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# **AIR QUALITY CRITERIA AND GUIDES FOR URBAN AIR POLLUTANTS**

**Report of a WHO Expert Committee**

WORLD HEALTH ORGANIZATION

GENEVA

1972

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AND GUIDES FOR URBAN AIR POLLUTANTS**

*Geneva, 5-11 April 1972*

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# **AIR QUALITY CRITERIA AND GUIDES FOR URBAN POLLUTANTS**

## **Report of a WHO Expert Committee**

### **1. INTRODUCTION**

A WHO Expert Committee on Air Quality Criteria and Guides for Urban Air Pollutants met in Geneva from 5 to 11 April 1972. Dr B. H. Dieterich, Director, Division of Environmental Health, opened the meeting on behalf of the Director-General. He called attention to a resolution adopted by the Twenty-Third World Health Assembly in 1970, expressing the wish that "due consideration should be given to the effect of water, soil, food and air pollution, noise and other environmental factors harmful to human health, and to the need for the establishment of environmental health criteria, guidelines for preventive measures, and methods of determining priorities and allocating resources based on health problems and needs in both developing and developed countries."

He further commented that WHO has received many requests from both developed and developing countries for guidance as to the levels of ambient air pollutants that constitute hazards to health. Many of these countries lack the expertise, equipment, and time to develop their own scientific foundation for the establishment of standards and legislation. Of course, considerations other than effects on health must also be taken into account by the governments concerned in setting standards, e.g., economic and technical resources, administrative and legislative traditions, and socioeconomic considerations.

Dr Dieterich also recalled that an Inter-Regional Symposium organized by WHO in October 1970 had recommended that a meeting of experts be convened for the purpose of proposing air quality criteria and guides<sup>1</sup> for such common urban air pollutants as oxides of sulfur, suspended particulates, carbon monoxide, oxidants, and oxides of nitrogen.

#### **1.1 Background information**

The earth's atmosphere is finite, and its capacity to cleanse itself, although as yet imperfectly understood, seems to be limited, at least in

<sup>1</sup> For definitions of these terms, see *Wld Hlth Org. techn. Rep. Ser.*, 1964, No. 271, p. 13.

some places and with respect to certain pollutants. As the world population grows and industry expands to make more and increasingly diverse products, the emission of some pollutants will inevitably increase. Increased emissions have already on several occasions and in several places led to ground level concentrations that were associated with dramatic rises in mortality and morbidity. There is abundant evidence that high levels of air pollutants may be generally harmful to human beings, and there is no evidence that pollutants at any level are beneficial. For most environmental factors, such as heat, humidity, noise, etc., acceptable levels have been established, but none have yet been established for air pollution. The World Health Organization<sup>1</sup> has therefore devoted much thought and effort to the necessary task of assessing the health effects of pollution such as it exists now and as it may develop in the future. Epidemiological factors were reviewed by Lawther et al. in 1962.

The present Expert Committee was conscious of the desirability and difficulty of making an exhaustive survey of all published evidence extant. Nevertheless, it recognized that many countries urgently require advice so that they may take prudent action, based on existing knowledge, to safeguard their populations from the adverse effects of pollution on health, both by developing standards and by setting long-term goals.

Although air pollution is difficult to define, the following working definition was developed by an earlier committee<sup>2</sup>: the term air pollution is "limited to the situations in which the outdoor ambient atmosphere contains materials in concentrations which are harmful to man or to his environment." This definition has been adopted in the present report, which is restricted to a discussion of 5 pollutants. In accordance with the definition, only outdoor pollution is considered, although it is noted that pollution of the air inside dwellings may be important and requires further study. Likewise, there is little discussion of the complex problems raised by the fact that gross variations in ground pollutant levels are sometimes caused by changes in the weather. Variability due to atmospheric conditions makes it difficult to relate long-term average values (e.g., mean annual concentrations) to short-term peaks, which may be more instrumental in producing certain effects. This question has been considered in detail elsewhere by Katz (1969).

Various types of data may be used in the development of air quality criteria and guides. Experimental techniques, for example, can yield valuable information. Some experts would interpret any deviation from the "normal" values of physiological, biochemical, or behavioural indices as undesirable rather than adaptive and would prefer to fix standards

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<sup>1</sup> *Wld Hlth Org. techn. Rep. Ser.*, 1958, No. 157; 1964, No. 271; 1968, No. 406; 1970, No. 439; *Wld Hlth Org. Monogr. Ser.*, 1961, No. 46.

<sup>2</sup> *Wld Hlth Org. techn. Rep. Ser.*, 1958, No. 157.



below which no damage at all could be expected. The use of human volunteers and animals as models for studying these very slight effects of suspected pollutants is especially helpful, as has been discussed by Rjazanov (1965). At the clinical level, one may evaluate the results of experiments with various pollutants in normal subjects to determine at what concentration they produce clinically undesirable effects. Experiments of this type are useful but are limited in value by the important fact that ethical considerations preclude studies on sick persons (see section 2.1). In the epidemiological approach, one studies the effect of fluctuations in ambient air pollution on the whole population or on selected and defined groups such as children, the elderly, or the ill, who may be particularly sensitive to the action of the pollutants suspected of causing ill effects. Acute, subacute, and chronic effects may be studied epidemiologically, but it must be recalled that many factors other than pollution may make it difficult to interpret mortality and morbidity variations since the effects of air pollution are closely linked to other factors including temperature and socioeconomic status. The prevalence of infection, smoking, therapeutic measures, population migration, malnutrition, and stress may pose serious problems of interpretation. Special difficulties beset those who study the response to pollution in a work environment. Working people are a specially selected and fit subgroup, and differ in obvious respects from the general population, which includes the very young, the elderly, and the sick. Nonetheless, such studies deserve critical appraisal rather than uncritical rejection.

Serious attempts have been made with limited information to reach a consensus of opinion as to the action that should be taken to protect people from pollution. Unfortunately, it must be recognized that this approach to the problem in no way guarantees that such action will necessarily confer the protection sought. Air pollution is an extremely complex matter. The association between a pollutant and illness or death may be accidental rather than causal; the concentrations of many pollutants, some unmeasured or unidentified, often go hand in hand; and any urban population will contain some people in such a precarious state of health that they will succumb to any stress from which they could not reasonably be protected.

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## 2. SULFUR OXIDES AND SUSPENDED PARTICULATES

Sulfur oxides and suspended particulates, here considered together, often have common origins in that both may be produced by the burning of fossil fuels. Sulfur dioxide is produced by the combustion of sulfur compounds present as impurities in many coals and heavy oils. Some of the sulfur in these fuels may be further oxidized during the combustion process to give sulfur trioxide; about 5% of the sulfur may be emitted in this form. Some sulfur in coal is retained as sulfate in the ash.

Particulate matter requires careful definition since its effects are determined not only by dose but also by its chemical composition and physical form. Similarly, the methods for measuring and expressing the concentration of suspended particulates in the air depend to a critical extent on the properties of these pollutants. When carbon-containing fuel is burned inefficiently, smoke is produced which may be almost pure carbon, as in the case of a poorly adjusted diesel engine, or a complex aerosol of tar droplets such as that produced by poor burning and destructive distillation of coal. Minute particles of mineral ash may be carried into the air by flue gases. Fog and mist contain droplets the size of which may be stabilized by salts. Particulate pollution may also be formed from gaseous pollutants. Sulfur dioxide may be oxidized to form sulfuric acid and, later, sulfate.

Many highly diverse particles are emitted by industrial processes other than fuel combustion. In addition, not all particulate pollution is man-made. Salt aerosols derived from sea spray are widespread; many parts of the world are exposed to airborne dusts which can produce gross concentrations of particles; and spores, pollens, moulds, and other organic materials are widely distributed.

It is important to realize that particulate and gaseous ambient pollutants are rarely if ever found in isolation. It follows, therefore, that unless the effect sought is highly specific, the use of epidemiological techniques will seldom result in the attribution, with any degree of certainty, of an observed effect to a specific pollutant. The task may be made easier by the fact that, in some parts of the world, pollution by smoke is decreasing much faster than is pollution by sulfur dioxide.

Most of the epidemiological studies published, including those referred to in this document, have been concerned with particles produced by burning fossil fuels. The data must therefore be extrapolated with care to situations where oxides of sulfur may occur in combination with other more specific pollutants arising from industrial, natural, or other sources. In addition, the overall exposure of individuals or groups may be difficult to assess because of different exposures at home (Biersteker et al., 1965) or in high apartment buildings (Gubernskij et al., 1969).

## 2.1 Experimental studies

The results of animal studies suggest that the addition of various particles to sulfur dioxide may potentiate or enhance its effect (Amdur & Underhill, 1968). There is no convincing evidence from experimental work on healthy human beings that particles added to inhaled sulfur dioxide potentiate its effects in short-time exposures (Frank et al., 1964). There is, however, an important limitation on the experimental method in that, for ethical reasons, experiments must be conducted only on fit subjects, and any negative results of such experiments must not be used to exonerate pollutants, since the same doses might have produced serious effects had they been administered to patients with severe impairment of respiratory, cardiovascular, or other functions.

Particle size is of obvious biological importance: in general it may be assumed that particles need to be smaller than about 5  $\mu\text{m}$  in order to penetrate and be deposited in the alveoli (Task Group on Lung Dynamics . . . , 1966). The importance of larger particles, however, must not be underestimated, since their impaction in the upper respiratory tract may cause irritation, reflex bronchoconstriction, and hypersecretion of mucus.

By and large, animal data on exposure to sulfur oxides and particulates do not lend themselves to a quantification of risk to human health, even though they are important for elucidating mechanisms of action and as such are indispensable for a total evaluation of risk (Amdur, 1969, 1970; Buštueva, 1961; Sidorenko & Susarova, 1955; US PHS Dept of HEW, 1965; Yelfimova & Gusev, 1969). For the time being, we are forced to rely on data on human beings in assessing the concentrations of these pollutants that may be harmful.

## 2.2 Effects on man

There have been some reports of studies that can be used to establish dose-response relationships or associations for sulfur dioxide and suspended particulates. These studies are limited in number, however, and at present there is little information available concerning the effect of varying one of these pollutants while the other is kept constant. Unfortunately, a variety of measurement methods have been used in such studies to date, making comparison of the data more difficult. For this reason, the results must be considered a first approximation.

In view of the above it is imperative that, until cause-effect relationships become established, sulfur dioxide and locally associated smoke and suspended particulates be considered as indices rather than as necessarily the specific pollutants causing the effects.

### 2.2.1 *Acute effects*

Epidemiological techniques have been used in attempts to evaluate the separate or combined effects of sulfur dioxide and particulates. In London, implementation of the Clean Air Act has greatly reduced pollution by particulate matter, and this reduction has been accompanied by a much smaller drop in sulfur dioxide concentrations. Lawther et al. (1970) studied the association of daily levels of smoke and sulfur dioxide with the reported state of health of patients with respiratory disease. The value of the experiment was limited by the fact that, over the years, episodes of severe pollution became very rare. Nevertheless, it was found that the patients no longer responded to peaks of sulfur dioxide that formerly, in association with higher particulate concentrations, produced a reaction. It was not possible to discriminate with any degree of certainty as to which pollutant, if either, was exerting the dominant effect. The technique may, however, have been too insensitive to be useful at this level. Some evidence in support of the beneficial effects of reducing the particulates has come from another study in London, in which a group of men were followed prospectively. Fletcher (1967) reported on a group of men aged 30–60 working in London who had been followed from 1961 to 1966. The mean volume of sputum steadily declined, even in those men who had not changed either their type or their level of smoking. This reduction in expectoration was associated with a decrease in the mean smoke concentrations in 7 sampling sites in London. The drop in mean winter smoke from approximately 420  $\mu\text{g}/\text{m}^3$  in 1959 to approximately 100  $\mu\text{g}/\text{m}^3$  in 1965 was not accompanied by a decrease of corresponding magnitude for sulfur dioxide, which fell only from 300 to 260  $\mu\text{g}/\text{m}^3$ .

### 2.2.2 *Chronic effects*

In any discussion of the effects of air pollution on people a distinction must be made between acute, subacute, and chronic effects. With very high concentrations such as have occurred in London, England, the Meuse Valley (Belgium), Donora, Pa., New York, the Ruhr, Osaka, and Rotterdam, the immediate effects were clearly manifest in terms of mortality or increased morbidity, especially among those already ill, old, or otherwise enfeebled. Less easily detected are the possible long-term consequences of exposure to episodes of high pollutant concentrations; these may include the subsequent development of chronic bronchitis. While there is no doubt that fluctuations in pollution less dramatic than those seen in London and elsewhere can aggravate existing cardiac and respiratory disease, the possibility must also be considered that chronic exposure to sulfur dioxide and particulate matter may play a part in the development of chronic respiratory disease. Epidemiological methods

are generally more useful than experimental techniques in the study of this important problem.

Selected studies on the chronic effects of pollution, as indicated by levels of sulfur dioxide and suspended particulates, are reviewed below. These studies incidentally illustrate some of the problems involved in the establishment of dose-effect relationships. Difficulties are primarily encountered in standardizing measurement techniques and in making adequate allowance for factors other than pollution, such as smoking, age, sex, and socioeconomic and meteorological variables.

*Chronic effects on adults.* Holland and coworkers (Holland & Reid, 1965; Holland & Stone, 1965; Holland et al., 1965) studied outdoor postal and telephone workers in the United Kingdom and the USA and found a gradation of respiratory disease symptoms across pollutant levels, particularly in the 50-59 year old category. These differences persisted when the various smoking categories were examined. The authors reported lower levels of pulmonary function, i.e., forced expiratory volume in 1 second ( $FEV_{1.0}$ ) and peak expiratory flow rates (PEFR), in the areas of higher pollution. Holland et al. (1965) indicated in this study of an occupational group that, ideally, random samples of populations should be used for international comparisons where there are gradations of air pollution.

Reid et al. (1964) attempted such a comparison, using data taken from the general practitioners' study in Great Britain (College of General Practitioners, 1961) and from a survey in Berlin, N.H., USA (Ferris & Anderson, 1962). The effects of air pollution were then examined by age group and by sex. When simple bronchitis, defined as phlegm production for 3 months of the year for 3 years, was present, standardizing for cigarette smoking removed all evidence of an effect of air pollution for both males and females. A more severe form of chronic bronchitis, characterized by phlegm production, exacerbations of colds settling in the chest, and shortness of breath when walking on level ground at one's own pace, did show an association with air pollution for both males and females. It is not known whether differences in socioeconomic or ethnic status might have been factors.

Ferris & Anderson (1964) studied a random sample from Chilliwack, B.C., Canada, a community with little air pollution. They noted no statistically significant differences between the respiratory symptoms of this community and those of the more polluted Berlin, N.H., USA, when they standardized for age and cigarette smoking, although the rates for Berlin tended to be higher than for Chilliwack. Tests of pulmonary function ( $FEV_{1.0}$  and PEFR) after standardization for age, height, sex, and smoking category did show some differences and in a number of instances these were statistically significant. It seems reasonable to

interpret the results from the Berlin and Chilliwack surveys as indicating that slight changes in respiratory symptoms and pulmonary function are related to pollutant levels.

A study by van der Lende (1969) in The Netherlands indicated that airways obstruction was not associated with air pollutions although cough and phlegm production were. As van der Lende pointed out, however, his study may have been confounded by the presence of allergens, such as spores or bacteria, in the agricultural region. Perhaps two different aspects of the problem were actually being studied: sulfur dioxide and particulates (type not identified) in the industrialized areas, and allergens in the agricultural community, where there was little apparent air pollution. Other countries should be aware of this possibility and determine whether they are dealing with a similar situation.

*Chronic effects on children.* Studies of children have also generated useful information for guides. Douglas & Waller (1966) carried out a retrospective-prospective study on a cohort identified as those children born in the first week of March 1946. The children were followed medically by health visitors and school doctors, and on admission to hospital. Level of disease and degree of symptoms were recorded. Air pollutant concentrations were estimated from the amount of coal consumed in a given area. From national data these investigators developed 4 levels of air pollution. An increased occurrence of lower respiratory tract infections was noted in areas with increased air pollution.

Lunn et al. (1967) studied young children in their first year at school. The children were given medical examinations, the parents were questioned concerning the previous health of their children, and FEV<sub>0.75</sub> and forced vital capacity (FVC) were measured. Socioeconomic factors were also considered. These particular families were stable, and there had been little or no migration into or out of the specific communities. Air pollution was measured for the 3 years of the study; it was found that there was an increased amount of chronic upper and lower respiratory tract infection in the more polluted areas. A follow-up study 3 years later by Lunn et al. (1970) disclosed that the reduction of smoke levels in Sheffield had been accompanied by a reduction in the differences between the groups of children.

Holland et al. (1969) collected data on children attending school in 4 areas of Kent, England. Two were predominantly urban and 2 were rural. Smoke and sulfur dioxide were measured in 3 of the areas, and information on crowding, population density, and housing was collected. Four factors emerged as having an important association with decreased PEFR and respiratory symptoms: place of residence was most significant, a previous history of respiratory disease was next in importance, and social class and family size were of least importance. The

effects of these 4 factors seemed to be simply additive and accounted for only 10–15% of the total variation.

Colley & Reid (1970) surveyed children 6–10 years of age living in urban and rural areas in England and Wales respectively. The prevalence of respiratory disease was assessed in the autumn, prior to the peak of winter pollution. In both areas there was a gradient across levels of pollution, but for comparable levels of pollution the rates were higher in Wales. The reasons for the increased prevalence of respiratory symptoms in the rural area were not clear, although it was suggested that it might be related to the fact that solid fuel consumption is high in Wales.

Further information was provided by a study by Ferris (1970) of school absences and pulmonary function tests in first and second graders (6–7 years of age) assigned to different schools in Berlin, N.H., USA. No difference in absences was noted between the different schools despite some variation in air pollutant levels. The students in the school with the greatest amount of pollution did, however, have decreased pulmonary function.

More than 10 years of study of the effects of air pollution on the health of primary-school children in Japan (Toyama & Nakamura, 1964) also yielded evidence of a positive association or relationship between pollution and health effects: a significant increase of air-flow resistance and an increase in school absences were demonstrated with higher pollutant levels.

Biersteker & Van Leeuwen (1970) raised the question of whether good housing is able to protect schoolchildren, as they failed to find a gradient in peak flow rates in two districts in Rotterdam with different pollution levels.

### 2.2.3 *Annoyance reactions*

The social awareness of pollution associated with particulate matter has been studied in a few areas. The results from a number of studies have been compiled in a document on particulate matter (US Dept of HEW, 1969), including data from a study carried out by Schusky (1966) in St Louis, Mo., USA, where “annoyance reactions” were associated with various levels of pollution. Similar studies should be performed in other countries, for it may well be that such reactions will, in the future, be the critical effects on which criteria for the protection of public health will be based. Since “annoyance reactions” have a large cultural component, these limits may vary from country to country and should be determined on a local basis.

### 2.3 **Effects on vegetation**

Sensitive vegetation is severely damaged by sulfur dioxide levels at the low end of the range of those known to cause deterioration in patients

with pulmonary disease. This effect is due to the synergistic action of sulfur dioxide with low levels of ozone or nitrogen dioxide. Significant damage to plants of economic importance occurs at levels well below those causing annoyance in man.

## 2.4 Evaluation

The Committee evaluated the studies discussed above, as well as other reports in the literature, and, drawing on their experience and judgement, identified the levels associated with certain observed effects. It should be emphasized that most of these effects were associated with the presence of both suspended particulates and sulfur dioxide in temperate climates and at relatively low altitudes, where both pollutants occur simultaneously from the burning of fossil fuel. The application of these results to higher altitudes or to other climatic conditions seems reasonable as a first approximation. It is important, however, that further studies be done under such conditions to determine whether these criteria actually do apply, particularly in areas with high natural dust levels associated with low sulfur oxide concentrations, or whether they need to be adapted to local geographic and/or demographic characteristics.

In interpreting the data given below and in Table 1, one must bear in mind that a numerical value associated with a given effect does not mean that all exposed individuals will be thus affected. There is no valid information available that permits precise quantification of this risk. Usually, the proportion of the population that may be expected to be affected is small. The values simply indicate that effects were

TABLE 1. EXPECTED HEALTH EFFECTS OF AIR POLLUTION ON SELECTED POPULATION GROUPS \*

Pollutant	Excess mortality and hospital admissions	Worsening of patients with pulmonary disease	Respiratory symptoms	Visibility and/or human annoyance effects
SO <sub>2</sub> <sup>a</sup>	500 µg/m <sup>3</sup> (daily average)	500-250 µg/m <sup>3</sup> <sup>b</sup> (daily average)	100 µg/m <sup>3</sup> (annual arithmetic mean)	80 µg/m <sup>3</sup> (annual geometric mean)
smoke <sup>a</sup>	500 µg/m <sup>3</sup> (daily average)	250 µg/m <sup>3</sup> (daily average)	100 µg/m <sup>3</sup> (annual arithmetic mean)	80 µg/m <sup>3</sup> (annual geometric mean) <sup>c</sup>

\* The Committee specifically urged that this table should not be considered independently of the accompanying text (see section 2.4).

<sup>a</sup> British Standard Practice (Ministry of Technology, 1966). Values for sulfur dioxides and suspended particulates apply only in conjunction with each other. They may have to be adjusted when translated into terms of results obtained by other procedures.

<sup>b</sup> These values represent the differences of opinion within the Committee.

<sup>c</sup> Based on high-volume samplers.



reported and that the number of those affected was large enough to be statistically different from the number affected in control groups.

An excess mortality in the general population has been noted when the levels of suspended particulates and sulfur oxides have both exceeded  $500 \mu\text{g}/\text{m}^3$  for 24 hours. This increase has been observed largely in the susceptible groups of the population, namely those with cardiac or pulmonary disease. These levels have also been associated with increased hospital admissions.

In the following discussion of morbidity changes due to pollution, it must be emphasized that morbidity represents a spectrum of effects ranging from functional change to the development of chronic disease.

From studies of the daily variations in the reported condition of bronchitic patients, aggravation appears to be associated with pollutant levels of  $500 \mu\text{g}$  of sulfur dioxide per  $\text{m}^3$  and  $250 \mu\text{g}$  of smoke per  $\text{m}^3$ , occurring together over 24 hours. According to unpublished preliminary findings reported to the Committee from India and the USA, persons with respiratory disease may exhibit increased symptoms when the levels of sulfur dioxide and particulates both reach  $250 \mu\text{g}/\text{m}^3$  (particulates measured by high-volume samplers). Higher frequencies of upper respiratory symptoms, increased prevalence of lower respiratory tract disease, and lower levels of pulmonary function may be found in children living in areas where the annual average levels for smoke and sulfur oxides are both above  $100 \mu\text{g}/\text{m}^3$ .

The visual range, sometimes called visibility, is reduced by air pollutants. Such changes occur when sulfur oxide and particulate concentrations are in excess of  $100\mu\text{g}/\text{m}^3$  in the presence of a relative humidity of 50% or greater and warm ambient temperature. One survey indicated that in areas where the annual geometric mean level of total suspended particulates was  $80 \mu\text{g}/\text{m}^3$  30% of persons reported awareness of reduced visibility and 10% reported annoyance. Similarly, 20% of people living in areas where the average level was  $120 \mu\text{g}/\text{m}^3$  also reported annoyance. The Committee recognizes that in the future annoyance may become a critical issue.

The above-mentioned levels of suspended particulates and sulfur oxides at which effects appear may vary in different localities depending on the chemical and physical characteristics of these pollutants and the presence of other pollutants. More refined methods for measuring and characterizing sulfur oxides together with suspended particulate matter — as well as other pollutants — are needed in addition to the procedures currently in use and should be developed.

Different methods of measuring sulfur dioxide and suspended particulate matter have been used in the various studies on health effects, making comparison difficult. The results of the high-volume samples and the results from spot samples are a case in point. In the present report,

concentrations of suspended particulate matter are expressed in terms of the British Standard Method based on the soiling of filter paper (Ministry of Technology, 1966) because it was used in several studies that have been considered by the Committee, but this does not imply that it should be accepted as the standard reference method. The Committee recommends that steps be taken to develop standard reference methods as soon as possible in order to facilitate comparison of the results obtained with the various techniques in use.

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### 3. CARBON MONOXIDE

The principal source of carbon monoxide (CO) is vehicles powered by petrol engines. Industrial plants and other operations that result in incomplete combustion of carbonaceous fuels are also important sources. Other significant and often forgotten sources of the gas are cigarette smoke (mainstream smoke contains up to 4% of CO) and domestic heating appliances which, if poorly flued, can produce high and often lethal CO concentrations indoors.

#### 3.1 Kinetics of carbon monoxide haemoglobin-reaction

When inhaled, CO combines with haemoglobin, whose vital function is to transport oxygen. Since CO has an affinity for haemoglobin some 240 times that of oxygen, the prime result of this reversible combination is to decrease the capacity of the blood to transport oxygen from the lung to the tissues. Oxygen transport capacity is further reduced by the fact that the presence of CO in the blood impairs the dissociation of oxyhaemoglobin.

The relationship between the concentrations of carboxyhaemoglobin (COHb) and oxyhaemoglobin in the blood and air can be derived from the Haldane equation, using the constants obtained by Forbes et al. (1945). In practice, carboxyhaemoglobin concentrations in the blood depend on the CO concentrations in the air breathed, duration of exposure, and pulmonary ventilation, which in turn is determined largely by the activity of the subject. When the concentration of CO in the ambient air is below that which would be in equilibrium with the blood, the subject of course exhales CO. Similarly, in a person absorbing CO, the time required to reach a given level will also depend on the initial concentration in the blood. About 3 hours are needed at rest for the COHb to reach 50% of the equilibrium value, but the rate of elimination is increased by exercise and by raising the partial pressure of oxygen of the inspired air. Table 2

TABLE 2. RELATIONSHIP BETWEEN AMBIENT CO CONCENTRATIONS, EXPOSURE TIME, AND LEVELS OF CARBOXYHAEMOGLOBIN<sup>a</sup>

Ambient CO		Carboxyhaemoglobin level (%) at		
mg/m <sup>3</sup>	ppm	1 hour	8 hours	equilibrium
117	100	3.6	12.9	16.0
70	60	2.5	8.7	10.0
35	30	1.3	4.0	5.0
23	20	0.8	2.8	3.3
12	10	0.4	1.4	1.7

<sup>a</sup> Assumes an average individual engaging in light activity and with an initial "basal" value.

indicates the relationships between selected ambient concentrations of CO, time of exposure, and blood levels. The assumption is made that initial CO saturation is virtually zero or "basal" and that the subject is engaged in light activity.

In any discussion of the relevance of CO as an air pollutant, note must be taken of the fact that the gas is naturally present in the blood in concentrations up to 0.8% of carboxyhaemoglobin as a result of catabolic processes (Sjöstrand, 1949; Coburn et al., 1969) and is often present in high concentrations in the blood of smokers who inhale tobacco smoke. Lawther & Commins (1970) and Goldsmith (1970) have discussed the influence of air pollution and cigarette smoking on blood carboxyhaemoglobin levels. CO concentrations of over 15% saturation have been found in smokers. It is important to realize that exposure to CO in the air does not necessarily raise the level in the blood. For example, continuous exposure to 25 ppm of CO will eventually result in 4% saturation, irrespective of the initial concentration in the blood: a person with an initial saturation of less than 4% will absorb the gas, while a smoker with an initial saturation greater than 4% will excrete it until he has reached equilibrium at 4%, provided he does not continue smoking.

### 3.2 Effects on man

#### 3.2.1 Effects on psychomotor function

The biological effects of CO were intensively reviewed at a meeting held under the auspices of the New York Academy of Sciences (*Ann. N.Y. Acad. Sci.*, 1970). Many subtle biochemical effects were discussed, but for the purposes of the present discussion it is sufficient to consider the acute and chronic effects that may be due simply to the relative hypoxia caused by the gas.

Early work was concerned with the forensic aspects of exposure to CO and with the definition of its toxic effects in industrial situations.

It has been established conclusively that 20% carboxyhaemoglobin saturation levels may cause symptoms and impair performance. In general, symptoms such as lassitude and headache are not reported until saturations are considerably in excess of 10% (Lindgren, 1961). Headache and impaired coordination, for example, have been noted with concentrations exceeding 10% saturation (Stewart et al., 1970). In addition, considerable attention has been given to the possible relevance of carboxyhaemoglobin concentrations too low to cause symptoms. McFarland et al. (1944) showed that visual threshold at low levels of illumination was increased at 5% saturation. Later work by Schulte (1963) and Beard & Wertheim (1967) reportedly demonstrated impairment of perception and performance at these levels. These studies, although widely quoted, were marred by technical defects in experimental method. Bender et al. (1971), using a single-blind technique, exposed volunteers to either 100 ppm of CO or ambient air for 2½ hours, followed by a battery of psychological tests. At a saturation of 7.2% a significant decrease was claimed in visual perception, manual dexterity, and the ability to learn and perform certain "intellectual" tasks. Guest et al. (1970), using a double-blind technique, studied critical flicker fusion and auditory flutter fusion thresholds after the administration of 500 ppm of CO for 1 hour (or 8% saturation) to 20 volunteers and found no effect, although these thresholds were increased by 60 mg of phenobarbital alone. Stewart et al. (1970) investigated the effects of various levels of carboxyhaemoglobin on many performance and perception tests and demonstrated that values of 15–20% saturation were associated with headache and impairment of manual coordination. Studies by Hosko (1970) have shown that visual evoked responses are modified when carboxyhaemoglobin levels exceed 20% but that spontaneous EEG activity remains unaffected until saturation approaches 33%.

In assessing the significance of these tests of psychomotor function, one must remember that it is unwise to consider CO in isolation. Concentrations too low to yield measurable results when administered alone may in future studies be shown to exert effects on subjects who have previously taken alcohol, sedatives, antihistamines, or hypotensive drugs.

### 3.2.2 *Effects on cardiovascular system*

The effects of CO on the cardiovascular system and on the oxygenation of tissues other than those of the central nervous system may be important. Chevalier et al. (1966) studied the reactions of healthy nonsmokers to CO and demonstrated an increase in oxygen debt with exercise at saturations of about 4%. Decreased arterial and mixed venous  $P_{O_2}$  in men with about 9% carboxyhaemoglobin saturation levels has been reported by Ayres et al. (1965, 1969), who claim that this might explain cardiac changes and the observed increase in oxygen debt. Ayres et al. (1970) found that

coronary arteriovenous oxygen differences were uniformly increased and coronary artery blood flow was accelerated when carboxyhaemoglobin was raised to 5–10% saturation; in addition, significant myocardial changes were seen in patients with carboxyhaemoglobin above 6% saturation. Obviously, the general population contains patients with already impaired myocardial function to whom a lowering of oxygen saturation would be harmful. Extrapolation on a quantitative basis is difficult but, in view of these acute effects, it must be conceded that there are patients with many diseases who obviously would not tolerate any further hypoxia.

Thus far, there has been no discussion of the possible role of CO in the genesis of disease, as opposed to the exacerbation of existing illness or impaired function. The work of Astrup et al. (1967) on rabbits may be of great importance in clarifying the etiology of some cardiovascular diseases. These investigators found an increased incidence of atheroma in cholesterol-fed rabbits that were also exposed to CO, and the changes produced in the vessel walls led them to suggest that the observed relationship between cardiovascular disease and cigarette smoking may well be explained in terms of chronic or repeated exposure to CO. Subsequent to these animal studies, an examination of 1000 factory workers chosen at random in Copenhagen (Kjeldsen, 1969) disclosed a clear relationship between high carboxyhaemoglobin concentrations after smoking and the occurrence of arteriosclerotic disease.

### 3.3 Effects on vegetation

CO has no adverse effects upon vegetation at the concentrations usually measured.

### 3.4 Evaluation

While it is generally agreed that individuals should be protected against continuous carboxyhaemoglobin levels of approximately 4% or over, the formulation of an air quality guide is fraught with difficulties. Many members of the population already have blood levels in excess of 4% as a result of smoking. Other smokers have concentrations under 4% but could reach this saturation level on relatively brief exposure. Since equilibration at 4% would be produced by constant inhalation of 25 ppm, this concentration is obviously undesirable. Usually, however, there are marked spatial and temporal variations in CO concentration and, as has already been stated, time is needed to reach equilibrium if the initial blood CO is low. Table 3 lists 3 levels of ambient CO and the time required for the blood to reach 4% saturation. A saturation level of 4% has been selected since levels above this appear to increase the risk for patients with cardiovascular disease.

TABLE 3. CO CONCENTRATIONS REQUIRED TO REACH 4% CARBOXYHAEMOGLOBIN LEVELS\*

Ambient CO <sup>a</sup>		Time
mg/m <sup>3</sup>	ppm	hours
29	25	24
35	30	8
117	100	1

\* The Committee specifically urged that this table should not be considered independently of the accompanying text (see Section 3.4).

<sup>a</sup> Light activity at sea level with initial "basal" values is assumed. Above 4% carboxyhaemoglobin levels, there may be increased risk for patients with cardiovascular disease.

It can be seen that the time required to reach equilibrium depends to a large extent on whether the subject has acquired CO from smoking or other sources before his exposure to ambient air. Whether susceptible persons or smokers are the proper subjects for protection is difficult to decide. One is confronted by a similar dilemma when defining the fraction of the population that must receive absolute protection at all costs, for it is obvious that in any urban community there will be some patients *in extremis* to whom any stress will prove ultimately intolerable.

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#### 4. PHOTOCHEMICAL OXIDANTS

The oxidizing type of air pollution is found in many urban areas. It results from the chemical combination of reactive hydrocarbons with nitrogen oxides in the sunlight, producing ozone, peroxyacyl nitrates, aldehydes, and other complex chemical compounds. The hydrocarbons originate primarily in petrol and motor vehicle exhaust, while the nitrogen oxides are emitted not only from motor vehicles but from stationary combustion sources as well. The photochemical oxidants considered are ozone ( $O_3$ ), peroxyacyl nitrates (PAN), and other oxidizing products of the complex atmospheric reaction that are measured by various methods but expressed as  $O_3$ . As with other pollutants, epidemiological methods are usually needed to determine whether photochemical oxidants, in the concentrations that occur in communities, have any adverse effects on human health.

##### 4.1 Effects on man

###### 4.1.1 Acute effects: mortality

No causal association was demonstrated in studies in Los Angeles between increased mortality and "alert days" when oxidant levels ranged from 0.50 to 0.90 ppm (US Dept HEW, 1970).

###### 4.1.2 Acute effects on respiratory system

A statistically significant association was demonstrated between asthmatic attacks and days on which oxidant levels were above  $500 \mu\text{g}/\text{m}^3$  (0.25 ppm), but no such association could be detected for days with mean levels of  $260 \mu\text{g}/\text{m}^3$  (0.13 ppm) (Schoettlin & Landau, 1961). The number of persons who had attacks on days on which plant damage occurred at official monitoring stations was significantly greater than on other days. There was no significant correlation between the number of persons affected and levels of carbon monoxide and particulates.

Persons with chronic pulmonary disease were found by Motley et al. (1959) to have a decrease in pulmonary function when exposed to photochemical smog. The measured oxidant levels were in the range of  $390\text{--}1370 \mu\text{g}/\text{m}^3$  (0.2–0.7 ppm). An improvement in pulmonary function was obtained when filtered air was administered, but function again



decreased upon re-exposure to unfiltered air. In a similar study, Rokaw & Massey (1962) found that exposure of such individuals to an oxidant level of  $120 \mu\text{g}/\text{m}^3$  (0.06 ppm) produced no further impairment of pulmonary function.

Schoettlin (1962) studied males over 60 in the Los Angeles area. Half of the study population had chronic respiratory disease and were matched by age and smoking categories with control males. It was found that in the respiratory disease group 11% of the variation in symptoms (cough, sputum, shortness of breath, wheezing) could be accounted for by the variation in mean oxidant levels, while 17% could be explained by the variation in maximum oxidant. In the controls, these values were 8% and 4% respectively. When the amount of oxidant precursors was determined by passing the ambient air through an irradiation chamber, 30% of the variation in symptoms could be explained by this parameter as compared with 4% for the controls. The threshold oxidant levels for the above associations ranged from 60 to  $1350 \mu\text{g}/\text{m}^3$  (0.03–0.69 ppm).

Three other studies failed to demonstrate any effects of oxidants on health (McMillan et al., 1969; Wayne & Wehrle, 1969; Pearlman et al., 1971). These negative results may be valid, but it is also possible that they may have been due to the insensitivity of the tests, inadequate sample size, and the limited range of relatively low oxidant levels studied.

#### *4.1.3 Athletic performance*

The effects of oxidants on athletic performance were investigated by Wayne et al. (1967), who compared the change in running times of long-distance runners over their previous competition times in the Los Angeles area. They found that if during the hour before the event the oxidant level was above  $200 \mu\text{g}/\text{m}^3$  (0.1 ppm) a significant number of the runners had increased running times, that is, poorer results, whereas the oxidant values 3 or 2 hours before the competition, or during the event, had no such association. Other air pollutants, such as oxides of nitrogen, carbon monoxide, and suspended particulates, were also measured but did not show any association with decreased performance at the levels recorded.

Smith (1965) studied the effect of PAN on oxygen consumption in 32 male college students at rest and during a 5-minute exercise on a bicycle ergometer. Only one level of PAN was used,  $1480 \mu\text{g}/\text{m}^3$  (0.3 ppm), which is rather high compared with the usual ambient levels; the workload was  $900 \text{ kg}/\text{m}^2$  per min. He found that oxygen uptake was increased over the control value at work but not at rest.

#### *4.1.4 Annoyance*

The acute effects of the oxidizing type of air pollution are well known to anyone who has been exposed to photochemical oxidants. Eye, nose,

and throat irritation is evident. Reasonably precise dose-response curves have been developed for eye irritation (Richardson & Middleton, 1958) but attempts to establish curves for the other symptoms have not been very successful, perhaps because of the complex nature of this type of pollution and its mechanism of action.

#### 4.1.5 *Chronic effects on man*

Although acute changes in concentration have been shown to affect man, the association of chronic respiratory disease with oxidant levels has yet to be demonstrated.

### 4.2 Experimental studies

In laboratory studies based on human subjects, the inhalation of ozone at a concentration of 1180–1570  $\mu\text{g}/\text{m}^3$  (0.6–0.8 ppm) for 2 hours has produced a decrease in diffusing capacity for CO, decreased dynamic compliance, decreased vital capacity and decreased FEV<sub>0.75</sub> (see section 2), as compared with pre-exposure values (Young et al., 1964; Silverman et al., 1970).

Animal experiments in which several species were pre-exposed to ozone in concentrations that occur in ambient air (160  $\mu\text{g}/\text{m}^3$ , i.e., 0.08 ppm or above) have shown that such exposure enhances the susceptibility of the animals to infective aerosols (Coffin et al., 1968; Gardner et al., 1971).

Although such effects have not been reported in human beings, they should be taken into account in establishing guides, since the basic mechanisms affected by ozone and possibly the other oxidant gases are common to all mammalian species.

Whether human beings are capable of developing tolerance to ozone, as has been reported in animals (Stokinger & Scheel, 1962), is not known. In this connexion, however, a distinction needs to be made between acute and chronic effects. Recent evidence suggests that tolerance to ozone in laboratory animals may be chiefly instrumental in preventing death from acute pulmonary oedema, whereas the subtle alterations of pulmonary defence as well as the occurrence of chronic lesions from low-level exposure over long periods of time are unaffected by tolerance (Coffin & Gardner, 1971). Since these are the changes that may be expected from exposure to ambient levels of oxidant, it would be unwise at this point to rely on the tolerance mechanism for human protection.

### 4.3 Effects on vegetation

Sensitive vegetation is damaged at levels below those known to cause annoyance and eye irritation, but over longer exposure times.

#### 4.4 Evaluation

Table 4 presents the Expert Committee's judgement concerning the levels associated with various effects on human beings that are appropriate for use as air quality guides.

TABLE 4. EXPECTED HEALTH EFFECTS OF PHOTOCHEMICAL OXIDANTS ON VULNERABLE GROUPS \*

Increased mortality	Increased asthmatic attacks	Pulmonary dysfunction	Annoyance and eye irritation
Not reported to date	250 µg/m <sup>3</sup> <sup>a</sup> 1 hour	200 µg/m <sup>3</sup> 1 hour	200 µg/m <sup>3</sup> 1 hour

\* The Committee specifically urged that this table should not be considered independently of the accompanying text.

<sup>a</sup> Oxidant as measured by neutral buffered KI method and expressed as ozone.

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## 5. NITROGEN DIOXIDE

Nitrogen dioxide ( $\text{NO}_2$ ) and nitric oxide ( $\text{NO}$ ) are often referred to collectively as nitrogen oxides, or  $\text{NO}_x$ .  $\text{NO}$  is emitted by both motor vehicles and stationary combustion sources, while  $\text{NO}_2$  originates in chemical and nitration industries and occurs in conjunction with the photochemical oxidant process. Most studies on the effects of the oxides of nitrogen have focused on  $\text{NO}_2$ , as the activity of  $\text{NO}$  is not yet well defined. Future studies may well reveal that it is more important than  $\text{NO}_2$ .

It should be emphasized that  $\text{NO}_2$  may have distinct biological effects apart from those associated with the photochemical pollution complex. The gas can exist as a primary pollutant in areas unaffected by photochemical oxidant pollution and should therefore be considered as a discrete pollutant requiring independent criteria and guides.

### 5.1 Effects on man

A study of second-grade (6–8 years of age) schoolchildren in Chattanooga, Tenn., USA, comparing 2 “low” control areas and 2 “high” pollution areas (one for  $\text{NO}_2$  and one for suspended particulates) showed that the reported  $\text{FEV}_{0.75}$  values were significantly higher in the control areas than in the “high”  $\text{NO}_2$  area. The levels of suspended particulates and  $\text{SO}_2$  did not seem to account for the health effects. Another finding was that the incidence of acute respiratory illness in the schoolchildren, their siblings, and their parents was significantly greater in the “high”  $\text{NO}_2$  area (Shy et al., 1970a, 1970b; Pearlman et al., 1971). This area also had an increased occurrence of lower respiratory tract infections in the sample of neonates who had been exposed 2–3 years; those exposed one year or less did not show such an increase.

The average  $\text{NO}_2$  level of  $190 \mu\text{g}/\text{m}^3$  (0.10 ppm) in the “high” area was exceeded on 40%, 18%, and 9% of the days respectively at the three monitoring stations; in the control areas this level was exceeded on 17% of the days at only one station.

### 5.2 Experimental studies

Exposures of selected animal species to the levels of  $\text{NO}_2$  occurring in ambient air have been followed by cellular alterations and changes in structural elements in the lungs as well as increased mortality from infectious aerosols (Ehrlich & Henry, 1968; Freeman et al., 1968; Gardner et al., 1969; Blair et al., 1969; Henry et al., 1970).

### 5.3 Evaluation

The published data on the effects of nitrogen oxides on human health are limited. While biological activity in animals and plants at low concentrations has been demonstrated, the Committee believes that there is insufficient information upon which to base specific air quality guides at this time.

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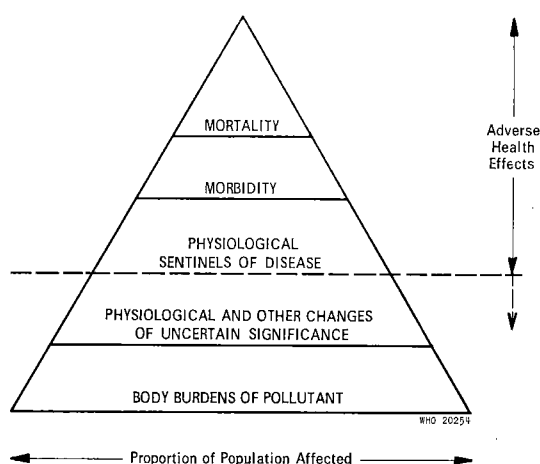
## 6. THE ADMINISTRATIVE USE OF AIR QUALITY CRITERIA AND GUIDES

### 6.1 Basic concepts

The relationship between human disease and exposure to pollution is neither simple nor fully understood. Death and disease represent only the extreme end of a whole spectrum of responses (Fig. 1). Further, some groups within the population may be especially sensitive to environmental factors, particularly the very young, the very old, those afflicted with disease, and those exposed to other toxic materials or stresses.

When using air quality criteria and guides to evaluate risks and set standards, one should ideally have available a complete set of dose-response curves for the different air pollutants, for different effects, and for the different types of populations exposed. This requirement, however, has not yet been satisfied for any single substance, and it is even further from being met for combinations of substances often found in the ambient air.

Despite this drawback, the Committee did agree that certain levels of air pollutants, in their best judgement, were associated with adverse effects on health. Owing to the uncertainty of the dose-response relationships, the use of a safety factor is prudent even when standards are derived

FIG. 1. SCHEMATIC SPECTRUM OF BIOLOGICAL RESPONSE TO POLLUTANT EXPOSURE <sup>a</sup>

<sup>a</sup> Based on a diagram in United States Congress Document No. 92-241, 1972.

from air quality guides. The magnitude of such a safety factor will depend upon many considerations. These may be political considerations with the main emphasis on cost-benefit analysis; they may be related to the significance and reliability of the data, including whether the experimental evidence was obtained from animals or human beings; or they may depend on the specific effect against which protection is sought—mortality or some lesser effect.

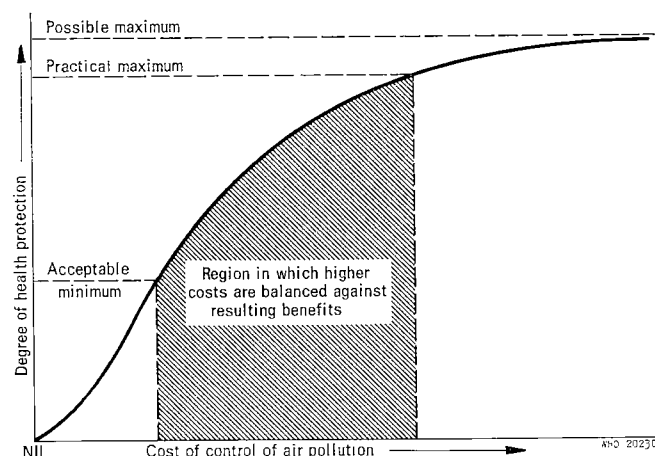
Such standards may vary from country to country and within a country over the course of time. The development of national air pollution standards <sup>1</sup> should include both standards to be met in a few years and long-term goals. In some countries in the immediate future, it may be necessary to base standards on tolerable levels, with intermediate goals of preventing illness and death in susceptible subgroups of the population. Certainly, the long-term goal should be to protect against all effects relevant to human health, including somatic and genetic change, and a safety factor should be adopted to ensure such protection. This means that pollutant levels should be as low as possible and the number of persons exposed as small as possible. It should be pointed out, however, that the concept of a standard to protect the population from significant harm is a statistical one, necessarily based on an aggregate of individual cases of significant harm. Obviously, the adoption of such a standard may not confer such protection on every individual.

<sup>1</sup> *Standards of environmental quality* are guides that have been adopted by governments and other competent authorities and therefore have legal force. In some contexts, however, standards may include recommendations that need not be rigidly enforced (*Wld Hlth Org. techn. Rep. Ser.*, 1970, No. 439, p. 37).

## 6.2 Health protection and air pollution control costs

Fig. 2 is a schematic diagram presenting several of the principal factors in the decision-making process concerning air pollution control. It indicates that the degree of health protection attained is a function of pollution control costs. The minimum acceptable level of health protection is, at the very least, that level necessary to protect from death; and, as stated above, the intermediate standards adopted for air pollution control should certainly protect from illness as well.

FIG. 2. SCHEMATIC REPRESENTATION OF DEGREE OF HEALTH PROTECTION AS A FUNCTION OF COST OF AIR POLLUTION CONTROL



The degree of health protection to be selected above the minimum acceptable level is a matter for political decision. The appropriate authorities must decide on the level of health protection desirable for their society. Increments of health protection above the minimum acceptable level are generally purchased at ever-increasing increments in control costs. Furthermore, the costs of the control programme are directly related to the deadline by which it is to be operational; for example, it is more expensive to achieve the desired goals in 3 years than in 10 years. The zone in which increased health protection (benefit) is obtained at increasing control costs (the cross-hatched area in Fig. 2) is also the region of social decision-making. The level of protection desired must of course take into account the existing air pollution effects, but other considerations are also important, including general social, cultural, and economic factors, as well as the magnitude of other health problems.

Another issue faces environmental administrators responsible for making recommendations for the control of sulfur oxides and suspended

particulates: the ratio between these pollutants varies from country to country, and there is no information documenting equivalent effects for the various concentrations of the two pollutants.

### **6.3 Relationship between standards with different averaging times**

In this report, effects are described as related to pollutant concentrations measured over short periods and over longer periods, indicating that different exposure times may be associated with different effects. This raises a problem for the air pollution control agency, for it must be sure that the air quality standards adopted will protect from the effects of both short-time and long-time exposures. To solve this problem one must know, for example, the relationship between the annual mean 24-hour value and the daily 24-hour values. If the effects against which protection is sought are known to be produced by exposure for 24 hours or less, then any control measure stipulating an annual mean 24-hour value must take note of the variations expected and state the number of days per year on which the specified concentrations may be reached. Similarly, if the undesirable effect is caused by exposure to the annual mean 24-hour value, then the stipulated 24-hour value must make allowance for the expected variations so that the annual mean is not exceeded. Further study of this question would be desirable.

The development of national air quality standards requires that work on air quality guides be concerned not merely with tolerable levels of pollutants but with desirable air quality as well. Although in the immediate future it may be necessary to base standards on tolerable levels, the long-term goal should be to achieve a desired air quality. Man should be able not only to survive but also to enjoy life.

## **7. SELECTION OF SHORT-TERM AND LONG-TERM GOALS**

### **7.1 Short-term goals**

The terms of reference for the Committee were primarily to formulate air quality criteria and guides for some urban air pollutants. These data have been presented in the preceding sections and may be used by countries wishing to set air quality standards. The general philosophy for the interpretation of air quality criteria and guides with a view to developing standards has been discussed in section 6; and it has been stressed that standards, particularly those chosen as short-term goals, may evolve differently in different countries depending on the exposure conditions, the socioeconomic situation, and the importance of other



health problems. On the basis of present knowledge, the Committee felt that it could only make the general statement that severe effects are obviously to be avoided.

## 7.2 Long-term goals

The situation is somewhat different for long-term goals. Without giving priority to the adverse effects of air pollutants over other health problems, it is the opinion of the Committee that exposure to the air pollutants discussed in this report should be kept as low as possible, as the subthreshold levels are not well defined at present and probably will not be defined with any great degree of certainty for a long time to come.

Since knowledge concerning the health effects of the pollutants discussed above decreases sharply as their concentrations drop, any forecast of the possible effects of levels below those given in the preceding sections must necessarily be speculative. Drawing on the information available, however, it is possible to set a level between these concentrations and the natural background level that the Committee would like to see adopted as an ultimate goal, with the hope that this intermediate level would be unlikely to produce any ill effects at all. Taking into consideration all the evidence available to it, the Committee reached the consensus that, in the light of present knowledge, the following recommendations could be offered as long-term goals intended to prevent undesirable effects from the air pollutants under discussion (Table 5). It emphasized, however, that these are tentative recommendations subject to change as and when more data on dose-response relationships within different populations become available.

TABLE 5. RECOMMENDED LONG-TERM GOALS \*

Pollutant and measurement method		Limiting level
<i>Sulfur oxides</i> <sup>a</sup> — British Standard Procedure <sup>b</sup>	annual mean	60 $\mu\text{g}/\text{m}^3$
	98 % of observations <sup>c</sup> below	200 $\mu\text{g}/\text{m}^3$
<i>Suspended particulates</i> <sup>a</sup> — British Standard Procedure <sup>b</sup>	annual mean	40 $\mu\text{g}/\text{m}^3$
	98 % of observations <sup>c</sup> below	120 $\mu\text{g}/\text{m}^3$
<i>Carbon monoxide</i> — nondispersive infrared <sup>b</sup>	8-hour average	10 $\text{mg}/\text{m}^3$
	1 hour maximum	40 $\text{mg}/\text{m}^3$
<i>Photochemical</i> — oxidant as measured by neutral buffered KI method expressed as ozone	8-hour average	60 $\mu\text{g}/\text{m}^3$
	1-hour maximum	120 $\mu\text{g}/\text{m}^3$

\* The Committee specifically urged that this table should not be considered independently of the accompanying text (see section 7.2).

<sup>a</sup> Values for sulfur oxides and suspended particulates apply only in conjunction with one another.

<sup>b</sup> Methods are not those necessarily recommended but indicate those on which these units have been based. Where other methods are used an appropriate adjustment may be necessary.

<sup>c</sup> The permissible 2 % of observations over this limit may not fall on consecutive days.

## 8. RECOMMENDATIONS

(1) Governments should establish and keep under review national air quality standards as part of their programmes for air pollution control. The air quality standards should aim primarily at the protection of health; in the Committee's opinion, the levels of air pollutants and the corresponding effects discussed in the present report may provide useful guidance toward the attainment of this objective.

(2) Long-term goals along the lines of those suggested in Table 5 should also be set, and the progress made towards achieving them should be periodically reviewed in the context of socioeconomic development and other public health problems.

(3) In establishing air quality standards, in addition to health effects governments should give consideration to the impact on climate, vegetation, animal life, and materials, as well as on the aesthetic quality of the environment. These effects have significant social, cultural, and economic implications, and are sometimes more sensitive indicators of air quality than are the effects on health.

(4) In addition to stating the concentration limit for the pollutant, air quality standards should specify methods of measurement, the average time over which concentrations should be measured, and the frequency with which the limit may be exceeded. A detailed plan for the implementation of standards should be developed at the same time, including appropriate monitoring schemes to permit assessment of the exposure of the population.

(5) In view of the inadequacy of presently available data, more studies should be performed on specific populations subject to comparatively high or low exposure to pollutants, using comparable study designs and uniform methods for measuring pollutant levels and biological response. It is also recommended that WHO stimulate international cooperative research in those parts of the world where unusual air pollution patterns now exist and assist Member Countries in conducting epidemiological studies to obtain comparable data on the health effects of air pollutants.

(6) Further experimental work should be conducted on human volunteers and animals in order to clarify the mechanism of action of environmental pollutants and to develop new and more reliable indices for use in epidemiological studies.

(7) WHO should take the initiative in developing and adopting reference methods for the measurement of air pollutants and for epidemiological methodology. This should help to eliminate some of the

difficulties experienced in comparing and evaluating available data on health effects.

(8) An appropriate mechanism should be developed to enable WHO, on a continuing and systematic basis, to collect information on the health effects of environmental pollutants, to review the information for its relevance to criteria and guides for environmental and air quality standards, and to survey new technological processes and new chemicals in order to identify possible environmental pollutants.

(9) WHO should publish detailed critical reviews for each pollutant.

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