BRITISH COLUMBIA HYDRO AND POWER AUTHORITY

HAT CREEK PROJECT

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CRITERIA FOR NATIONAL AIR QUALITY OBJECTIVES

SULPHUR DIOXIDE, SUSPENDED PARTICULATES, CARBON MONOXIDE, OXIDANTS (OZONE) AND NITROGEN DIOXIDE

Reports to the Federal–Provincial Committee
on Air Pollution (1971 and 1973)

by

The Subcommittee on Air Quality Objectives

November 1976

ISSUED UNDER THE AUTHORITY OF THE HON. ROMEO LEBLANC, MINISTER OF FISHERIES AND THE ENVIRONMENT

PREFACE

Clear evidence exists of the direct relationship between high air pollution levels and human health; however, this relationship becomes uncertain at low levels. Because, of this evidence Canada's Department of National Health and Welfare, like health departments in various parts of the world, was instrumental in developing the federal role in the control of air pollution.

Late in 1969, the Department of National Health and Welfare established an Air Pollution Control Division as part of its Environmental Health Directorate. For better coordination of air pollution control and abatement activities in Canada, an ad hoc Federal-Provincial Committee on Air Pollution was also established.

At its first meeting in December 1969, the Federal-Provincial Committee agreed to the development of National Air Quality Objectives and on the need of an expert subcommittee to undertake this task. This subcommittee was established in November 1970, and undertook the studies reported in this document.

With the creation of the Department of the Environment in 1971, responsibilities for air pollution control were transferred to the Air Pollution Control Directorate of the Environmental Protection Service. The Federal-Provincial Committee on Air Pollution was formally recognized by the Minister of the Environment as an advisory body. Under the Clean Air Act, proclaimed in November 1971, the Subcommittee on National Air Quality Objectives is continuing its activities under the aegis of the Committee.

The Subcommittee members are pleased to acknowledge the generous assistance and advice of Mr. A. Grignon, who also provided secretarial services on behalf of the Air Pollution Control Division.

AVANT-PROPOS

Nous avons de trés bonnes preuves que des niveaux élevés de pollution atmosphérique affectent la santé des humains. Cette relation de cause à effet devient toutefois plus difficile à établir à des niveaux inférieurs. C'est pourquoi le ministère canadien de la Santé nationale et du Bien-être spcoa;, tout comme les organes compétents de plusieurs autres pays, a contribué à établir le rôle fédéral dans la lutte contre la pollution de l'air.

Vers la fin de 1969, le ministère de la Santé nationale s'est donné une Direction de la lutte contre la pollution atmosphérique, intégrée à sa Direction de l'hygiène du milieu. Par ailleurs, pour favoriser la coordination des activités visant à réduire et à prévenir cette pollution au pays, un comité spécial fédéral-provincial a été créé.

A sa première réunion, en décembre 1969, ce comité s'est dit favorable à l'élaboration d'objectifs nationaux pour la qualité de l'air; il a, en outre, souhaité la création d'un sous-comité d'experts pour s'acquitter de cette tâche. Créé en novembre 1970, ce dernier a immédiatement entrepris les études dont fait état le présent document.

A la création du ministère de l'Environnement, en 1971, le mandat de lutte contre la pollution de l'air est passé à la Direction générale de la pollution atmosphérique du Service de la protection de l'environnement. Le nouveau ministère a, en outre, officiellement reconnu le Comité.

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Maximum acceptable limit	1 h	900	0 34		35	30	160	0.08	400	0.21
	8 h				15	13				
	24 h	300	0.11	120			50	0.025	200	0.11
	1 yr	60	0.02	70			30	0.015	100	0.05
Maximum desirable limit	1 h	450	0.17		15	13	100	0.05		
	8 h				6	5				
	24 h	150	0.06				30	0.015		
	1 yr	30	0.01	60			20	0.01	60	0.03

Conversion factors used to convert mass units to volume units of concentration (at 25°C and 760 mm Hg).

Sulphur dioxide: 3.82×10^{-4}
Carbon monoxide: 0.87
Oxidants (ozone): 5.1×10^{-4}
Nitrogen dioxide: 5.32×10^{-4}

1 INTRODUCTION

At their second meeting, held November 18, 1970, the Federal-Provincial Committee on Air Pollution recommended the establishment of an expert subcommittee for the purpose of developing air quality objectives.

The Subcommittee on National Air Quality Objectives was convened on March 17 and 18, 1971, and accepted the following terms of reference:

1. To list air contaminants for which National Air Quality Objectives (NAQO) should be prepared and to decide the order in which these contaminants should be considered.

2. To determine the criteria for establishing limits (as defined in the Clean Air Act) at which control action will be required.

3. To recommend the concentrations of specific contaminants at which such action will be required: i.e., to define for specific contaminants, in numerical terms, demarcation lines in relation to scientific effects on the basis of best available knowledge.

4. To report the scientific findings to the Federal-Provincial Committee on Air Pollution in the form of recommendations.

The following contaminants, listed in order of priority, have been reviewed and discussed by all members of the Subcommittee to date:

Sulphur dioxide
Suspended particulates
Carbon monoxide
Oxidants (ozone)
Nitrogen dioxide

For the purpose of recommending numerical values for concentration limits in the acceptable and desirable ranges, the following definitions were used:

The Maximum Acceptable Level is intended to provide adequate protection against effects on soil, water, vegetation, materials, animals, visibility, personal comfort and well-being. It represents the realistic objective today for all parts of Canada. When this level is exceeded, control action by a regulatory agency is indicated.

Clarification. The maximum acceptable level is that which will most directly concern control agencies in their day-to-day operations. When this level is exceeded control action is indicated. This level is intended to provide *adequate* protection for all parts of the environment. It corresponds in concept to the U.S. Federal Secondary Air Quality Standards. It also corresponds in concept to the present Ontario Ambient Air Quality Criteria. In any federal pronouncements or publications on this topic the maximum acceptable evel would be given the most prominence.

The Maximum Desirable Level defines the long-term goal for air quality and provides a basis for an antidegradation policy for the unpolluted parts of the country and for the continuing development of control technology.

Clarification. The maximum desirable level is based on a concept which is somewhat unique in the field of air pollution. It has, however, been applied in various ways in the radiation field, in drinking water standards, and by the World Health Organization for health standards. The concept of a long-term goal for air quality provides guidance for land-use planners and for the continuing development of control technology. At levels below the maximum desirable limit there would, in essence, be 'no effect' on any receptor. Persuasion and financial incentives would be the principal methods used to attain this objective.

2 SULPHUR DIOXIDE

In setting recommended concentration limits, the following factors are deemed pertinent based on the present available information:

2.1 Effects

2.1.1 ***Animal.*** Studies show that sulphur dioxide has a toxic effect on animals at concentrations considerably higher than might be anticipated in even severely polluted atmospheres. It is difficult, however, to extrapolate these data meaningfully for lower concentrations.

It is apparent from a review of numerous toxicity studies (1) that animals exhibit a higher resistance to sulphur dioxide than man; therefore, animal toxicity has not been considered a factor in limiting concentrations.

2.1.2 ***Human.*** Interpretation of the effects of sulphur dioxide on man must take into account that 'short period-high concentration' effects are derived from laboratory studies considering a single pollutant, whereas 'long period-low concentration' effects are derived from morbidity and mortality studies where the synergistic effects of other pollutants such as particulates are considered.

2.1.3 ***Vegetation.*** At high concentrations, plants are more susceptible to sulphur dioxide damage than man; however, phytotoxicity is affected by numerous factors including temperature, relative humidity, soil moisture, light intensity, nutrient supply and age of plant tissue.

2.1.4 ***Materials.*** The effect of sulphur dioxide on materials has been documented quite extensively on a qualitative basis; however, little quantitative data is available on short-term effects.

2.1.5 ***Compounding Effects.*** It must also be remembered that sulphur dioxide may oxidize in the ambient atmosphere in the presence of sufficient moisture to form sulphuric acid mist which brings about a three to fourfold potentiation of the irritant response and an increased deleterious effect on all forms of life and materials.

The effects of sulphur dioxide and associated suspended particulates at various concentrations are summarized in Table 2.

2.2 Background Level

The background level of sulphur dioxide is essentially nil.

TABLE 2 SUMMARY OF THE EFFECTS OF SULPHUR DIOXIDE AND ASSOCIATED SUSPENDED PARTICULATES

Effects	Concentration	
	Sulphur dioxide	Associated suspended particulates
Vegetation		
- injury to white pine (2)	90 $\mu g/m^3$ (0 034 ppm) annual avg.	
Materials		
- corrosion of mild, low-carbon steel (3)	70 $\mu g/m^3$ (.027 ppm) annual avg.	
Human Health		
- Increased absence from work, industrial workers (4)	250 $\mu g/m^3$ (0.096 ppm) 24-h avg.	200 $\mu g/m^3$, 24-h avg.
- Accentuation of symptoms in patients with chronic lung disease (5)	600 $\mu g/m^3$ (0.23 ppm) 24-h avg.	300 $\mu g/m^3$ (smoke) 24-h avg.
- Sharp rise in illness rates among bronchitics older than 54 years (6)	715 $\mu g/m^3$ (0.27 ppm) 24-h avg.	150 $\mu g/m^3$ (partic) 24-h avg.
- Increased hospital admissions (7)	300 $\mu g/m^3$ - 500 $\mu g/m^3$ (0.11-0.19 ppm) 24-h avg.	low particulates
- Increased mortality (7, 8, 9)	500 $\mu g/m^3$ (0.19 ppm) 24-h avg. 715 $\mu g/m^3$ (0.27 ppm) 24-h avg. 1500 $\mu g/m^3$ (0.57 ppm) 24-h avg.	low particulates 750 $\mu g/m^3$ (smoke) 6 cohs (soiling index)
- Increased frequency of respiratory disease symptoms & lung disease (10)	105-265 $\mu g/m^3$ (.040-.101 ppm) 24-h avg.	185 $\mu g/m^3$ 24-h avg.
- Increased frequency & severity of respiratory disease among school children (11)	130 $\mu g/m^3$ (0.050 ppm) ann. avg.	100-130 $\mu g/m^3$ annual avg.
- Increased mortality: bronchitis, lung cancer (12)	115 $\mu g/m^3$ (0.044 ppm) annual average	160 $\mu g/m^3$ annual avg.
- Odour easily detected (13)	7.8 mg/m^3 (3.0 ppm)	
- Detection by taste (13)	78 - 2.62 mg/m^3 (.3 - 1.0 ppm)	

NOTE: For suspended particulates annual average means geometric mean.
For SO_2 concentrations annual average means arithmetic mean.

2.3 Recommended Limits for Sulphur Dioxide (1971)

Maximum Desirable Limits

1-h avg:	450 $\mu g/m^3$ (0.17 ppm)
24-h avg:	150 $\mu g/m^3$ (0.06 ppm)
1-yr avg:	30 $\mu g/m^3$ (0.01 ppm)

Criteria: *No health effects.*
Nil background values.

Maximum Acceptable Limits

1-h avg:	900 $\mu g/m^3$ (0.34 ppm)
24-h avg:	300 $\mu g/m^3$ (0.11 ppm)
1-yr avg:	60 $\mu g/m^3$ (0.02 ppm)

Criteria: *Vegetation effects at 90 $\mu g/m^3$ (0.034 ppm) annual avg(2).*
Materials effects at 70 $\mu g/m^3$ (0.027 ppm) annual avg(3).
Increased hospitalization above 300 $\mu g/m^3$ (0.11 ppm) 24-h avg(7).

NOTE:
- Arithmetic mean values are used unless otherwise noted.
- When considering health, limits suggested for sulphur dioxide should be taken in conjunction with the limits suggested for particulates. Values for either one or the other should be used as the applicable limit.
- See Table 2 summarizing information on health effects of sulphur dioxide and particulates on health.

3 SUSPENDED PARTICULATES

3.1 Properties and Occurrence of Suspended Particulates

'Suspended particulate' is a general term which applies to a wide variety of solid or liquid particles of a size and configuration such that they tend to remain suspended in the air and can thus be drawn into the respiratory passages.

By general agreement, the term is restricted to particles under 100μ in diameter. In the present context, it excludes substances with a known specific toxic effect (arsenic, asbestos, benzo-a-pyrene, beryllium, sulphuric acid mist, etc.) and refers only to those which are, chemically, relatively inert. In general, only those below 5μ in diameter can penetrate to the deeper regions of the lung.

In accepting 'suspended particulate' as a descriptive term, one must bear in mind that the actual composition of the material, its physical state and size distributions may profoundly affect its action.

3.2 Effects

Particulate material is the most commonly perceived form of pollution, often manifesting itself by interference with visibility, a soiling effect on materials, or as a respiratory irritant.

In cases of occupational exposure, workers may be exposed to relatively high concentrations without obvious effect. As a general air pollutant, particulate is usually thought of in association with sulphur dioxide. The particulate could be toxic in itself, it could be a vehicle for carrying sulphuric acid mist into the lungs, or it might somehow interfere with normal pulmonary hygiene -- the self-cleansing mechanisms of the lung.

In laboratory experiments, it is necessary to use high concentrations of particulate, 1000 $\mu g/m^3$ or more to obtain reproducible effects (9).

Since the results of animal experiments cannot be applied to man, we look mainly to epidemiological studies for information about the effect of particulates on health. These may occur as an increase in mortality or morbidity or an exaggeration of symptoms in those who are already ill.

Many studies have noted such responses to pollution but none of these provide clear evidence of health effects when particulate alone is below 300 $\mu g/m^3$ for a 24-hour average (10).

Some studies suggest that there is a health effect when the *annual* average is above 130 $\mu g/m^3$ and this is noted in reference 14. In this and other such studies, it is always postulated that there is a concomitant presence of sulphur dioxide at the same or a higher concentration (130 $\mu g/m^3$ in this case).

3.3 **Discussion.** In view of the above, the selected limits have been based on interference with visibility and on nuisance effects which do occur at lower particulate concentrations. References 15 and 16 give an indication of the proportion of people who might complain of pollution when the particulate level is 60 $\mu g/m^3$ and 200 $\mu g/m^3$. Reference 17 deals with the effect of particulates on visibility.

It will be noted that, under Maximum Desirable Limits, no 24-hour level value has been assigned. This is purposely omitted because of the fact that any number lower than the 24-hour 'acceptable' (i.e. 120 $\mu g/m^3$) could easily be exceeded at any time because of natural phenomena (e.g. windstorm).

3.4 **Recommended Limits for Suspended Particulates (1971)**

Maximum Desirable Limits

1-yr avg: 60 $\mu g/m^3$

Criteria: Background values: 40-70 $\mu g/m^3$ annual avg (18).
Social awareness of pollution: 50-60 $\mu g/m^3$ (15).
Short-term value not given; see Section 3.3.

Maximum Acceptable Limits

24-h avg: 120 $\mu g/m^3$
1-yr avg: 70 $\mu g/m^3$

Criteria: Visibility reduced below 5 miles when particulate exceeds 100 $\mu g/m^3$ (17).
Increased frequency and severity of lower respiratory disease among children where annual smoke concentration and SO_2 both exceeded 130 $\mu g/m^3$ (14).

NOTE:
- When considering health, the limits suggested for particulates should be taken in conjuncation with those for SO_2; values for either one or the other should be used as the applicable limit.
- The above limits do not apply to chemically-active particulates.
- Information on size distribution and nature of particulate material should be available.
- One-year average to be expressed as annual geometric mean.
- See Table 2 summarizing information on health effects of SO_2 and particulates.

4 CARBON MONOXIDE

The following discussion of carbon monoxide is based on a review of *Air Quality Criteria for Carbon Monoxide* (19) and of papers published in the available literature since January 1970. Pertinent references are summarized in the Appendix.

4.1 Properties and Occurrence of Carbon Monoxide

Carbon monoxide (CO) is an odourless, colourless, tasteless gas with a specific gravity of 0.968 relative to air. The amount measurable in relatively unpolluted air (background value) ranges between 0.029-1.15 mg/m^3 (19). Carbon monoxide concentrations in ambient air correspond to man's pattern of activities (for example, automobile traffic) and meteorological factors (atmospheric stability), causing diurnal, weekly and seasonal variations.

Carbon monoxide is also produced endogenously in man and animals and by marine hydrozoa and vegetation. Endogenous production in man occurs as a by-product of haeme catabolism and amounts to 0.42 ± 0.07 ml/h (20). Larger amounts are produced in haemolytic disease states.

Carbon monoxide combines with haemoglobin to form carboxyhaemoglobin (COHb). Carboxyhaemoglobin is formed 200-250 times more readily, mole for mole, than the corresponding oxygen-haemoglobin compound, oxyhaemoglobin, resulting in a displacement of oxygen and, hence, an interference with the oxygen carriage to tissues. Endogenously produced carbon monoxide results in a biological background level of 0.5% COHb(19). Exposure to known concentrations of carbon monoxide in air will give rise to reasonably predictable levels of COHb in blood (21). Carboxyhaemoglobin concentrations increase with cigarette smoking and correlate with carbon monoxide variation in the ambient air (22).

Carbon monoxide not only displaces oxygen from haemoglobin (a tissue hypoxia-producing effect) but interferes with the release or unloading of oxygen at the tissue level. Uptake of carbon monoxide by blood increases, and equilibrium between gas mixtures and COHb concentrations is reached more rapidly with increasing concentrations of carbon monoxide, increasing length of exposure and increasing ventilation rates.

4.2 Effects

The *acute* effects of carbon monoxide at high doses are well documentec (23). Less extensively documented are the effects of lower concentrations such as those found in urban environments. A number of studies report the acute or subacute physiological effects of low COHb blood levels and the possible effects of low ambient air concentrations of carbon monoxide on mortality and morbidity.

The studies of Ayres et al (24) indicate quite convincingly that potentially serious adverse effects may occur in patients with coronary artery disease when blood COHb levels reach 5% or more.

The decrease in the oxygen-carrying capacity of the blood represented by such levels may prove lethal to a small group among this sensitive section of the population. With regard to the additional load imposed by such levels of COHb (or hypoxia) it is of interest to note that Chevalier (25), studying the effect of carbon monoxide on the degree of oxygen debt produced in response to exercise, found that there was a significant increase in oxygen debt when related to the total increased oxygen uptake at blood COHb levels of 3.95% ± 1.87%; and that the study of Brody and Colburn (26) indicates that there is an additive effect when patients with existing degrees of oxygen lack (other forms of heart ailment or emphysema) are exposed to carbon monoxide.

That concentrations of carbon monoxide in the ambient air may also have an effect on mortality was suggested by Cohen et al (27) who found an increased mortality due to myocardial infarctions during weeks when community carbon monoxide levels averaged more than 10 ppm. This suggestion was recently repeated in a paper by Hexter and Goldsmith (28). The effects of similar concentrations of carbon monoxide on morbidity are uncertain.

The occurrence of *chronic* effects has been suggested by the results of animal studies in which injury to the arterial wall was induced in rabbits by 10%-15% COHb for eight weeks and the uptake of cholesterol was enhanced (29,30): and by studies in dogs in which the occurrence of electrocardiographic changes accompanied by evidence suggesting degeneration of the heart muscle have been demonstrated following COHb levels of up to 12% for six weeks (31). Changes in the morphology of the brain and central nervous system in dogs at approximately 20% COHb for eleven weeks were also reported (32) but the latter could not be distinguished from those attributable to cerebral anoxia secondary to coexistent myocardial damage. Recently, significant losses of trace elements at the subcellular level were demonstrated in a pilot study by Mazaleski et al (33) carried out on rats (50 ppm; twelve weeks), animals otherwise relatively resistant to the effects of carbon monoxide. Trace element effects, however, diminished towards the end of the exposure period. Correlation of carbon monoxide and hormonal disturbances have also been reported (34) together with effects on enzyme systems (35).

Chronic health effects in humans have been suggested by a number of case studies. However, studies of workers occupationally exposed to carbon monoxide over long periods at concentrations higher than those found in urban atmospheres failed to demonstrate such effects (36, 37).

The effect of carbon monoxide on *performance* has been studied extensively. Decreased performance has been well demonstrated in rats (38, 39).

Human data pertaining to decreased performance at low COHb blood values have been controversial. Shulte's work (40) suggested that an effect could be detected at a COHb level of more than 2% and at least 5% the limit of accuracy of the method used for the determinations of blood COHb concentrations. Horvath et al (41) found a highly significant decrease in vigilance among nonsmokers at average COHb levels of 6.6%, but no decrement among smokers.

Beard and Wertheim (38) produced results which suggested an impairment of time-interval discrimination in a group of nonsmokers at extrapolated blood values of approximately 2.5% COHb

(COHb values not being available). Stewart et al (42) and Mikulka (43) were unable to confirm measurable impairment of performance at such low COHb levels. There were, however, differences in design of the experiments carried out by the above investigators (group setting as opposed to individual setting; single blind as opposed to double blind studies etc.). In addition, insufficient emphasis appeared to have been placed on environmental factors such as those affecting motivation and attention to task performance (including factors in the laboratory environment and the interest or states of fatigue and boredom on the part of the participant). Nevertheless, the most definitive studies, at present, are those of Stewart et al. These cast doubt upon the significance of human performance effects at levels below 5% COHb concentration in blood.

Physiological or performance effects which appear to have been confirmed include those of McFarland et al (44) demonstrating an increased visual threshold following exposure to carbon monoxide (increasing with COHb levels from 5% 20%); and of Halperin et al (45) who detected impairment of visual function at COHb levels between 4% - 5%.

The lowered oxygen pressures at higher altitudes tend to aggravate the effect of carbon monoxide. Although the general effects of altitude and carbon monoxide exposure are similar, physiological responses differ (46).

Experimental evidence suggests that some degree of adaptation may occur through such mechanisms as increased haemoglobin, haematocrit red blood cell values (47) and possibly increased circulating blood volume. Human data are inconclusive.

Vegetation damage due to carbon monoxide is not anticipated at the average concentrations found in urban atmospheres.

4.3 Discussion

(a) The available information indicates that COHb saturation levels of 5% and over can produce effects in man which may affect performance and pose additional physiological stress on patients with chronic heart and respiratory disease.

(b) Evidence of significant adverse effects at COHb saturation levels of 2%-4% is less well established.

(c) No detrimental effects have been noted at COHb saturation levels below 2%.

4.3.1 Interpretation. In arriving at national air quality objectives for carbon monoxide, the following guidelines were used:

a. In order to protect all sensitive groups of the population (except perhaps on a theoretical basis extremely small groups of cardiac patients) ambient air concentrations of carbon monoxide should be such that COHb levels do not exceed 5% saturation in nonsmokers.

b. In view of repeated suggestions of a significant effect on performance at COHb saturation levels between 2.5% and 5%, it is reasonable to apply a safety factor to the definite effect level of 5% COHb and to aim at COHb saturation levels below 3% in nonsmokers.

In interpreting the above in terms of acceptable limits, the following equation described by Larsen (48) was modified for use:

$$C_{COHb} = a\ c_{co}\ t + E$$

where

C_{COHb} = degree of COHb saturation

a = activity factor
- (i) = 0.018 under conditions at rest with a tidal volume at 6 l/min
- (ii) = 0.048 under conditions of light to moderate work with a tidal volume of 18 l/min
- (iii) = 0.1 under conditions of heavy work (arbitrarily selected)

C_{co} = concentration of CO in air in ppm

t = length of exposure in hours with maximum of 12 hours at which time equilibrium conditions are assumed to be operative

E = 0.5% COHb representing endogenous CO production

Note:

In adapting the activity factors to the periods selected for monitoring purposes, factors of 0.075 and 0.025 were selected as appropriate for the 1-hour and 8-hour average concentrations respectively. This was done on the assumption that moderate to strenuous activity was possible and would provide for most situations during the 1-hour period, and that for the 8-hour period the selected activity factors represented conditions of resting to moderate activity.

4.4 Recommended Limits for Carbon Monoxide (1971)

Maximum Desirable Limits

1-h avg:	15 mg/m^3 (13 ppm)
8-h avg:	6 mg/m^3 (5 ppm)
Criteria:	Repeated suggestions re deleterious effects of COHb values below 3% (38, 28).

Maximum Acceptable Limits

1-h avg:	35 mg/m^3 (30 ppm)
8-h avg:	15 mg/m^3 (13 ppm)
Criteria:	Physiological stress on patients with heart disease: COHb more than 5% (24). Impairment in performance of some psychomotor tests: 5% COHb (40). Impairment of visual acuity: 5% COHb (44, 45). Increased metabolic cost of heavy work; O_2 debt increases 14% at 3.95% COHb (25).

NOTE: Eight-hour average is an arithmetic mean.

5 OXIDANTS (OZONE)

5.1 Properties and Occurrence of Oxidants

Total oxidants include all atmospheric oxidants which can be measured by oxidation of iodide ions. Oxidants in the atmosphere comprise ozone, organic peroxy-nitrogen compounds and nitrogen dioxide. Ozone and peroxy-nitrates are secondary products resulting from the chemical interaction of primary pollutants (nitrogen dioxide and reactive hydrocarbons) in the presence of intense sunlight. Thus, nitrogen dioxide not only takes part in the formation of photochemical oxidants but is also formed from nitric oxide in a photochemical reaction. Only about 10% of the nitrogen dioxide appears as equivalent ozone in the measurement of total oxidants. Under some circumstances it can make up an appreciable fraction of total oxidants (49). During periods of high photochemical activity, ozone is the major oxidant present (90% or more of the total oxidizing capacity). Because adverse effects on the health of man, animals and vegetation can be expected on high-oxidant days the activity of atmospheric oxidants is attributed primarily to the activity of the ozone content. Control of the precursors (nitrogen oxides and reactive hydrocarbons) will automatically reduce the production of ozone and other oxidants by photochemical reaction.

Oxidant (ozone) levels not much lower than those known to be toxic occur naturally in the atmosphere. For other contaminants such as sulphur dioxide, the detectable level is approximately the same as the background concentration. Ozone, however, can occur in appreciable quantities in both rural and forested areas remote from major sources of the precursor ingredients of the photochemical reaction. In the forest, terpenes emitted by coniferous foliage, and nitrogen oxides released from the soil, can contribute to the photo-oxidation process. A layer of ozone, about .20-.25 ppm in concentration, surrounds the earth at a distance of about 15 miles. Convection currents may at times bring some of this ozone into the troposphere.

Studies conducted at Chalk River, Ontario, 200 miles north of Toronto, showed that the natural background level of oxidants during the growing season of 1965 was 0.011 ppm (arithmetic mean). The maximum concentration measured was 0.06 ppm with 99.5% of the measurements (15-min averages) being less than 0.05 ppm (64).

5.2 Effects

Some of the effects of oxidants discussed in this section are summarized in Table 3.

5.2.1 ***Materials.*** Rubber cracks when subjected to an ozone concentration of 0.02 ppm for 1 hour.

Degradation of cellulose, resulting in weakened fabrics and fading of colour in dyed materials, can occur at ambient concentrations of oxidants. Effects on materials have been observed in urban atmospheres but little is known about exact concentrations of oxidants and exposure times required.

TABLE 3 SUMMARY OF THE EFFECTS OF OXIDANTS

Effect	Oxidant concentration and exposure time
Materials	
- Rubber cracking (53)	0.02 ppm (ozone), 1h
Vegetation	
- Slight injury under sensitive conditions to:	
Bean (51)	0.15 ppm (ozone), 0.5h
Tomato (54)	0.08 ppm (ozone), 1h
Tobacco (55)	0.05 ppm (ozone), 4h
Bean (51)	0.03 ppm (ozone), 8h
Peanut (56)	0.02 ppm (ozone), 24h
Bacteria	
- >90% mortality to *Streptococcus salivarius* (57)	0.025 ppm (ozone), 0.5h
Animal	
- Increased susceptibility of mice to bacterial infection (58)	0.08 ppm (ozone), 3h
Human	
- Odour detection (59)	0.02 - 0.05 ppm (ozone), few min.
- Increased airway resistance (60)	0.10 ppm (ozone), 1h
- Threshold limit value	0.10 ppm (ozone), 8h/day, 5 day week
- Impairment of performance (student athletes) (61)	0.07 ppm (oxidants), 1h
- Eye irritation (62)	0.10 ppm (oxidants), instantaneous
- Increase in asthma attacks (63)	0.15 ppm (oxidants), 1h

5.2.2 ***Vegetation.*** The concentration-time dosages of ozone that will cause slight injury to sensitive vegetation grown under the most sensitive conditions are listed in Table 3. There is considerable genetic variability in response to oxidants. The threshold limits given in Table 3 are for extremely sensitive plant species and varieties which are not representative of the total population of that species.

In addition to visible injury produced at the levels of ozone shown, there may be subtle manifestations. Physiological responses such as reduction in yield, quality, and growth of plants may occur in the absence of visible markings.

The combined effect of two or more gases may lower the threshold levels of the individual contaminants required to cause injury to vegetation. The following combinations of gases have been found to act synergistically on tobacco plants:

0.027 ppm ozone + 0.24 ppm sulphur dioxide for 2 h (50)
0.03 ppm ozone + 0.10 ppm sulphur dioxide for 4 h (51)

5.2.3 ***Human.*** The conclusions of the United States Public Health Service document, *Air Quality Criteria for Photochemical Oxidants* (52), set 0.07 ppm for 1 hour as the lowest limit for health effects. In order to comply with regulations for establishing air quality standards individual states must set standards to allow for a margin of safety with regard to health effects. Thus, New York State recently proposed a change in their standard for total oxidants from 0.15 ppm for 1 hour to 0.06 ppm for 1 hour. On January 30, 1971, the Environmental Protection Agency (EPA) proposed a United States National Air Quality Standard (primary and secondary) of 125 $\mu g/m^3$ (0.06 ppm) maximum 1-hour concentration for photochemical oxidants. The enforcement of this standard would be difficult because, as explained earlier, the background concentration can occasionally equal the prescribed dosage. On April 30, 1971, EPA published a more realistic standard of 160 $\mu g/m^3$ (0.08 ppm) as a maximum 1-hour concentration not to be exceeded more than once a year.

5.3 Discussion

The maximum desirable limits were set as a long-term national goal for all parts of Canada. Concentrations of oxidants (ozone) below these limits in combination with other contaminants should not affect living organisms. Materials are affected by natural ambient air levels of oxidants.

Difficulties may be encountered in meeting the maximum desirable limits at certain times of the year. Studies conducted (64) on the natural background levels of atmospheric oxidants have shown that these levels can be appreciable and that they fluctuate constantly. In the forests at Chalk River, Ontario, the 24-hour average concentration of oxidants varied from slightly under 0.01 ppm to slightly over 0.04 ppm. Diurnal fluctuations showed oxidant levels at night usually less than 0.01 ppm, increasing in the morning hours to a peak of 0.03 ppm around noon and then decreasing in the evening hours. Measurements were made over three years, 1965 to 1967, and the maximum oxidant concentration recorded was 0.06 ppm for 4 hours which occurred between 1500 and 1900 hours on two occasions, June 27, 1965, and August 17, 1967. In 1965, 99.5% of the ambient air measurements (15-min averages) were less than 0.05 ppm for oxidants (Figure 1). The arithmetic mean for 6868 measurements was 0.011 ppm.

Vegetation is not susceptible to injury by dosages less than 0.08 ppm ozone for 1 hour (51,54). Impairment of student performance occurred when oxidant levels between 0.067 and 0.163 ppm existed in the hour before an athletic event (61). These health effects have not been corroborated and because background levels can approach the lower concentration a realistic maximum acceptable limit for oxidants (ozone) would be 0.08 ppm for 1 hour.

In this paper the term 'ozone' or 'oxidant' has been used where appropriate. In the past, atmospheric oxidants were measured by oxidation of iodide ions and ambient air measurements of oxidants reflected the total oxidant content. In laboratory 'effects' experiments (health, vegetation, bacteria, animals and materials) pure ozone was used. As it is now possible to measure atmospheric ozone specifically, by a chemiluminescent method, it would be prudent to establish National Air Quality Objectives for both total oxidants and oxidants (ozone).

5.4 Recommended Limits for Oxidants (Ozone) (1971)

Maximum Desirable Limits

1-h avg:	100 $\mu g/m^3$ (0.05 ppm)
24-h avg:	30 $\mu g/m^3$ (0.015 ppm)
1-yr avg:	20 $\mu g/m^3$ (0.01 ppm)

Criteria: No known acute or subacute health effects. Minimal materials effects (rubber cracking) at 0.02 ppm for 1 h (53). Vegetation effects - slight injury to peanut plants at 0.02 - 0.03 ppm for 24 h to 48 h (56). Synergistic effects of ozone in combination with other substances causing vegetation damage, e.g., tobacco: 0.027 ppm ozone + 0.024 ppm SO_2 for 2 h (50), 0.03 ppm ozone + 0.10 ppm SO_2 for 4 h (51).

Maximum Acceptable Limits

1-h avg:	160 $\mu g/m^3$ (0.08 ppm)
24-h avg:	50 $\mu g/m^3$ (0.025 ppm)
1-yr avg:	30 $\mu g/m^3$ (0.015 ppm)

Criteria: Above minimum level for physiological effects (impairment of performance of student athletes at 0.07 ppm for 1 h (61) but natural background concentrations approach this level. Vegetation damage, e.g. tomato plants: 0.08 ppm for 1 h (54). Animal experimental data indicating increased susceptibility of mice to bacterial infection: 0.08 ppm ozone for 3 h (58).

Background Levels: Maximum 0.06 ppm. Under 0.05 ppm 99.5% of the time. Annual arithmetic average 0.011 ppm. 24-h and 1-yr averages are arithmetic means (64).

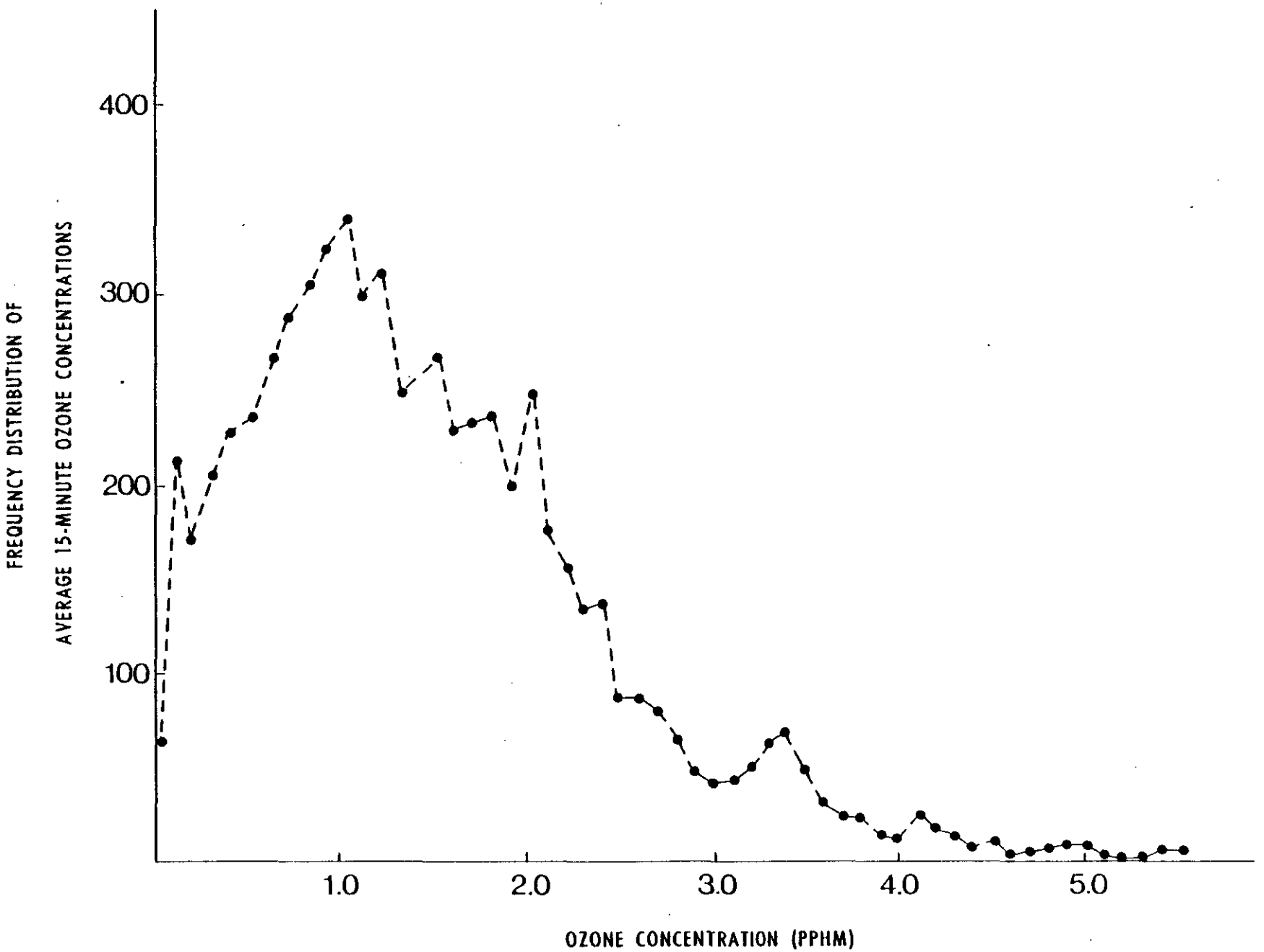

FIGURE 1 BACKGROUND CONCENTRATIONS OF OZONE IN FOREST AREAS (64)

6 NITROGEN DIOXIDE

EPA's publication *Air Quality Criteria for Nitrogen Oxides* (65) and other reference material was reviewed to assist in arriving at a proposal for National Air Quality Objectives with respect to nitrogen dioxide at the Maximum Desirable and Maximum Acceptable Levels.

6.1 Properties and Occurrence of Nitrogen Oxides

Various nitrogen compounds are present in the atmosphere and stem from both natural and man-made sources. It is significant to note that the balance between total pollutant and natural emissions of nitric oxide (NO) and nitrogen dioxide (NO_2) is in the ratio of almost 1:15, the natural emissions in the form of nitric oxide being much greater than the pollutant emissions (66). This does not mean, however, that emissions of nitrogen oxides (NO_x) from man-made sources are unimportant.

Of the nine atmospheric forms of nitrogen which include the gases N_2O, NO, NO_2, N_2O_4 N_2O_3, and N_2O_5 and the ammonium (NH_4+), nitrite (NO_2-), and nitrate (NO_3-) aerosols, the most significant pollutants emitted by man's activities are nitric oxide and nitrogen dioxide. By convention their sum is termed nitrogen oxides.

Nitric oxide is a colourless, odourless gas, slightly soluble in water, while nitrogen dioxide is an orange-brown gas, with a characteristic pungent odour. Nitrogen dioxide is much more toxic and irritating than nitric oxide. It reacts with water to form nitric acid (HNO_3) and either nitrous acid (HNO_2) or nitric oxide.

Nitrogen oxides originate chiefly in combustion processes in which the nitrogen and oxygen in the combustion air are subjected to temperatures in excess of 1093 °C and in which the combustion gases are quenched rapidly enough to prevent dissociation back to nitrogen and oxygen. The major oxide in combustion emissions is nitric oxide. A small fraction of nitric oxide is converted to nitrogen dioxide by reaction with oxygen during the exhaust dilution process. This oxidation is chiefly a result of a photochemical interaction between nitrogen oxides and hydrocarbons.

The significance of nitrogen oxides, particularly nitrogen dioxide, to air pollution cannot be overemphasized, since they affect human health, suppress growth of vegetation, and cause corrosion of metals. Nitrogen dioxide being a highly coloured gas, reduces atmospheric visibility and is responsible for a portion of the brownish discolouration of air masses over and near urban areas.

Further reduction in visibility is due to oxidation of sulphur dioxide to sulphur trioxide, accelerated by the presence of nitrogen dioxide. The most significant factor for pollutant-induced decrease in atmospheric visibility is, however, photochemical smog which consists of aerosols formed from certain hydrocarbons in the presence of nitrogen oxides and sunlight.

Section 5 of this report indicates that one of the consequences of sunlight-induced reactions in polluted ambient atmospheres is the formation of new compounds termed oxidants. These oxidants,

mainly ozone, have undesirable effects not only on human health but also on vegetation, materials and welfare (67). The rate of formation and maximum concentration of oxidants is a complex function of the concentration of hydrocarbons, the concentrations of nitric oxide and nitrogen dioxide, the structure of hydrocarbons, the light intensity, and the temperature. Nitrogen oxides together with hydrocarbons are oxidant precursors. The reaction products are responsible for adverse effects such as powerful eye irritation (68) and production of phytotoxicants which cause substantial crop loss (69).

Even though nitric oxide is a relatively nontoxic gas, it tends to convert to nitrogen dioxide quite rapidly in the atmosphere with consequences of the kind mentioned above. Control of man-made nitrogen oxide emissions is therefore of prime importance to clean air, particularly because of possible buildup during adverse weather conditions.

As mentioned earlier the total amount of nitrogen oxides generated by natural sources exceeds the amount from man-made sources by a factor of about 15. Natural scavenging processes (66) keep background levels in nonurban areas low. Typical concentrations of nitrogen dioxide reported (70) vary from 1.8 $\mu g/m^3$ - 7.1 $\mu g/m^3$. In urban areas, however, the levels are much higher, because pollutants are added faster than the scavenging processes can control them. Observed levels of nitrogen oxides are summarized in Table 4. It will be noted that mean annual concentrations range from 17 $\mu g/m^3$ to a maximum of 113 $\mu g/m^3$ with a peak concentration of 1436 $\mu g/m^3$ recorded in Edmonton during 1968.

At CAMP (Continuous Air Monitoring Program) stations, in the United States, the arithmetic average nitrogen dioxide concentrations during 1968 ranged from 40 $\mu g/m^3$ in St. Louis to 95 $\mu g/m^3$ in Chicago and Washington (71). The annual average ratio of nitrogen dioxide to nitrogen oxides varied from 40% to 60%. Applied to the measured Canadian levels of nitrogen oxides, these percentages yield the estimated nitrogen dioxide concentrations shown in Table 4. This table reveals that the Canadian levels are similar to those of major American cities.

The reported (72) maximum nitrogen dioxide concentration for a period of 5 minutes in Los Angeles from 1962 through 1968 was 2387 $\mu g/m^3$. Other major American cities exhibited 5-minute peak values ranging from 677 $\mu g/m^3$ to 1635 $\mu g/m^3$. Further details on American levels can be obtained from reference 71.

Reference was previously made to the work of Robinson and Robbins (66) who studied the cycles of the various nitrogen compounds through the atmospheric environment. It was indicated that the scavenging of nitrogen dioxide and ammonia occurs both through aerosol formation and by surface reactions with vegetation and other materials.

The residence time of nitrogen dioxide based on the atmospheric nitrogen cycle derived in the presence of moisture was estimated at only 3 days; however, the half-life of 1 ppb of nitrogen dioxide in the atmosphere in the presence of 5 ppb ozone was found to be about 2 weeks. The authors stated:

"From an air pollution standpoint the natural scavenging processes are probably not rapid enough to affect the hour-to-hour concentrations in urban areas. However, there would appear to be little reason to expect any long-term buildup of these nitrogen compounds in the global environment".

TABLE 4 OBSERVED LEVELS OF NITROGEN OXIDES (NO_x) IN CANADIAN CITIES

City		Mean annual concentration ($\mu g/m^3$)	Peak concentration ($\mu g/m^3$)	Estimated mean annual NO_2 concentrations ($\mu g/m^3$)
Windsor	1969	38	282	15 - 23
	1970	75		30 - 45
Sarnia	1969	36		14 - 22
	1970	57		10 - 14
Hamilton	1970	75		30 - 45
Toronto	1967	71		28 - 43
	1968	71		28 - 43
	1969	66	526	26 - 40
	1970	113		45 - 68
Calgary (4 mo)	1969	66	972	26 - 40
Edmonton	1966	23	649	9 - 14
	1967	17	818	7 - 10
	1968	24	1436	10 - 14
	1969	24	1021	10 - 14
Westmount*	1969			75
Montreal*	1969			70
	1971			26
	1972			26

NOTE: NO_x values are expressed in terms of NO_2.
*The levels are those for nitrogen dioxide only.

The reference to long-term buildup was not qualified. One should stress, however, that we are dealing with urban situations where at times, due to adverse atmospheric conditions, unduly high buildups of nitrogen oxides over several days could have significant effects on health.

6.2 Effects

6.2.1 Materials. Investigators have found that certain dyed cellulosics fade under high humidity conditions during the drying cycle in gas-fired home dryers. Nitrogen dioxide levels in the dryers ranged from 1100 to 3700 $\mu g/m^3$ (73). Similar effects at lower concentrations cannot be ruled out.

It has been concluded that corrosion of metals is promoted by the presence of anions, principally nitrates, in accumulated dust. Nitrates in urban atmospheres have been identified as end products of photochemical reactions between nitrogen oxides and hydrocarbons. Available information on urban areas shows average airborne nitrate particulate concentrations of 3.0 and 3.4 $\mu g/m^3$, corresponding to annual average gaseous nitrogen oxides levels of 124 and 158 $\mu g/m^3$, respectively (73).

6.2.2 Vegetation. Plant damage such as leaf abscission and decreased yield were reported after Navel oranges were continuously exposed for 8 months to nitrogen dioxide at 470 $\mu g/m^3$ (74). Besides the direct effect of nitrogen dioxide one must consider the plant damage caused by oxidants or other toxicants.

It has been stated that exposure of plants to oxidants need not be frequent to cause substantial damage. One episode is enough to severely damage or ruin an entire crop. Nitrogen oxides need to be controlled because of their specific effects and because they participate in producing oxidants (75). Synergism of other pollutants in community atmospheres is also an important factor contributing to plant damage. A variety of tobacco was damaged by laboratory exposure beginning at 1300 $\mu g/m^3$ of sulphur dioxide alone or 3760 $\mu g/m^3$ of nitrogen dioxide alone, but it was also damaged by exposure to a concentration of 260 $\mu g/m^3$ sulphur dioxide plus 190 $\mu g/m^3$ nitrogen dioxide for 4 hours (75).

6.2.3 Animals. The lowest nitric oxide concentration shown to be harmful to date is 470 $\mu g/m^3$. Structural changes in lung collagen were reported after exposures of 4 h/day for 6 days in the case of rabbits (76). Investigators have further reported that nitrogen dioxide reduces the ability of an animal to combat bacterial lung infections (77).

6.2.4 . Humans.

Experimental Exposures. The odour of nitrogen dioxide was perceived by less than half of a group of young, healthy males when exposed to 225 $\mu g/m^3$, while more than half immediately sensed the odour at the 415 $\mu g/m^3$ level. All of the subjects perceived the odour at the 835 $\mu g/m^3$ level (78).

The additive effects of nitrogen dioxide and sulphur dioxide on pulmonary function have only been studied at relatively high concentrations. It is significant to note that sulphur dioxide increased the inspiratory and expiratory flow resistance immediately after inhalation, while nitrogen dioxide showed a maximum resistance 30 minutes after the end of the 10-minute exposure. A mixture of nitrogen dioxide and sulphur dioxide confirmed the bimodal increase in resistance (79).

Nitrogen dioxide appears to exert its primary toxic effect on the lungs and can be associated with increased susceptibility to respiratory infection and emphysematous changes (80).

Epidemiological Exposures. A unique study was undertaken in the Chattanooga area in response to histological, biochemical, and clinical studies implicating nitrogen dioxide as a possible potentiator of respiratory disease in man. The Chattanooga study is considered to support that evidence. Results have been discussed in detail by Shy et al (81, 82). It was concluded that the ventilatory performance of second grade school children in the high-nitrogen dioxide exposure area was significantly lower than that of children in the control areas. Further, illness-incidence rates for each family segment in the high-nitrogen dioxide area were both consistently and significantly higher than incidence rates in the two control areas throughout the entire study period. The increased incidence of acute respiratory disease was observed when the 24-hour nitrogen dioxide concentration, measured over 6 months, was between 117 and 205 $\mu g/m^3$. Exposure to nitrogen dioxide and to suspended particulates appears to be the most probable explanation for the observed increase in respiratory illness rates.

A retrospective study of acute lower respiratory illness among infants and first and second graders was made by Pearlman et al (83). Parents were asked to report the frequency of croup, bronchitis, pneumonia and asthma, during the 3-year period from July 1966 through June 1969. Sensitivity and specificity exceeded 67% for each clinical diagnosis. Excess bronchitis was noted among those residing in the high-exposure area. This greater frequency of acute bronchitis was observed when the mean 24-hour nitrogen dioxide concentration, measured over 6 months, was between 118 $\mu g/m^3$ and 156 $\mu g/m^3$ (84).

The Pearlman study confirmed Shy's observation that excessive acute respiratory illness can be found among children living in nitrogen dioxide - polluted areas.

Results of the Chattanooga studies imply that any site that exhibits a concentration of 113 $\mu g/m^3$ or greater, exceeds the Chattanooga health effect-related nitrogen dioxide value.

In the Chattanooga study only nitrogen dioxide concentrations were measured because this gas was considered the major nitrogen oxide attributable to one major source: the manufacture of trinitrotoluene (TNT) which employs large amounts of nitric acid. Results collected in that area by the United States army during 1967 and 1968 show that the ratio of NO:NO_2 is about 0.38 meaning that nitrogen dioxide concentrations are more than twice those of nitric oxide.

6.3 Discussion

Larsen (85) has stated that cumulative damage for some pollutants may be approximately proportional to the total number of pollutant molecules to which a person has been exposed. This total is the air pollution dosage and is equal to concentration times exposure time. For instance, most of the nitrogen oxides inhaled remain in the lungs and react with the lung or its contents. This implies that long-term exposure even at very low concentrations could have an adverse health effect; however, no precise information on such effects is available at this writing.

The proposed National Air Quality Objectives for Nitrogen Dioxide are based in part on nuisance effects observed as odour perception in experimental exposures and on the epidemiological studies described above. The Chattanooga study, spanning 3 years, is the most authoritative

epidemiological investigation carried out to date. In a critical review of the National Air Quality Standards of the United States, Heuss et al (86) had major objections about the measurement of nitrogen dioxide by the Jacobs-Hochheiser method and the interpretation of the medical results. Barth et al (87) have more than adequately defended their conclusions on the subject. Based on recent studies of the reference method (EPA) for the analysis of nitrogen dioxide, Hauser and Shy in a "Position Paper" (88) reported new facts about the methodology used in the Chattanooga study and concluded: "The several independent methods available to estimate nitrogen dioxide exposure during the Chattanooga health study do not support a revision of the National Air Quality Standard for Nitrogen Dioxide". This last paper discusses the nonlinear varying collection efficiency of the Jacobs-Hochheiser method with varying nitrogen dioxide concentrations and the positive interference of nitric oxide for some concentrations. Applying these corrections, the results of the Chattanooga study would give values 11% higher.

Further consideration has been given to synergistic effects with photochemical oxidants and sulphur dioxide, and to the possibility that some of the available analytical data may tend to be lower than actual values. In other words, it has been necessary to compromise to some extent, with a view of revising the limits if and when more extensive data become available.

The numerical values for the recommended national air quality objectives are based primarily on the following considerations:

Maximum Desirable Limits

1 year at 60 $\mu g/m^3$ is about ten times the maximum nitrogen dioxide natural background concentration and concentrations more than double this value have been found in some small urban centres where no adverse health effects have ever been reported.

Maximum Acceptable Limits

1 hour at 400 g/m^3 is slightly below the 415 $\mu g/m^3$ level immediately perceptible to the majority of young, healthy people (78) and is about one-tenth the value at which increase in both inspiratory and expiratory flow resistance occurs immediately after exposure (79) in the presence of equal concentrations of sulphur dioxide.

4 hours at 190 $\mu g/m^3$ NO_2 and 260 $\mu g/m^3$ SO_2 causes plant injury.

24 hours at 200 $\mu g/m^3$ no evidence of any health effects even in the presence of 260 $\mu g/m^3$ SO_2.

1 year at 100 $\mu g/m^3$ is slightly below the ranges shown to have adverse long term health effects in the Chattanooga investigations (81, 82, 83).

In the absence of other systematic studies similar to the Chattanooga investigations and with the knowledge of synergistic effects, it is recommended, that the following National Air Quality Objectives be adopted.

6.4 Recommended Limits For Nitrogen Dioxide (1973)

Maximum Desirable Limits

1-yr avg.	60 $\mu g/m^3$
Criteria:	No known acute human health effects. Animals and vegetation are generally more resistant than humans. No adnormal effects observed on materials.

Maximum Acceptable Limits

1-h avg.	400 $\mu g/m^3$
24-h avg.	200 $\mu g/m^3$
1-y avg.	100 $\mu g/m^3$
Criteria:	Immediate odour perception by a majority of young, healthy people at 415 $\mu g/m^3$. Adverse health effects from long-term exposure at levels in excess of 100 $\mu g/m^3$ (81, 82, 83). Lowest concentration shown to be harmful to animals is 470 $\mu g/m^3$ (76). Plant damage as the result of synergistic effects on exposure to a mixture of 190 $\mu g/m^3$ of NO_2 and 260 $\mu g/m^3$ of SO_2 each for 4 h (75).

APPENDIX - EFFECTS OF CARBON MONOXIDE, SUMMARY

A. SUMMARY OF PERTINENT HUMAN DATA - PREVIOUSLY REPORTED IN U.S. CRITERIA DOCUMENT

Environmental conditions	Effect	Comment	Reference
1. 35 mg/m^3 (30 ppm) for up to 12 h.	Equilibrium value of 5% blood COHb is reached in 8-12 h; 80% of this equilibrium value, (4% COHb) is reached within 4 h.	Experimental exposure of nonsmokers. Theoretical calculations suggest exposure to 23 (20 ppm) and 12 mg/m^3 (10 ppm) would result in COHb levels of about 3.7% and 2%, respectively, if exposure was continuous for 8 h or more.	Smith (21)
2. 58 mg/m^3 (50 ppm) for 90 min.	Impairment of time-interval discrimination in nonsmokers.	Blood COHb levels not available, but anticipated to be about 2.5%. Similar blood COHb levels expected from exposure to 10-17 mg/m^3 (10-15 ppm) for 8 h or more. Single blind procedure.	Beard and Wertheim (38)
3. 115 mg/m^3 (100 ppm) intermittently through a facial mask.	Impairment in performance of some psychometor tests at a COHb level of 5%.	Similar results may have been observed at lower COHb levels, but blood measurements were not accurate.	Schulte (40)
4. High concentrations of CO were administered for 30-120 s, and then 10 min was allowed for washout of alveolar CO before blood COHb was measured.	Exposure sufficient to produce blood COHb levels above 5% has been shown to place a physiologic stress on patients with heart disease.	Data rely on COHb levels produced rapidly after short exposure to high levels of CO; this is not necessarily comparable to exposure over a longer time period or under equilibrium conditions.	Ayres et al (24)
5. 24 levels of exposure; less than 1 ppm to more than 500 ppm; Duration: 1-24 h.	50 ppm: COHb at 8 h = 5.1% at 12 h = 7.0% at 22 h = 8.5% at 24 h = 7.9% 100 ppm: COHb - 11-13% no effect on performance. Time interval estimations not affected at COHb several fold higher than demonstrated in 2 above. At 20% COHb objective evidence of intoxication. Also changes in visual evoked response marked at 30% COHb.	Healthy subjects (18). Only 3 smokers who abstained for period of study. CO excretion studies reported in companion paper (37). COHb and CO breath concentrations during and following exposures. Double blind procedure.	Stewart et al. (42) (see also Arch. Env. Health, 21: 154, Aug. 1970).

6.	0, 58 and 144 mg/m^3 (0, 50, 125 ppm) for 3 h	No effect on tests of time estimation, tracking task or vestibular function at COHb levels of 2 98% and 6 64%.	Nine nonsmokers aet. 19–22 controlled for training effect. Double blind.	Mikulka et al. (43)
7	CO gas mixtures with different percentages O_2 to simulate various altitudes.	Visual threshold increased as COHb levels increased from 5%–20%. Effects of CO hypoxia and altitude equivalent.	Trained male subjects 16–25 yrs. Fingerprick samples for determining COHb – at beginning and during test.	McFarland et al. (44)
8.	Measured amounts of pure CO ranging from 100–300 ml; through mask for 10–15 min.	COHb levles up to 20% observed. Measurable impairment in visual function detectable when COHb concentrations reached 4%–5%: greater degrees of impairment at higher concentrations.	Four healthy males aet 16–25 yrs.	Halperin et al (45)
9	CO: 5750 mg/m^3 (5000 ppm) in compressed air; inhaled for 2 5 to 3.5 min.	Increased oxygen debt at COHb levels of 3.95% ± 1.87% COHb.	COHb determined before and after exposures. Study of 10 nonsmokers. Sham exposures and double blind techniques not used.	Chevalier et al (25)
10.	Ambient atmosphere concentrations; in high (9–16 mg/m^3 (8–14 ppm)) and low CO pollution areas.	Significant correlations for weekly myocardial infarction case fatality rates and ambient CO levels during the week of admission.	Individuals hospitalized with acute cardiovascular disease; 35 hospitals in Los Angeles county in 1958. 3080 admissions involving separate calculations for high and low CO pollution areas. Factors other than CO exposure, such as medical care practices etc. were operative. Study requires confirmation.	Cohen et al. (27)

8. SUMMARY OF SELECTED RECENT PUBLICATIONS RE EFFECTS OF CARBON MONOXIDE

Environmental conditions	Effect	Comment	Reference
a. Human Studies			
1. 20 ppm, 1 h avg 10 ppm for a few days Over 5% COHb	COHb level attained requires 8 h at CO concentration of 5 ppm or less. Aggravation of patients with coronary thrombosis. Psychomotor impairment; accelerated death of patients with anaemia and impaired cirulatory function of important organs.	Used by Japan as guide-lines for standards. Considerations include time required to desorb CO from system (Report not yet available).	Expert Committee (89) Report
2. CO at 0, 26, 111 ppm	Nonsmokers had highly *significant decrement in* vigilance while breathing 111 ppm CO (average COHb of 6.6%). Smokers exhibited no decrement.	Fifteen males aet. 21-32 *yrs; ten nonsmokers*. COHb determined after 60 and 135 min of exposure. Full report not yet available.	Horvath et al. (41)
3. CO inhalation	Tissue hypoxia produced by CO may be accentuated by marked degrees of arterial hypoxemia in patients with heart and lung disease.	Five normal subjects; two with right to left shunts; and patients. Full report not yet available.	Brody and Coburn (26)
4. *Transport workers* exposed to 30-60 mg/m^3 CO.	*COHb among smokers* 3.9%-9.3%. COHb among nonsmokers 0.0%-5.5%. Percentage abnormal blood counts increasing with length of service. Hypochromia thought to be characteristic.	*140 bus drivers, 92 bus* conductors, and 245 truck drivers. Full report not yet available.	*Szollosi et al.* (90)
5. Inhalation of CO	Significant increase in *blood fibrinolytic* activity but no significant difference in fibrinolytic inhibitor levels.	Eight men with asbestosis, aet. *40-55 yrs. Full* report not yet available.	Menon and Pumford (91)
6. 24 h exposure to CO	COHb approx. 20%. Fall of 2.3 DPG (intraerythrocytic diphosphoglycerate)	Eight human subjects. Suggested explanation of why smokers with obliterating arterial diseases have higher oxygen affinity - contrary to Haldane effect.	Astrup (92)

7.	Ambient atmosphere concentrations in Los Angeles (basin averages). Highest weekly average concentration 20.2 ppm; lowest 7.3 ppm	CO as air pollutant associated with statistically highly significant excess mortality. Cyclic variation and maximum temperature were the main contributors.	Total number of deaths for each day from Jan. 1, 1962 to Dec. 31, 1965 and temperature with basin averages for CO and oxidant concentrations for the week preceding the study as variables. Findings re mortality attributed to arteriosclerotic heart disease similar and consistent with findings of Cohen.	Hexter and Goldsmith (28)
b.	Animal Studies			
1.	50 ppm CO, 5 h per day, 5 days per week (rats)	Consistent trace metal loss of five metal species studies (Zn, Co, Cu, Fe, Mg) Zn migrates out of liver and to lesser degree out of kidney, heart and lungs. Relatively massive quantities of cobalt lost from rat liver mitochondrial fractions. Indicates an overall reduction of cellular respiration and ATP production.	These subcellular effects demonstrated with exposure to so-called 'no effect' Threshold Limit Value. Tendency to return to normal after 3 month's exposure.	Mazaleski et al. (33)
2.	CO at 1 and 2 mg/m^3, SO_2 at 0.15 and 4 mg/m^3 CO + SO_2 at 2 & 4 mg/m^3 respectively. Female rats; all for 72 days.	Studies of estrous cycle and fertility revealed hormonal disturbances (in all cases excepting SO_2 at 0.15 mg/m^3) indicative of increased functioning of the pituitary body.	Fran Aptic abstract. Paper not yet available.	Mamatsushvili (34)
3.	Lethal doses of CO (also four other pollutants); mice	Female mice much more resistant to CO poisoning. Castration increases resistance to CO in males but decreases resistance in females.	Male and female mice in groups of ten. Full report not yet available.	Stupfel and Bonfey (93)
4.	60-150 ppm for 4 days; rats	Activity of B(a) P-hydroxylase slowly but significantly decreased	Authors comment re possible favourable retention of unchanged B(a)P in lung tissue. Paper not yet available.	Rondia and Gielen (35)

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