ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[AD-FRL-2866-6]

Review of the National Ambient Air Quality Standards for Carbon Monoxide

AGENCY: Environmental Protection Agency.

ACTION: Final rule.

SUMMARY: In 1971, identical primary and secondary national ambient air quality standards (NAAQS) for carbon monoxide (CO) were promulgated at levels of 9 parts per million (ppm), 8-hour average, and 35 ppm, 1-hour average, neither to be exceeded more than once per year (36 FR 8186). In accordance with sections 108 and 109 of the Clean Air Act, EPA has reviewed and revised the criteria upon which the existing NAAQS for CO are based, and has reviewed those standards to determine if revisions to the standards are appropriate. On August 18, 1980, EPA proposed certain changes in the standards based on the scientific knowledge reported in the revised criteria document for CO (45 FR 55066). Today's notice announces EPA's decision not to revise the existing primary (health) standards at this time and revokes the secondary (welfare) NAAQS for CO. EPA plans to review several ongoing human health effect studies upon their completion and, if warranted, will reexamine this decision at that time.

EFFECTIVE DATE: This action is effective October 15, 1985.

ADDRESSES: A docket (Number OAQPS 79-7) containing information relating to EPA's review of the CO standards, is available for public inspection and copying between 8:00 a.m. and 4:00 p.m., on weekdays, at EPA's Central Docket Section, West Tower Lobby, Gallery I, Waterside Mall, 401 M Street SW., Washington, D.C. A reasonable fee may be charged for copying.

Availability of Related Information

The final revised criteria document, "Air Quality Criteria for Carbon Monoxide" (EPA-600/8-79-022, October 1979; NTIS PB 81-244840, $17.00 paper and $4.50 microfiche), an addendum to the criteria document, "Revised Evaluation of Health Effects Associated with Carbon Monoxide Exposure" (EPA-600/8-83-033F, August 1984; NTIS PB 85-103471, $10.00 paper and $4.50 microfiche) and the final staff paper, "Review of the NAAQS for Carbon Monoxide: Reassessment of Scientific and Technical Information" (EPA-450/5-84-004, July 1984; NTIS PB 84-231315, $10.00 paper and $4.50 microfiche) are available from: U.S. Department of Commerce, National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, Virginia 22161.

FOR FURTHER INFORMATION CONTACT: Mr. Michael Jones, Strategies and Air Standards Division (MD-12), Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711, Telephone: (919) 541-5531 (FTS 629-5531).

SUPPLEMENTARY INFORMATION:

Background

Legislative Requirements Affecting This Action

Two sections of the Clean Air Act govern the establishment, review, and revision of NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify pollutants which may reasonably be anticipated to endanger public health or welfare and to issue air quality criteria for them. These air quality criteria are to reflect the latest scientific information useful in indicating the kind and extent of all identifiable effects on public health or welfare that may be expected from the presence of the pollutant in the ambient air.

Section 109(a) (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as one the attainment and maintenance of which in the judgment of the Administrator, based on the criteria and allowing for an adequate margin of safety, is requisite to protect the public health. The secondary standard, as defined in section 109(b)(2), must specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on the criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of the pollutant in the ambient air.

The courts have upheld EPA's interpretation that the requirement for an adequate margin of safety for primary standards is intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It is also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *Lead Industries Association* v. *EPA*, 647 F.2d 1130, 1154 (D.C. Cir. 1980), *cert. denied*, 101 S. Ct. 621 (1980); *American Petroleum Institute* v. *Costle*, 665 F.2d 1176, 1177 (D.C. Cir. 1981), *cert. denied*, 102 S. Ct. 1737 (1982). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards with an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been clearly demonstrated to be harmful, but also to prevent lower pollutant levels that he finds pose an unacceptable risk of harm, even if that risk is not precisely identified as to nature or degree.

In evaluating such risks for the purpose of providing an adequate margin of safety, EPA has considered such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. Given that the "margin of safety" requirement by definition comes into play where no conclusive showing of harm exists, such factors, which involve unknown, qualitatively defined, or only partially quantified risks, have inherent limits as guides to action. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. *Lead Industries Association* v. *EPA, supra*, 647 F.2d at 1161-62.

The courts, however, have endorsed a reading of the Act that sets strict limits on the factors EPA may consider in providing an adequate margin of safety for primary standards. The leading judicial decisions state that the economic and technological feasibility of attaining primary standards are not to be relied upon in setting them, even in the context of a margin of safety. *Lead Industries Association* v. *EPA, supra*, 647 F.2d at 1148-1151; *American Petroleum Institute* v. *Costle, supra*, 665 F.2d at 1185, 1190. Such factors may, however, be considered to a degree in the development of State plans to implement the standards.

Section 109(d) of the Act (42 U.S.C. 7409(d)) requires periodic review and, if appropriate, revision of existing criteria and standards. If, in the Administrator's judgment, the Agency's review and revision of criteria make appropriate the proposal of new or revised standards, such standards are to be revised and promulgated in accordance with section 109(b). Alternatively, the Administrator may find that revision of the standards

is inappropriate and conclude the review by leaving the existing standard(s) unchanged. The process by which EPA has reviewed the original criteria and standards for carbon monoxide (CO) under section 109(d) is described in a later section of this notice.

States are primarily responsible for assuring attainment and maintenance of ambient air quality standards. Under section 110 of the Act (42 U.S.C. 7410), States are to submit to EPA for approval State implementation plans (SIPs) that provide for the attainment and maintenance of such standards through control programs directed to sources of the pollutants included. Other federal programs provide for nationwide reductions in emissions of these and other air pollutants through the federal motor vehicle control program, which involves controls for automobile, truck, bus, and motorcycle under Title II of the Act (42 U.S.C. 7501 to 7534), and through the development of new source performance standards for various categories of stationary sources under section 111 (42 U.S.C. 7411).

Original CO Standards, Revision of the Criteria Document, and Proposed Revisions of the Standards

Original CO Standards. On April 30, 1971, the Environmental Protection Agency (EPA) promulgated NAAQS for CO under section 109 of the Clean Air Act (36 FR 8186). Identical primary and secondary standards were set at levels of 9 ppm, 8-hour average, and 35 ppm, 1-hour average, neither to be exceeded more than once per year. The scientific and medical bases for these standards are described in the document, "Air Quality Criteria for Carbon Monoxide" (DHEW, 1970), published by the U.S. Department of Health, Education, and Welfare in March 1970. The primary standards set in 1971 were based largely on work by Beard and Wertheim (1967) suggesting that low-level CO exposures resulting in carboxyhemoglobin (COHb) levels of 2 to 3 percent were associated with impairment of ability to discriminate time intervals, a central nervous system effect.

Revision of the Criteria Document. On December 1, 1978, EPA announced that it was in the process of reviewing and updating the 1970 document, "Air Quality Criteria for Carbon Monoxide," and called for information that might be helpful in revising the document (43 FR 56250). In the process of developing the revised criteria document, EPA provided a number of opportunities for review and comment by organizations and individuals outside the Agency. Two successive drafts of a revised criteria document, prepared by EPA's Environmental Criteria and Assessment Office (ECAO), were made available for external review, and EPA received approximately 30 written comments on these drafts. The external review drafts of the criteria document were also reviewed by the Clean Air Scientific Advisory Committee (CASAC)[1] Subcommittee on Carbon Monoxide on January 30–31, 1979 and June 14–15, 1979. These meetings were open to the public and were attended by individuals and representatives of organizations who provided critical reviews and new information for consideration. A summary of EPA's responses to the comments on the two external review drafts of the criteria document has been placed in the public docket (Docket No. ECAO–CD–78–3). Transcripts of the two CASAC meetings have also been placed in the docket.

The CASAC prepared a "closure" memorandum (Hovey, 1979) to the Administrator indicating its satisfaction that the final draft of the criteria document was scientifically adequate for standard-setting purposes. The closure memorandum, dated October 9, 1979 also outlines major issues addressed by the CASAC's and CASAC's recommendation concerning those issues. A summary of the issues raised by CASAC and the general public prior to proposal is contained in the proposal notice (45 FR 55080).

On the basis of a careful review of scientific information contained in the revised Criteria Document (EPA, 1979a) EPA's Office of Air Quality Planning and Standards (OAQPS) prepared a Staff Paper (EPA, 1979a) in which several key considerations were identified as major factors to be considered in the possible revision of the CO standards. A draft of the Staff Paper was provided to the CASAC, made available to the public, and reviewed by the CASAC CO Subcommittee on June 14–15, 1979.

Proposed Revisions of the Standards. The principal findings of the Staff Paper are summarized in the August 18, 1980 proposal notice (45 FR 55066). As discussed in this notice and in the revised Criteria Document, the Beard and Wertheim (1967) study, the prinicipal basis for the 1971 primary standards, is no longer considered a sound scientific basis for the standards. However, medical evidence accumulated since 1970 indicated at the time of the 1980 proposal that aggravation of angina pectoris and other cardiovascular diseases could occur at COHb levels as low as 2.7 to 2.9 percent. Assessment of this and other medical evidence led EPA to propose: (1) retaining the 8–hour primary standard level of 9 ppm, (2) revising the 1-hour primary standard level from 35 ppm to 25 ppm, (3) revoking the existing secondary CO standards (since no adverse welfare effects have been reported at or near ambient CO levels), (4) changing the form of the primary standards from deterministic to statistical (i.e., EPA proposed to allow one expected exceedance of each standard level per year), (5) adopting a daily interpretation for exceedances of the primary standards, so that exceedances would be determined on the basis of the number of days on which the 8- or 1-hour average concentrations are above the standard levels (45 FR 55066). The proposal notice set forth in more detail the rationale for these and other proposed revisions of the CO NAAQS and background information related to the proposal.

Developments Subsequent to Proposal

Following proposal, EPA held two public meetings to receive comments on the proposed standard revisions. Meetings were held in Washington, D.C. on October 2, 1980 and Denver, Colorado on October 10, 1980; transcripts are available in the docket (Docket No. OAQPS 79–7). The CASAC CO Subcommittee also met on November 15, 1980 to review the notice of proposed rulemaking (45 FR 55066) with EPA officials, and the CASAC met on November 17, 1981 to hear a status report on the regulation. The public was invited to both CASAC meetings (45 FR 73790 and 46 FR 53210) and transcripts of the meetings have been placed in the docket (Docket No. OAQPS 79–7).

On June 18, 1982 EPA announced (47 FR 26407) an additional public comment period to address several key issues concerning the 1980 proposal and technical documents related to the review of the CO standards. These issues included: (1) role of the Aronow (1981) study, (2) consideration of a multiple exceedance 8–hour standard, (3) the technical adequacy of the revised draft sensitivity analysis on the Coburn model predictions of blood carboxyhemoglobin (COHb) levels, and (4) the technical adequacy of the revised exposure analysis. The CASAC met on July 6, 1982 to provide its advice on these issues. CASAC's recommendations arising from that meeting are summarized in an August

[1] CASAC is a standing committee of scientists and engineers external to the Federal government established under Section 109 of the Clean Air Act to advise the Administrator on the scientific basis for ambient air quality standards.

31, 1982 letter to the Administrator (Friedlander, 1982) which has been placed in the public docket (Docket No. OAQPS 79-7, IV-H-41).

The 1980 proposal was based in part on the evaluation by EPA staff and CASAC in 1979 of several health studies conducted by Dr. Wilbert Aronow. EPA concluded at that time, based in part on the Aronow studies, that COHb levels of 2.7-3.0 percent represent a health concern for individuals with angina and other types of cardiovascular disease. In March 1983 EPA learned that the Food and Drug Administration (FDA) had raised serious questions regarding the technical adequacy of several studies conducted by Dr. Aronow on experimental drugs, leading FDA to reject use of the Aronow drug studies data. While there was then no direct evidence that similar problems might exist for Dr. Aronow's CO studies, EPA concluded that an independent examination of these studies was advisable prior in a final decision on the CO NAAQS.

An expert committee was convened and met with Dr. Aronow to discuss his studies and to examine the limited available data and records from his 1981 CO study. In its report, the Committee (chaired by Dr. Steven M. Horvath, Director of the Institute of Environmental Stress, University of California-Santa Barbara) concluded that EPA should not rely on Dr. Aronow's data due to concerns regarding the research which substantially limit the validity and usefulness of the results (Horvath et al., 1983). In early June 1983, EPA received a detailed reply (Aronow, 1983) from Dr. Aronow disputing, but not effectively refuting, the major points raised in the "Horvath Committee" report.

Addendum to the 1979 Criteria Document and Staff Reassessment. On August 18, 1983, EPA announced (48 FR 37519) the availability of an external review draft of a document entitled "Revised Evaluation of Health Effects Associated with Carbon Monoxide Exposure: An Addendum to the 1979 Air Quality Criteria Document for Carbon Monoxide" (hereafter cited as Addendum). The Addendum reevaluates the scientific data base concerning health effects associated with exposure to CO at or near ambient exposure levels in light of the Horvath Committee's recommendations concerning Dr. Aronow's studies and taking into account new findings reported beyond those reviewed in the 1979 Criteria Document.

On September 16, 1983 EPA announced the availability of a draft staff paper, "Review of the NAAQS for Carbon Monoxide: 1983 Reassessment of Scientific and Technical Information" (hereafter cited as Staff Reassessment) and solicited public comment on the draft paper (48 FR 41608). The Staff Reassessment, prepared by the OAQPS, provided the staff's assessment of how the scientific data reviewed in the Addendum might be used in selection of final CO standards. CASAC held a public meeting on September 25, 1983 to review both the draft Staff Reassessment. In addition to comments from CASAC members, representation of several organizations also provided critical review of both EPA documents. A transcript of the CASAC meeting has been placed in the public docket (OAQPS 79-7).

The CASAC sent a closure letter to the Administrator on May 17, 1984 which concluded that both the Addendum and Staff Reassessment "represent a scientifically balanced and defensible summary of the current basis of our knowledge of the health effects literature for this pollutant" (Lippmann, 1984). The closure letter, which also discusses major issues addressed by the CASAC and CASAC's recommendations concerning those issues, has been placed in the public docket (Docket No. OAQP 79-7, IV-K-25).

On August 9, 1984, EPA announced (49 FR 31923) the availability of the final Addendum (EPA, 1984b) and final Staff Reassessment (EPA, 1984a) which were revised to reflect public and CASAC comments. Both final documents are available from the address given earlier in the Availability of Related Information Section of this notice. Where there are differences between the 1979 Criteria Document and 1980 proposal assessment of the health effects evidence and the more recent EPA documents, the final Addendum and final Staff Reassessment represented the Agency's interpretation as of their issuance. In the August 9, 1984 notice (49 FR 31923) EPA also reviewed the basis for EPA's proposal to revise the CO standards and solicited additional public comment.

Carbon Monoxide and Human Health Effects

Control of human exposures to sources of CO is important because CO, when inhaled, can enter the bloodstream and disrupt the delivery of oxygen to the body's tissues. A continous and adequate flow of oxygen to the body's tissues is essential to maintain normal health. After being inhaled into the lungs, oxygen normally enters the bloodstream and is delivered, via the circulating blood, to the body's energy-consuming organs, tissues, and cells. Once in the tissue cells, oxygen reacts with nutrients to form energy. This energy is essential to the normal functioning of cells, tissues, and organs.

Humans have specialized molecules in blood and other tissue cells for ensuring a large, continuous flow of oxygen to the tissues. The most important chemical substance is "heme," an iron-containing substance that has a special affinity for oxygen and that combines with proteins to form hemo-proteins. Hemoglobin, a highly specialized hemo-protein contained in red blood cells, binds oxygen to the lungs to form oxyhemoglobin (O_2Hb) and delivers the oxygen to all other tissues where it is released for use.

Unfortunately, CO can also bind to hemoglobin, forming carboxyhemoglobin (COHb), with an affinity that is 200 to 300 times greater than that of oxygen. For this reason, small elevations of CO in ambient air that enters the lungs can result in a relatively large displacement of oxygen from the carrier hemoglobin.

Mechanisms of CO Toxicity

The two principal sources of CO that contribute to levels of CO in the human body are: (1) endogenous CO production from the normal breakdown of hemoglobin constituents in the body and (2) inhalation of exogenous CO from ambient and non-ambient (e.g., occupational and indoor) exposures.[2] Endogenous CO production contributes roughly 0.3 to 0.7 percent of hemoglobin saturation (0.3 to 0.7 percent COH).

Presently, the most important mechanism of CO toxicity at low-level CO exposures is thought to be hypoxia. This mechanism generally involves diffusion of exogenous CO through the lungs into the blood with resultant formation of COHb. Such an increase in COHb levels disrupts normal delivery of oxygen to the tissues in two ways. First, CO displaces oxygen on the hemoglobin carrier so that the hemoglobin has a reduced capacity to carry oxygen through the blood to the tissues (EPA, 1979b). Second, CO makes it more difficult for the oxygen that is still transported by hemoglobin to be released at the tissue (EPA, 1979b). Under these conditions, the tissues must operate at lower than normal levels of oxygen, a condition known as hypoxia, and this amount of oxygen may be inadequate to meet the energy needs of the tissues. The effects of CO on the cardiovascular system, central nervous system, and other systems are thought

[2] The contribution of direct smoking to levels of CO in the body is not a part of this rulemaking.

to be directly related to this reduction in the ability of the blood to deliver oxygen to these systems and the resultant oxygen deficiency in the tissues themselves.

Other possible mechanisms of toxicity have been discussed (Coburn, 1979; EPA, 1984b). These alternative mechanisms involve the binding of CO to such intracellular hemoproteins as cytochrome oxidase, myoglobin, tryptophan deoxygenase, and tryptophan catalase. Because the affinity of CO for these proteins is much less than for hemoglobin, it is unlikely that they play a major role in CO hypoxia. However, in tissues with a high oxygen gradient between blood and tissue, it is likely that the interaction of CO with these proteins in these tissues may play a role in CO toxicity. This is particularly true of myoglobin in heart muscle cells. Myoglobin appears to facilitate the movement of oxygen through muscle cells and, as a short-term oxygen reservoir in muscle, it releases oxygen during sudden increased metabolic activity. The affinity of myoglobin for CO is about 40 times greater than the affinity of myoglobin for oxygen. The ratio of CO content in heart muscle to CO content in blood is approximately three. Therefore, given human exposure to CO, substantial amounts of CO may be stored in heart muscle. In situations of sudden, severe stress or exertion, the heart muscle's immediate oxygen supply may be inadequate, even in healthy persons and particularly in persons with a history of disease. This mechanism could provide theoretical support for experimental evidence of myocardial ischemia (in which part of the heart muscle is deprived of oxygen), such as electrocardiographic irregularities and decrements in work capacity discussed later in this notice. It is not known whether binding of CO to myoglobin is related to health effects observed at COHb levels as low as 4–5 percent (EPA, 1979b).

In summary, disruption of the normal transport of oxygen by formation of COHb in the blood appears to be the primary mechanism of CO toxicity. In addition, EPA concludes that regardless of the mechanism of toxicity, COHb levels provide a meaningful and useful physiological marker of the body's CO burden and exposure to exogenous sources of CO (EPA, 1948b).

Health Effects As A Result of CO Exposure

Exposure to high levels of exogenous CO can result in headache, dizziness, drowiness, nausea, vomiting, collapse, coma, or death, depending on how much the flow of oxygen to the body is impeded (EPA, 1979b). The most notorious effects of CO are severe functional losses and death, which can result from automobile or truck emissions trapped in the enclosed interior of a vehicle or garage. In this case, so much hemoglobin is bound with CO that a severely inadequate supply of oxygen is delivered to the tisses; the cause of death is severe hypoxia (or oxygen deficiency). Exposure to lower levels of CO (i.e., levels closer to ambient air levels) is associated with effects on several different organ systems. Because the cardiovascular system (including the heart) and the central nervous system (including the brain) are always active and have a continuous need for oxygen, even when the body is at rest, these organ systems are particularly sensitive to reduced supplies of oxygen.

Cardiovascular System. The heart and blood vessels are critical to human survival because they pump and carry, respectively, blood containing oxygen and essential nutrients to all organs, tissues, and cells of the body, including the brain and the heart muscle itself. Because the heart has almost no ability to function without oxygen, nor the ability (except in situations of clear heart muscle hypoxia) to increase extraction of oxygen from blood, the human body has compensatory mechanisms for ensuring that in low-oxygen situations, the heart continues to receive whatever oxygen is available (EPA, 1979b). These compensatory mechanisms include increasing heart rate, blood pressure, and blood flow to maximize the amount of oxygen available to the muscle (EPA, 1979b). Although these compensatory changes have not been associated experimentally with damage to the cardiovascular system, it is possible that the added stress on the body due to the functioning of the compensatory mechanisms themselves may result in damage to the heart muscle or vasculature (EPA, 1979b). The potential damage includes those effects associated with elevated blood pressure, primarily an increased risk of damage to the arteries, and elevated heart rate, which further increases the heart muscle's demand for oxygen. However, if the coronary arteries supplying the heart muscle are unable to compensate for CO exposure, parts of the heart muscle may receive amounts of oxygen inadequate for normal activity.

As a result, EPA is concerned that CO exposure may increase the risk of the following effects on the cardiovascular system:

- Local myocardial ischemia (in which a part of the heart muscle is deprived of oxygen);
- Aggravation of angina pectoris;
- Heart attack (myocardial infarction), including heart attacks leading to sudden death;
- Reduced exercise and physical work capacity; and
- Enhanced development of arteriosclerosis and coronary artery disease.

Associations between low-level CO exposure and angina and reduced work capacity are supported by human experimental evidence discussed later in this notice. Angina pectoris (or simply "angina") is a group of symptoms of pressure and pain in the chest resulting from a transient episode of insufficient oxygen supply to a portion of the heart muscle (EPA, 1948b). The pain probably results from an accumulation of unoxygenated metabolic products that irritate nerve endings in the heart muscle. The intensity of pain varies from mild chest discomfort to severe and incapacitating distress. Although angina, by definition falls short of inducing death of part of the heart muscle, it can cause serious discomfort, and each attack carries some risk of heart attach. Acute coronary insufficiency, coronary failure, and myocardial infarction result when an episode of ischemia is more prolonged and severe.

An individual with an underlying coronary insufficiency is at risk of having an angina attack under any condition that places an additional demand on the heart. CO exposures can place an added demand on the heart, and, therefore, would appear to increase the risk of having an angina attack, and may increase the risk of a more severe attack. There is some experimental evidence of reduced time to onset of angina attack and increased duration of angina attack associated with low COHb levels (Anderson et al. 1973).

A heart attack is usually a precipitous and frequently fatal consequence of coronary heart disease, which usually results from a sudden reduction or cessation of a significant portion of the blood and oxygen flow normally reaching the heart. Very little is known about the specific factors which induce or precipitate heart attack or sudden death. However, any agent that reduces the supply of oxygen available to the heart muscle in an individual with pre-existing heart disease is suspect as a precipitating factor for heart attacks and sudden death. A relation between elevated COHb and these cardiac effects is plausible in light of what is known about CO toxicity and possible

pathological mechanisms of heart disease.

EPA has reviewed the available epidemiological data relating to hypotheses of an association between elevated COHb or CO exposure and the risk of heart attack, sudden death, and progression of atherosclerosis and has found them to be inconclusive (EPA, 1979b). EPA would like to see further epidemiological research in this area, although, admittedly, these questions will be extremely difficult to resolve with absolute certainty because of the multitude of experimental confounding factors, the multitude of risk factors for heart disease, the variability in human response, and the technical difficulty in investigating them.

Nonetheless, from a theoretical standpoint, associations between CO and these coronary effects are possible. For this reason, and because the health effects in question are very serious, EPA believes that it is prudent to take early signs of cardiovascular effects and other potential, but not well understood, cardiovascular effects very seriously.

Central Nervous System. Elevated COHb levels can reduce the oxygen available to parts of the central nervous system (CNS), including the brain. It appears that the body can compensate, to some extent, by increasing the blood flow through the brain's vasculature (EPA, 1979b). However, in some circumstances compensation may not always occur, leaving the brain with a reduced amount of available oxygen. In addition, or alternatively, prolonged compensation may damage the brain's blood vessels (EPA, 1979b).

When elevated COHb reduces the amount of oxygen delivered to the brain to a level which is below critical needs, two types of damage may result. First, severe acute oxygen deprivation of sufficient duration and intensity can result in a stroke (the damage or death of brain cells (neurons)). Loss of mature brain cells is irreversible since the CNS is not thought capable of replacement of neurons. Depending upon the duration of oxygen deficit to the brain, the effects may range from unconsciousness and convulsions, brain swelling and protrusion, to death of parts of the brain or death of the individual. Individuals who recover often have significant deficits in brain function. Second, repeated episodes of impaired oxygen supply would be expected to damage the blood-brain barrier and possibly cause structural damage resulting in the inability of the CNS to transmit information.

At lower COHb levels there is evidence of neurobehavioral effects: impaired learning ability, reduced vigilance (ability to detect small changes in a subject's environment), decreased manual dexterity, impaired performance of complex tasks, and disturbed sleep activity (EPA, 1984b). Although the potential influence of ambient CO levels on drivers has received only slight attention, suggestive but not conclusive evidence has indicated that drivers in fatal auto accidents often have elevated COHb levels (Yabroff et al., 1974). There is no evidence with which to determine whether these effects at lower CO levels are associated with pathological changes to the CNS or with reduced oxygen tension in the CNS. Due to redundancy in structure of the CNS, functional changes (e.g., behavior changes) may not be observed until the structural damage is great. Observation of such effects may be more likely in the case of a severe reduction in oxygen or in an individual who has a history of an impaired oxygen delivery system.

Basis for Decision Not To Revise the Primary Standards

The current primary NAAQS for CO are 10 mg/m^3 (9 ppm), 8-hour average and 40 mg/m^3 (35 ppm), 1-hour average, neither of which is to be exceeded more than once per year. As indicated above, the Act requires review of the existing criteria and ambient air quality standards for CO and other pollutants every five years. During the current standard review for CO, EPA has considered whether it should retain the existing CO primary standards, repropose the same or different standards, or promulgate the revisions proposed in August 1980. For the reasons discussed below, the Administrator has concluded that there is no need to revise the existing primary CO standards at this time.

As indicated above, section 109(b)(1) of the Clean Air Act requires EPA to set primary standards, based on the air quality criteria which, in the Administator's judgment, are requisite to protect the public health with an adequate margin of safety. The legislative history of the Act makes clear the Congressional intent to protect sensitive persons who in the normal course of daily activity are exposed to the ambient environment. Air quality standards are to be established with reference to protecting the health of a represenative, statistically related, sample of persons comprising the sensitive group rather than a single person in such a group.

EPA's objective in reviewing primary standards, therefore, has been to determine whether new or revised standards are appropriate, based on the existing scientific evidence, assessment of the uncertainties in this evidence, understanding of underlying biological mechanisms, and the need to make a reasonable provision for scientific and medical knowledge yet to be acquired, in order to protect sensitive population groups with an adequate margin of safety. As for other ambient standard pollutants, none of the evidence presented in the 1979 Criteria Document or Addendum shows a clear threshold for adverse health effects of CO. Rather, there is a continuum, ranging from CO levels at which health effects are undisputed, through levels at which many, but not all scientists generally agree that health effects have been convincingly shown, down to levels at which the indication of health effects are less certain and more difficult to identify. This does not necessarily mean that there is no threshold, other than zero, for CO related health effects; it simply means that no precise threshold can be identified with certainty based on existing scientific evidence. Thus, in setting a standard, EPA is unable to append an exact margin of safety to a known threshold effect level. Rather, setting a standard with an adequate margin of safety is necessarily a public health policy judgment that must take into account both the known continuum of effects, understanding of the underlying biological mechanisms of effects, and any gaps and uncertainties in the existing scientific data base.

In determining whether revision of the primary CO standards is appropriate, EPA has made assessments and judgments in the following areas:

1. Identification of reported effect levels and associated exposure duration that scientific research has linked to health effects in healthy and sensitive persons.

2. Characterization of scientific uncertainties in the health effects evidence and judgments concerning which effects are important to consider in reviewing or setting primary standards.

3. Description of population groups believed to be most sensitive to CO and estimates of the size of those groups.

4. The estimated number of sensitive persons that would be exposed to elevated CO levels upon attainment of a given standard and the uncertainties in these exposure estimates.

5. The uncertainties in estimating carboxyhemoglobin (COHb) levels that result from exposures to CO.

6. Consideration of CO standard levels and averaging times that provide an adequate margin of safety based on CO levels and exposure periods that may affect sensitive population groups,

taking into account the various uncertainties.

Based on the assessment of relevant scientific and technical information in the Criteria Document and the Addendum, the Staff Reassessment outlines a number of key factors to be considered in each of the above areas. Both the staff and CASAC recommended that the Administrator focus consideration on a discrete range of policy options in each area. In most respects, the Administrator has adopted the recommendations and supporting reasons contained in the Staff Reassessment, the most recent CASAC closure letter (Lippmann, 1984) and applicable portions of the earlier Staff Paper (EPA, 1979b) and CASAC closure letters (Hovey, 1979; Friedlander, 1982). Rather than reiterating those discussions at length, the following discussion of the final decision focuses primarily on those considerations that were most influential in the Administrator's selection of a particular option, or that differ in some respect from considerations that influenced the staff and/or CASAC recommendations.

Assessment of Health Effects Evidence

The Staff Reassessment, which has been placed in the public docket (Docket No. OAQPS 79-7, IV-A-10), presents an assessment by OAQPS staff of the key health effect studies contained in the 1979 Criteria Document and in the Addendum and other critical scientific and technical issues relevant to the review of the existing CO standards.

TABLE 1.—LOWEST OBSERVED EFFECT LEVELS FOR HUMAN HEALTH EFFECTS ASSOCIATED WITH LOW LEVEL CARBON MONOXIDE EXPOSURE

Effects	COHb concentration (percent)[a]	References
Statistically significant decreased (~3-7%) work time to exhaustion in exercising young healthy men.	2.3-4.3	Horvath et al., 1975, Drinkwater et al., 1974, Raven et al., 1974.
Statistically significant decreased exercise capacity (i.e., shortened duration of exercise before onset of pain) in patients with angina pectoris.	2.9-4.5	Anderson et al., 1973.
Statistically significant decreased maximal oxygen consumption and exercise time during strenuous exercise in young healthy men.	5-5.5	Klein et al., 1980, Stewart et al., 1978, Weiser et al., 1978.
No statistically significant vigilance decrements after exposure to CO.	Below 5	Haider et al., 1976, Winneke, 1974, Christensen et al., 1977, Benignus et al., 1977, Putz et al., 1976.
Statistically significant impairment of vigilance tasks in healthy experimental subjects.	5-7.6	Horvath et al., 1971, Groll-Knapp et al., 1972, Fodor and Winneke, 1972, Putz et al., 1976.
Statistically significant diminution of visual perception, manual dexterity, ability to learn, or performance in complex sensorimotor tasks (such as driving).	5-17	Bender et al., 1971, Schulte, 1973, O'Donnell et al., 1971, McFarland et al., 1944, McFarland, 1973, Putz et al., 1976, Salvatore, 1974, Wright et al., 1973, Rockwell and Weir, 1975, Rummo and Sartanis, 1974, Putz et al., 1979, Putz, 1979.
Statistically significant decreased maximal oxygen consumption during strenuous exercise in young healthy men.	7-20	Ekblom and Huot, 1972, Pirnay et al. 1971, Vogel and Gleser, 1972.

[a] The physiologic norm (i.e., COHb levels resulting from the normal breakdown of hemoglobin and other heme-containing materials) has been estimated to be in the range of 0.3 to 0.7 percent (Coburn et al., 1963).

Table 1 is a summary of key clinical studies reporting human health effects associated with low-level exposures to CO. This table is based on evidence discussed in the 1979 Criteria Document (EPA, 1979a) and in the Addenum (EPA, 1984b) but excludes a series of studies by Dr. Aronow for reasons given below. The table is included primarily as an aid for the following discussion and should be used only in conjunction with qualifying statements made in the 1979 Criteria Document, in the Addenum, in the Staff Reassessment, or in this notice regarding the technical merits of each study.

Cardiovascular Effects. In reviewing the primary standards, a principal area of concern to the Administrator has been evidence linking aggravation of angina and other cardiovascular diseases to CO exposures. Angina patients who have been exposed to low levels of CO while resting have subsequently exhibited, during exercise, reduced time to onset of angina (Anderson et al., 1973). Increased duration of angina attacks has also been reported (Anderson et al., 1973). In proposing to revise the CO standards, EPA viewed aggravation of angina, as reported above, to be an adverse health effect (45 FR 55066) and CASAC concurred with EPA's judgment on this matter.

One controlled human exposure study (Anderson et al. 1973) reported that experimental subjects with angina exhibited statistically significant reduced time to onset of exercise-induced angina after exposure to low levels of CO resulting in mean COHb levels of 2.9 (range 1.3-3.8 percent) and 4.5 percent (range 2.8-5.4 percent). In the same study, it was reported that subjects experienced statistically significant increases in duration of angina attacks during exercise at a mean COHb level of 4.5 percent. Some concerns have been raised about the study findings due to ambiguities regarding the design and conduct of the study and the small number of subjects (N=10) examined. However, as discussed in the Addendum and in a subsequent section of this notice, a reevaluation of the Anderson et al. (1973) study, addressing major points of concern, found that the study provides reasonably good evidence for the hastening of angina occurring in angina patients at COHb levels of 2.9 to 4.5 percent.

In similar studies Aronow and Isbell (1973) and Aronow (1981) reported decreased time to onset of angina for exercising subjects with reported COHb levels in the range of 2 to 3 percent. In addition, Aronow et al. (1974) reported that subjects with peripheral vascular disease had reduced time to onset of leg pain at similar COHb levels. As discussed earlier in this notice, however, an expert committee convened to conduct an independent review of these CO studies expressed considerable concern about the validity of the result reported and concluded that EPA should not rely on Dr. Aronow's data (Horvath et al., 1983).[3] As stated earlier in this notice, Dr. Aronow provided EPA a detailed reply disputing, but not effectively refuting, the substance of the concerns raised by the Committee (Aronow, 1983). The Committee also recommended that EPA pursue and support both current in-house and independent scientific research to resolve the issues raised by the reevaluation of Dr. Aronow's CO studies. Because EPA shares the concerns raised by the committee and by some public comments, the Administrator has not considered Dr. Aronow's studies in his decision on the CO primary standards.

[3] The Committee's concerns included (1) apparent failure to maintain a "double blind" approach as reported in the publications, (2) raw data were lost or discarded, (3) available data were of poor quality, (4) quality control for COHb measurements was nonexistent or inadequate, and (5) there appeared to be discrepancies between hospital record descriptions of patient diagnosis and those reported in Dr. Aronow's publications (Horvath et al., 1983).

Another cardiovascular effect of concern is the possible detrimental effect of increased blood flow that occurs as a compensatory response to CO exposure (Ayres et al., 1969; Ayres et al., 1970; and Ayres et al., 1979). This effect may be related to coronary damage or cerebrovascular effects at very high blood flow rates due to the added stress on the cardiovascular system. However, community epidemiological studies have been inconclusive. In particular, based on EPA's evaluation of the Goldsmith and Landau (1968), Kurt et al. (1978), and Kurt et al. (1979) epidemiological studies, the relationship between CO exposures and effects such as mortality from myocardial infarction (heart attack), sudden death due to arteriosclerotic heart disease, and cardiorespiratory complaints remains in question.

Maximum aerobic capacity and exercise capacity are indirect measures of cardiovascular capacity which have been reported to be reduced in several carefully conducted studies involving normal healthy adults exposed to CO. A linear decline in maximum aerobic capacity for healthy individuals was reported for COHb levels ranging from 5–20 percent in a series of studies (Stewart et al., 1978; Weiser et al., 1978; and Ekblom and Huot, 1972). Horvath et al. (1975) found decreases in maximum aerobic capacity when COHb levels were 4.3 percent and that COHb levels of 3.3 and 4.3 percent reduced work time to exhaustion by 4.9 and 7.0 percent, respectively. The effects of lower CO exposure levels in healthy individuals have also been investigated under conditions of short-term, maximum exercise duration. In a series of studies involving different CO exposure levels, two alternative ambient temperatures (25°C and 35°C), and two different healthy adult populations (young and middle-aged), no statistically significant reduction in maximum aerobic capacity was observed for either group when CO exposures were compared with the clean air control. However, small (less than or equal to 5 percent) decreases in absolute exercise time were consistently observed in the non-smoking subjects whose COHb levels reached 2.3 and 2.5 percent (Drinkwater et al., 1974; Raven et al., 1974). These effects, while not as serious as aggravation of angina, are still a matter of concern since they have been found to occur in healthy individuals and such effects might impair the normal activity of more sensitive populations. Therefore, these effects have been considered in judging which CO standards provide an adequate margin of safety.

Central Nervous System Effects. Numerous studies (Putz et al., 1976; Bender et al., 1971; Schulte, 1973; O'Donnel et al., 1971; McFarland et al., 1944; McFarland, 1973; Salvatore, 1974; Wright et al., 1973; Rockwell and Weir, 1975; and Rummo and Sarlanis, 1974) have reported effects on the central nervous system for CO exposures resulting in COHb levels in the range 5–17 percent. The range of effects reported included impairment of vigilance, visual perception, manual dexterity, learning ability, and performance of complex tasks. While one study (Beard and Grandstaff, 1975) has suggested that vigilance effects may occur at levels as low as 1.8 percent COHb, several other studies (Haider et al., 1976; Winneke, 1976; Christensen et al., 1977; Benignus et al., 1977; and Putz et al., 1976) have not found any vigilance decrements below 5 percent COHb. Measurement methods used to detect effects may have been too insensitive to detect changes in the latter vigilance studies. Based on the assessment of the above mentioned studies, the Addendum concluded that, at least under some conditions, small decrements in vigilance occur at 5 percent COHb. A series of studies (Putz et al., 1976; Putz et al., 1979; and Putz 1979) has also found that 5 percent COHb produced decrements in compensatory tracking, a hand-eye coordination task. EPA considers hand-eye coordination effects as well as decrements in vigilance to be important since these functions are components of more complex tasks, such as driving, and reduced alertness or visual sensitivity could lead to increased vehicular accidents.

Fetal Effects. Based on limited animal toxicology data, the 1979 Criteria Document and the Addendum (EPA, 1984b) suggest that CO may produce effects on the fetus or newborn. CO from long-term or intermittent exposures is taken up and eliminated more slowly in the fetus than in the mother, so that a maternal exposure can result in mean fetal COHb concentrations well above maternal COHb levels (EPA, 1979b). Elevated COHb levels may lead to interference with fetal tissue oxygenation during development. Because the fetus may be developing at or near critical tissue oxygenation levels, even exposures to moderate levels of CO may produce deleterious effects on the fetus such as reduced birth weight, increased newborn mortality, and lower behavioral activity levels (Longo, 1977), although this remains to be demonstrated along with pertinent dose-response relationships (EPA, 1984b). Evidence from smoking mothers is suggestive of similar fetal and newborn effects due to CO exposures (Peterson, 1981). While the fact that cigarette smoke contains substances other than CO prevents a direct quantitative application of the results of these studies in setting the CO primary standards, these studies do suggest the need for caution in protecting unborn children from such potentially deleterious effects of CO exposures.

As discussed in the Addendum and Staff Reassessment, research on sudden infant death syndrome (SIDS) recently has suggested a possible link between ambient CO levels and increased incidence of SIDS (Hoppenbrowers et al., 1981). In response to this study, Goldstein (1982) has suggested that indoor sources of CO, as well as other pollutants (e.g., nitrogen dioxide, lead), may be at least as important as ambient CO in causing SIDS and that several covariates (e.g., seasonal trends) may be responsible for the associations reported. Because the number of potentially confounding factors makes finding an association between CO and SIDS extremely difficult, further confirmation is needed before any causal relationship can be inferred (EPA, 1984a; EPA, 1984b).

Sensitive Population Groups

In EPA's judgment, the available health effects data identify persons with angina or other types of cardiovascular disease (e.g., history of heart attack or peripheral vascular disease) as the groups at greatest risk from low-level, ambient exposures to CO. Based on 1980 census data (DOC, 1980) and earlier U.S. National Health Examination Survey data (DHEW, 1975), this group is estimated to number approximately 8.7 million individuals. As discussed previously, the concern for persons with cardiovascular disease results from the fact that their condition is due to an insufficient oxygen supply to cardiac tissue, so that persons with this condition have an inadequate oxygen reserve capacity and an impaired ability to compensate for the effects of excess CO.

The Addendum and Staff Reassessment also identify several other groups as likely to be particularly sensitive to low-level CO exposures based on EPA's review of the health effects evidence. These groups include: (1) persons with chronic respiratory disease (e.g., bronchitis, emphysema, and asthma), (2) elderly individuals, especially those with reduced

cardiopulmonary function, (3) fetuses and young infants, and (4) individuals suffering from anemia and those with abnormal hemoglobin types that affect oxygen carrying capacity or transport in the blood. In addition, individuals taking certain medications or drinking alcoholic beverages may be at greater risk for CO-induced effects based on some limited evidence suggesting interactive effects between CO and some drugs. Visitors to high altitude locations are also expected to be more vulnerable to CO health effects due to reduced levels of oxygen in the air they breathe. Finally, individuals with some combination of the disease states or conditions listed above (e.g., individuals with angina visiting a high altitude location) may be particularly sensitive to low-level CO exposures, although there is no experimental evidence to confirm this hypothesis.

For many of the groups cited above there is little specific experimental evidence to clearly demonstrate that they are indeed at increased risk for CO-induced health effects. However, it is reasonable to expect that individuals with preexisting illnesses or physiological conditions which limit oxygen absorption into blood or its transport to body tissues would be more susceptible to the hypoxic (i.e., oxygen deficiency) effects of CO. Since no human experimental evidence exists that identifies CO effect levels for these other groups, however, EPA is considering the possible effects of CO on these groups only in its determination of an adequate margin of safety.

Uncertainty in Estimating COHb Levels

The health effect studies discussed above report the effects observed at varying COHb levels. In order to make decisions about ambient CO standards based on these studies, it is necessary to estimate the ambient concentrations of CO that are likely to result in COHb levels at or near those observed in the studies. A model known as the Coburn equation (Coburn et al., 1965) has been developed to estimate COHb levels resulting from CO concentrations as a function of time and various physiological factors (e.g., blood volume, endogenous CO production rate). Table 2 presents "baseline" estimates (using typical values for the various physiological parameters) of COHb levels expected to be reached by adult nonsmokers exposed to various constant concentrations of CO for either 1 or 8 hours based on the Coburn model.

TABLE 2.—PREDICTED COHb RESPONSE TO EXPOSURE TO CONSTANT CO CONCENTRATIONS ASSUMING TYPICAL VALUES FOR PHYSIOLOGICAL PARAMETERS

[Percent COHb Based on Coburn Equation[a] Exposure Time

CO (ppm)	1 hour exposure		8 hours exposure	
	Intermittent rest/light activity	Moderate activity	Intermittent rest/light activity	Moderate activity
7.0	0.7	0.7	1.1	1.1
9.0	0.7	0.8	1.4	1.4
12.0	0.8	0.9	1.7	1.8
15.0	0.9	1.1	2.1	2.2
20.0	1.1	1.3	2.7	2.9
25.0	1.2	1.5	3.4	3.6
35.0	1.5	2.0	4.6	4.9
50.0	2.0	2.7	6.4	6.9

[a]Assumed parameters for non-smoking adults: alveolar ventilation rates = 10 liters/min (intermittent rest/light activity) and 20 liters/min (moderate activity); hemoglobin = 15 g/100 ml (normal male); altitude = sea level; initial COHb level = 0.5 percent; endogenous CO production rate = 0.007 ml/min; blood volume = 5500 ml; Haldane constant (measure of affinity of hemoglobin for CO) = 218; lung diffusivity for CO = 30 ml/min/torr.

TABLE 3.—PERCENTAGE OF NON-SMOKING ADULTS WITH CARBOXYHEMOGLOBIN GREATER THAN OR EQUAL TO SPECIFIED PEAK VALUE WHEN EXPOSED TO AIR QUALITY ASSOCIATED WITH ALTERNATIVE EIGHT-HOUR CARBON MONOXIDE STANDARDS [a,b,c]

Peak COHb percent	9 ppm, 8-hr			12 ppm, 8-hr		
	Low pattern	Mid-range pattern	High pattern	Low pattern	Mid-range pattern	High pattern
3.7						<0.01
3.5						0.01
3.3						0.1
3.1					<0.01	0.6
2.9			<0.01		0.01	2
2.7			0.02	<0.01	0.2	9
2.5		<0.01	0.2	0.01	2	36
2.3		0.02	2	0.2	12	84
2.1	<0.01	0.4	10	4	49	100
1.9	0.05	5	53	36	88	100
1.7	3	35	98	91	99	100
1.5	39	88	100	100	100	100
1.3	97	100	100	100	100	100
1.1	100	100	100	100	100	100

[a]COHb responses to fluctuating CO concentrations were dynamically evaluated using the Coburn model prediction of the COHb level resulting from one hour's exposure as the initial COHb level for the next hour. The series of 1-hour CO concentrations used were from 20 sets of actual air quality data. Each pattern was proportionally rolled back or up so that its peak 8-hour average CO concentration equalled the level of the 8-hour standard. Of the 20 selected patterns, results from 3 patterns are presented here. The low pattern tends to give the lowest peak COHb levels, the midrange pattern tends to give a midrange value, and the high pattern tends to give the highest value.

[b]Haldane constant = 218. Alveolar ventilation rate = 10 liters/min. Altitude = 0.0 ft.

[c]The estimation of distributions for each of the physiological parameters used in the Coburn model and the Monte Carlo procedure used to generate these estimates are discussed in Appendix C of the Sensitivity Analysis (Biller and Richmond, 1982).

There are, however, at least two uncertainties involved in estimating COHb levels resulting from exposure to CO concentrations. First, for each of the physiological parameters used in the Coburn model, there is a distribution of values in the general population. These variations are sufficient to produce noticeable deviations from the COHb levels in Table 2, which as noted above were predicted using a set of typical physiological parameter values. Second, predictions based on exposure to constant CO concentrations inadequately represent the responses of individuals exposed to widely fluctuating CO concentrations that typically occur in ambient exposure situations.

As discussed in the proposal preamble (45 FR 55066), EPA attempted to represent these uncertainties in a draft Sensitivity Analysis (EPA, 1980). This analysis used the Coburn model to examine the effects of fluctuating CO concentrations and variations in physiological parameters on COHb estimates. Since proposal, EPA has revised the Sensitivity Analysis to address concerns raised in several public comments and placed the report (Biller and Richmond, 1982) in the docket (QAQPS 79-7, IV-A-8). Table 3 presents estimates of the distribution of COHb levels in the adult non-smoking population based on variations in physiological parameters upon exposure to three different patterns of CO levels which just meet alternative 9 ppm and 12 ppm CO standards. The estimates given in Table 3 and others contained in the Sensitivity Analysis report (Biller and Richmond, 1982) are based on the assumption that the entire adult population is exposed to CO levels just meeting a given standard.

The impact of fluctuating air quality levels on COHb uptake can be roughly estimated by comparing the "baseline" result of a constant 9 ppm exposure for 8 hours (1.4 percent COHb from Table 2) with selected results shown in Table 3. For a "typical" (50th percentile) adult exposed to several different air quality patterns that result in the same maximum 8-hour dose (i.e., 9 ppm, 8-hour average), the results shown in Table 3 indicate that COHb levels ranging from approximately 1.4 to 1.9 percent can be reached. A similar comparison of the results for air quality with a 12 ppm, 8-hour average peak exposure indicates that fluctuating CO levels can increase the peak COHb value from that observed with a constant CO exposure by up to 0.7 percent OCHb (i.e., from 1.7 percent COHb in Table 2 to 2.4 percent COHb for the 50th percentile in the high pattern column of Table 3).

The Sensitivity Analysis results in Table 3 also illustrate the effect of using distributions of values for each physiological parameter rather than just a single representative set of values in applying the Coburn model. For any given air quality pattern, the effect of using the distribution of values for each physiological parameter is to generate a distribution of peak COHb levels that the population would reach. For example, the results in Table 3 show that 95 percent of the population is estimated to be within ±0.3 percent COHb of the median adult value after exposure to the midrange pattern with a peak 9 ppm, 8-hour average. This variation in COHb levels attained is one further source of uncertainty that is being considered in judging which standards provide an adequate margin of safety.

Since proposal, EPA has made considerable improvements in its exposure analysis methodology which, unlike the Sensitivity Analysis, treats movement of people and variation of CO concentration levels through time and space. EPA believes that the revised Exposure Analysis described below, represents the best available tool for estimating the percentage of the population that would reach various CO concentrations and COHb levels upon attainment of alternative CO standards. Since the Exposure Analysis model simulates the exposure of individuals on an hourly basis and simulates actual air quality patterns, the impact of fluctuating CO levels is largely taken into account in the Exposure Analysis results. The results of the revised Sensitivity Analysis are useful, however, in characterizing the uncertainties resulting from variations in physiological parameters in the population which at this time are not fully accounted for in the Exposure Analysis.

Exposure Analysis Estimates

EPA's revised exposure analysis report and addendum, "The NAAQS Exposure Model (NEM) Applied to Carbon Monoxide," (Johnson and Paul, 1983; Paul and Johnson, 1985) contain estimates of the numbers and percentages of urban American adults that would be exposed to various ambient CO levels if alternative 8-hour CO standards were just attained. In addition, estimates have been made of the percentage of this population that would exceed selected COHb levels each year. These latter estimates were derived by applying the Coburn model, which relates patterns of CO exposure to resultant COHb levels, to the exposure model outputs using a typical set of physiological parameters for adult men and a separate set of physiological parameters for adult women.

In contrast to the Sensitivity Analysis, the Exposure Analysis simulates pollutant concentrations and the activities of people with regard to time, place, and exercise level. In the exposure model, the population is represented by a set of "cohorts" (i.e. age-occupational groups that tend to "track together" in time and space). For each hour of the year each cohort is located in one of five "microenvironments." A microenvironment is a general physical location such as indoors-at-home or inside a transportation vehicle.

CO levels in each of the microenvironments are estimated by the use of multiplicative "transformation factors," which relate CO levels recorded at the nearest monitor to estimate CO levels for each microenvironment. This method is used since attainment of a standard is defined in terms of the monitoring system. Values of the multiplicative transformation factors that would give the best exposure estimates are uncertain. The values used for these factors were estimated making use of the available literature on (1) indoor and inside-motor-vehicle air pollution and (2) statistical analyses of monitoring data (e.g., how ambient values change with height and distance from a monitor). A more detailed description of the approach, input data, and assumptions used to derive exposure estimates appears in the Exposure Analysis reports (Johnson and Paul, 1983; Paul and Johnson, 1985).

The exposure analysis model described above was applied to four urban areas: Chicago, Los Angeles, Philadelphia, and St. Louis. Exposure estimates for the adult population living in urban areas in the United States were obtained primarily by associating each urban area in the United States having a population greater than 200,000 with one of the four cities mentioned above. The association was made on the basis of geographic proximity to one of the base areas, average wind speed, peak CO concentrations, observed climate, and general character of the area.

TABLE 4.—CUMULATIVE PERCENT OF ADULT POPULATION IN URBAN AREAS WHOSE COHb Levels Would Exceed Specified COHb Values Upon Attainment of Alternative 8-Hour Standards [a,b,c]

COHb level exceeded (percent)	8-Hour standards		
	9 ppm 1 expected exceedance	12 ppm 1 expected exceedance	15 ppm 1 expected exceedance
3.0		<.01	1.1
2.9		.02	2.5
2.7		0.1	5.9
2.5		0.8	9.7
2.3	<0.1	4.1	14
2.1	0.1 (<.01)[d]	8.6	20

[a] Projected cardiovascular and peripheral vascular disease population in all urbanized areas in the United States for 1987 is 5,240,000 adults.
[b] These exposure estimates are based on air quality distributions which have been adjusted to just attain the given standards.
[c] These exposure estimates are based on best judgments of microenvironment transformation factors. Projections for one urban area based on lower and upper estimates of the microenvironment transformation factors are provided in the Exposure Analysis report (Johnson and Paul, 1983).
[d] Exposure estimate for current 9 ppm, 8-hour average deterministic standard.

Table 4 provides estimates for 1987 of the percentage of the adult population living in urban areas who would exceed various COHb levels upon attainment of three alternative 8-hour standards. For example, less than 0.01 percent of the adult population in urban areas is estimated to exceed 2.1 percent COHb due to CO exposures associated with attainment of the current (deterministic) 9 ppm standard, and approximately 20 percent of the adult population is estimated to exceed 2.1 percent COHb upon attainment of a 15 ppm standard with 1 expected exceedance allowed per year. It should be noted that the estimates given in Table 4 are based on air quality distributions which have been adjusted to just attain the given standards.

Several factors make the accuracy of the nationwide exposure estimates uncertain. They include: (1) the paucity of information on several of the needed inputs (e.g., some of the microenvironment multiplicative transformation factors) and (2) the fact

that nationwide estimates were extrapolated from only four urbanized areas. In addition, the results of the Coburn Model Sensitivity Analysis, discussed previously, suggest that the use of two representative sets of physiological parameters (one for men and one for women), rather than the full distributions of the physiological parameters for the population, in applying the Coburn model to derive COHb estimates introduces some further uncertainty, although the uncertainty about such inputs as the microenvironment multiplicative transformation factors is much larger. Exposure Analysis report (Johnson and Paul, 1983) describes some limited sensitivity analysis runs for one urbanized area to give a rough idea of the range of possible COHb levels that would be reached.

Decision on the Primary Standards

The Staff Reassessment and 1984 CASAC findings and recommendations set forth a framework for considering whether revision of the primary CO standards is appropriate at this time. The discussion that follows relies heavily on that framework and on applicable supporting material in the 1979 Criteria Document, 1984 Addendum, 1979 Staff Paper, and earlier CASAC closure letters (Hovey, 1979; Friedlander, 1982).

Based on its assessment of scientific evidence discussed previously in this notice, EPA concludes that adverse health effects clearly occur, even in healthy individuals, at COHb levels in the range of 5 to 20 percent. EPA also remains concerned that adverse health effects may be experienced by large numbers of sensitive individuals with COHb levels in the range 3.0 to 5.0 percent. On this point, CASAC similarly concluded after reviewing the scientific literature (not including the Aronow studies), "that the critical effects level for NAAQS-setting purposes is approximately 3 percent COHb (not including a margin of safety)" (Lippmann, 1984).

In addition to concurring with EPA that cardiovascular effects are likely to occur at approximately 3 percent COHb (Anderson et al., 1973), the CASAC also indicated that several studies (Drinkwater et al., 1974; Raven et al., 1974; and Davies and Smith, 1980) reporting physiological effects in the range 2.3–2.8 percent COHb lend support to concerns about low level CO exposures and should be considered in determining whether a given standard provides an adequate margin of safety. As discussed previously, the Administrator has excluded Dr. Aronow's studies from consideration in his decision on the CO primary standards.

Based on the exposure analysis results summarized previously in Table 4 of this notice, 8-hour single exceedance CO standards in the range 9 to 12 ppm are estimated to keep more than 99 percent of the non-smoking cardiovascular population below 3.0 percent COHb. Different standards within this range would, of course, provide different levels of protection. For example, the current 9 ppm, 8-hour average standard is estimated to keep more than 99.9 percent of the adult cardiovasular population below 2.1 percent COHb. A 12 ppm, 8-hour standard is estimated to keep more than 99 percent of the population below 2.5 percent COHb. The 2.5 percent COHb level is in the range where physiological effects of concern to EPA and CASAC have been reported.

In considering whether revision of the existing primary CO standards is appropriate at this time, the Administrator has considered uncertainties regarding the lowest levels of COHb at which adverse health effects may occur, as well as uncertainties about the levels of COHb likely to result from CO exposure at the levels associated with attainment of alternative standards. More specifically, the Administrator considered the following factors and sources of uncertainty, discussed in the Staff Reassessment (EPA, 1984), in his decision:

1. Human susceptibility to health effects and the levels at which these effects occur vary considerably among individuals, and EPA cannot be certain that experimental evidence has accounted for the full range of susceptibility. In addition, for ethical reasons, clinical investigators have generally excluded from their studies individuals who may be especially sensitive to CO exposure, such as those with a history of myocardial infarction or multiple disease states (e.g., both angina and anemia). Another factor is that, once the Aronow et al. studies are excluded, there are no human exposure studies investigating effects on individuals with angina at COHb levels below the 2.9 percent level reported by Anderson et al. (1973).

2. There is some animal study evidence indicating that there may be detrimental effects on fetal development (e.g., reduced birth weight, increased newborn mortality, and behavioral effects) associated with CO exposure. Similar types of effects have also been found in studies examining effects of maternal smoking on human fetuses. However, it is not possible at this time to sort out the confounding influence of other components of tobacco smoke in causing the effects observed in the human studies. While human exposure-response relationships for fetal effects remain to be determined, these findings denote a need for caution in evaluating the margin of safety provided by alternative CO standards.

3. Other groups that may be affected by ambient CO exposures but for which there is little or no experimental evidence include: anemic individuals (over 3 million individuals), persons with chronic respiratory diseases (about 14 million individuals), elderly individuals (over 24.7 million individuals over 65 years old), visitors to high altitude locations, and individuals on certain medications. The levels of COHb that might produce adverse effects for each of these groups is uncertain. However, elevated COHb levels in even a small percentage of this very large potentially sensitive population would translate to a significant number of individuals. The large size of the potentially sensitive population, then, argues for standards providing a greater margin of safety.

4. There are a number of uncertainties regarding the uptake of CO, including those related to the accuracy of the Coburn equation, in assessing variations in the population due to differing physiological parameters and exposure to varying air quality patterns.

5. Several factors contribute to uncertainties about the exposure estimates of the expected number of individuals achieving various COHb levels upon attainment of alternative standards. These factors include: the paucity of information on several of the needed inputs, the fact that nationwide estimates were extrapolated from only four urbanized areas, and the use of two representative sets (one for men and one for women) of physiological parameters (e.g., blood volume) rather than distributions of physiological parameters in applying the Coburn model to derive COHb estimates in the exposure analysis. As indicated in the Staff Reassessment (pp. 18–21), some individuals with physiological parameters that maximize uptake of COHb, if exposed to certain patterns of air quality attaining a 12 ppm, 8-hour standard, would likely exceed 3.0 percent COHb. Consequently, the Agency is concerned that a 12 ppm, 8-hour standard may not provide an adequate margin of safety. EPA is continuing its CO exposure research efforts, which should lead to future

improvements in its exposure analysis and a better capability to assess the accuracy of the exposure estimates (Johnson, 1984; Hartwell et al., 1984).

6. There is uncertainty regarding adverse health effects that may result from very short duration, high-level CO exposures (the bolus effect). As discussed in the proposal notice (45 FR 55077), existing air quality data indicate that attainment of a 9 ppm, 8-hour averaging time standard should limit the magnitude of short-term peak concentrations.

7. There is some concern about possible interactions between CO and other pollutants, although there is little experimental evidence to document such interactions at this time.

8. Given the unsettled nature of the scientific information bearing on the level of the standard, EPA believes that there is much to be said for a policy that would maintain the status quo until the data become more adequate. That is particularly so given the absence of the information indicating with some assurance that the present standard is set at an incorrect level. For the reasons stated elsewhere in this preamble, the available information falls short of supporting such finding.

The CASAC concurred with the ranges of 9 to 12 ppm for the 8-hour and 25 to 35 ppm for the 1-hour primary standards recommended in the Staff Reassessment and characterized them as scientifically defensible standard ranges. They recommended that the Administrator consider choosing standards that maintain approximately the same level of protection as that afforded by the current standards (Lippman, 1984). In making its recommendation the CASAC cited the uncertainties associated with the scientific data base and margin of safety concerns and its belief" . . . that where the scientific data, as in this case, are subject to large uncertainties, it is desirable for the Administrator to consider a greater margin of safety than the numerical values of COHb generated by the Coburn equation might otherwise suggest" (Lippmann 1984).

Several ongoing human health effect studies are being conducted by EPA and other groups to investigate the effect of CO on aggravation of angina. Subjects in these studies are being exposed to CO levels that result in blood COHb levels in the range 2.0 to 4.0 percent. One study is being conducted by EPA, several are being conducted at the request of EPA by the Health Effects Institute (HEI), and another study is being sponsored by the California Air Resources Board (CARB). The Health Effects Institute is an independent organization which sponsors research on automotive pollutants and is jointly funded by EPA and automobile manufacturers. The peer-reviewed results of these studies are expected by December 1986. EPA will be initiating development of a new criteria document for CO in Fiscal Year 1986. Thus, the Agency should be in a good position to expeditiously assess the results from the above health studies, as well as other scientific evidence published since completion of the Addendum.

Given the uncertainty in the current data base and the fact that new studies are in progress, the Administrator considered deferring a final decision on the CO standards until these new studies have been completed. While this might allow the Administrator to base the decision on better information, the Clean Air Act appears to require periodic decisions on NAAQS notwithstanding any uncertainty in the data base. Deferring this decision would imply that the Agency has inadequate evidence to select an appropriate course of action at this time. However, both EPA and the CASAC have concluded that, even without the Aronow studies, a sufficient scientific data base exists upon which to base a decision. Given these findings, and the concern that a deferral of the decision could be perceived as a signal to relax or delay ongoing control programs, the Administrator has concluded that final action now on the CO standards is warranted and justified.

In reaching a final decision, the Administrator has focused primarily on the level of protection the CO standards should provide. As previously noted, after reassessing the available scientific data and EPA staff recommendations, the CASAC has recommended that the CO standards provide protection approximately equal to the current standards. Based on EPA's assessment of the scientific data, the Administrator concurs with this recommendation. Retention of the current standards would, of course, satisfy this objective.

As discussed above, the standards proposed in August 1980 would also provide approximately the same level of protection as that afforded by the current standards. In addition, these standards would provide some technical changes that the Agency believes would be advantageous. However, the proposed changes would require some modification in the way attainment of the standards is determined. Given the uncertainties in the existing data base and the possibility that the results of the new studies in progress might warrant further revisions within a few years, the Administrator has concluded that it would not be prudent to promulgate the proposed revisions at this time, particularly when they would alter the manner in which attainment is determined.

In short, the Administrator has concluded that it would not be prudent to defer a decision on the CO standards pending the results of the new research mentioned above, that the standards should provide approximately the same level of protection as that afforded by the current standards, and that disruption of ongoing control programs should be minimized. Given these considerations, the Administrator has concluded that the best course of action is not to revise the CO primary standards at this time. Together with the Administrator's decision to rescind the secondary standards (discussed below), this decision completes the current review of the NAAQS for CO under section 109(d). The ongoing studies conducted by the Agency, by the HEI, and by the CARB (all discussed above) will, of course, be considered in the next 5-year review cycle of the CO criteria and standards. Upon completion of this series of studies, the Agency plans to review the results with the CASAC and assess the need for any revisions in the primary standards. If revisions in the standards seem appropriate, the Agency will act expeditiously to revise the standards.

Welfare Effects and the Secondary Standards

Carbon monoxide is a normal constituent of the plant environment. Plants can both metabolize and produce CO. This may explain the fact that relatively high levels of CO are necessary before damage occurs to vegetation. The lowest level for which significant effects on vegetation have been reported is 100 ppm for 3 to 35 days. The effect observed in this study was an inhibition of nitrogen fixation in legumes. Since CO concentrations of this magnitude are rarely if ever observed in the ambient air, it is very unlikely that any damage to vegetation will occur from CO air pollution. No other effects on welfare have been associated with CO exposures at or near ambient levels. Because no standards appear to be requisite to protect the public welfare from any known or anticipated adverse effects from ambient CO exposures, EPA is rescinding the existing secondary standards.

Significant Harm Levels

Section 303 of the Clean Air Act authorizes the Administrator to take

certain emergency actions if pollution levels in an area constitute "an imminent and substantial endangerment to the health of persons." EPA's regulations governing adoption and submittal of SIP's contain a provision (40 CFR 51.16) that requires the adoption by States of contingency plans to prevent ambient pollutant concentrations from reaching specified significant harm levels. The existing significant harm levels for CO were established in 1971 (36 FR 24002) at the following levels and averaging times:

50 ppm—8-hour average
75 ppm—4-hour average
125 ppm—1-hour average

Exposure under these conditions could result in a widespread blood COHb concentration of 5 to 10 percent, the range EPA has determined would cause significant harm. On the basis of EPA's reassessment of the earlier data and assessment of more recent medical evidence, no modifications are being made to the existing significant harm designations. EPA's assessment of the medical evidence on exposure to higher CO concentrations that could lead to significant harm is contained in the 1979 Criteria Document (EPA, 1979b) and in the Addendum (EPA), 1984b).

Summary of Public Comments and Agency Responses

Overview of Comments

The following discussion summarizes in general terms the comments received, at various times since the 1980 proposal, from the public and from Federal and State agencies on the levels of the primary standards and on the related issue of multiple exceedance standards. Significant comments on all aspects of the CO proposal and Agency responses to these comments are summarized by category later in this section. A more detailed description of individual comments and Agency responses is contained in the public docket (OAQPS 79–7).

Comments on 1980 Proposal. Of the written comments received during the initial comment period (which closed November 24, 1980) that expressed some opinion on the level of the proposed 8-hour standard, 7 out of 11 favored EPA's proposed standard or a more stringent standard. Environmental group comments endorsed the proposed standard or recommended tightening of the standard, industry groups favored relaxation of the standard, and local and State agency comments supported the proposed standard or some relaxation of the standard.

Several comments were received on the Agency's proposal to lower the 1-hour standard to 25 ppm. Of the 18 written comments which expressed an opinion on the 1-hour standard, 8 comments favored the proposed standard level, 5 comments urged retention of the existing 35 ppm standard level, and 3 comments recommended revocation of the 1-hour standard. There also was one comment favoring further tightening of the 1-hour standard and one comment favoring a relaxation of the standard. Those favoring revocation of the 1-hour standard argued that the 1-hour standard was redundant since attainment of the 8-hour standard would effectively prevent 1-hour average levels from exceeding either 25 or 35 ppm.

Comments on the proposed revisions were received from five Federal agencies. Four of these agencies neither supported nor opposed the proposed standards. The Department of Commerce urged EPA to maintain the existing 35 ppm, 1-hour standard. The Council on Wage and Price Stability suggested, through the Regulatory Analysis Review Group, that the proposed standards should be based on cost-effectiveness analysis and that if costs were considered, the proposed standards might be excessively stringent.

Comments on Subsequent Notices. As discussed earlier in this notice, EPA announced an additional public comment period on June 18, 1982 to address several key issues, including the Agency's consideration of multiple expected exceedance 8-hour CO standards (47 FR 26407). Of the comments that expressed an opinion on the issue of multiple expected exceedances, 12 out of 23 favored the use of the multiple expected exceedances approach for ambient standards. While State and local governments' comments were almost evenly divided on the question, all industrial groups supported the concept and all environmental groups opposed multiple exceedance standards. Comments from private citizens were evenly divided for and against multiple exceedance standards.

During the period from November 24, 1980 through August 6, 1982 (the close of the second comment period), of those commenting on whether the 8-hour standard should be relaxed, 10 out of 45 favored relaxation. Most of the States (12 of 14) opposed relaxation, while 3 of 5 local governments favored relaxation. As in the earlier comment period, all industry comments favored relaxation while all environmental groups opposed relaxation.

Subsequent to the close of the second public comment period, the State and Territorial Air Pollution Program Administrators (STAPPA) submitted a resolution, endorsed by a majority of State air pollution agencies, which expressed several reservations about a multiple exceedance CO standard. The STAPPA resolution recommended promulgation of single exceedance CO standards.

EPA solicited additional public comment on the draft Staff Reassessment which evaluated the scientific data in view of the diminished value of Dr. Aronow's CO studies on September 16, 1983 (48 FR 41608). Of the 13 comments received in response to this notice that expressed an opinion on the levels of the standards, 7 favored retaining the current primary standards, 2 favored the proposed standards (i.e., 9 ppm for the 8-hour averaging time and 25 ppm for the 1-hour averaging time), and 4 favored relaxation of the 8-hour standard to 12 ppm or higher. Those supporting the proposed or current standards included an environmental group, several State environmental agencies, two health scientists, and one local environmental agency. Those favoring relaxation of the primary 8-hour standard included two from the automotive industry and two local environmental agencies.

On August 9, 1984, EPA summarized the basis for EPA's proposed revisions to the CO standards, announced availability of the final Addendum (EPA, 1984b) and Staff Reassessment (EPA, 1984a), and solicited additional public comment (49 FR 31923). Of the 7 comments received that expressed an opinion on the levels of the standards, 2 favored retaining the current primary standards, 2 favored the proposed primary standards (i.e., 9 ppm, 8-hour and 25 ppm, 1-hour), and 3 favored relaxation of the 8-hour standard to 12 ppm or higher. Two commentors argued that EPA should defer final action on the CO standards until the Health Effects Institute's CO research was completed.

Summary of Significant Comments and Agency Responses

Significant comments are summarized and responded to by category below.

I. Health Effects Criteria and Selection of the Primary Standards

A. Definition of An Adverse Health Effect

Comment: It is questionable whether a reduction in the time to onset of an angina attack following exercise is adverse.

Agency Response: EPA concludes that aggravation of angina represents an

adverse health effect. As explained in the proposal preamble (45 FR 55066), EPA considers such aggravation of angina to be an adverse health effect for several reasons. First, it may result in cardiovascular damage, which is unquantifiable using present technology. Second, aggravation of angina may be the first in a series of progressively more serious symptoms that accompany cardiovascular disease. At low levels of oxygen deprivation, angina patients experience symptoms of chest pain described above. Coronary insufficiency is a more serious symptom that occurs at greater levels of oxygen deprivation. This symptom is sometimes accompanied by changes in enzyme levels and electrocardiographic irregularities. The most serious effect in this continuum of effects is myocardial infarction. In addition to longer and more intense pain, myocardial infarction is accompanied by irreversible heart damage (death of myocardial cells) as indicated by enzyme level changes and electrocardiographic alterations. Finally, because aggravation of angina may be the first in a series of symptoms that may lead to permanent heart damage, EPA considers aggravation of angina an adverse effect and an indicator that more serious effects may occur in some individuals at the same COHb levels. EPA's judgment has been supported by the CASAC and CASAC CO Subcommittee (Hovey, 1979; Lippmann, 1984).

Comment: Scientific research on CO suggests that there is no clear threshold level at which adverse health effects begin. Rather, there appears to be an increase in severity of potential health effects with increasing levels of CO. The fact that a no-effects level cannot be identified may lead to a standard near zero. The current evidence can support standards other than those proposed by EPA.

Agency Response: EPA agrees that there is a continuum of effects consisting of COHb levels at which health effects are certain, through levels at which scientists can generally agree that medically significant effects have been convincingly shown, and down to levels at which health effects are less certain, are harder to identify, and the medical significance of which are typically disputed. In short, the present scientific evidence does not permit selection of an undisputed value for a primary standard. Rather, the Administrator in selecting a standard must make a judgment as to the level of physiological response that should be considered adverse and the relative acceptability of various degrees of uncertainty that any given level is low enough to prevent known and possible adverse health effects.

B. Scientific Validity of the Human Experimental Studies

Comment: Work of Dr. Wilbert Aronow is suspect because results are too consistent, data are not available for evaluation, the study was not double blind, and the subjects tested were not a representative set of angina patients. The Horvath Committee (Horvath et al., 1983) recommended that EPA not rely on Dr. Aronow's studies in setting the CO standards.

Agency Response: The Administrator agrees with the Horvath Committee that there are very serious technical questions about the validity of Dr. Aronow's CO studies and, therefore, is not considering the results of these studies in his decision.

Comment: The Anderson et al. (1973) study should be eliminated from consideration because Anderson et al. reported a COHb level of only 4.5 percent after a four hour, 100 ppm CO exposure, whereas other investigators have generally reported 8–10 percent COHb for similar exposures.

Agency Response: During the Anderson et al. (1973) study, subjects were allowed breaks and, thus, were exposed to CO intermittently, not continuously, for 4 hours. This would cause COHb levels to be substantially lower than for the continuous exposure periods used in other studies.

Comment: The Anderson et al. (1973) study should not be considered for drawing conclusions about lowest observed effect levels because of the (1) small number of subjects, (2) questionable double-blind conditions, (3) missing data for some subjects, (4) lack of dose-response relationships for onset and duration of angina in some subjects, (5) reliance on subjective responses, and (6) inadequate presentation of statistical procedures.

Agency Response: EPA has carefully reviewed the Anderson et al. (1973) study and the above criticisms of the study. A detailed response is contained in a February 20, 1985 memorandum prepared by EPA's scientists in the Office of Research and Development which has been placed in the public docket (OAQPS 79–7, IV–B–2). EPA does not challenge the proposition that the Anderson study has limitations that must be weighed in assessing the study's value for standard setting. Very few, if any, studies are perfect and thus conclusively demonstrate precise pollutant thresholds for the entire range of sensitive persons under various environmental conditions. However, the Agency does not agree with the commenters' characterization of the Anderson study limitations nor with the commenters' conclusion that those limitations are grounds for totally excluding the study from an assessment of the health risks of CO. After carefully evaluating the scientific merit of the study and considering the advice provided by the CASAC, EPA has concluded that the Anderson study should be considered in selecting a CO standard with an adequate margin of safety.

EPA's responses to the major points of concern about the Anderson et al. (1973) study are briefly summarized below:

1. EPA readily acknowledges that the subject sample size is small and that the study would be strengthened if the number of subjects had been greater. However, the sample size is adequate to treat the data statistically and large enough to use in making statistical inferences regarding effects and causal agents. These inferences are routinely made by clinical investigators conducting studies with relatively few subjects and treatment replications. In addition, even though only a small number of subjects were tested, they do represent some portion of the national population suffering from angina.

2. Because of the explicit precautions taken by the author to ensure the integrity of such a protocol, EPA rejects the speculative assertion that the study was not double blind. Specifically, a "double blind" study is one where both the administering investigator and patient are unaware of the nature of any treatment. EPA has contacted the author (Docket ECAO CD 78–3, IIA–J–3) who reaffirmed the study protocol which is consistent with the definition of double blind. The author cited the published study which states:

> A 5-day, double-blind exposure protocol was followed—Only the investigator administering the gas knew which concentration of CO was being used. The patient, technician, and the investigator conducting the exercise test were all unaware of the exposure condition. A curtain separated the gas tanks from the patient—On completion of the five exercise tests for each subject, the ECG records were assigned a random code number and interpreted by an investigator who had not been present and had no knowledge of the exposure sequence.

3. Because of the explicit double blind protocol practiced in the study, there is no basis for concluding that the two subjects who missed one day of exposure had in some way gained knowledge of the exposure conditions due to their missing one of the exposure

sessions. EPA rejects this assertion as illogical and unfounded.

4. The fact that the effects reported were not dose-related in the Anderson et al. study is not surprising given the small range of exposure concentrations, the fact that the exposures were close to the lower limits for CO-related effects, and the small number of subjects. However, EPA has not used this or any other study to develop a dose-response relationship which would show different levels of subject response over a specific range of administered CO doses. EPA has argued that the Anderson study does suggest an increased risk of aggravation of angina for sensitive individuals who have elevated COHb levels.

5. The fact that the endpoint measured by Anderson et al., namely time to onset of angina pian, is subjective in nature is no basis for ignoring this serious and important effect as carefully articulated in the proposal preamble (45 FR 55069):

> EPA considers such aggravation of angina to be an adverse health effect for several reasons. First, it may result in cardiovascular damage, which is unquantifiable using present technology. Second, aggravation of angina may be the first in a series of progressively more serious symptoms that accompany cardiovascular disease. At low levels of oxygen deprivation, angina patients experience symptoms of chest pain described above. Coronary insufficiency is a more serious symptom that occurs at greater levels of oxygen deprivation. This symptom is sometimes accompanied by changes in enzyme levels and electrocardiographic irregularities. The most serious symptom in this continuum of effects is myocardial infarction. In addition to longer and more intense pain, myocardial infarction is accompanied by irreversible heart damage (death of myocardial cells) as indicated by enzyme level changes and electrocardiographic alterations. Finally, because aggravation of angina may be the first in a series of symptoms that may lead to permanent heart damage, EPA considers aggravation of angina an adverse effect and an indicator that more serious effects may occur in some individuals at the same COHb levels.

6. EPA has reanalyzed the data from the Anderson et al. study using a more conservative multivariate analysis which tests each health endpoint separately for purposes of inference (see Appendix A in the Addendum; EPA 1984b). EPA believes that, had Anderson et al. originally used this technique, the effect of time to onset of angina would have been statistically significant ($p=0.014$ at the 97.5 percent confidence level) but that duration of angina experience would not have been statistically significant ($p=0.11$ at the 97.5 percent confidence level).

In conclusion, EPA agrees with the CASAC statement,

> . . . that it is important to replicate such a study, but the notion that a study has no validity until it's been replicated is flawed. Based upon its current knowledge of how the study was conducted, CASAC presumes that double blind protocols were, in fact, observed and that discrepancies between observed and predicted COHb levels are not as great or as serious as originally suggested. In summary, while CASAC treats the Anderson et al. study with caution, it can find no substantive reason at this time to dispute the reported values, and it recommends that the Agency not disregard its findings (Lippmann, 1984).

The Agency continues to believe that the Anderson et al. findings can be appropriately interpreted as providing reasonably good evidence of exacerbation of angina symptoms occurring in some segments of the population at approximately 2.9 to 4.5 percent COHb.

Comment: Psychological factors contribute to angina attacks, making it difficult to reproduce experiments linking CO and angina.

Agency Response: Although psychological factors may contribute to angina attacks, it has been reasonably shown in the study by Anderson et al. that exposure to CO does contribute independently to the aggravation of angina. Given the double-blind design of this study, there is no reason to suppose that the subjects were exposed to different psychological factors on experimental CO exposure days than they were on clean air control days.

C. Margin of Safety

Comment: EPA has proposed CO standards with an inappropriate margin of safety. The margin of safety was criticized as being either inadequate or excessive.

Agency Response: The decision regarding an adequate margin of safety is a judgment which must be made by the Administrator after weighing all the medical evidence bearing on the effects of CO. The factors to be taken into account include inconclusive evidence as well as findings from studies that are considered definitive and not subject to challenge. The Administrator has considered uncertainties regarding the lowest levels of COHb at which adverse health effects may occur, as well as uncertainties about the levels of COHb likely to result from CO exposure at the levels associated with attainment of alternative standards. More specifically, the Administrator considered the factors and sources of uncertainty, discussed earlier in the Decision on the Primary Standards section of this notice and in the Staff Reassessment (EPA, 1984).

EPA has examined the health protection afforded by alternatives to the current CO primary standards. For example, attainment of a 12 ppm, 8-hour CO standard is estimated to keep more than 99 percent of the population below 2.5 percent COHb. The 2.5 percent COHb level, however, is in the range where physiological effects of concern to EPA and CASAC have been reported. In addition, as indicated in the Staff Reassessment (pp. 18–21), some individuals with physiological parameters that maximize uptake of COHb if exposed to certain patterns of air quality attaining a 12 ppm, 8-hour standard would exceed 3.0 percent COHb. Consequently, the Administrator is concerned that a 12 ppm, 8-hour standard may not provide an adequate margin of safety. There is no credible scientific evidence that suggests more stringent CO primary standards than the current standards are required to protect public health with an adequate margin of safety.

D. Coburn Model Sensitivity Analysis of COHb Levels

Comment: The term used in the Coburn model analysis for inspired CO pressure was mistakenly interpreted as a pressure in dry ambient air. The term was intended to refer to CO pressure in air saturated with water vapor at body temperature.

Agency Response: EPA agrees with the comment and has revised all of the COHb estimates used in this notice and in the final COHb Sensitivity Analysis (Biller and Richmond, 1982) to reflect this change.

Comment: The parameters utilized in the EPA COHb Sensitivity Analysis were not representative of the medical literature. Modeling efforts of this kind should choose conservative values for physiological parameters in order to err on the side of protecting public health.

Agency Response: The values selected for the physiological parameters used in the Sensitivity Analysis were carefully reviewed with members of the scientific community and with consultants to the CASAC and were judged to be reasonable estimates. EPA does not agree that the most conservative values should be selected for the physiological parameters because this would introduce an additional margin of safety into an analysis attempting to estimate the effects of alternative standards. The Sensitivity Analysis does use a range of values for the various physiological parameters.

Comment: The draft Sensitivity Analysis does not adequately treat

differences in physiological parameters between men and women.

Agency Response: In an Appendix to the revised Sensitivity Analysis (Biller and Richmond, 1982), men and women are modelled separately and appropriate physiological parameters are selected for each sex based on a review of the scientific literature.

E. Exposure Estimates for Sensitive Population Groups

Comment: Various technical factors in the exposure analysis either systematically underestimate or overestimate exposures that would occur upon attainment of alternative standards. The exposure analysis should not be used in selecting the primary standards until all the recommended changes are incorporated into the analysis.

Agency Response: EPA has carefully reviewed these comments and separated them into three categories. The first category includes comments with which EPA agrees and for which changes have been made in the exposure analysis. The revised exposure analysis estimates are similar in magnitude, after taking these comments into account, to the original exposure estimates.

A second category of comments suggested changes that EPA agrees would refine the exposure assessment methodology, but which are not feasible at this time because of limitations on the resources and time available for this standard review. EPA will incorporate these comments into its plan for improving its exposure assessment capability. While the exact impact of these refinements cannot be clearly predicted at this time, EPA agrees with CASAC's conclusion that the exposure analysis is "acceptable given the current state-of-the-art of the scientific community's ability to model physiological and other parameters related to this pollutant" (Friedlander, 1982).

A third category consists of comments where EPA disagrees with the suggested changes. Detailed responses to these comments are provided in the docket (Docket OAQPS 79-7, V-C-1). EPA recognizes that there is uncertainty about the accuracy to the exposure estimates, but it believes that the revised estimates are reasonable given the state-of-the-art of exposure assessment.

While future refinements in the methodology and availability of additional data will undoubtedly reduce the degree of uncertainty in the exposure estimates, EPA believes that the current Exposure Analysis provides a useful tool to estimate the number of sensitive individuals who would be exposed to various COHb levels upon attainment of alternative standards.

II. Form of the Primary Standards

Comment: Some commenters favored retaining the current deterministic form; others supported either EPA's 1980 proposal to adopt single-expected-exceedance (statistical form) or the concept of multiple-expected-exceedances (statistical form) primary CO standards. Those in favor of adopting a statistical form argued that it would minimize the impact of unusual meteorological events on control strategies with minimal reductions in health protection offered by the standard. Those opposed to a multiple-expected-exceedances standard expressed concerns about the increased health risks to the sensitive population and the ability of the public to understand the basis for allowing multiple excursions of the standard levels.

Agency Response: Given the concerns expressed by the vast majority of State air pollution control agencies and others that a single-exceedance standard, either deterministic or statistical, more closely reflects the health basis for the standard and is more readily implemented and understood by the general public, EPA has decided to retain the single exceedance format for the primary standards. While EPA believes that adopting a statistical, single-exceedance standard would be a technical improvement, EPA is deferring any such change in the primary CO standards until the uncertainties regarding the health effects basis for the standards are better resolved.

III. Miscellaneous

A. Natural Background

Comment: It has been reported that 93% of the CO in the atmosphere is produced by vegetation and the oceans and, therefore, anthropogenic sources of CO should be of little concern.

Agency Response: Although much of the CO produced globally may be of natural origin, the elevated CO levels found in urban areas and in rural areas near roadways are principally a result of human activities involving the combustion of fossil fuels.

B. Stating Standards in ppm and Not mg/m^3

Comment: EPA should state the revised CO standards solely in parts per million (ppm) rather than mg/m^3. Changing to ppm would (1) avoid confusion as to the level of the standards at different altitudes, (2) provide additional protection for high altitude areas, and (3) be consistent with health and monitoring data using the Federal Reference Methods which are reported in ppm.

Agency Response: EPA is retaining both systems of units but is indicating that ppm is the preferred system of units. Section 50.3 specifies that measurements of air quality are to be corrected to standard conditions (i.e., 25 degrees centigrade and sea level pressure), the only difference between ppm and mg/m^3 (at standard conditions) is a scale factor that does not vary with altitude. That is, if a measurement is expressed in ppm units or mg/m^3 units (at standard conditions) the values will differ by a factor of 0.875 regardless of altitude. This conversion factor is specified in Appendix D of 40 CFR Part 50.

Revisions to Part 50 Regulations

Because EPA has decided not to revise the CO primary standards, there are no substantive changes to the Part 50 regulations concerning these standards. EPA is revising the text of the standards slightly, however, to make them clearer and more understandable to the public. EPA is also explicitly stating several data handling conventions that have traditionally been used in EPA's data systems and guidance. These changes are not intended to redefine the standard but simply to reduce potential ambiguity and to formalize existing data handling conventions. The specific points are the 75 percent data completeness rule that EPA has used when computing 8-hour averages and the number of significant figures retained when making comparisons with the levels of the standard. These changes were discussed in the proposal notice. EPA is also changing to ppm units as the preferred units as discussed in the proposal notice. Finally, EPA is also rescinding the secondary standards for CO, as proposed, because there is no evidence of welfare related effects at or near ambient levels.

Part 51 Regulations and SIP Development

This action does not modify the existing Part 51 regulations. Current guidance to State and local air pollution control agencies on how to interpret air quality data for purposes of attainment decisions and SIP development is contained in "Guidelines for the Interpretation of Air Quality Standards" (EPA, 1977).

Regulatory and Environmental Impacts

Regulatory Impact Analysis

As has been noted, the Clean Air Act specifically requires that NAAQS be based on scientific criteria relating to the level that should be attained to protect public health and welfare adequately. The courts have endorsed a reading of the Act that excludes reliance on the cost or feasibility of achieving such a standard in determining the level of the primary standards. In response to Executive Order 12291, EPA has prepared a regulatory impact analysis (RIA). However, EPA's analysis, "Regulatory Impact Analysis for Carbon Monoxide," has not been considered in determining the standard levels in this final notice. The document is available from the address given earlier in the For Further Information Contact section of this notice until supplies are exhausted and from the NTIS whose address is given in the Addressees section of this notice.

Both the RIA and this final notice were submitted to the Office of Management and Budget (OMB) for review under Executive Order 12291. Any written comments from OMB and any written EPA responses to those comments are available for public inspection at EPA's Central Docket Section, Docket No. OAQPS 79-7, West Tower Lobby, Gallery I, Waterside Mall, 401 M Street SW., Washington, D.C.

Environmental Impacts

Environmental impacts associated with control of CO emission have been examined in the Environmental Impact Statement, which is available in the docket (OAQPS 79-7, IV-A-13) or from EPA at the address given earlier in the For Further Information Contact Section of this notice. This analysis indicates that control strategies required to attain the standards will have minimal adverse impacts on other environmental media.

Impact on Small Entities

The Regulatory Flexibility Act (5 U.S.C. 601 et seq.) requires that all federal agencies consider the impacts of final regulations on small entities, which are defined to be small businesses, small organizations, and small governmental jurisdictions. EPA's analysis pursuant to this Act is summarized in a section of the final report, "Cost and Economic Assessment of Alternative National Ambient Air Quality Standards for Carbon Monoxide" (EPA, 1985). A NAAQS for CO by itself has no direct impact on small entities. However, it requires each State to design and implement control strategies for those areas not in attainment. Three possible sources of impacts on small entities include (1) the FMVCP for cars and trucks, (2) I&M programs, and (3) stationary source control programs. FMVCP requirements are largely established by statute or by regulatory provisions not directly related to the levels of these standards. In addition, they fall primarily on automobile manufacturers, none of which are classified as small businesses. Additionally, the incremental cost of CO control, which is passed on to purchasers of motor vehicles—including small entities—is a small fraction of the purchase price and, thus, the impact to these purchasers should be negligible.

I&M programs for CO control may have a slight negative economic impact on small entities, but may also have a positive economic impact on some small entities. The estimated per vehicle average annual cost for the CO portion of an I&M program is estimated to be $3.50 for the inspection fee and $19 for repairs to failed vehicles. (In those few areas needing an I&M program for CO only, these estimates would be doubled.) These costs should not impose a significant negative economic impact on small entities. On the other hand, some small entities such as gas stations and garages will be repairing failed vehicles resulting in a net increase in receipts due to a CO I&M program. In addition, if a decentralized I&M program is implemented using small businesses to inspect motor vehicles, then their net receipts will also increase due to receipt of the inspection fee, most of which they retain. (The remainder goes to the governmental unit sponsoring the area-wide I&M program.)

Finally, only the largest stationary sources of CO appear to need to implement controls to attain the CO standards that were analyzed. These sources are among the largest facilities within their standard industrial class, and therefore are not likely to be small entities.

Based on the analysis summarized above, EPA concludes that no small entity group will be significantly negatively affected due to retention of the primary CO NAAQS. Therefore, pursuant to 5 U.S.C. 605(b) the Administrator certifies that this regulation will not have a significant economic impact on a substantial number of small entities.

Impact on Reporting Requirements

There are no reporting requirements directly associated with this action. There are reporting requirements associated with related sections of the Act, particularly sections 107, 110, 160, and 317 (42 U.S.C. 7407, 7410, 7460, and 7617), however, there are no changes in reporting requirements associated with this final action since the current primary standards are being retained.

Federal Reference Method

The measurement principle and calibration procedure applicable to reference methods for measuring ambient CO concentrations to determine compliance with applicable CO standards are not affected by this action. The measurement principle and the calibration procedure for CO are set forth in Appendix C of 40 CFR Part 50. Reference methods, as well as equivalent methods, for monitoring CO are designated in accordance with 40 CFR Part 53. A list of all methods designated by EPA as reference or equivalent methods for measuring CO is available from any EPA Regional Office, or from EPA, Department E (MD-76), Research Triangle Park, N.C. 27711.

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List of Subjects in 40 CFR Part 50

Air pollution control, Carbon monoxide, Ozone, Sulfur oxides, Particulate matter, Nitrogen dioxide, Lead.

Dated: September 5, 1985.

Lee M. Thomas,

Administrator.

PART 50—NATIONAL PRIMARY AND SECONDARY AMBIENT AIR QUALITY STANDARDS

For the reasons set forth in the preamble, EPA amends Chapter I, Part 50, of Title 40 of the Code of Federal Regulations as follows:

1. The authority citation for Part 50 continues to read as follows:

Authority: Sec. 109 and 301(a), Clean Air Act, as amended (42 U.S.C. 7409, 7601(a)).

2. Section 50.8 is revised to read as follows:

§ 50.8 National primary ambient air quality standards for carbon monoxide.

(a) The national primary ambient air quality standards for carbon monoxide are:

(1) 9 parts per million (10 milligrams per cubic meter) for an 8-hour average concentration not to be exceeded more than once per year and

(2) 35 parts per million (40 milligrams per cubic meter) for a 1-hour average concentration not to be exceeded more than once per year.

(b) The levels of carbon monoxide in the ambient air shall be measured by:

(1) a reference method based on Appendix C and designated in accordance with Part 53 of this chapter, or

(2) an equivalent method designated in accordance with Part 53 of this chapter.

(c) An 8-hour average shall be considered valid if at least 75 percent of the hourly average for the 8-hour period are available. In the event that only six (or seven) hourly averages are available, the 8-hour average shall be computed on the basis of the hours available using six (or seven) as the divisor.

(d) When summarizing data for comparision with the standards, averages shall be stated to one decimal place. Comparison of the data with the levels of the standards in parts per million shall be made in terms of integers with fractional parts of 0.5 or greater rounding up.

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