Air Quality Protection in the West (November 27-28)

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Health Effects of Air Pollutants (Sulfur Dioxide, Ozone, and Carbon Monoxide)
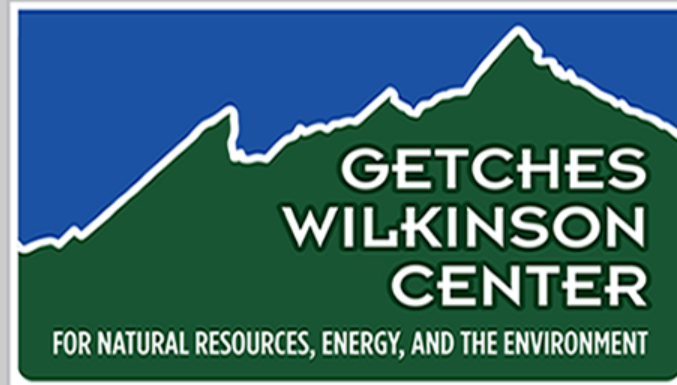
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HEALTH EFFECTS OF AIR POLLUTANTS
(Sulfur dioxide, ozone, and carbon monoxide)

Robert A. Bethel, M.D.
Department of Medicine
National Jewish Center for
Immunology and Respiratory Medicine
Denver, Colorado

AIR QUALITY PROTECTION IN THE WEST

Robert A. Bethel
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(Sulfur dioxide, ozone, and carbon monoxide)

Abstract

Air pollutants can have marked effects on health. These effects generally depend on the concentration of the pollutant in ambient air, the time of exposure, and the rate of ventilation during exposure. It is difficult to determine the long term effects of low concentrations of air pollutants because many pollutants are present simultaneously and the effects of low concentrations may occur over long periods of time. On the other hand, the short term effects of air pollutants in controlled exposures are easy to determine and have been fairly well defined. Sulfur dioxide causes asthma attacks. Persons who have asthma are especially sensitive to inhaled SO_2, which may cause typical asthma attacks with wheezing, cough, and shortness of breath. Some epidemiologic studies also suggest that inhaled particulate sulfates contribute to mortality in some American cities. Ozone affects chiefly airways and lungs. It may decrease respiratory mechanical function and cause throat dryness, chest tightness, substernal pain, cough, wheeze, and shortness of breath. Ozone also affects athletic performance and alters airway function in numerous ways. Ambient carbon monoxide has its greatest effects on persons with cardiovascular disease. In these persons carbon monoxide may induce angina pectoris or myocardial infarction ("heart attack") and may significantly decrease exercise tolerance.

Ambient air quality has improved considerably since passage of the Federal Clean Air Act about two decades ago. Nevertheless, current air quality standards sometimes fail to protect persons from the health effects of air pollutants. For instance, the lack of a short term standard for sulfur dioxide allows concentrations of SO_2 that may cause significant asthma attacks.

Whereas indoor air pollution is recognized as a problem itself, ambient concentrations of SO_2, ozone, and carbon monoxide are generally considerably less indoors than outdoors. A sensitive person can diminish the effects of these three pollutants by remaining indoors and by refraining from exertion during times of high pollution.

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I. Sulfur Dioxide (SO_2)

A. Sources:

1. <u>Fossil fuels</u>. Sulfur dioxide is released into air by the burning of sulfur-containing fossil fuels, such as coal or shale oil. Since all living matter contains sulfur and since sulfur is concentrated in the fossilization process, fossil fuels may contain large quantities of sulfur. Prime Virginia coal contains 1 - 2.5% sulfur and midwestern coal about 6%. Thus, burning coal to generate electricity, to generate energy in industrial processes, or to heat homes releases staggering amounts of SO_2. Those parts of the country that generate electricity by hydroelectric, geothermal, and nuclear power generally have lower ambient SO_2 concentrations than those that burn large amounts of coal. The refining processes that generate gasoline remove sulfur from crude oil, and so automobiles do not contribute greatly to SO_2 pollution.

2. <u>Mineral Smelters</u>. Sulfur dioxide is also produced in large quantities by mineral smelters which release sulfur dioxide from the sulfate and sulfite salts of the

minerals smelted. As most smelters are located away from large cities, some rural areas have the highest concentrations of ambient sulfur dioxide.

3. Natural sources. Volcanos and oceans also release SO_2.

B. Effects of SO_2 on Asthma:

Inhaled sulfur dioxide constricts airways and decreases airflow. Sufficient concentrations constrict airways even in healthy persons; however, low concentrations constrict airways in persons who have asthma. Sulfur dioxide causes typical asthma attacks with wheezing, chest tightness, and shortness of breath. These effects are of greatest concern where ambient concentrations of SO_2 are highest, where persons exercise and thus inhale larger doses of SO_2, and where persons exposed to SO_2 have more severe asthma. The determinants of SO_2-induced airway constriction include:

1. Concentration of ambient SO_2. In the concentration ranges encountered in ambient air, the greater the concentration of inhaled SO_2 after a threshold the greater the airway constriction.

2. Minute ventilation. The dose of SO_2 delivered to airways depends on the volume of air breathed. Therefore, increased ventilation during exercise increases the dose of SO_2. When breathed through a mouthpiece at rest, 5 ppm SO_2 causes airway constriction in healthy persons, but 1 ppm SO_2 causes similar airway constriction in persons who have asthma. When exercise increases minute ventilation, as little as 0.50 ppm SO_2 causes airway constriction in freely breathing asthmatics. Even 0.25 ppm SO_2 causes small degrees of airway constriction in some asthmatics.

3. Degree of airway reactivity. Persons who have asthma are more sensitive to SO_2 than heathy persons. Among asthmatics, the response of airways to inhaled SO_2 probably depends on the severity of asthma.

4. <u>Distribution of inhaled air between the nose and mouth</u>. Since SO_2 is a polar molecule which is highly water soluble, the moist mucous membranes of the nose absorb it efficiently from inhaled air, even at high airflow rates. On the other hand, because airflow through the mouth has less turbulence and less contact with moist membranes than in the nose, SO_2 is absorbed less efficiently. The dose of SO_2 that passes the throat to cause airway constriction depends on the distribution of airflow between the nose and mouth. During exercise, both oral and total ventilation increase; and greater quantities of SO_2 are delivered to the lower airways.

5. <u>Humidity and temperature</u>. The degree of bronchoconstriction induced by SO_2 also depends on the temperature and humidity of air. In persons who have asthma, some concentrations of SO_2 cause greater airway constriction when inhaled in cold, dry air than when inhaled in warm, moist air. This effect is probably due to the additive constrictor effects of SO_2 and cold, dry air. But it may also be due to the drying effect of cold, dry air on mucous membranes, which may allow greater passage of SO_2 through the nose to lower airways. Thus, SO_2 may cause greater airway constriction in cold, dry climates (such as the Rocky Mountain West) than in warm, moist ones. Also, SO_2 may interact with other agents that cause airway constriction in asthmatics such as fog, ozone, NO_2, particulates, cigarette smoke, and pollens.

C. Effects of SO_2 on mortality:

Epidemiologic studies have documented a number of air pollution disasters and have shown that severe air pollution causes significant health effects, especially in persons who have cardiovascular or lung disease or who are very old or very young. For example, in the Meuse River Valley of Belgium in December 1930, industrial emissions were trapped by an atmospheric thermal inversion. Thousands became ill and 60 died. In Donora, Pennsylvania, in October 1948, industrial emissions were

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similarly trapped by a thermal inversion. Six thousand of 14,000 residents became ill; the death rate increased from two expected to twenty during the period. In London in December 1952, soft coal was widely used for heating. An inversion trapped the pollutants produced by coal burning and industrial processes and caused 4,000 excess deaths in five days. In December 1962, 750 deaths were attributed to another inversion in London. In all these disasters, air pollution affected most severely the elderly and those who were already ill with heart and lung disease. More than one pollutant was responsible for the disasters, but the sulfur oxide-particulate complex is most often implicated as a causative factor. Other epidemiologic studies have indicated that as much as 4-6% of all mortality in selected American cities in 1980 was due to sulfur oxide-particulate complex pollution (4). However, the conclusions of these latter studies are controversial and require further investigation.

II. Ozone (O_3)

A. Sources:

1. Photochemical reaction. Ozone is formed by a complex photochemical reaction that requires organic vapors, nitrogen oxides, and sunlight. Automobile emissions are one of the important sources of organic vapors and nitrogen oxides. Therefore, where automobile traffic is heavy and where there is much sunlight, ozone air pollution is a problem. Since ozone is highly reactive, its concentration at ground level drops markedly in the evening.

2. Stratospheric ozone. Stratospheric ozone can intrude into the troposphere, especially in the spring when the stratospheric-tropospheric air exchange is greatest. The current concern about ozone depletion in arctic areas relates to stratospheric ozone.

Ozone concentration for three days
(Arvada)
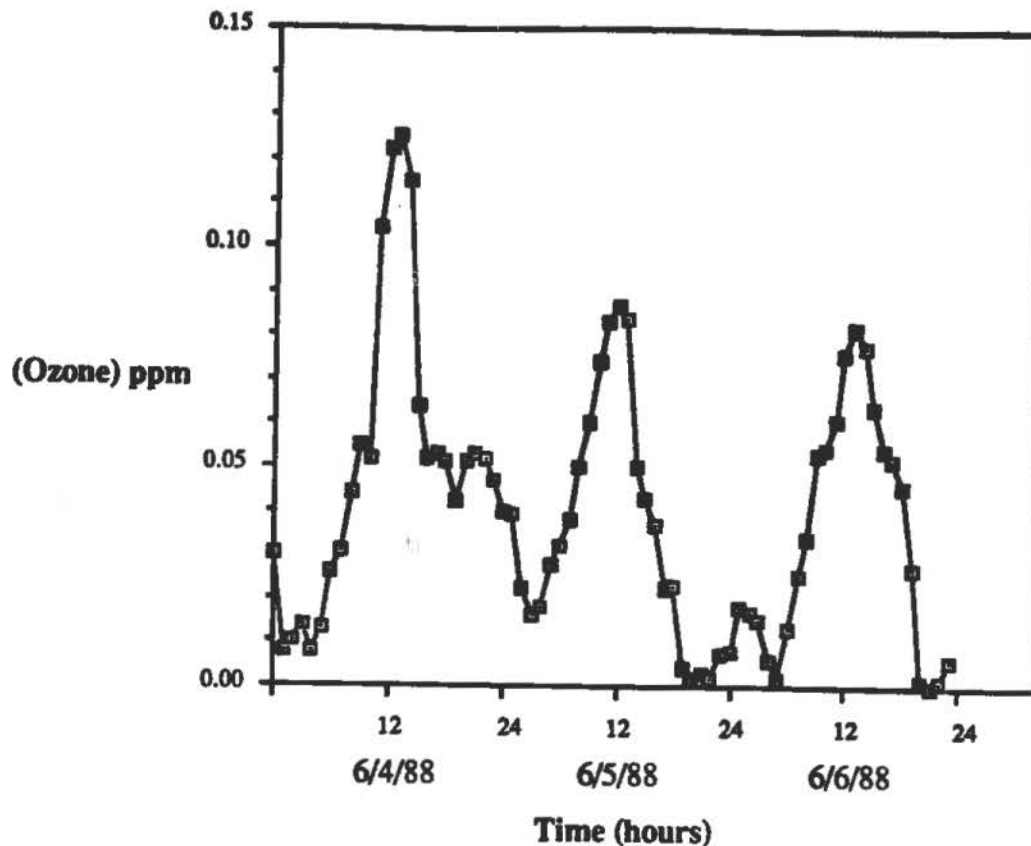
Data obtained by the Colorado Air Quality
Control Division during three of the worst days
for ozone pollution in 1988.

B. **Acute effects of ozone:**

No group of persons with pre-existing illness have been identified as having increased sensitivity to ozone. Therefore, attention is directed at the effects of ozone on healthy persons who exercise regularly outdoors, as a primary population of concern. Ozone affects chiefly airways and lungs:

1. <u>Effects on respiratory mechanical function</u>. Ozone causes concentration-dependent decrements in exhaled volumes and flow rates during forced expiratory maneuvers. The decrements increase with increasing depth of breathing (exercise) during exposure and are additive over several hours. The decrements in forced expiratory volume in 1 second (FEV_1) after 6.6 hours of exposure to 0.120 ppm average 13.6%.
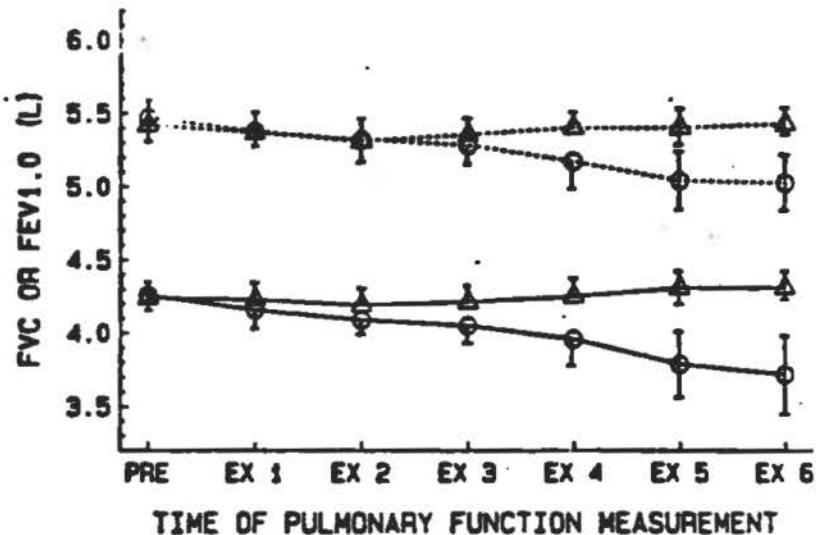
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Figure 4. Mean ± S.E. of forced vital capacity and forced expired volume in one second after each of six exercise periods. Dashed lines are FVC and solid lines are $FEV_{1.0}$; triangles represent air exposures and circles ozone exposures. From: Folinsbee et al.[37] JAPCA 1988; 38:28
Ozone concentration 0.120 ppm.

2. Symptoms. Adults exercising heavily while breathing 0.120 ppm ozone develop throat dryness, chest tightness, substernal pain, cough, wheeze, pain on deep inspiration, shortness of breath, dyspnea, lassitude, malaise, headache, and nausea.

3. Effects on athletic performance. Controlled exposure studies of heavily exercising competitive runners have demonstrated decreased function at 0.200-0.300 ppm ozone (2). Another study exposed ten young male adult endurance athletes to 0.120, 0.180, and 0.240 ppm ozone while exercising moderately for 30 minutes, followed by strenuous exercise for an additional 30 minutes. All ten subjects completed the protocol while breathing filtered air. However, 1, 5, and 7 of them could not complete the protocol while breathing 0.120, 0.180, and 0.240 ppm ozone (2).

4. Effects on airway reactivity. Reactivity of airways refers to their bronchoconstrictor sensitivity to inhaled irritants. Ozone increases the reactivity of airways in a concentration-dependent fashion. Exposure of subjects to 0.120 ppm ozone for 6.6 hours will approximately double the reactivity of airways.

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5. Effects on airway permeability. Inhaled ozone injuries the airway epithelium and decreases its barrier function. The permeability of the epithelium is thus increased. This effect may explain the increase in airway reactivity to inhaled irritants induced by ozone.

6. Effects on airway inflammation. Inhaled ozone can induce inflammation of the airways and cause the influx of cells and the release of chemical mediators of inflammation. This response depends on the concentration of ozone inhaled and the time of exposure. 0.100 ppm ozone, inhaled for 6.6 hours, induces significant airway inflammation.

7. Effect on particle clearance. Changes in the ability of the deep lung to clear deposited particles occur even before significant changes in respiratory function. Changes in airway particle clearance alter the defense mechanisms of the lungs and may alter the response to infectious agents.

8. Effects on lung infectivity. Increased susceptibility to bacterial infection has been reported in mice at 0.080-0.100 ppm ozone for a single 3-hour exposure.

C. Chronic effects of ozone exposure:

Since single exposures lasting for an hour or more at current peak ambient ozone levels produce measurable biologic responses in healthy humans, and since there is a high probability that one high ozone day will be followed by several more, it is important to know the extent to which the effects of ozone accumulate or ·progress over multiple days. Repetitive daily exposures, at a level which produces a functional response upon single exposure, result in an enhanced response on the second day, with diminishing responses on days 3 and 4, and virtually no response by day 5. This functional adaptation to exposure disappears about a week after exposure ceases (2). The adaptation phenomenon has led some people to conclude that transient functional decrements are not important health effects. On the other hand, recent

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research in animals has shown that persistent damage to lung cells accumulates even as functional adaptation takes place.

1. Controlled laboratory exposure. Studies at Rancho Los Amigos Hospital in southern California have found that subjects have greater functional decrements in lung function in the spring than in the fall following a summer of natural ozone exposure. These findings suggest natural adaptation to ozone.

2. Epidemiologic studies. Epidemiologic studies suggest that chronic ozone exposures do affect baseline respiratory function and cause respiratory function to decrease over time more rapidly than occurs normally.

Table 1. Annual change in lung function (males).

Population	FEV_1 (ml)	FVC (ml)
Tucson, AZ	-29	-30
Lancaster, CA	-46	-51
Glendora, CA	-48	-60

From ref 2. FEV_1 = forced expiratory volume in one second, FVC = forced vital capacity. Tucson exceeded a one-hour O_3 concentration of 0.120 ppm only once in 1981, 1982, and 1983. In Lancaster in 1985, there were 58 days with one-hour O_3 maxima greater than 0.120 ppm. In Azusa, adjacent to Glendora, there were 117 days in 1985 with one-hour maxima greater than 0.120 ppm.

III. Carbon Monoxide (CO)

A. Sources:

Carbon monoxide is produced by the incomplete oxidation (burning) of carbon containing substances.

1. <u>Exhaust from motor vehicles</u>. Exhaust from motor vehicles accounts for approximately 60% of total CO emissions per year. Tail pipe CO concentrations range from 0.5 to 7% depending on the year of automobile manufacture, the state of engine tuning, and the fuel burned. Concentrations of 25 ppm are encountered on expressways in major metropolitan areas during peak traffic periods. During weather inversions, CO concentrations may exceed 100 ppm.

2. <u>Stationary sources, industrial processes, and solid waste disposal</u>. These sources account for about 20% of total CO emission.

3. <u>Improperly vented hot water heaters, furnaces, space heaters, and fireplaces</u>.

4. <u>Tobacco smoke</u>. Most cigarette smokers who smoke one pack per day have a carboxyhemoglobin (COHb) saturation of 5-6% (% of hemoglobin bound by CO). Two to three pack per day smokers average 7-9% saturation, and heavy cigar smokers may have saturations of 20%. The COHb saturation from tobacco smoking adds to that from other CO sources.

5. <u>Paint stripper</u>. The basic ingredient is methylene chloride which is metabolized to CO. A 3 hour exposure to paint stripper vapor in a well ventilated room can cause COHb saturations of 8-16%.

6. <u>Normal metabolism of hemoglobin</u>. Metabolism of hemoglobin causes endogenous production of CO and accounts for the normal COHb saturation of 0.4-0.7%. The progesterone phase of the menstrual cycle and some drugs, such as phenobarbital and diphenylhydantoin, increase hemoglobin metabolism and increase CO production.

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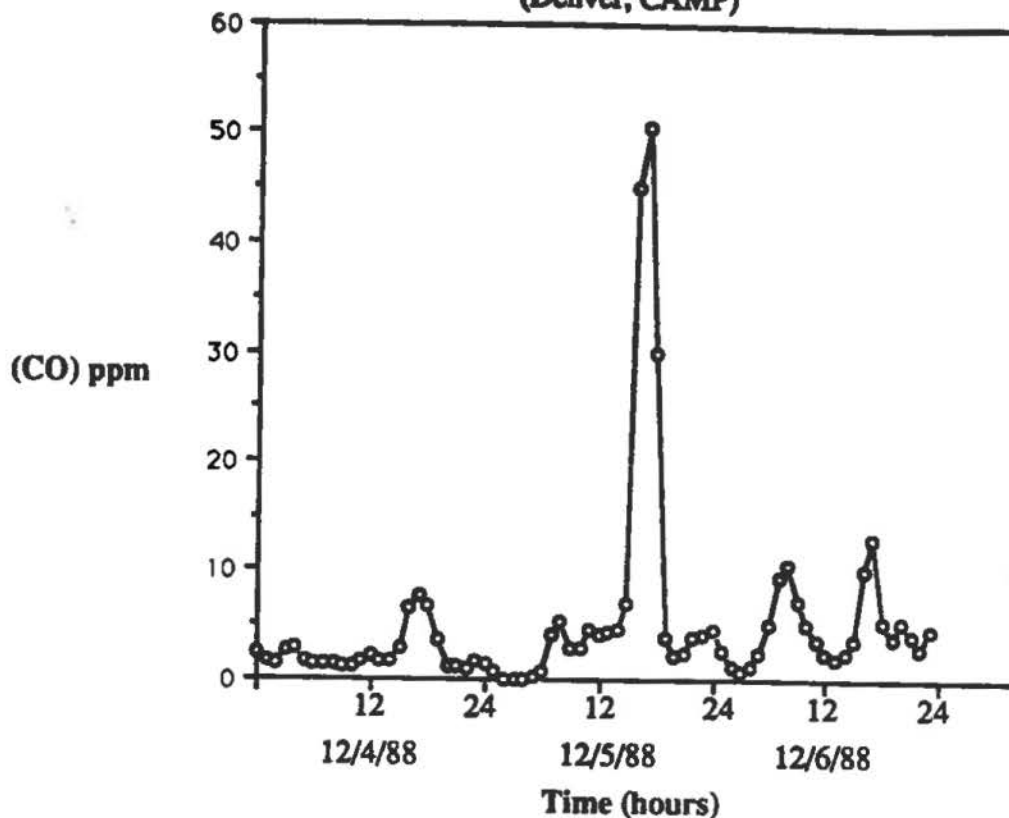
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Carbon monoxide concentration over three days
(Denver, CAMP)

Data obtained by the Colorado Air Quality
Control Division during three of the worst days
for CO pollution in 1988.

B. Pathophysiology:

Mechanism of action. Carbon monoxide combines avidly with hemoglobin, the
molecule which carries oxygen in the blood. The affinity of hemoglobin for carbon
monoxide is approximately 200-250 times that for oxygen. Therefore, oxygen is
displaced. The pathology of carbon monoxide poisoning is basically that of poor
oxygen delivery to vital organs. The heart and the brain are most significantly af-
fected. Carbon monoxide also shifts the oxyhemoglobin dissociation curve so that
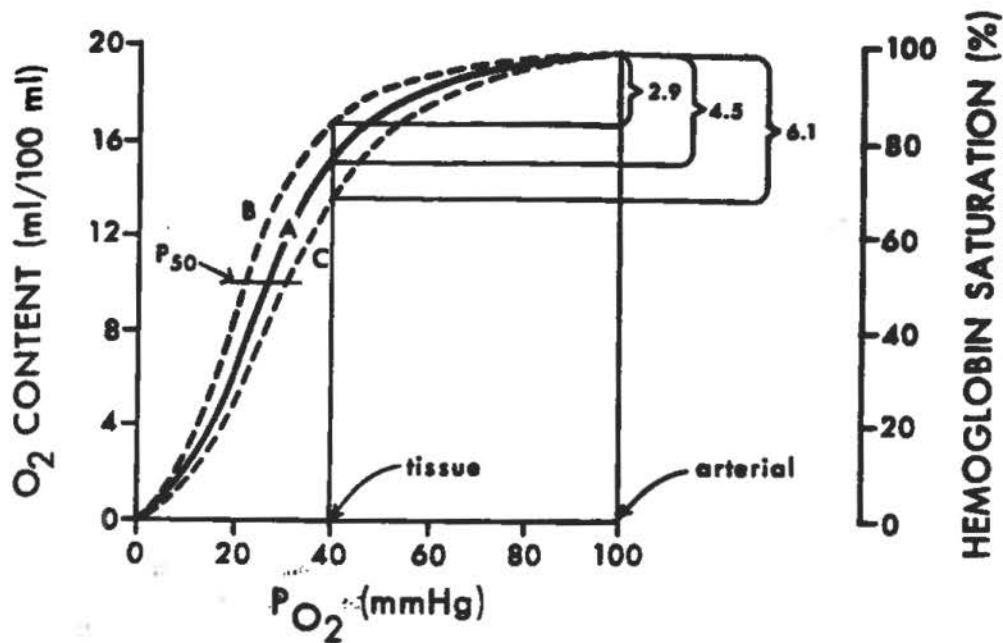hemoglobin releases oxygen less readily to peripheral tissues.

Figure 7–8. Schematic diagram showing the effects of increases and decreases in O_2 affinity on the amount of O_2 available at the Po_2 values prevailing in arterial blood and at the tissues. P_{50} = Po_2 at which hemoglobin saturation is 50 per cent. Hemoglobin concentration is assumed for convenience to be 14.9 gm/100 ml; therefore, O_2 content at 100 per cent saturation is 20 ml/100 ml. Curve A = normal blood; curve B = blood with increased affinity (decreased P_{50}); curve C = blood with decreased affinity (increased P_{50}). For further discussion, see text.

From: Murray JF. The Normal Lung. WB Saunders, Philadelphia, 1986. CO shifts the oxyhemoglobin curve to the left (B).

C. Absorption and elimination:

Carbon monoxide is rapidly absorbed through the lungs. Some variables that determine the percentage of COHb formed when a person breathes CO include:

1. Concentration of inhaled carbon monoxide

2. Duration of exposure

3. Minute ventilation

4. Partial pressure of oxygen (more COHb is formed at higher altitudes where the partial pressure of oxygen is less than at sea level).

Most CO is eliminated unchanged from the lungs. The biological half-life in healthy sedentary adults at sea level is 4-5 hours. When a person breaths 100% oxygen the biological half-life is decreased to 80 minutes. If a person is placed in a hyperbaric chamber with 100% oxygen at 3 atmospheres of pressure, the biologic half-life decreases to 23.5 minutes.

D. Acute toxicity:

Table 2. Carboxyhemoglobin equilibrium at an atmospheric pressure of 1

ppm CO inhaled	COHb Saturation (%)		
	15% O_2	18% O_2	21% O_2
1	0.62	0.46	0.38
5	1.61	1.19	0.99
10	2.81	2.08	1.75
25	6.25	4.68	3.94
35	8.41	6.33	5.34
50	11.47	8.71	7.38
70	15.24	11.69	9.96
100	20.34	15.83	13.57
300	43.14	35.84	31.81
500	55.80	48.17	43.69
700	63.85	56.53	52.05
1000	71.62	65.01	60.78

Table 3. Human response to various concentrations of carboxyhemoglobin

Blood saturation COHb (%)	Response of healthy adult[a]	Response of patient ill with severe heart disease
0.3-0.7	Normal range due to endogenous CO production; no known detrimental effect	
1-5	Selective increase in blood flow to certain vital organs to compensate for reduction in oxygen-carrying capacity of the blood	Patient with advanced cardiovascular disease may lack sufficient cardiac reserve to compensate
5-9	Visual light threshold increased	Less exertion required to induce chest pain in patients with angina pectoris
16-20	Headache; visual-evoked response abnormal	May be lethal for patients with severely compromised cardiac function
20-30	Throbbing headache; nausea; fine manual dexterity abnormal.	
30-40	Severe headache; nausea and vomiting; syncope	
50-60	Coma; convulsions	
67-70	Lethal if not treated	

[a] Exposure to CO in concentrations in excess of 50,000 ppm can result in a fatal cardiac arrhythmia and death before the carboxyhemoglobin saturation is significantly elevated.

Infants and persons with cardiovascular disease, anemia, lung disease and increased metabolic rate have an increased susceptibility to the toxic effects of CO.

E. Chronic toxicity:

The toxicity of carbon monoxide is due to decreased delivery of oxygen to vital tissues. Exposures to carbon monoxide that cause significant central nervous system deficits of oxygen cause residual impairment of memory, vision, hearing, and speech. However, repeated acute exposures to carbon monoxide, none of which result in sufficient oxygen deficit to produce permanent injury at the time, do not cause chronic disease. It is unlikely that exposure to ambient concentrations of CO causes permanent neurologic injury. One's body compensates, to a certain extent, to chronic exposure to carbon monoxide (such as in cigarette smoking) by increasing circulating red blood cell mass, which increases oxygen carrying capacity.

F. Effects on cardiovascular function:

The response of a healthy person to increased carboxyhemoglobin saturations is to increase cardiac output and blood flow to specific organs to compensate for the decreased oxygen carrying capacity of the blood. Persons who have cardiovascular disease often cannot increase cardiac output. Thus, they are more vulnerable to the decreased oxygen carrying capacity of their blood. Additionally, persons with coronary artery disease, in whom oxygen delivery to heart tissues is already compromised, are unable to increase blood flow in response to increased carboxyhemoglobin concentrations. Significant cardiac oxygen deficit may result with consequent angina pectoris or myocardial infarction. Persons with advanced coronary artery disease and angina pectoris have their exercise tolerance significantly decreased when carboxyhemoglobin saturation is as low as 5%.

G. Effects on cognitive function:

The effect of low carboxyhemoglobin concentrations on arithmetic problem solving, vigilance testing, and driving performance is controversial. The ability to perform complex tasks requiring both judgement and motor coordination is probably not affected adversely by COHb saturations below 10%.

IV. National Ambient Air Quality Standards

National ambient air quality standards

Pollutant	Averaging times	Standard
SO_2	24 h	0.14 ppm
	Annual	0.03 ppm
O_3	1 h	0.120 ppm
CO	1 h	35 ppm
	8 h	9 ppm

Compliance with the National Ambient Air Quality Standards has improved considerably over the past 20 years. However, in some circumstances the standards may not entirely protect the health of sensitive persons. For instance, the lack of a short term standard for sulfur dioxide means that SO_2 concentrations are allowed to be considerably above the standard during parts of the day as long as the overall daily average is less than 0.14 ppm. SO_2 often is carried in plumes emitted from the top of smoke stacks. The plumes may touch down to ground level and cause rapid and considerable increases in SO_2 concentrations. Point sources that produce SO_2 and that are compliant with air quality standards may cause ground level SO_2 concentrations in excess of 1 ppm at times.

V.   Protection from the health effects of air pollutants

The protection of sensitive persons from the adverse effects of air pollutants needs to continue to be directed primarily at decreasing levels of ambient air pollution. For the present, however, during thermal inversions or during other periods when air pollution is severe, persons who have asthma, other chronic lung disease, or cardiovascular disease can protect themselves somewhat by remaining indoors where concentrations of most air pollutants are often lower than outdoors. If one can keep windows closed and use central heating and air conditioning, the indoor environment is likely to be considerably cleaner than that outdoors. Since the effects of air pollutants depend on minute ventilation, sensitive persons should avoid exercising in polluted air.