year) means are reported to be in the range of 13–18 $\mu g/m^3$. Although adverse effects on health cannot be entirely ruled out below these levels, the annual average WHO AQG value represents that concentration of $PM_{2.5}$ that has not only been shown to be achievable in large urban areas in highly devel-

oped countries, but also the attainment of which is expected to significantly reduce the health risks.

Besides the guideline value, three interim targets (IT) are defined for $PM_{2.5}$ (see Table 1). These have been shown to be achievable with successive and sustained abatement measures. Countries may find these interim targets particularly helpful in gauging progress over time in the difficult process of steadily reducing population exposures to PM.

An annual mean $PM_{2.5}$ concentration of 35 μg/m³ was selected as the IT-1 level. This level corresponds to the highest mean concentrations reported in studies of long-term health effects, and may also reflect higher but unknown historical concentrations that may have contributed to observed health effects. This level has been shown to be associated with significant mortality in the developed world.

The IT-2 interim level of protection is set at 25 μg/m³ and relies, as its basis, on the studies of long-term exposure and mortality. This value is greater than the mean concentration at which effects have been observed in such studies, and is likely to be associated with significant health impacts from both long-term and daily exposures to $PM_{2.5}$. Attainment of this IT-2 value would reduce the health risks of long-term exposure by about 6% (95% CI, 2–11%) relative to the IT-1 value. The recommended IT-3 level is 15 μg/m³ and places even greater weight on the likelihood of significant effects associated with long-term exposures. This value is close to the mean concentrations that are reported in studies of long-term exposure and provides an additional 6% reduction in mortality risk relative to the IT-2 value. Corresponding AQGs and interim targets are also recommended for PM_{10} (Table 1). This is because a $PM_{2.5}$ guideline alone would not provide protection against the harmful effects of coarse PM (the fraction between 10 and 2.5 μm). However, the quantitative evidence on coarse PM is considered insufficient to derive separate guidelines. In contrast, there is a large body of literature on effects of short-term exposures to PM_{10}, which has been used as a basis for the development of WHO AQGs and interim targets for 24-hour concentrations of PM (see below).

Table 1

WHOair quality guidelines and interim targets for particulate matter: annual mean concentrations[a]

	PM_{10} (μg/m³)	$PM_{2.5}$ (μg/m³)	Basis for the selected level
Iinterim target-1 (IT-1)	70	35	These levels are associated with about a 15% higher long-term mortality risk relative to the AQG level.
Interim target-2 (IT-2)	50	25	In addition to other health benefits, these levels lower the risk of premature mortality by approximately 6% [2–11%] relative to theIT-1 level.
Interim target-3 (IT-3)	30	15	In addition to other health benefits, these levels reduce the mortality risk by approximately 6% [2-11%] relative to the -IT-2 level.
Air quality guideline (AQG)	**20**	**10**	These are the lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to long-term exposure to $PM_{2.5}$

[a] The use of $PM_{2.5}$ guideline value is preferred.

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 the WHO AQGs and interim targets, it is 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. It is recommended that countries with areas not meeting the 24-hour guideline values undertake immediate action to achieve these levels in the shortest possible time.

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 10 μg/m³ (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 10 μg/m³ (Cohen et al., 2004), very similar in fact to that derived for Asian cities (0.49% per 10 μg/m³)(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 10 μg/m³ increment in the daily concentration. Therefore, a PM_{10} concentration of 150 μg/m³ would be expected to translate into roughly a 5% increase in daily mortality, an impact that would be of significant concern, and one for which immediate mitigation actions would be recommended. The IT-2 level of 100 μg/m³ would be associated with approximately a 2.5% increase in daily mortality, and the IT-3 level with a 1.2% increase (Table 2). For PM_{10}, the AQG for the 24-hour average is 50 μg/m³, and reflects the relationship between the distributions of 24-hour means (and its 99th percentile) and annual average concentrations.

Table 2

WHO air quality guidelines and interim targets for particulate matter: 24-hour concentrations[a]

	PM_{10} (μg/m³)	$PM_{2.5}$ (μg/m³)	Basis for the selected level
Interim target-1 (IT-1)	150	75	Based on published risk coefficients from multi-centre studies and meta-analyses (about 5% increase of short-term mortality over the AQG value).
Interim target-2 (IT-2)	100	50	Based on published risk coefficients from multi-centre studies and meta-analyses (about 2.5% increase of short-term mortality over the AQG value).
Interim target-3 (IT-3)*	75	37.5	Based on published risk coefficients from multi-centre studies and meta-analyses (about 1.2% increase in short-term mortality over the AQG value).
Air quality guideline (AQG)	**50**	**25**	Based on relationship between 24-hour and annual PM levels.

[a] 99th percentile (3 days/year).

* For management purposes. Based on annual average guideline values; precise number to be determined on basis of local frequency distribution of daily means. The frequency distribution of daily $PM_{2.5}$ or PM_{10} values usually approximates to a log-normal distribution.

Ultrafine particles (UF), i.e. particles smaller than 0.1 μm 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.

Ozone

Guideline

O_3: 100 $\mu g/m^3$ 8-hour mean

Rationale

Since the publication of the second edition of the WHO Air quality guidelines for Europe (WHO, 2000) which sets the guideline value for ozone levels at 120 $\mu g/m^3$ for an 8-hour daily average, little new information about the health effects of ozone has been obtained from either chamber studies or field studies. 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 120 $\mu g/m^3$ 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 120 $\mu g/m^3$ to 100 $\mu g/m^3$ (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 100 $\mu g/m^3$ over that when ozone levels are at a baseline level of 70 $\mu g/m^3$ (the estimated background ozone level; see Table 3). There is some evidence that long-term exposure to ozone may have chronic effects but it is not sufficient to recommend an annual guideline.

Ozone is formed in the atmosphere by photochemical reactions in the presence of sunlight and precursor pollutants, such as the oxides of nitrogen (NOx) and volatile organic compounds (VOCs). It is destroyed by reactions with NO, and is deposited to the ground. Several studies have shown that ozone concentrations correlate with various other toxic photochemical oxidants arising from similar sources, including the peroxyacyl nitrates, nitric acid and hydrogen peroxide. Measures to control tropospheric ozone levels focus its precursor gas emissions, but are likely to also control the levels and impacts of a number of these other pollutants.

Hemispheric background concentrations of tropospheric ozone vary in time and space but can reach 8-hours average levels of around 80 $\mu g/m^3$. These arise from both anthropogenic and biogenic emissions (e.g. VOCs from vegetation) of ozone precursors and downward intrusion of stratospheric ozone into the troposphere. Indeed, the proposed guideline value may occasionally be exceeded due to natural causes.

As ozone concentrations increase above the guideline value, health effects at the population level become increasingly numerous and severe. Such effects can occur in places where concentrations are currently high due to human activities or are elevated during episodes of very hot weather.

The 8-hour IT-1 level for ozone has been set at 160 $\mu g/m^3$ at which measurable, though transient, changes in lung function and lung inflammation have been recorded in controlled chamber tests

in healthy young adults undertaking intermittent exercise. 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. Furthermore, chamber studies provide little information about repeated exposures. Based on time-series evidence, exposures at the IT-1 level are associated with an increase in the number of attributable deaths brought forward of 3–5% (see Table 3).

At 8-hour concentrations exceeding 240 $\mu g/m^3$, 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 (see Table 3).

Table 3

WHO air quality guideline and interim target for ozone: 8-hour concentrations

	Daily maximum 8-hour mean ($\mu g/m^3$)	Basis for selected level
High levels	240	Significant health effects: substantial proportion of vulnerable populations affected.
Interim target-1 (IT-1)	160	Important health effects; does not provide adequate protection of public health. Exposure to this level of ozone is associated with: • physiological and inflammatory lung effects in healthy exercising young adults exposed for periods of 6.6 hours; • health effects in children (based on various summer camp studies in which children were exposed to ambient ozone levels). • an estimated 3–5% increase in daily mortality[a] (based on findings of daily time-series studies).
Air quality guideline (AQG)	**100**	Provides adequate protection of public health, though some health effects may occur below this level. Exposure to this level of ozone is associated with: • an estimated 1–2% increase in daily mortality[a] (based on findings of daily time-series studies). • Extrapolation from chamber and field studies based on the likelihood that real-life exposure tends to be repetitive and chamber studies exclude highly sensitive or clinically compromised subjects, or children. • Likelihood that ambient ozone is a marker for related oxidants.

[a] Deaths attributable to ozone. Time-series studies indicate an increase in daily mortality in the range of 0.3–0.5% for every 10 $\mu g/m^3$ increment in 8-hour ozone concentrations above an estimated baseline level of 70 $\mu g/m^3$.

Nitrogen dioxide

Guidelines

NO_2: **40 $\mu g/m^3$ annual mean**
200 $\mu g/m^3$ 1-hour mean

Rationale

As an air pollutant, nitrogen dioxide (NO_2) has multiple roles, which are often difficult or sometimes impossible to separate from one another:

i. Animal and human experimental studies indicate that NO_2 – at short-term concentrations exceeding 200 $\mu g/m^3$ – 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.

ii. 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.

iii. 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.

The current WHO guideline value of 40 $\mu g/m^3$ (annual mean) was set to protect the public from the health effects of gaseous NO_2. The rationale for this was that because most abatement methods are specific to NO_x, they are not designed to control other co-pollutants, and may even increase their emissions. If, however, NO_2 is monitored as a marker for complex combustion-generated pollution mixtures, a lower annual guideline value should be used (WHO, 2000).

Long-term exposures

There is still no robust basis for setting an annual average guideline value for NO_2 through any direct toxic effect. 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 40 $\mu g/m^3$. 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.

Taken together, the above findings provide some support for a lowering of the current annual NO_2 guideline value. However, it is unclear to what

extent the health effects observed in epidemiological studies are attributable to NO_2 itself or to the other primary and secondary combustion-related products with which it is typically correlated. Thus it can be argued that the available scientific literature has not accumulated sufficient evidence to justify revising the existing WHO AQG for annual NO_2 concentrations. Nevertheless, since NO_2 concentrations in ambient air are routinely measured but those of other correlated combustion-derived pollutants are not, it seems reasonable to retain a prudent annual average limit value for NO_2. Such a limit allows for the fact that there may be direct toxic effects of chronic NO_2 exposure at low levels. In addition, maintaining the annual guideline value may help to control complex mixtures of combustion-related pollution (mainly from road traffic)

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 500 μg/m³. Although the lowest level of NO_2 exposure to show a direct effect on pulmonary function in asthmatics in more than one laboratory is 560 μg/m³, studies of bronchial responsiveness among asthmatics suggest an increase in responsiveness at levels upwards from 200 μg/m³.

Since the existing WHO AQG short-term NO_2 guideline value of 200 μg/m³ (1-hour) has not been challenged by more recent studies, it is retained.

In conclusion, the guideline values for NO_2 remain unchanged in comparison to the existing WHO AQG levels, i.e. 40 μg/m³ for annual mean and 200 μg/m³ for 1-hour mean.

Sulfur dioxide

Guidelines

SO_2: 20 μg/m^3 24-hour mean
500 μg/m^3 10-minute mean

Rationale

Short-term exposures

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. 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. Because short-term SO_2 exposure depends very much on the nature of local sources and the prevailing meteorological conditions, it is not possible to apply a simple factor to this value in order to estimate corresponding guideline values over longer time periods, such as one hour.

Long-term exposures (over 24-hours)

Early estimates of day-to-day changes in mortality, morbidity or lung function in relation to 24-hour average concentrations of SO_2 were necessarily based on epidemiological studies in which people are typically exposed to a mixture of pollutants. As there was little basis for separating the contributions of individual pollutants to the observed health outcomes, prior to 1987, guideline values for SO_2 were linked to corresponding values for PM. This approach led to the setting of an AQG value for SO_2 of 125 µg/m^3 as a 24-hour average, after applying an uncertainty factor of 2 to the lowest-observed-adverse-effect level (WHO, 1987). In the second edition of the WHO *Air quality guidelines for Europe* (WHO, 2000), it was noted that later epidemiological studies documented separate and independent adverse public health effects for PM and SO_2, and this led to a separate WHO AQG for SO_2 of 125 µg/m^3 (24-hour mean). The latest evidence to emerge includes a study conducted in Hong Kong (Hedley et al., 2002) where a major reduction in the sulfur content of fuels has been achieved over a very short period of time. This has been linked to substantial reductions in health effects (e.g. childhood respiratory disease and all-age mortality). Recent time-series studies on hospital admissions for cardiac disease in Hong Kong and London, produced no evidence of a threshold for health effects at 24-hour SO_2 concentrations in the range of 5–40 µg/m^3 (Wong et al., 2002). Twenty-four hour SO_2 levels were significantly associated with daily mortality rates in 12 Canadian cities, which had an average concentration of only 5 µg/m^3 (the highest mean SO_2 level was below 10 µg/m^3) (Burnett et al., 2004). In the American Cancer Society (ACS) study (see Particulate matter), significant associations between SO_2 and mortality were observed for the 1982–1998 cohort in 126 United States metropolitan areas, in which the mean SO_2 concentration recorded was 18 µg/m^3, and the highest mean, 85 µg/m^3 (Pope et al., 2002). If there were a threshold for effects in either of these two studies, it would have to be very low.

There is still considerable uncertainty as to whether SO_2 is the pollutant responsible for the observed adverse effects or whether it is a surrogate for ultrafine particles or some other correlated substance. Both Germany (Wichmann et al., 2000) and the Netherlands (Buringh, Fisher & Hoek, 2000) have experienced a strong reduction in SO_2 concentrations over a decade, but although mortality also decreased with time, the association between SO_2 and mortality was not judged to be

causal in either case the fall in mortality and was instead attributed to a similar time trend in a different pollutant (PM).

In consideration of: a) the uncertainty of SO_2 in causality; b) the practical difficulty of attaining levels that are certain to be associated with no effects; and c) the need to provide a greater degree of protection than that provided by the present AQG, and assuming that reduction in exposure to a causal and correlated substance is achieved by reducing SO_2 concentrations, there is a basis for revising the 24-hour guideline for SO_2 downwards adopting a prudent precautionary approach to a value of 20 $\mu g/m^3$.

Table 4

WHO air quality guidelines and interim targets for SO_2: 24-hour and 10-minute concentrations

	24-hour average ($\mu g/m^3$)	10-minute average ($\mu g/m^3$)	Basis for selected level
Interim target-1 (IT-1)[a]	125	–	
Interim target-2 (IT-2)	50	–	Intermediate goal based on controlling either motor vehicle emissions, industrial emissions and/or emissions from power production. This would be a reasonable and feasible goal for some developing countries (it could be achieved within a few years) which would lead to significant health improvements that, in turn, would justify further improvements (such as aiming for the AQG value).
Air quality guideline (AQG)	**20**	**500**	

[a] Formerly the WHO Air Quality Guideline (WHO, 2000).

An annual guideline is not needed, since compliance with the 24-hour level will assure low annual average levels. These recommended guideline values for SO_2 are not linked to those for PM. Since the revised 24-hour guideline may be quite difficult for some countries to achieve in the short term, a stepped approach using interim goals is recommended (see Table 4). For instance, a country could move towards compliance with the guideline by controlling emissions from one major source at a time, selecting from among motor vehicle sources, industrial sources and power sources (which would achieve the greatest effect on SO_2 levels for the lowest cost), and follow this up with monitoring of public health and SO_2 levels for health effect gains. Demonstrating health benefits should provide an incentive to mandate controls for the next major source category.

References

Buringh E, Fischer P, Hoek G (2000). Is SO_2 a causative factor for the PM-associated mortality risks in the Netherlands? *Inhalation Toxicology*,12(Suppl.):S55–S60.

Burnett RT et al. (2004). Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities. *Archives of Environmental Health,* 59:228–236.

Cohen A et al. (2004). Mortality impacts of urban air pollution. In: Ezzati M et al., eds. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Geneva, World Health Organization:1353–1434.

Dockery DW et al. (1993). An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine,* 329:1753–1759.

Hedley AJ et al. (2002). Cardiorespiratory and all-cause mortality after restrictions on sulfur content of fuel in Hong Kong: an intervention study. *Lancet,* 360:1646–1652.

HEI (2000). *Reanalysis of the Harvard Six-Cities study and the American Cancer Society study of particulate air pollution and mortality. A special report of the Institute's Particle Epidemiology reanalysis Project*. Cambridge, MA, Health Effects Institute.

HEI International Oversight Committee (2004). *Health effects of outdoor air pollution in developing countries of Asia: a literature review.* Boston, MA, Health Effects Institute (Special Report No. 15).

Jerrett M (2005). Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiology*,16:727–736.

Katsouyanni K et al. (2001). Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. *Epidemiology*, 12:521–531.

Pope CA et al. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *American Journal of Respiratory and Critical Care Medicine*, 151:669–674.

Pope CA et al. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 287:1132–1141.

Samet JM et al. (2000). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. *Research Reports of the Health Effects Institute*, 94:5–70.

Wong CM et al. (2002). A tale of two cities: effects of air pollution on hospital admissions in Hong Kong and London compared. *Environmental Health Perspectives*, 110:67–77.

WHO (1987). *Air quality guidelines for Europe*. Copenhagen, World Health Organization Regional Office for Europe, 1987 (WHO Regional Publications, European Series No. 23).

WHO (2000). *Air quality guidelines for Europe, 2nd ed.* Copenhagen, World Health Organization Regional Office for Europe, 2000 (WHO Regional Publications, European Series No. 91).

Wichmann HE et al. (2000). *Daily mortality and fine and ultrafine particles in Erfurt, Germany. Part 1: Role of particle number and particle mass.* Cambridge, MA, Health Effects Institute (Research Report No. 98).

The WHO air quality guidelines are designed to offer guidance in reducing the health impacts of air pollution. Based on a review of the accumulated scientific evidence, the revised guideline values for the most common air pollutants are presented in this document. These guidelines are applicable across all WHO regions and inform policy-makers considering various options for air quality management in different parts of the world about the targets for air quality.