Policy Assessment for the Review of the Carbon Monoxide National Ambient Air Quality Standards

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Policy Assessment for the Review of the Carbon Monoxide National Ambient Air Quality Standards

U.S. Environmental Protection Agency
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An earlier draft of this document was formally reviewed by the Clean Air Scientific Advisory Committee (CASAC) and made available for public comment. This final document has been informed by the expert advice and comments received from CASAC, as well as by public comments.

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1 INTRODUCTION

1.1 PURPOSE

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the carbon monoxide (CO) national ambient air quality standards (NAAQS). The overall plan and schedule for this review were presented in the *Plan for Review of the National Ambient Air Quality Standards for Carbon Monoxide* (IRP; USEPA, 2008b).[1] The IRP identified key policy-relevant issues to be addressed in this review as a series of questions that frame our consideration of whether the current NAAQS for CO should be retained or revised.

This Policy Assessment (PA), prepared by staff in EPA's Office of Air Quality Planning and Standards (OAQPS), is intended to help "bridge the gap" between the relevant scientific information and assessments and the judgments required of the EPA Administrator in determining whether, and if so, how it is appropriate to revise the NAAQS for CO. This PA presents factors relevant to EPA's review of the current primary (health-based) standards and consideration of a secondary (welfare-based) standard. It focuses on both evidence- and risk-based information in evaluating the adequacy of the current CO NAAQS and in identifying potential alternative standards for consideration. In this PA, we consider the scientific and technical information available in this review as assessed in the *Integrated Science Assessment for Carbon Monoxide* (henceforth referred to as the ISA, USEPA, 2010a), prepared by EPA's National Center for Environmental Assessment (NCEA), and the *Quantitative Risk and Exposure Assessment for Carbon Monoxide - Amended* (henceforth referred to as the Risk and Exposure Assessment document or REA, USEPA, 2010b). In so doing, we focus on information that is most pertinent to evaluating the basic elements of NAAQS: indicator,[2] averaging time, form,[3] and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the CO standards.

While this PA should be of use to all parties interested in the CO NAAQS review, it is written with an expectation that the reader has some familiarity with the technical discussions contained in the ISA (USEPA, 2010a) and the REA (USEPA, 2010b).

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[1] As described below in section 1.2.3, the schedule for this review is governed by a court order.

[2] The "indicator" of a standard defines the chemical species or mixture that is to be measured in determining whether an area attains the standard.

[3] The "form" of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. For example, the form of the annual $PM_{2.5}$ NAAQS is the 3-year average of the weighted annual mean $PM_{2.5}$ concentrations, while the form of the current 8-hour CO NAAQS is the second-highest 8-hour average in a year.

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify certain pollutants that meet specified criteria, including emissions which "may reasonably be anticipated to endanger public health and welfare" and whose presence "in the ambient air results from numerous or diverse mobile or stationary sources" and to issue air quality criteria for them. Air quality criteria are to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in ambient air . . ."

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants listed 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 such criteria and allowing an adequate margin of safety, are requisite to protect the public health."[4] A 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 such 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."[5]

The requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was 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, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), cert. denied, 455 U.S. 1034 (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 that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been

[4] The legislative history of section 109 indicates that a primary standard is to be set at the "maximum permissible ambient air level…which will protect the health of any [sensitive] group of the population," and that for this purpose "reference should be made to a representative sample of persons comprising the sensitive group rather than a single person in such group." S. Rep. No.91-1196, 91st Cong., Sess. 10 (1970)

[5] Welfare effects as defined in section 302(h) (42U.S.C. 7602(h)) include, but are not limited to, "effects in soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effect on economic values on personal comfort and well-being."

demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, the EPA considers 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. 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.

In setting standards that are "requisite" to protect public health and welfare, as provided in section 109(b), EPA's task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, EPA may not consider the costs of implementing the standards. See generally *Whitman v. American Trucking Associations*, 531 U.S. 457, 465-472, 475-76 (2001).

Section 109(b)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate . . . ." Section 109(d)(2) requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate . . . ." Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.

1.2.2 Previous Reviews

EPA initially established NAAQS for CO, under section 109 of the Act, on April 30, 1971. The primary standards were established to protect against the occurrence of carboxyhemoglobin levels in human blood associated with health effects of concern. The standards were set at 9 parts per million (ppm), as an 8-hour average and 35 ppm, as a 1-hour average, neither to be exceeded more than once per year (36 FR 8186). In the 1971 decision, the Administrator judged that attainment of these standards would provide protection of public health with an adequate margin of safety and would also protect against known and anticipated adverse effects on public welfare, and accordingly set the secondary (welfare-based) standards identical to the primary (health-based) standards.

In 1985, EPA concluded its first periodic review of the criteria and standards for CO (50 FR 37484). In that review, EPA updated the scientific criteria upon which the initial CO standards were based through the publication of the 1979 *Air Quality Criteria Document for*

Carbon Monoxide (AQCD; USEPA, 1979a) and prepared a Staff Paper (USEPA, 1979b), which, along with the 1979 AQCD, served as the basis for the development of the notice of proposed rulemaking which was published on August 18, 1980 (45 FR 55066). Delays due to uncertainties regarding the scientific basis for the final decision resulted in EPA's announcing a second public comment period (47 FR 26407). Following substantial reexamination of the scientific data, EPA prepared an Addendum to the 1979 AQCD (USEPA, 1984a) and an updated Staff Paper (USEPA, 1984b). Following review by CASAC (McClellan, 1991, 1992), EPA announced its decision not to revise the existing primary standard and to revoke the secondary standard for CO on September 13, 1985, due to a lack of evidence of effects on public welfare at ambient concentrations (50 FR 37484).[6]

On August 1, 1994, EPA concluded its second periodic review of the criteria and standards for CO by deciding that revisions to the CO NAAQS were not warranted at that time (59 FR 38906). This decision reflected EPA's review of relevant scientific information assembled since the last review, as contained in the 1991 AQCD (USEPA, 1991) and the 1992 Staff Paper (USEPA, 1992). Thus, the primary standards were retained at 9 ppm with an 8-hour averaging time, and 35 ppm with a 1-hour averaging time, neither to be exceeded more than once per year (59 FR 38906).

EPA initiated the next periodic review in 1997 and held a workshop in September 1998 to review and discuss material to be contained in the AQCD. On June 9, 1999, CASAC held a public meeting to review the first draft AQCD and to provide a consultation on a draft exposure analysis methodology document. Comments from CASAC Panel members and the public on the AQCD were considered in a second draft AQCD, which was reviewed at a CASAC meeting, held on November 18, 1999. After revision of the second draft AQCD, the final 2000 AQCD (U.S. EPA, 2000) was released in August 2000. EPA put this review on hold when Congress requested that the National Research Council (NRC) review the impact of meteorology and topography on ambient CO concentrations in high altitude and extreme cold regions of the U.S. The NRC convened the Committee on Carbon Monoxide Episodes in Meteorological and Topographical Problem Areas, which focused on Fairbanks, Alaska as a case-study.

A final report, "Managing Carbon Monoxide Pollution in Meteorological and Topographical Problem Areas," was published in 2003 (NRC, 2003) and offered a wide range of recommendations regarding management of CO air pollution, cold start emissions standards, oxygenated fuels, and CO monitoring (see Appendix A). Following completion of the NRC report, EPA did not conduct rulemaking to complete the review.

[6] EPA concluded in 1985 that "no standards appear to be requisite to protect the public welfare from any known or anticipated adverse effects from ambient CO exposures" (50 FR 37494).

1.2.3 The Current Review

On September 13, 2007, EPA issued a call for information from the public (72 FR 52369) requesting the submission of recent scientific information on specified topics. A workshop was held on January 28–29, 2008 (73 FR 2490) to discuss policy-relevant scientific and technical information to inform EPA's planning for the CO NAAQS review. Following the workshop, a draft IRP (USEPA, 2008a) was made available in March 2008 for public comment and was discussed by the CASAC via a publicly accessible teleconference consultation on April 8, 2008 (73 FR 12998; Henderson, 2008). EPA made the final IRP available in August 2008 (USEPA, 2008b).

In preparing the CO ISA, NCEA held an authors' teleconference in November 2008 with invited scientific experts to discuss preliminary draft materials prepared as part of the ongoing development of the CO ISA and its supplementary annexes. The first draft ISA (USEPA, 2009a) was made available for public review on March 12, 2009 (74 FR 10734) and reviewed by CASAC at a meeting held on May 12-13, 2009 (74 FR 15265). A second draft ISA (USEPA, 2009b) was released for CASAC and public review on September 23, 2009 (74 FR 48536), and it was reviewed by CASAC at a meeting held on November 16-17, 2009 (74 FR 54042). The final ISA was released in January 2010 (USEPA, 2010a).

In May 2009, OAQPS released a draft planning document, the draft Scope and Methods Plan (USEPA, 2009c), for consultation with CASAC and public review at the CASAC meeting held on May 12-13, 2009. Taking into consideration comments on the draft Plan from CASAC (Brain, 2009) and the public, OAQPS staff developed and released for CASAC review and public comment a first draft REA (USEPA, 2009d), which was reviewed at the CASAC meeting held on November 16-17, 2009. Subsequent to that meeting and taking into consideration comments from CASAC (Brain and Samet, 2010a) and public comments on the first draft REA, a second draft REA (USEPA, 2010c) was released for CASAC review and public comment in February 2010, and reviewed at a CASAC meeting held on March 22-23, 2010. Drawing from information in the final CO ISA and the second draft REA, a draft PA (USEPA, 2010d) was released in early March for CASAC review and public comment at the same meeting. Taking into consideration comments on the second draft REA and the draft PA from CASAC (Brain and Samet, 2010b, 2010c) and the public, staff completed the quantitative assessments which are presented in the final REA (USEPA, 2010b) and discussed in this PA.

The schedule for completion of this review is governed by a court order resolving a lawsuit filed in March 2003 by a group of plaintiffs who alleged that EPA had failed to perform its mandatory duty, under section 109(d)(1), of completing a review of the CO NAAQS within

the period provided by statute.[7] The court order that governs this review, entered by the court on November 14, 2008 and amended on August 30, 2010, provides that EPA will sign, for publication, notices of proposed and final rulemaking concerning its review of the CO NAAQS no later than January 28, 2011 and August 12, 2011, respectively.

1.3 CURRENT AIR QUALITY

This section provides a general overview of the current air quality conditions to provide context for this consideration of the current standards for carbon monoxide. A more comprehensive discussion of air quality information is provided in the ISA (ISA, sections 3.2 and 3.4), and a more detailed discussion of aspects particularly relevant to the exposure assessment is provided in the REA (REA, chapter 3).

1.3.1 Sources to Ambient Air

Carbon monoxide in ambient air is formed primarily by the incomplete combustion of carbon-containing fuels and by photochemical reactions in the atmosphere. As a result of the combustion conditions, CO emissions from large fossil-fueled power plants are typically very low because optimized fuel consumption conditions make boiler combustion highly efficient. In contrast, internal combustion engines used in many mobile sources have widely varying operating conditions. Therefore, higher and more varying CO formation results from the operation of these mobile sources (ISA, section 3.2). As with previous reviews of the CO NAAQS, mobile sources continue to be a significant source sector for CO in ambient air, as indicated by national emissions estimates from on-road vehicles, which accounted for approximately half of the total CO emissions by individual source sectors in 2002 (ISA, Figure 3-1).[8]

National-scale anthropogenic CO emissions have decreased by approximately 45% between 1990 and 2005 (Figure 1-1), with nearly all of this national-scale reduction coming from reductions in on-road vehicle emissions (ISA, Figure 3-2; 2005 NEI[9]).

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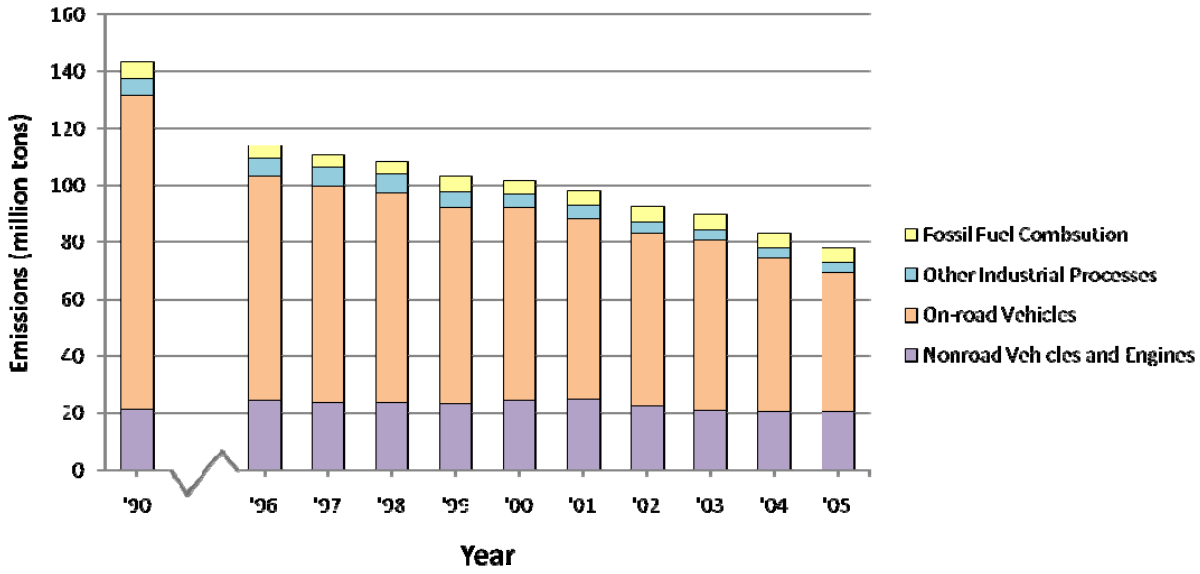
[7] *Communities for a Better Environment, et al. v. EPA* (No. 07-CV-03678, N.D. Cal.).

[8] EPA compiles CO emissions estimates for the U.S. in the National Emissions Inventory (NEI). The 2002 NEI provides the most recent publicly available CO emissions estimates for the U.S. that meet EPA's data quality assurance objectives. Estimates come from various sources and different data sources use different data collection methods, most of which are based on engineering calculations and estimates rather than measurements. Although these estimates are generated using well-established approaches, uncertainties are inherent in the emission factors and models used to represent sources for which emissions have not been directly measured. Uncertainties vary by source category, season and region (ISA, section 3.2.1).

[9] The emissions trends information here and in Figure 1-1are drawn from recently available 2005 National Emissions Inventory estimates (http://www.epa.gov/ttn/chief/net/2005inventory.html, Tier Summaries) and 1990 and other estimates, available at http://www.epa.gov/ttn/chief/net/critsummary.html Figure 3-2 from the ISA provides estimates through 2005.

Figure 1-1. Trends in anthropogenic CO emissions in the U.S. by source category for 1990 and 1996-2005.



The role of mobile source emissions is evident in the spatial and temporal patterns of ambient CO concentrations, which are heavily influenced by the patterns associated with mobile source emissions (ISA, chapter 3). In metropolitan areas of the U.S., due to their greater motor vehicle density relative to rural areas, mobile source contribution to all ambient CO emissions was estimated to be as high as 75% in the 2002 National Emissions Inventory (ISA, p. 3-2). When considering all mobile sources nationwide (non-road and on-road combined), the contribution to total ambient CO emissions nationally is over 80%. As an example, on-road mobile source emissions in urban Denver County, Colorado are estimated to be about 71% of total CO emissions and emissions from all mobile sources (on-road and non-road combined) are estimated to contribute about 98% (ISA, section 3.2). In contrast, on-road CO emissions are estimated to be just 20% of the total for rural Garfield County, Colorado[10] (ISA, section 3, Figure 3-6).

[10] The 2002 National Emissions Inventory estimate for on-road emissions in Garfield is 20,000 tons, and the total emissions from all sources is estimated to be 98,831 (99K) tons. Thus, in this example the on-road vehicles accounts for 20.2% of the total emissions (ISA, section 3, figure 3-6).

1.3.2 Ambient Monitoring Network

Ambient CO concentrations are measured by monitoring networks that are operated by state and local monitoring agencies in the U.S., which are typically funded in part by the EPA. The main network providing ambient data for use in comparison to the NAAQS is the State and Local Air Monitoring Stations (SLAMS) network. CO monitors are typically sited to reflect one of the following spatial scales:

- Microscale: Data represent concentrations in air volumes associated with area dimensions ranging from several meters up to 100 meters. Particularly for CO, microscale monitors have historically been sited 2 – 10 meters from a roadway. These microscale CO measurements have typically represented street canyon and traffic corridors.
- Middle scale: Data represent concentrations in air volumes associated with area dimensions ranging from 100 meters to 500 meters. Such measurements are analogous to CO concentrations representative of several city blocks.
- Neighborhood scale: Defines concentrations within some extended area of the city that has relatively uniform land use with dimensions in the 0.5 to 4.0 kilometers range. Such measurements are intended to represent extended portions of a city.

Currently, there are no minimum monitoring requirements for the number of CO monitoring sites, except in the new National Core (NCore) monitoring network, although continued operation of existing SLAMS CO sites is required until discontinuation is approved by the EPA Regional Administrator.[11] Further, in areas where SLAMS CO monitoring is ongoing, at least one site must be a maximum concentration site for that area under investigation.[12]

The complete NCore network, which will be comprised of multi-pollutant monitoring stations throughout the country, is required to be fully implemented by January 1, 2011. This network will consist of approximately 63 urban and 18 rural stations and will include some existing SLAMS sites that have been modified to include additional measurements. The majority of NCore stations will be sited, however, to represent neighborhood, urban, and regional scales, consistent with the NCore network design objective of representing exposure expected across urban and rural areas in locations that are not dominated by local sources (ISA, p. 3-21). Although NCore stations are intended to meet multiple monitoring objectives, they also provide data that are suitable for comparison to the NAAQS.

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[11] Prior to 2006, minimum CO monitoring requirements called for one "peak" concentration site (near a high traffic road or in an urban core) and one area- or community-wide monitor in any urban area over 500,000 population (44 FR 27558). In 2006, monitoring requirements for CO and other NAAQS pollutants were revised with the establishment of the National Core monitoring network (71 FR 61298).

[12] 40 CFR Appendix D to Part 58, section 4.2. *Carbon Monoxide (CO) Design Criteria*.

EPA has established federal reference methods (FRMs) and methods designated as equivalent (federal equivalent methods, FEMs) for use in ambient air sample collection and analysis to promote uniform enforcement of the air quality standards set forth under the CAA. Measurements for determinations of NAAQS compliance must be made with FRMs or FEMs. More than 95% of FRM or FEM CO monitors in use in the CO monitoring network during 2005-2007 had lower detectable limits (LDLs) of 0.5 ppm (ISA, Appendix A, Table A-8). Given the levels of the CO NAAQS (35 ppm, 1-hour; 9 ppm, 8-hour), a lower detectable limit (LDL) on the order of 0.5 ppm is well below the NAAQS levels and is therefore sufficient for demonstration of compliance.[13] However, with ambient CO levels now routinely near or below 1 ppm, however, a large percentage of the measurements from the CO monitoring network are below the LDL of conventional CO monitors, contributing greater uncertainty in a larger portion of the distribution of monitoring data (ISA, section 3.4.1). For example, more than half of the dataset of nationally reported hourly data for 2005-2007 analyzed in the ISA fell below the reported LDL of 0.5 ppm (ISA, p. 3-56). To reduce the uncertainty in monitoring data collected at these lower concentrations, a new generation of ambient CO monitors has been designed that provides improved sensitivity for measurements at or below the typical ambient CO levels measured in most urban and all rural locations. These sensitive, or so-called 'trace level,' CO monitors generally have LDLs on the order of 0.04 ppm. The number of active monitors employing such sensitive methods is increasing, primarily in association with the implementation of the NCore network.[14] The extent to which these trace-level monitors or other sensitive reference or equivalent monitoring methods become integrated into non-NCore SLAMS stations, however, will depend on the availability of funding for states to replace existing legacy CO monitors as well as the possibility that monitoring requirements for CO might either encourage or require such technological improvements.

[13] Among the 13 approved FRMs in use in the SLAMS network for which data were reported to EPA's Air Quality System (AQS) between 2005 and 2009, nine are "legacy" methods with a federal method detection limit (MDL) listed as 0.5 ppm.

[14] For example, four approved FRMs are newer, more sensitive methods with a federal MDL of 0.02 ppm and a growing body of ambient data from more sensitive CO instruments is becoming available. Testing performed by EPA on several such CO monitors in 2005 and 2006 demonstrated MDLs of approximately 0.017 – 0.018 ppm (17 – 18 ppb), slightly below the stated LDL of 0.02 – 0.04 ppm (ISA, section 3.4.1).

1.3.3 Ambient Monitoring Concentrations

In 2009, approximately 350 ambient monitoring stations across the U.S. reported continuous hourly averages of CO concentrations to EPA's Air Quality System.[15] For the most recent period for which air quality status relative to the CO NAAQS has been analyzed (2009), all areas of the U.S. meet both CO NAAQS.[16] Although one area of the country (Las Vegas, Nevada) is designated in non-attainment with the CO NAAQS, air quality in that area currently meets the standards. In two of the previous three data review periods (2005-2006 and 2006-2007), one area (Jefferson County, Alabama) has failed to meet the 8-hour standard. Large CO emissions sources in this area are associated with an integrated iron and steel facility. As shown in Figures 1-2 and 1-3 below, 2009 concentrations of CO at most currently operating monitors are well below the current standards, with just a few locations having concentrations near the controlling 8-hour standard of 9 ppm as a second maximum 8-hour average.[17]. Of the monitoring sites with extensive records[18] for 2007, sites in two counties reported second-highest 1-hour CO concentrations between 15.1 and 35.0 ppm and sites in five counties reported second-highest 8-hour CO concentration of 5.0 ppm or higher (ISA, section 3.5.1.1).

[15] http://www.epa.gov/ttn/airs/airsaqs/.

[16] The air quality status in areas monitored relative to the CO NAAQS is provided at http://www.epa.gov/air/airtrends/values.html.

[17] As the form of the CO 8-hour standard is not-to-be-exceeded more than once per year, the second highest 8-hour average in a year is the design value for this standard. Based on the current rounding convention, the standard is met if the CO concentrations over a year result in a design value at or below 9.4 ppm. More detailed information on CO NAAQS design values is available at http://www.epa.gov/airtrends/values.html.

[18] During the period 2005-2007, 291 out of 376 monitors sited in 243 different counties, cities or municipalities met the following dataset completeness criteria: 75% of the hours in a day, 75% of days in a calendar quarter and 3 complete quarters for 3 years (ISA, section 3.4.2.2). An exception was made for monitors in U.S. EPA Region 10 for which two rather than three complete quarters were considered to meet the criteria (ISA, p. 3-20).

Figure 1-2. Second maximum 8-hour average concentrations of CO at monitors nationally, 2009.

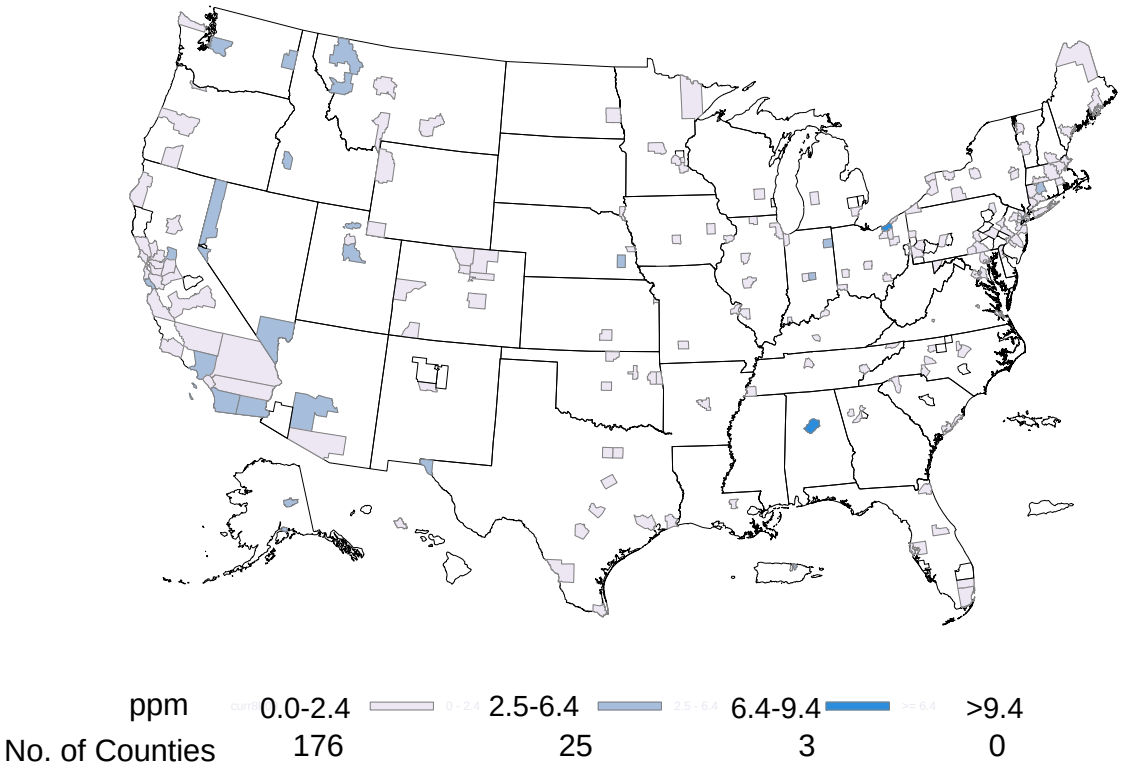
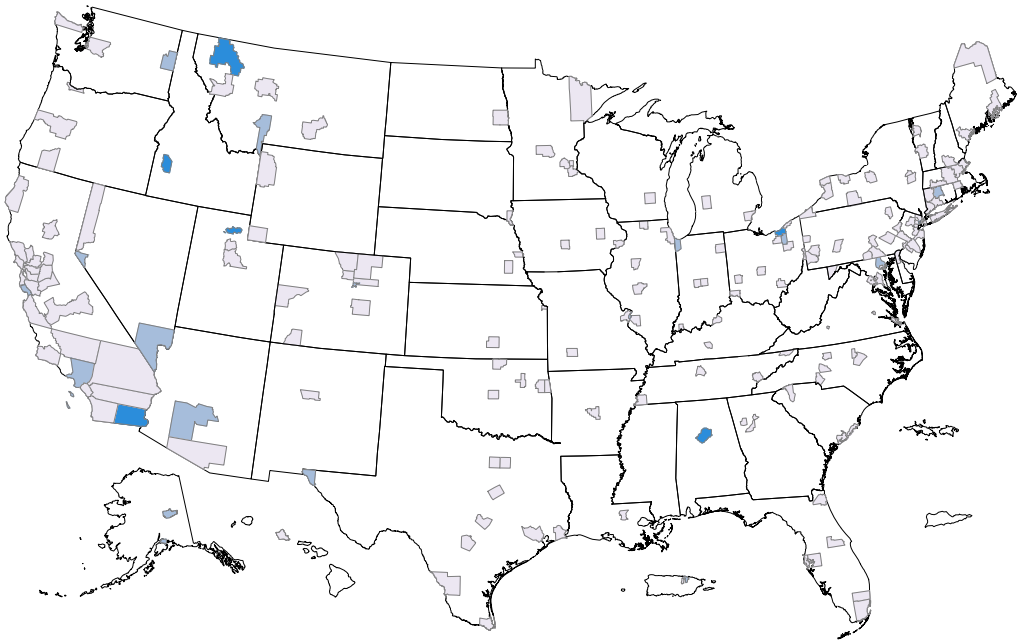


Figure 1-3. Second maximum 1-hour average concentrations of CO at monitors nationally, 2009.



ppm	0.0-4.4	4.5-9.4	9.4-35.4	>35.4
No. of Counties	180	17	7	0

The current levels of ambient CO across the U.S. reflect the steady declines in ambient concentrations that have occurred over the past several years. Both the second highest 1-hour and 8-hour concentrations have significantly declined since the last review (Figures 1-4 and 1-5). At the set of sites across the U.S. that have been continuously monitored since 1990 the average second highest 8-hour and 1-hour concentrations have declined by nearly 70%.

Figure 1-4. Trends in CO concentration (second maximum 8-hour average) in the U.S., 1990-2009. The white line indicates average across the sites; ninety percent of sites have concentrations below the top line, while ten percent of sites have concentrations below the bottom line.

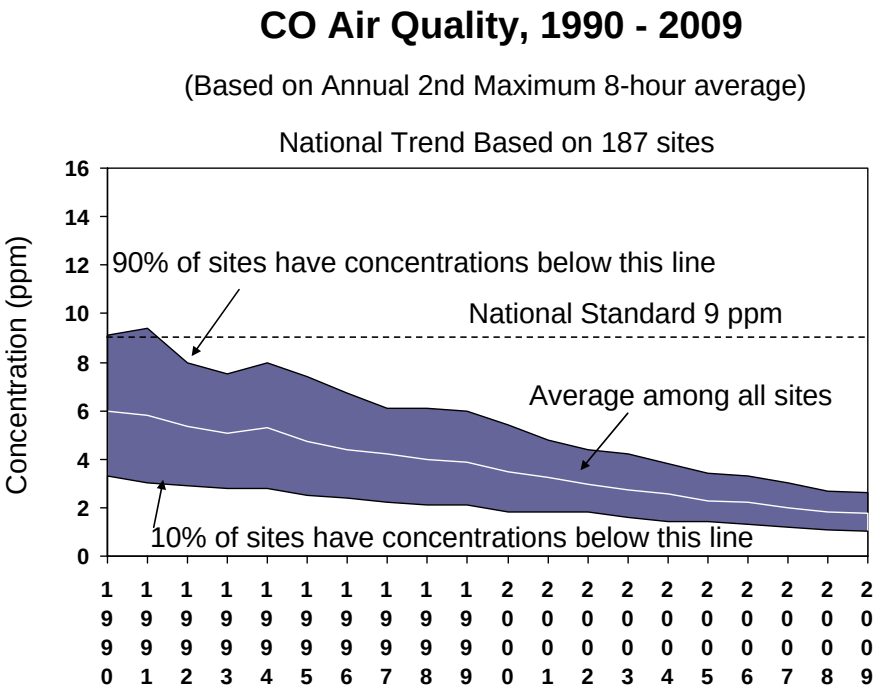
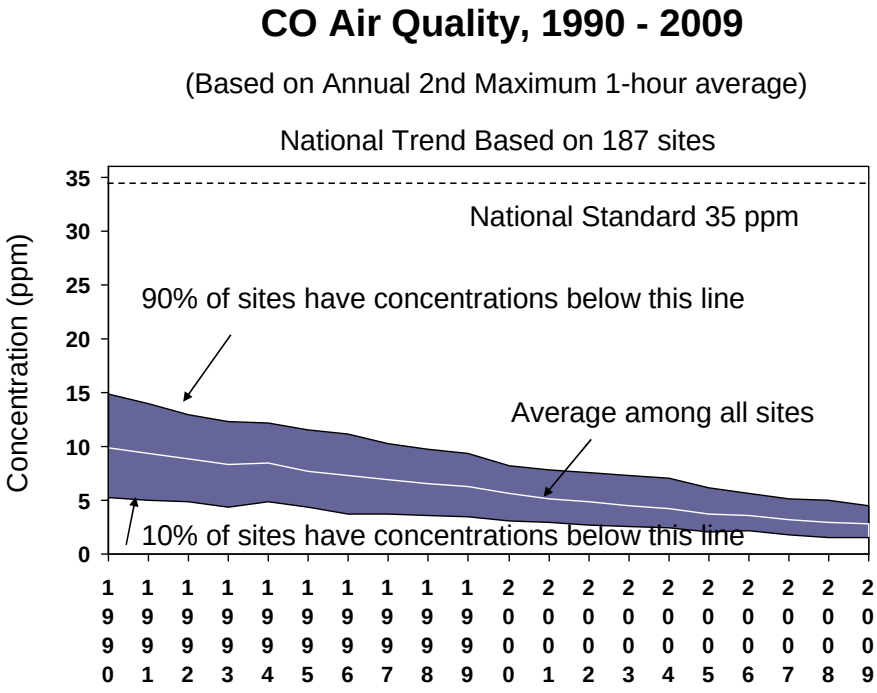


Figure 1-5. Trends in CO concentration (second maximum 1-hour average) in the U.S., 1990-2009. The white line indicates average across the sites; ninety percent of sites have concentrations below the top line, while ten percent of sites have concentrations below the bottom line.



1.4 GENERAL APPROACH AND ORGANIZATION OF THE DOCUMENT

This PA includes staff's evaluation of the policy implications of the scientific evidence reviewed in the ISA and the results of quantitative analyses based on that evidence. Taken together, this information informs staff conclusions and the identification of a range of policy options for consideration in addressing public health and welfare effects associated with ambient CO.

Following this introductory chapter, chapter 2 focuses on review of the primary standards for CO, presenting background information on the rationale for previous reviews and the approach followed in the current review. Chapter 2 discusses the adequacy of the current standards, taking into account evidence- and risk-based considerations, and includes staff conclusions on adequacy. Chapter 2 also discusses potential alternative standards for consideration, focusing on indicator, averaging time, form, and level, and includes staff conclusions on alternative standards for consideration. Finally, chapter 3 discusses information relevant to staff's consideration of a secondary standard for CO.

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2 REVIEW OF THE PRIMARY STANDARDS FOR CARBON MONOXIDE

This chapter presents staff conclusions for consideration in deciding whether the existing primary standards for carbon monoxide (CO) should be revised and, if so, what revisions are appropriate. The current primary CO standards include a 1-hour and an 8-hour standard to protect public health from exposure to CO. In evaluating the current primary NAAQS we have reviewed, and as appropriate, updated, a series of key policy-relevant issues presented in the *Plan for Review of the National Ambient Air Quality Standards for Carbon Monoxide* (USEPA, 2008a, section 3.1). The answers to these policy-relevant questions will inform decisions on whether, and if so, how to revise the primary standards for CO.

Following a background section regarding considerations in the previous review, the discussion in this chapter focuses on two central issues related to: (1) the adequacy of the current CO standards and (2) what potential alternative standards, if any, should be considered in this review. Within each of these broad areas, a series of questions are addressed. The four basic elements of the NAAQS (indicator, averaging time, level, and form) are considered collectively in evaluating the health protection afforded by the current or any potential alternative standards.

2.1 APPROACH

For the purposes of this Policy Assessment (PA), staff has drawn from EPA's assessment and integrated synthesis of the scientific evidence presented in the *Integrated Science Assessment for Carbon Monoxide* (USEPA, 2010a; henceforth referred to as the ISA) and 2000 *Air Quality Criteria Document for Carbon Monoxide* (USEPA, 2000; henceforth referred to as the 2000 AQCD) and on the analyses presented in the Risk and Exposure Assessment (USEPA, 2010b; henceforth referred to as the REA). The evidence-based discussions presented in this chapter draw upon evidence from epidemiologic studies, controlled human exposure studies, and toxicological studies evaluating short- or long-term exposures to CO, as discussed in chapter 5 of the ISA, with supporting evidence related to dosimetry and potential mode of action as presented in chapters 4 and 5 of the ISA, respectively, as well as the integration of evidence across each of these disciplines, as presented in chapter 2 of the ISA. The exposure/risk-based discussions have drawn from the quantitative health analyses for CO presented in the REA. Together the evidence-based and risk-based considerations have informed our conclusions related to the adequacy of the current CO standards and alternative standards that are supported by the currently available scientific evidence.

In presenting a range of primary standard options for consideration, we note that the final decision is largely a public health policy judgment. A final decision must draw upon scientific

information and analyses about health effects and risks, as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on the recognition that the available health effects evidence generally reflects a continuum, consisting of ambient levels at which scientists generally agree that health effects are likely to occur, through lower levels at which the likelihood and magnitude of the response become increasingly uncertain. This approach is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that primary standards be set at a zero-risk level, but rather at a level that avoids unacceptable risks to public health.

The following subsections include background information on the approach used in the previous review of the CO standards (section 2.1.1) and also a discussion of the approach for the current review (section 2.1.2).

2.1.1 Approach Used in the Previous Review

The current primary standards for CO are set at 9 parts per million (ppm) as an 8-hour average and 35 ppm as a 1-hour average, neither to be exceeded more than once per year. These standards were initially set in 1971to protect against the occurrence of carboxyhemoglobin (COHb) levels that may be associated with effects of concern (36 FR 8186). Reviews of these standards in the 1980s and early 1990s identified additional evidence regarding ambient CO, CO exposures, COHb levels, and associated health effects (USEPA, 1984a, 1984b; USEPA, 1991; USEPA, 1992; McClellan, 1991, 1992). Assessment of the evidence in those reviews led the EPA to retain the existing standards without revision (59 FR 38906).

The 1994 decision to retain the primary standards without revision was based on the evidence published through 1990 and reviewed in the 1991 AQCD (USEPA, 1991), the 1992 Staff Paper assessment of the policy-relevant information contained in the AQCD and the quantitative exposure assessment (USEPA, 1992), and the advice and recommendations of CASAC (McClellan 1991, 1992). At that time, as at the time of the prior NAAQS review (50 FR 37484), COHb levels in blood were recognized as the most useful estimates of exogenous CO exposures and to serve as the best biomarker of CO toxicity for ambient-level exposures to CO. Consequently, COHb levels were used as the indicator of health effects in the identification of health effect levels of concern for CO (59 FR 38909).

In reviewing the standards in 1994 the Administrator first recognized the need to determine the COHb levels of concern "taking into account a large and diverse health effects database." The more uncertain and less quantifiable evidence was taken into account to identify the lower end of this range to provide an adequate margin of safety for effects of clear concern. To consider ambient CO concentrations likely to result in COHb levels of concern, a model solution to the Coburn-Forster-Kane (CFK) differential equation was employed in the analysis of CO exposures expected to occur under air quality scenarios related to just meeting the current 8-hour CO NAAQS, the controlling standard (USEPA, 1992).[1] Key considerations in this approach are described below.

Carboxyhemoglobin Levels of Concern and Margin of Safety

The assessment of the science that was presented in the 1991 AQCD (USEPA, 1991) indicated that CO is associated with effects in the cardiovascular system, central nervous system (CNS), and the developing fetus. Additionally, factors recognized as having potential to alter the effects of CO included exposures to other pollutants, some drugs and some environmental factors, such as altitude. Cardiovascular effects of CO, as measured by decreased time to onset of angina and to onset of significant electrocardiogram (ECG) ST-segment depression[2] were judged by the Administrator to be "the health effects of greater concern, which clearly had been associated with CO exposures at levels observed in ambient air" (59 FR 38913).

Based on the consistent findings of response in patients with coronary artery disease[3] across the controlled human exposure evidence (Adams et al., 1988; Allred et al., 1989a, 1989b, 1991; Anderson et al., 1973; Kleinman et al., 1989, 1998; Sheps et al., 1987[4]) and discussions of

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[1] Air quality analyses of CO levels in the U.S. consistently demonstrate that meeting the 8-hour standard results in 1-hour maximum concentrations well below the corresponding 1-hour standard.

[2] The ST-segment is a portion of the electrocardiogram, depression of which is an indication of insufficient oxygen supply to the heart muscle tissue (myocardial ischemia). Myocardial ischemia can result in chest pain (angina pectoris) or such characteristic changes in ECGs or both. In individuals with coronary artery disease, it tends to occur at specific levels of exercise. The duration of exercise required to demonstrate chest pain and/or a 1-mm change in the ST segment of the ECG were key measurements in the multicenter study by Allred et al (1989a, 1989b, 1991).

[3] Coronary artery disease (CAD), often also called coronary heart disease or ischemic heart disease is a category of cardiovascular disease associated with narrowed heart arteries. Individuals with this disease may have myocardial ischemia, which occurs when the heart muscle receives insufficient oxygen delivered by the blood. Exercise-induced angina pectoris (chest pain) occurs in many of them. Among all patients with diagnosed CAD, the predominant type of ischemia, as identified by ST segment depression, is asymptomatic (i.e., silent). Patients who experience angina typically have additional ischemic episodes that are asymptomatic (2000 AQCD, section 7.7.2.1). In addition to such chronic conditions, CAD can lead to sudden episodes, such as myocardial infarction (ISA, p. 5-24).

[4] Statistical analyses of the data from Sheps et al., (1987) by Bissette et al (1986) indicate a significant decrease in time to onset of angina at 4.1% COHb if subjects that did not experience exercise-induced angina during air exposure are also included in the analyses.

adverse health consequences in the 1991 AQCD and the 1992 Staff Paper, at the CASAC meetings and in the July 1991 CASAC letter, the Administrator concluded that "CO exposures resulting in COHb levels of 2.9-3.0 percent (CO-Ox) or higher in persons with heart disease have the potential to increase the risk of decreased time to onset of angina pain and ST-segment depression" (59 FR 38913). Two of the five key studies were given particular emphasis in the 1991 AQCD to indicate the basis for conclusions regarding lowest observed-effect levels of COHb in patients with exercise-induced ischemia, in terms of measured COHb and its representation in terms of increase from baseline COHb on the order of 1.5 to 2.2% (USEPA, 1991, pp. 1-11 to 1-12; Allred et al., 1989a, 1989b, 1991; Anderson et al., 1973).

While EPA and CASAC recognized the existence of a range of views among health professionals on the clinical significance of the responses observed in the clinical studies, CASAC noted that the dominant view was that they should be considered "adverse or harbinger of adverse effect" (McClellan, 1991) and EPA recognized that it was "important that standards be set to appropriately reduce the risk of ambient exposures which produce COHb levels that could induce such potentially adverse effects" (59 FR 38913) as those occurring at COHb levels of 2.9-3.0% (CO-Ox) representing an increase on the order of 1.5 to 2.2 percent above baseline (59 FR 38913; USEPA, 1991, p. 1-12; USEPA, 1992, pp. 20-22). In further considering additional results from the controlled human exposure evidence as well as other aspects of the available evidence and uncertainties regarding modeling estimates of COHb formation and human exposure to COHb levels in the population associated with attainment of a given CO NAAQS, the Administrator recognized the need to extend the range of COHb levels for consideration in evaluating whether the current CO standards provide an adequate margin of safety to those falling between 2.0 to 2.9 (59 FR 38913). Factors considered in recognizing this margin of safety included the following (59 FR 38913).

- Uncertainty regarding the clinical importance of cardiovascular effects associated with exposures to CO that resulted in COHb levels of 2 to 3 percent. Although recognizing the possibility that there is no threshold for these effects even at lower COHb levels, the health significance of the small changes observed for ST-segment depression at 2.0 COHb (Allred et al., 1989a,b) was described as appearing to be "relatively trivial" (59 FR 38913).
- Findings of short-term reduction in maximal work capacity measured in trained athletes exposed to CO at levels resulting in COHb levels of 2.3 to 7 percent.
- The potential that the most sensitive individuals have not been studied, the limited information regarding the effects of ambient CO in the developing fetus, and concern about visitors to high altitudes, individuals with anemia or respiratory disease, or the elderly.

- Potential for short term peak CO exposures to be responsible for impairments (impairment of visual perception, sensorimotor performance, vigilance or other CNS effects) which could be a matter of concern for complex activities such as driving a car, although these effects had not been demonstrated to be caused by CO concentrations in ambient air.
- Concern based on limited evidence for individuals exposed to CO concurrently with drugs (e.g., alcohol), during heat stress, or co-exposure to other pollutants.
- Uncertainties, described as "large," that remained regarding modeling COHb formation and estimating human exposure to CO which could lead to overestimation of COHb levels in the population associated with attainment of a given CO NAAQS.
- Uncertainty associated with COHb measurements made using CO-Ox which may not reflect COHb levels in angina patients studied, thereby creating uncertainty in establishing a lowest effects level for CO.

Based on these considerations of the evidence, the Administrator identified a range of COHb levels of concern extending from 2.9% at the upper end down to 2% at the lower end and concluded that "evaluation of the adequacy of the current standard should focus on reducing the number of individuals with cardiovascular disease from being exposed to CO levels in the ambient air that would result in COHb levels of 2.1 percent". She additionally concluded that standards that "protect against COHb levels at the lower end of the range should provide an adequate margin of safety against effects of uncertain occurrence, as well as those of clear concern that have been associated with COHb levels in the upper-end of the range" (59 FR 48914).

Estimation of Population Exposures

To estimate CO exposures and resulting COHb levels that might be expected under air quality conditions that just met the current standards, an analysis of exposure and associated internal dose in terms of COHb levels in the population of interest in the city of Denver, Colorado was performed (59 FR 38906; USEPA, 1992). That analysis indicated that if the 9 ppm 8-hour standard were just met, the proportion of the nonsmoking population with cardiovascular disease experiencing a daily maximum 8-hour exposure at or above 9 ppm for 8 hours decreased by an order of magnitude or more as compared to the proportion under then-existing CO levels, down to less than 0.1 percent of the total person-days in that population. More specifically, upon meeting the 8-hour standard, EPA estimated that less than 0.1% of the nonsmoking cardiovascular-disease population would experience a COHb level greater than or equal to 2.1%. A smaller percentage of the at-risk population was estimated to exceed higher

COHb levels.[5] Based on these estimates, the Administrator concluded that "relatively few people of the cardiovascular sensitive population group analyzed will experience COHb levels ≥ 2.1 percent when exposed to CO levels in absence of indoor sources when the current standards are attained." The analysis also took into account that certain indoor sources (e.g., passive smoking, gas stove usage) contributed to total CO exposure and EPA recognized that such sources may be of concern for such high risk groups as individuals with cardiovascular disease, pregnant women, and their unborn children but concluded that "the contribution of indoor sources cannot be effectively mitigated by ambient air quality standards" (59 FR38914).

Decision Regarding Adequacy of the Standards

Based on consideration of the evidence and the quantitative results of the exposure assessment, the Administrator concluded that revisions of the current primary standards for CO were not appropriate at that time (59 FR 38914). The Administrator additionally concluded that both averaging times for the primary standards, 1 hour and 8 hours, be retained. The 1-hour and 8-hour averaging times were first chosen when EPA promulgated the primary NAAQS for CO in 1971. The selection of the 8-hour averaging time was based on the following: (a) most individuals' COHb levels appeared to approach equilibrium after 8 hours of exposure, (b) the 8-hour time period corresponded to the blocks of time when people were often exposed in a particular location or activity (e.g., working or sleeping), and (c) judgment that this provided a good indicator for tracking continuous exposures during any 24-hour period. The 1-hour averaging time was selected as better representing a time period of interest to short-term CO exposure and providing protection from effects which might be encountered from very short duration peak exposures in the urban environment (59 FR 38914).

2.1.2 Approach for the Current Review

To evaluate whether it is appropriate to consider retaining the current primary CO standards, or whether consideration of revisions is appropriate, we adopted an approach in this review that builds upon the general approach used in the last review and reflects the broader body of evidence and information now available. As summarized above, the Administrator's decisions in the previous review were based on an integration of information on health effects associated with exposure to ambient CO; expert judgment on the adversity of such effects on individuals; and policy judgments as to when the standard is requisite to protect public health

[5] In the 1992 assessment, the person-days (number of persons multiplied by the number of days per year exposed) and person-hours (number of persons multiplied by the number of hours per year exposed) were the reported exposure metrics. Upon meeting the 8-hour standard, it was estimated that less than 0.1% of the total person-days simulated for the nonsmoking cardiovascular-disease population were associated with a maximum COHb level greater than or equal to 2.1% (USEPA, 1992; Johnson et al., 1992).

with an adequate margin of safety, which were informed by air quality and related analyses, quantitative exposure and risk assessments when possible, and qualitative assessment of impacts that could not be quantified.

In conducting this assessment, we draw on the current evidence and quantitative assessments of exposure pertaining to the public health risk of ambient CO. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since the last review, including the current ISA and the 2000 AQCD (USEPA, 2010; USEPA, 2000), as well as current and preceding quantitative exposure/risk assessments (USEPA 2010b; Johnson et al., 2000; USEPA 1992). As was the case at the time of the last review, the best characterized health effect associated with CO levels of concern is hypoxia (reduced oxygen availability) induced by increased COHb levels in blood (ISA, section 5.1.2). Accordingly, CO exposure is of particular concern for those with impaired cardiovascular systems, and the most compelling evidence of cardiovascular effects is that from a series of controlled human exposure studies among exercising individuals with coronary heart disease (CHD) also referred to as coronary artery disease (CAD) (ISA, sections 5.2.4 and 5.2.6). Additionally available in this review are a number of epidemiological studies that investigated the association of cardiovascular disease-related health outcomes with concentrations of CO at ambient monitors. To inform our review of the ambient standards, we performed a quantitative exposure and dose modeling analysis that estimated COHb levels associated with different air quality conditions in simulated at-risk populations in two U.S. cities. Thus, in developing conclusions in this review as discussed in sections 2.2 and 2.3 below, we have taken into account both evidence-based and exposure/risk-based considerations framed by a series of key policy-relevant questions.

2.2 ADEQUACY OF THE CURRENT STANDARD

In considering the adequacy of the current CO standards, the overarching question we consider is:

- **Does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and REA, support or call into question the adequacy of the protection afforded by the current CO standards?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative exposure/risk analyses to address this question, we have focused on a series of more specific questions, posed within sections 2.2.1 and 2.2.2 below. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since the last review which has been critically analyzed and characterized in the 2000 AQCD and more recently in the ISA.

2.2.1 Evidence-based Considerations

In considering the evidence with regard to the issue of adequacy of the current standard, we address a series of questions that focus on policy-relevant aspects of the evidence beginning with the health effects associated with CO exposure, followed by the use of COHb levels as the indicator of CO exposures and biomarker for characterizing the potential for health effects associated with exposures to ambient CO, and then the identification of the populations most susceptible to the effects of CO. We next consider the evidence regarding the levels of CO in ambient air associated with health effects and the important uncertainties associated with the evidence.

- **Does the current evidence alter our conclusions from the previous review regarding the health effects associated with exposure to CO?**

The current evidence continues to support our conclusions from the previous review regarding key health effects associated with CO exposure. The best characterized effect of CO continues to be related to the binding of CO to blood Hb to form increased levels of COHb (ISA, sections 4.1 and 5.1.2) and the primary focus is on associated cardiovascular effects (ISA, section 5.2). In the scientific assessment for the current review, a likely causal relationship is judged to exist between relevant CO exposures[6] and cardiovascular effects (ISA, section 2.5.1) which is similar to conclusions in the last review. The evidence for effects on the central nervous system, birth outcomes and developmental effects, and respiratory effects, in some cases expanded from that which was available at the time of the last review, is judged to be suggestive

[6] Relevant CO exposures are defined in the ISA as "generally within one or two orders of magnitude of ambient CO concentrations" (ISA, section 2.5).

of a causal relationship with relevant CO exposures (ISA, section 2.5). These overall findings, additional details of which are described below, are consistent with and extend in some ways conclusions drawn from the health effects evidence in the last review.

The long-standing body of evidence that has established many aspects of the biological effects of CO continues to contribute to our understanding of the health effects of ambient CO. Binding to heme proteins and the alteration of their function is the common mechanism underlying biological responses to CO. Upon inhalation, CO diffuses through the respiratory zone (alveoli) to the blood where it binds to Hb, forming COHb. Accordingly, inhaled CO elicits various health effects through binding to, and associated alteration of the function of, a number of heme-containing molecules, mainly Hb (see e.g., ISA, section 4.1). The best characterized health effect associated with CO levels of concern is hypoxia (reduced oxygen availability) induced by increased COHb levels in blood and decreased oxygen availability to critical tissues and organs, specifically the heart (ISA, section 5.1.2). Consistent with this, medical conditions that affect the biological mechanisms to compensate for this effect (e.g., vasodilation and increased coronary blood flow with increased oxygen delivery to the myocardium) can contribute to a reduced amount of oxygen available to key body tissues, potentially affecting organ system function and limiting exercise capacity (2000 AQCD, section 7.1).[7] The dose metric most commonly used as a bioindicator of exposure and health risk from CO is the level of COHb in the blood (1991 AQCD, 2000 AQCD, 2010 ISA).

The body of health effects evidence for CO has grown considerably since the review completed in 1994 with the addition of numerous epidemiological and toxicological studies (ISA; 2000 AQCD). This evidence provides additional detail and support to our prior understanding of CO effects and population susceptibility. For example, the currently available evidence expands on potential nonhypoxic mechanisms for CO effects, although the importance of such mechanisms at environmentally-relevant CO exposures is unclear (ISA, section 5.1.3). Most notably, the current evidence includes much expanded epidemiological evidence that is consistent with previous conclusions regarding cardiovascular disease-related susceptibility and may provide indications of air quality conditions that may be associated with ambient CO-related risk. In this review, the clearest evidence is available for cardiovascular effects. In the ISA, the evidence is characterized as to likelihood of causal relationships between exposure to ambient CO and specific health effects using an established framework (ISA, chapter 1). The major conclusion drawn in the ISA regarding the critical analysis of all available data on health effects of CO including the clinical and epidemiological evidence is that "Given the consistent and

[7] People with peripheral vascular diseases and heart disease patients often have markedly reduced circulatory capacity and reduced ability to compensate for increased circulatory demands during exercise and other stress (2000 AQCD, p. 7-7).

coherent evidence from epidemiologic and human clinical studies, along with biological plausibility provided by CO's role in limiting oxygen availability, it is concluded that a causal relationship is likely to exist between relevant[8] short-term CO exposures and cardiovascular morbidity" (ISA, p. 2-6, section 2.5.1). Additionally, as mentioned above, the ISA judges the evidence to be suggestive of causal relationships between relevant short- and long-term CO exposures and CNS effects, birth outcomes and developmental effects following long-term exposure, respiratory morbidity following short-term exposure, and mortality following short-term exposure (ISA, section 2.5, Table 2-1). The ISA concludes there is not likely to be a causal relationship between relevant long-term CO exposures and mortality (ISA, Table 2-1).

Similar to the previous review, results from controlled human exposure studies of individuals with coronary artery disease (Adams et al., 1988; Allred et al., 1989a, 1989b, 1991; Anderson et al., 1973; Kleinman et al., 1989, 1998; Sheps et al., 1987[9]) are the "most compelling evidence of CO-induced effects on the cardiovascular system" (ISA, Section 5.2). Additionally, the use of an internal dose metric, COHb, adds to the strength of the findings in these controlled exposure studies. As a group, these studies demonstrate the role of CO in increasing the susceptibility of people with CAD to incidents of exercise-associated myocardial ischemia. Toxicological studies described in the current review provide evidence of CO effects on the cardiovascular system, including electrocardiographic effects of 1-hour exposures to 35 ppm CO in a rat strain developed as an animal model of cardiac susceptibility (ISA, section 5.2.5.3). Further toxicological evidence is identified for other effects (e.g., aortic injury, microvascular permeability, vascular remodeling, ventricular hypertrophy) occurring at laboratory animal CO exposure levels ranging up to 200 ppm for multiple durations, including much longer than 1 hour (ISA, section 5.2.5).

Among the controlled human exposure studies, the ISA places principal emphasis on the study of CAD patients by Allred et al. (1989a, 1989b, 1991)[10] which was key to considerations from the previous review for the following reasons: 1) dose-response relationships were observed; 2) effects were observed at the lowest COHb levels tested (mean of 2-2.4% COHb[11]

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[8] Relevant CO exposures are defined in the ISA as "generally within one or two orders of magnitude of ambient CO concentrations" (ISA, section 2.5).

[9] See footnote 4 above.

[10] Other controlled human exposure studies of CAD patients (listed in Table 2-2 below, and discussed in more detail in the 1991 and 2000 AQCDs) similarly provide evidence of reduced time to exercise-induced angina associated with elevated COHb resulting from controlled short-duration exposure to increased concentrations of CO.

[11] These levels and other COHb levels described for this study below are based on GC analysis unless otherwise specified. Based on matched measurements available for CO-oximetry (CO-Ox) and gas chromatography (GC) in this study, staff note that CO-Ox measurements of 2.9 to 3.0 percent COHb appear to correspond to GC measurements on the order of 2% (Allred et al., 1991)

following experimental CO exposure), with no evidence of a threshold; 3) objective measures of myocardial ischemia (ST-segment depression) were assessed, as well as the subjective measure of decreased time to induction of angina; 4) measurements were taken both by gas chromatography (GC), which provides a more accurate measurement of COHb blood levels[12], and by CO-Ox; 5) a large number of study subjects were used; 6) a strict protocol for selection of study subjects was employed to include only CAD patients with reproducible exercise-induced angina.; and 7) the study was conducted at multiple laboratories around the U.S. This study evaluated changes in time to exercise-induced onset of markers of myocardial ischemia resulting from two short CO exposures targeted to result in mean study subject COHb levels of 2% and 4%, respectively (ISA, section 5.2.4). In this study, subjects (n=63) on three separate occasions underwent an initial graded exercise treadmill test, followed by 50 to 70-minute exposures under resting conditions to room air CO concentrations or CO concentrations targeted for each subject to achieve blood COHb levels of 2% and 4%. The exposures were to average CO concentrations of 0.7 ppm (room air concentration range 0-2 ppm), 117 ppm (range 42-202 ppm) and 253 ppm (range 143-357 ppm). After the 50- to 70-minute exposures, subjects underwent a second graded exercise treadmill test, and the percent change in time to onset of angina and time to ST endpoint between the first and second exercise tests was determined. For the two CO exposures, the average post-exposure COHb concentrations were reported as 2.4% and 4.7%, and the subsequent post-exercise average COHb concentrations were reported as 2.0% and 3.9%.[13]

Across all subjects, the mean time to angina onset for control ("room" air) exposures was approximately 8.5 minutes, and the mean time to ST endpoint was approximately 9.5 minutes (Allred et al., 1989b). Relative to room-air exposure that resulted in a mean COHb level of 0.6% (post-exercise), exposure to CO resulting in post-exercise mean COHb concentrations of 2.0% and 3.9% were observed to decrease the exercise time required to induce ST-segment depression by 5.1 ($p=0.01$) and 12.1% ($p<0.001$), respectively. These changes were well correlated with the onset of exercise-induced angina, the time to which was shortened by 4.2%

[12] As stated in the ISA, the gas chromatographic technique for measuring COHb levels "is known to be more accurate than spectrophotometric measurements, particularly for samples containing COHb concentrations < 5%" (ISA, p. 5-41). CO-oximetry is a spectrophotometric method commonly used to rapidly provide approximate concentrations of COHb during controlled exposures (ISA, p. 5-41). At the low concentrations of COHb (<5%) more relevant to ambient CO exposures, co-oximeters are reported to overestimate COHb levels compared to GC measurements, while at higher concentrations, this method is reported to produce underestimates (ISA, p.4-18).

[13] While the COHb blood level for each subject during the exercise tests was intermediate between the post-exposure and subsequent post-exercise measurements (e.g., mean 2.0-2.4% and 3.9-4.7%), the study authors noted that the measurements at the end of the exercise test represented the COHb concentrations at the approximate time of onset of myocardial ischemia as indicated by angina and ST segment changes. The corresponding ranges of CO-Ox measurements for the two exposures were 2.7-3.2% and 4.7-5.6%. In this document, we refer to the GC-measured mean of 2.0% or 2.0-2.4% for the COHb levels resulting from the lower experimental CO exposure.

(p=0.027) and 7.1% (p=0.002), respectively, for the two experimental CO exposures (Allred et al., 1989a, 1989b, 1991).[14] As at the time of the last review, while ST-segment depression is recognized as an indicator of myocardial ischemia, the exact physiological significance of the observed changes among those with CAD is unclear (ISA, p. 5-48).

No human clinical studies have been specifically designed to evaluate the effect of controlled short-term exposures to CO resulting in COHb levels lower than a study mean of 2% (ISA, section 5.2.6). However, an important finding of the multi-laboratory study was the dose-response relationship (discussed further in addressing a subsequent question below) observed between COHb and the markers of myocardial ischemia, with effects observed at the lowest increases in COHb tested, without evidence of a measurable threshold effect. As reported by the authors, the results comparing "the effects of increasing COHb from baseline levels (0.6%) to 2 and 3.9% COHb showed that each produced further changes in objective ECG measures of ischemia" implying that "small increments in COHb could adversely affect myocardial function and produce ischemia" (Allred et al., 1989b, 1991).

The epidemiological evidence has expanded considerably since the last review including numerous additional studies that are coherent with the evidence on markers of myocardial ischemia from controlled human exposure studies of CAD patients (ISA, section 2.7). The most recent set of epidemiological studies in the U.S. have evaluated the associations between ambient concentrations of multiple pollutants (i.e. fine particles or $PM_{2.5}$, nitrogen dioxide, sulfur dioxide, ozone, and CO) at fixed-site ambient monitors and increases in emergency department visits and hospital admissions for specific cardiovascular health outcomes including ischemic heart disease (IHD), myocardial infarction (MI), congestive heart failure (CHF), and cardiovascular diseases (CVD) as a whole (Bell et al., 2009; Koken et al., 2003; Linn et al., 2000; Mann et al., 2002; Metzger et al., 2004; Symons et al., 2006; Tolbert et al., 2007; Wellenius et al., 2005). Findings of positive associations for these outcomes with metrics of ambient CO concentrations are coherent with the evidence from controlled human exposure studies of myocardial ischemia-related effects resulting from elevated CO exposures (ISA, section 2.5.1; ISA, Figure 2-1).

Table 2-1 below presents studies reporting associations of ambient CO concentration estimates for several U.S. urban areas with hospital admissions for ischemic heart disease, cardiovascular disease or congestive heart failure described in the ISA (ISA, Figures 5-2, 5-5 and 5-4). Presented first in Table 2-1 are the U.S. studies reporting associations with ischemia-

[14] Another indicator measured in the study was the combination of heart rate and systolic blood pressure which provides a clinical index of the work of the heart and myocardial oxygen consumption, since heart rate and blood pressure are major determinants of myocardial oxygen consumption (Allred et al., 1991). A decrease in oxygen to the myocardium would be expected to be paralleled by ischemia at lower heart rate and systolic blood pressure. This heart rate-systolic blood pressure indicator at the time to ST-endpoint was decreased by 4.4% at the 3.9% COHb dose level and by a nonstatistically-significant, smaller amount at the 2.0% COHb dose level.

related health outcomes as the studied outcomes most explicitly consistent with the role of CO in limiting oxygen availability. In these studies, the ambient CO concentration averaging time for which health outcomes were analyzed varied from 1 hour to 24 hours, with the air quality metrics based on either a selected central-site monitor for the area or an average for multiple monitors in the area of interest. The study areas include the Atlanta, Georgia metropolitan statistical area, the greater Los Angeles, California, area and a group of 126 urban counties. Together the individual study periods spanned the years from 1988 through 2005. The risk estimates from these studies presented here indicate statistically significant positive associations were observed with ambient CO concentrations based on air quality for the day of hospital admission or based on the average of the selected ambient CO concentration metric across that day and 2 or 3 days previous (ISA, Figures 5-2 and 5-5). Many of the studies for these outcomes include same day or next day lag periods, which, as noted in the ISA "are consistent with the propose mechanism and biological plausibility of these CVD outcomes" (ISA, p. 5-40). Of the studies for which estimates shown in Table 2-1 are based on multi-day averages (the Atlanta studies and the California study by Mann et al., 2002), the California study by Mann et al., (2002) also observed a significant positive association with same day CO concentration.

Additionally presented in Table 2-1 are the U.S. studies reporting associations with hospital admissions for CHF, a condition that affects an individual's ability to compensate for reduced oxygen availability. One of the IHD outcome studies cited above also reported a significant association for ambient CO with hospital admissions for CHF (Linn et al., 2000), as did additional studies in Allegheny County (Pittsburgh) for 1987-1999 study period, and Denver for the months of July-August during 1993-1997 (Koken et al., 2003; Wellenius et al., 2005; ISA, pp. 5-31 to 5-33). The risk estimates presented for all three of these studies are based on the 24-hour CO concentration, with the California and Allegheny County studies' association with same-day air quality, while the association shown for the Denver study was with ambient CO 3 days prior to health outcome (Table 2-1).

Table 2-1. U.S. epidemiological studies for ischemic heart disease, cardiovascular disease and congestive heart failure.

Study Reference	Health Outcome [A]	Risk Estimate[B] (confidence interval) *Estimates are standardized within averaging times.*	CO Concentration Metric for Risk Estimated Presented	Assignment of Monitors to Study Subjects	Study Area	Study Time Period
U.S. studies of hospital admissions for coronary heart disease (ISA, Figure 5-2).						
Metzger et al., 2004	IHD	1.016 (0.999-1.034)	1-hour daily maximum, average for most recent 3 days	Single central residential monitor	20-county Atlanta area	1993-2000
Mann et al., 2002	IHD	1.0136 (1.0053-1.0220)	8-hour daily maximum, average for most recent 4 days	Residence grid centroid interpolated from 3 closest monitors	South Coast Air Basin, CA	1988-1995
	IHD+CHF	1.0304 (1.0135-1.0475)				
Linn et al., 2000	MI	1.020 (1.011-1.029)	24-hour average, same day	Average across all monitors	"	1992-1995
U.S. studies of hospital admissions for cardiovascular disease (ISA, Figure 5-5).						
Bell et al., 2009	CVD 65+	1.0096 (1.0079-1.0112)	1-hour daily maximum, same day	Average of monitors in county of residence	126 urban counties	1999-2005
Tolbert et al., 2007	CVD	1.016 (1.008-1.025)	1-hour daily maximum, average for most recent 3 days	Single central residential monitor	20-county Atlanta area	1993-2004
Metzger et al., 2004	CVD	1.017 (1.008–1.027)				1993-2000
Linn et al., 2000	CVD	1.016 (1.013-1.019)	24-hour average, same day	Average across all monitors	South Coast Air Basin, CA	1992-1995
U.S. studies of hospital admissions for congestive heart failure (ISA, Figure 5-4).						
Linn et al., 2000	CHF	1.013 (1.004-1.021)	24-hour average, same day	Average across all monitors	South Coast Air Basin, CA	1992-1995
Metzger et al., 2004	CHF	1.010 (0.988-1.032)	1-hour daily maximum, average for most recent 3 days	Single central residential monitor	20-county Atlanta area	1993-2000
Symons et al., 2006	CHF	1.08 (0.40-2.99)	8-hour daily maximum, average for most recent 4 days	Single central residential monitor	Baltimore area, MD	2002 (April-Dec)
Wellenius et al., 2005	CHF	1.0843 (1.0614-1.1077)	24-hour average, same day	Single area-wide representation based on 3 monitors	Allegheny county, PA	1987-1999
Koken et al., 2003	CHF	1.181 (1.002-1.393)	24-hour average, 3 days prior	Average across all monitors	Denver county, CO	1993-1997 (Jul-Aug)

A Hospital admissions for ischemic heart disease (IHD), myocardial infarction (MI), cardiovascular disease (CVD; Bell 2009 included only subjects 65 years old and above), and congestive heart failure (CHF).
B Unadjusted for other pollutants and standardized for 1 ppm increase in CO concentration for 1-hour metric, 0.75 ppm for 8-hour metric and 0.5 ppm for 24-hour metric.

As noted by the ISA, "[s]tudies of hospital admissions and ED visits for IHD provide the strongest [epidemiological] evidence of ambient CO being associated with adverse CVD outcomes" (ISA, p. 5-40, section 5.2.3; Linn et al., 2000; Mann et al., 2002; Metzger et al., 2004). With regard to studies for other measures of cardiovascular morbidity, the ISA notes that "[t]hough not as consistent as the IHD effects, the effects for all CVD hospital admissions (which include IHD admissions) and CHF hospital admissions also provide evidence for an association of cardiovascular outcomes and ambient CO concentrations" (ISA, section 5.2.3; Bell et al., 2009; Tolbert et al., 2007). While noting the difficulty in determining the extent to which CO is independently associated with CVD outcomes in this group of studies as compared to CO as a marker for the effects of another traffic-related pollutant or mix of pollutants, the ISA concludes that the epidemiological evidence, particularly when considering the copollutant analyses, provides support to the clinical evidence for a direct effect of short-term ambient CO exposure on CVD morbidity (ISA, pp. 5-40 to 5-41).

Additional epidemiological studies have evaluated associations of ambient CO with other cardiovascular effects since the last review. For example, preliminary evidence of a link between exposure to CO and alteration of blood markers of coagulation and inflammation in individuals with CAD or CVD has been provided by a few well conducted and informative studies (ISA, Table 5-6; Delfino et al., 2008; Liao et al., 2005). As noted by the ISA, however, further studies are warranted to investigate the role of these markers in prothrombotic events and their possible contribution to the pathophysiology of CO-induced aggravation of ischemic heart disease (ISA, section 5.2.1.8). Other epidemiological studies (including field and or panel studies) also provide some evidence of a link between CO exposure and heart rate and heart rate variability (ISA, section 5.2.1.1). With regard to the two of three studies reporting a positive association with heart rate, the ISA concluded that "further research is warranted" to corroborate the results, while the larger number of studies for heart rate variability parameters is characterized as having mixed associations (ISA, p. 5-15). Additionally, of the two studies of electrocardiogram changes indicative of ischemic events (ISA, section 5.2.1.2), one found no association and, in the other study, the association with CO did not remain statistically significant in multipollutant models, unlike the association with black carbon in that study (ISA, p. 5-16). A limited number of epidemiological studies (Bell et al., 2009; Linn et al., 2000) have investigated hospital admissions for stroke (including both hemorrhagic and ischemic forms) and generally report small or no associations with ambient CO concentrations (ISA, section 5.2.1.9, Table 5-8 and Figure 5-3).

At the time of the last review, there was evidence for effects other than cardiovascular morbidity, including neurological, respiratory and developmental effects. These findings include the following.

- With regard to neurological effects, acute exposures to CO have long been known to induce CNS effects such as those observed with CO poisoning, although limited and equivocal evidence available at the time of the last review included indications of some neurobehavioral effects to result from CO exposures resulting in a range of 5-20% COHb (2000 AQCD, section 6.3.2). No additional clinical or epidemiological studies are now available that investigated such effects of CO at ambient levels (ISA, section 5.3).
- With regard to potential effects of CO on birth outcomes and developmental effects, the potential vulnerability of the fetus and very young infant to CO was recognized during the 1994 review and in the 2000 AQCD. The CO-specific evidence available, however, included limited epidemiological analyses focused primarily on very high CO exposures associated with maternal smoking, and animal studies involving very high CO exposures (USEPA, 1992; 2000 AQCD). The 2000 AQCD concluded that typical ambient CO levels were unlikely to cause increased fetal risk (2000 AQCD, p. 6-44). The current review includes additional epidemiological and animal toxicological studies. The currently available evidence includes limited but suggestive epidemiologic evidence for a CO-induced effect on preterm-birth, birth defects, decrease in birth weight, other measures of fetal growth, and infant mortality (ISA, section 5.4.3). The available animal toxicological studies provide some support and coherence for these birth and developmental outcomes at higher than ambient exposures,[15] although a clear understanding of the mechanisms underlying potential reproductive and developmental effects is still lacking (ISA, section 2.5.3).
- With regard to respiratory effects, the 2000 AQCD concluded it unlikely that CO has direct effects on lung tissue, except at extremely high concentrations (2000 AQCD, p. 6-45). There is currently limited, suggestive evidence of an association between short-term exposure to CO and respiratory-related outcomes. Only preliminary evidence is available, however, regarding a mechanism that could provide plausibility for CO-induced effects (ISA, section 5.5.5.1).

Thus, while there is some additional evidence on neurological, respiratory and developmental effects, it remains limited.

In summary, rather than altering our conclusions from the previous review, the current evidence provides continued support and some additional strength to our previous conclusions regarding the health effects associated with exposure to CO and continues to indicate cardiovascular effects, particularly effects related to the role of CO in limiting oxygen availability, as those of greatest concern at low exposures.

[15] The lowest exposures eliciting an effect in the animal studies were exposures of 22 hours per day over about 14 prenatal days at a concentration of 12 ppm (ISA, Table 5-17).

- **Does the current evidence continue to support a focus on COHb levels as the most useful indicator of CO exposures and the best biomarker to characterize potential for health effects associated with exposures to ambient CO? Or does the current evidence provide support for a focus on alternate dose indicators to characterize potential for health effects?**

As discussed in both the 2000 AQCD (USEPA, 2000) and the ISA, the best characterized mechanism of action of CO is tissue hypoxia caused by binding of CO to hemoglobin to form COHb. Increasing levels of COHb with subsequent decrease in oxygen availability for organs and tissues are of concern in people with pre-existing heart disease who have compromised compensatory mechanisms (e.g., lack of capacity to increase blood flow due to the inability of coronary arteries to vasodilate in response to increased CO). The integrative review of health effects of CO indicates that "the clearest evidence indicates that individuals with CAD are most susceptible to an increase in CO-induced health effects" (ISA, section 5.7.8) and the evidence, including that from clinical studies described in addressing the previous question (regarding health effects associated with exposure to CO), continues to support levels of COHb as the most useful indicator of CO exposure that is related to the health effects of CO of major concern.

Apart from the impaired oxygen delivery to tissues related to COHb formation, the evidence also indicates cytotoxic effects independent of limited oxygen availability (2000 AQCD, section 5.9; ISA, section 5.1.3). These alternative mechanisms of CO-induced effects are primarily associated with CO's ability to bind heme-containing proteins other than hemoglobin and myoglobin, and involve a wide range of molecular targets and CO concentrations, as described in the 2000 AQCD (USEPA, 2000, section 5.8) and in the ISA (ISA, section 5.1.3). Older toxicological studies demonstrated that exposure to high concentrations of CO resulted in altered functions of heme proteins other than myoglobin and hemoglobin, potentially interfering with basic cell and molecular processes and leading to dysfunction and/or disease. More recent toxicological *in vitro* and *in vivo* studies have provided evidence of alteration of nitric oxide signaling, inhibition of cytochrome C oxidase, heme loss from protein, disruption of iron homeostasis and alteration of cellular reduction-oxidation status (ISA, section 5.1.3.2). The ISA notes that these mechanisms may be interrelated. The evidence for mechanisms related to alteration in ion channel activity and modulation of protein kinase signaling pathways is less well understood (ISA, section 5.1.3.2).

As noted in the ISA, "CO may be responsible for a continuum of effects from cell signaling to adaptive responses to cellular injury, depending on intracellular concentrations of CO, heme proteins and molecules which modulate CO binding to heme proteins" (ISA, section 5.1.3.3). New research based on this evidence for pathways other than those related to impaired oxygen delivery to tissues is needed to further understand these pathways and their linkage to

CO-induced effects in susceptible populations. Therefore, at this time, the evidence indicates that COHb continues to be the most useful and well-supported indicator of CO exposures and the best biomarker to characterize the potential for health effects associated with exposures to ambient CO.

- **Does the current evidence alter our understanding of populations that are particularly susceptible to CO exposures? Is there new evidence that suggest additional susceptible populations that should be given increased focus in this review?**

The term susceptibility has been used to recognize populations that have a greater likelihood of experiencing effects related to ambient CO exposure (ISA, section 5.7). Thus, susceptible populations are at greater risk of CO effects and are also referred to as *at-risk* in the discussion here. This increased likelihood of response to CO can potentially result from many factors, including pre-existing medical disorders or disease state, lifestage, gender, lifestyle or increased exposures (ISA, section 5.7).

The current evidence, while much expanded in a number of ways, continues to support our conclusions from the previous review regarding susceptible populations for exposure to ambient CO. In the 1994 review and the 2000 AQCD, the evidence best supported the identification of patients with CAD as a population at increased risk for low levels of CO (USEPA, 1992; 2000 AQCD). Other groups were also recognized as potentially susceptible in the 2000 AQCD based on consideration of the clinical evidence and theoretical work, as well as laboratory animal research (2000 AQCD, p. 7-6). These include fetuses and young infants; pregnant women; the elderly, especially those with compromised cardiovascular function; people with conditions affecting oxygen absorption, blood flow, oxygen carrying capacity or transport; people using drugs with central nervous system depressant properties or exposed to chemical substances that increase endogenous formation of CO; and people who have not adapted to high altitude and are exposed to a combination of high altitude and CO. For these potentially susceptible groups, little empirical evidence was available by which to specify health effects associated with ambient or near-ambient CO exposures (2000 AQCD, p. 7-6).

Based on the evidence from clinical studies also considered in the last review, with which the now much-expanded epidemiological evidence base is coherent, the population with pre-existing cardiovascular disease associated with limitation in oxygen availability continues to be the best characterized population at risk of adverse CO-induced effects, with CAD recognized as "the most important susceptibility characteristic for increased risk due to CO exposure" (ISA, section 2.6.1). An important factor determining the increased susceptibility of this population is their inability to compensate for the reduction in oxygen levels due to an already compromised cardiovascular system. Individuals with a healthy cardiovascular system (i.e., with healthy

coronary arteries) have operative physiologic compensatory mechanisms (e.g., increased blood flow and oxygen extraction) for CO-induced hypoxia and are unlikely to be at increased risk of CO-induced effects (ISA, p. 2-10).[16] In addition, the high oxygen consumption of the heart, together with the inability to compensate for the hypoxic effects of CO make the cardiac muscle of a person suffering with CAD a critical target for the hypoxic effects of CO.

In the current review, recognition of susceptibility of the population with pre-existing cardiovascular disease, such as CAD, is supported by the expanded epidemiological database, which includes a number of studies reporting significant increases in hospital admissions for IHD, angina and MI in relation to CO exposures (ISA, section 2.7). Further support is provided by epidemiologic studies (Mann et al., 2002; and Peel et al., 2007) of increased hospital admissions and emergency department visits for IHD among individuals with secondary diagnoses for other cardiovascular outcomes including arrhythmia and congestive heart failure (ISA, section 5.7), and toxicological studies reporting altered cardiac outcomes in animal models of cardiovascular disease (ISA, section 5.2.1.9).

Cardiovascular disease comprises many types of medical disorders, including heart disease, cerebrovascular disease (e.g., stroke), hypertension (high blood pressure), and peripheral vascular diseases. Heart disease, in turn, comprises several types of disorders, including ischemic heart disease (CHD or CAD, myocardial infarction, angina), congestive heart failure, and disturbances in cardiac rhythm (2000 AQCD, section 7.7.2.1). Types of cardiovascular disease other than those discussed above may also contribute to increased susceptibility to the adverse effects of low levels of CO (ISA, section 5.7.1.1). For example, some evidence with regard to other types of cardiovascular disease such as congestive heart failure, arrhythmia, and non-specific cardiovascular disease, although more limited for peripheral vascular and cerebrovascular disease, indicates that "the continuous nature of the progression of CAD and its close relationship with other forms of cardiovascular disease suggest that a larger population than just those individuals with a prior diagnosis of CAD may be susceptible to health effects from CO exposure" (ISA, p. 5-117).

Although there was little experimental data available at the time of the last review to adequately characterize specific health effects of CO at ambient levels for other potentially at-risk populations, several other populations were identified as being potentially more at risk of CO-induced effects due to a number of factors. These factors include pre-existing diseases that could inherently decrease oxygen availability to tissues, lifestage vulnerabilities (e.g., fetuses,

[16] The other well-studied individuals at the time of the last review were healthy male adults that experienced decreased exercise duration at similar COHb levels during short term maximal exercise. This population was of lesser concern since it represented a smaller sensitive group, and potentially limited to individuals that would engage in vigorous exercise such as competing athletes (1991 AQCD, section 10.3.2).

young infants or newborns, the elderly), gender, lifestyle, medications or alterations in the physical environment (e.g., increased altitude). This is consistent with the ISA conclusions in the current review which recognize other populations that may be potentially susceptible to the effects of CO as continuing to include: those with other pre-existing diseases that may have already limited oxygen availability or increased COHb production or levels, such as people with obstructive lung diseases, diabetes and anemia; older adults; fetuses during critical phases of development and young infants or newborns; commuters and those living near heavily traveled roadways; visitors to high-altitude locations; and people ingesting medications and other substances that enhance endogenous or metabolic CO production.

Preliminary evidence from epidemiological, controlled human exposure, and toxicological studies suggest that people with obstructive lung disease (e.g., COPD patients with underlying hypoxia, asthmatics) may be a susceptible population (ISA, section 5.7.1.2). Overall, the few available epidemiological studies have reported weak, positive associations between ambient CO and CVD hospital admissions for individuals with underlying COPD. Additionally, a controlled human exposure study of individuals with COPD reported that two patients experienced COPD exacerbation and a slight anti-inflammatory effect during CO exposures of 100-125 ppm for 2 hours (ISA, section 5.5.1.2). Other epidemiological studies (ISA, section 5.5.2.2) have reported weak associations in asthmatics, which constitute another population that can experience exercise-induced airflow limitation. Preliminary evidence was also shown in one animal toxicological study (Ghio et al., 2008), indicating mild pulmonary inflammation upon exposure to 50 ppm CO.

With regard to other potentially at-risk populations, there is also limited epidemiological data regarding the susceptibility of diabetics (ISA, section 5.7.1.3). Epidemiologic studies (Pereira Filho et al., 2008; Zanobetti and Schwartz, 2001) provide suggestive evidence that CVD patients with diabetes may be at greater risk of emergency department visits and hospital admissions than those without diabetes (ISA, section 5.7.1.3). Inferences may also be drawn from results from panel studies for individuals with metabolic syndrome[17] that observed associations between short-term exposures to CO and changes in heart rate variability parameters (ISA, section 5.2.1.1) and from a toxicological study providing evidence of vascular dysfunction associated with increases in endogenous CO in an animal model of metabolic syndrome (ISA, section 5.7.1.3). People with anemia who have reduced oxygen-carrying capacity and/or higher baseline COHb levels are an additional population considered to be potentially susceptible to the hypoxic effects of CO. However, there are no controlled human studies or epidemiological

[17] These patients share risk factors with diabetics; see ISA section 5.7.1.3.

studies that have specifically examined the CO-related health effects in individuals with anemia (ISA, section 2.6.1, p. 2-11).

Older adults (65+) have been considered as a potentially susceptible population to the effects of CO, primarily due to the increased prevalence of cardiovascular disease among this population when compared to all age groups or lifestages. There is limited epidemiologic evidence showing greater positive associations between short-term ambient CO concentrations and IHD or myocardial infarction (MI) hospital admissions among older adults as compared to all age groups or younger adults. The combination of this limited epidemiological data and the fact that older adults have a higher prevalence of CAD than the general population, indicates that older adults are a potentially susceptible population for increased health effects due to CO exposure (ISA, section 5.7.2.1).

The developing fetus and young infants or newborns have been considered to be potentially susceptible to CO exposures due to their altered CO kinetics. Although the effects of CO on maternal-fetal relationships are not well understood, fetuses are likely to have higher circulating COHb levels than the mother due to differences in uptake and elimination of CO from fetal Hb (AQCD 2000, section 7.7.1). Newborn infants are also potentially susceptible to CO-induced effects due to their comparatively higher oxygen consumption rate and lower oxygen-transport capacity than those of adults, which could potentially result in higher COHb levels (AQCD 2000, section 7.7.1). Data from laboratory animal studies on CO developmental toxicity suggest that prolonged exposure to high CO levels (>60 ppm) during gestation may produce reduction in birth weight, transient cardiomegaly and delayed behavioral development, or may disrupt the normal physiological roles of endogenous CO in the body (ISA, section 5.4.2.2). Multiple-day prenatal animal exposures to exposures at or above 12 ppm indicated effects on the developing auditory system (ISA, pp. 5-75 to 5-76). Limited epidemiological evidence suggests some association of short-term ambient CO exposure with pre-term birth and birth defects, and weak evidence suggests an association with reduction in birth weight and fetal growth, and infant mortality (ISA, section 5.7.2.2; 2000 AQCD, section 7.7.1), although a clear understanding of the mechanisms by which CO may induce those effects and at what exposure levels is lacking (ISA, section 5.4.3).

Gender has also been considered as a possible source for susceptibility. However, the evidence is inconclusive based on the limited epidemiological data and the gender-specific variability in CO endogenous production (ISA, section 5.7.3). Increased altitude and physical activity through their effect on CO uptake and elimination have also been considered to potentially influence susceptibility. For example, residents of low altitudes visiting high altitudes, especially the elderly and those with CAD (Leaf and Kleinman, 1996; Kleinman et al., 1998), may be at greater risk from added effects of ambient CO than adapted residents (ISA, p.

2-12). Other less certain susceptibility factors have been considered, such as use of medications that may alter CO production (ISA, p. 2-12). Lastly, people experiencing increased CO exposures, such as those spending relatively greater amounts of time in microenvironments with relatively higher levels of ambient CO have been considered to be potentially at risk for CO-induced effects (ISA, section 5.7.6).

As we recognize the potential susceptibility of the populations identified above, we also note the lack of information on specific COHb levels that may be associated with health effects in these other groups and the nature of those effects, as well as a way to relate the specific evidence available for the CAD population to these other populations.

In summary, the current evidence continues to support the identification of people with cardiovascular disease as having susceptibility to CO-induced health effects (ISA, 2-12), with those having CAD as the population with the best characterized susceptibility to CO-induced health effects (ISA, sections 5.7.1.1 and 5.7.8).[18] An important susceptibility consideration for this population is the inability to compensate for CO-induced hypoxia since individuals with CAD have an already compromised cardiovascular system. Included in this susceptible population are those with angina pectoris (cardiac chest pain), those who have experienced a heart attack, and those with silent ischemia or undiagnosed IHD (AHA, 2003). People with other cardiovascular diseases, particularly heart diseases, are also at risk of CO-induced health effects. We also recognize other populations potentially susceptible to CO-induced effects, most particularly those with other pre-existing diseases that may have already limited oxygen availability, increased COHb levels, or increased endogenous CO production, such as people with obstructive lung diseases, diabetes and anemia; however, information characterizing susceptibility for this population is limited.

[18] As recognized in the ISA, "Although the weight of evidence varies depending on the factor being evaluated, the clearest evidence indicates that individuals with CAD are most susceptible to an increase in CO-induced health effects" (ISA, p. 2-12).

- **Does the current evidence alter our conclusions from the previous review regarding the levels of CO in ambient air associated with health effects?**

At the time of the last review, EPA's conclusions regarding concentrations of CO in ambient air that might be associated with risk of health effects were drawn from the combined consideration of the evidence of COHb levels for which cardiovascular effects of concern had been reported and the results of an exposure and dose modeling assessment (59 FR 38906). As described in more detail in section 2.1.1 above, the key effects judged to be associated with CO exposures resulting from concentrations observed in ambient air were cardiovascular effects, as measured by decreased time to onset of exercise-induced angina and to onset of ECG ST-segment depression (59 FR 38913).

Levels of COHb that have been associated with different types of effects in clinical studies are summarized in Table 2-2 below. At the time of the last review, decreases in time to onset of exercise-induced angina (a symptom of myocardial ischemia) had been documented in multiple studies at post-exposure COHb levels ranging from 2.9 to 5.9% (CO-Ox), which represented incremental increases of 1.5-4.4% COHb from baseline (Adams et al., 1988; Allred et al., 1989a, 1989b, 1991; Anderson et al., 1973; Kleinman et al., 1989, 1998; Sheps et al., 1987[19]). The matched measurements available from Allred et al. (1989a, 1989b, 1991) of CO-Ox and gas chromatography, the method generally recognized to be the more accurate for COHb levels below 5% (ISA, section 5.2.4), indicate that CO-Ox measurements of 2.9 to 3.0% COHb generally correspond to GC measurements on the order of 2-2.4%.[20] Evidence of effects in other clinical study groups includes effects in subjects with cardiac arrhythmias and effects on exercise duration and maximal aerobic capacity in healthy adults. Among the studies of myocardial ischemia indicators in patients with CAD, the two studies involving the lowest experimental CO exposures (which resulted in average increases in COHb of about 1.5% over pre-exposure baseline) were Anderson et al. (1973)[21] and the more recent Allred et al. (1989a, 1989b, 1991) to which we give primary attention in this review (discussed in more detail above). Neither study provided evidence of a measurable threshold at the lowest experimental CO exposures and associated COHb levels assessed (mean of 2.0-2.4% COHb, GC). Allred et al. (1989a, 1989b, 1991) further reported a dose-response relationship between the increased COHb levels and the

[19] See footnote 4 above.

[20] In the lower CO exposure group, the post-exposure mean COHb was 3.21% by CO-Ox and 2.38% by GC, while the post-exercise mean COHb was 2.65% by CO-Ox and 2.00% by GC (Allred et al., 1989a, 1989b, 1991).

[21] The study by Anderson et al. (1973) did not use GC to measure COHb levels, and reported reduced exercise duration due to increased chest pain at CO exposures resulting in 2.9% COHb (CO-Ox), representing a 1.6% increase in average COHb levels over baseline.

response of the assessed indicators of myocardial ischemia (Allred et al., 1989a, 1989b, 1991). While this evidence informed our conclusions regarding COHb levels associated with health effects, the CO exposure concentrations employed in the studies to achieve these COHb levels were substantially above ambient concentrations. Thus, an exposure and dose assessment was performed to consider the COHb levels that might be attained as a result of exposures to ambient CO allowed under the current NAAQS, as described in section 2.1.1 above.

Table 2-2. Carboxyhemoglobin levels and reported effects in CAD patients and healthy adults resulting from short-term CO exposures.

Study Population	Effect	%COHb, mean		%COHb, increase over baseline, GC (CO-Ox)	Study
		GC	CO-Ox		
Patients with coronary artery disease[A]	Reduction in time to exercise-induced onset of myocardial ischemia following ~1-hour exposures - 5.1% decrease in time to ST- segment change, 4.2% decrease in time to angina - 12.1% decrease in time to ST- segment change, 7.1% decrease in time to angina	2.0-2.4[B] 3.9-4.7[B]	2.7-3.2[B] 4.7-5.6[B]	1.4-1.8[B] (1.4-2.0) 3.2-4.0[B] (3.5-4.4)	Allred et al., 1989a, 1989b, 1991
	Reduction in time to exercise-induced angina and/or ST segment changes following exposures of 1-4 hours	NA[C]	2.9-5.9	(1.4-4.2)	Adams et al., 1988; Anderson et al., 1973; Kleinman et al., 1989, 1998;Sheps et al,1987[D]
	Exercise-induced arrhythmia following 1-hour exposure	NA	5.3[E]	(3.5)[E]	Sheps et. al., 1990, 1991
Healthy adults	Reduction in exercise duration and/or maximal aerobic capacity	NA	3[F]-20	-	Adir et al., 1999; Drinkwater et al., 1974; Ekblom and Huot, 1972; Horvath, 1975; Raven et al., 1974; Weiser et al., 1978
	Inconsistent findings of behavioral effects (hand eye coordination, vigilance, continuous performance of critical tasks)	NA	5-20	-	Benignus et. al., 1987, 1990; Fodor and Winneke, 1972 ; Horvath et al., 1971; Putz et. al., 1976, 1979

[A] All studies involved subjects with reproducible exercise-induced angina.
[B] The values presented correspond to the study subject average levels post and pre-exercise (subsequent to CO exposure).
[C] NA = not available.
[D] Statistical analyses of the Sheps data by Bissette et al (1986) indicate a significant decrease in time to onset of angina at 4.1% COHb if subjects that did not experience exercise-induced angina during air exposure are also included in the analyses.
[E] Based on mean of pre-and post-exercise measurements for the 6% COHb group and increase from pre-exposure. The difference in COHb from the mean air exposure day level was 3.9%.
[F] The study of adults aged 40-57 (Raven et al., 1974) reported an effect on exercise duration at 2.3% COHb (CO-Ox), while the other studies, which were of adults younger than 35 reported effects at or above 3.3%.

Since the time of the last review, there have been no new controlled human exposure studies specifically designed to evaluate the effects of CO exposure in susceptible populations at COHb levels below 2%. Thus, similar to the last review, the multilaboratory study by Allred et al. (1989a, 1989b, 1991) continues to be the study that has evaluated cardiovascular effects of

greatest concern (i.e., reduced time to exercise-induced myocardial ischemia as indicated by ECG ST-segment changes and angina) at the lowest tested COHb levels (ISA, section 2.7). This study is also of particular importance in this review because it is considered the most rigorous and well designed study, presenting the most sensitive analysis methods (GC used in addition to CO-Ox) to quantify COHb blood levels. Key findings from that study with regard to levels of CO associated with health effects include the following:

- Short (50-70 minute) exposure to increased CO concentrations that resulted in increases in COHb to mean levels of 2.0% and 3.9% from baseline of 0.6% significantly reduced exercise time required to induce markers of myocardial ischemia in CAD patients (by about one half minute in the lower exposure).[22]
- The associated dose-response relationship between incremental changes in COHb and change in time to myocardial ischemia in CAD patients indicates a 1.9% and 3.9% reduction in time to onset of exercise-induced angina and ST-segment change, respectively, per 1% increase in COHb concentration from average baseline COHb of 0.6% without evidence of a measurable threshold.

In considering the clinical study evidence on COHb levels associated with effects to address the question regarding ambient CO concentrations associated with health effects, we have developed estimates of COHb associated with different air quality conditions using quantitative exposure and dose modeling, as was done at the time of the last review. Since the last review, there have been numerous improvements to the exposure and COHb models that we use to estimate exposure and dose for the current review. The results of modeling using these improved tools in the current review and our conclusions based on it with regard to the expectation for COHb levels of concern to occur in the at-risk population under differing air quality conditions are described in section 2.2.2 below.

As discussed in more detail above, a number of epidemiological studies of health outcome associations with ambient CO have been conducted since the last review. These include studies, identified in Table 2-1 above, that have reported associations with different ambient CO metrics (e.g., 1-hour and 8-hour averages, often as central-site estimates) derived from CO measurements at fixed-site ambient monitors in selected urban areas of the U.S. and cardiovascular endpoints other than stroke, particularly hospitalizations and emergency

[22] Relative to baseline (0.6% mean COHb), mean COHb levels of 2.0-2.4% and 3.9-4.7% were associated with decreases in time to onset of exercise-induced ST-segment changes of 5.1% ($p=0.01$) and 12.1%($p<0.001$), respectively. Similarly, the time to onset of exercise-induced angina was shortened by 4.2% ($p=0.027$) and 7.1% ($p=0.002$) for these two mean levels of COHb (Allred et al., 1989a, 1989b.1991). Across all subjects, the mean time to angina onset for baseline or control ("clean" air) exposures was approximately 8.5 minutes, and the mean time to ST endpoint was approximately 9.5 minutes, with the "time to onset" reductions of the two exposure levels being approximately one half and one minute, respectively for ST-segment change, and slightly less and slightly more than one half minute, respectively, for angina (Allred et al., 1989b).

department visits for specific cardiovascular health outcomes including IHD, CHF and CVD (Bell et al., 2009; Koken et al., 2003; Linn et al., 2000; Mann et al., 2002; Metzger et al., 2004; Symons et al., 2006; Tolbert et al., 2007; Wellenius et al., 2005). In general, these studies, many of which were designed to evaluate the effects of a variety of air pollutants, including CO, report positive associations, a number of which are statistically significant (ISA, sections 5.2.3 and 5.2.1.9; Table 2-1 above). The long-standing body of evidence for CO summarized above, including the well-characterized role of CO in limiting oxygen availability, lends biological plausibility to the ischemia-related health outcomes reported in the epidemiological studies, providing coherence between these studies and the clinical evidence of short-term exposure to CO and health effects.

In consideration of the evidence base for CO cardiovascular effects, Figure 2-1 below presents a conceptual model of the pathway from CO exposures to these effects. Figure 2-2 separately shows elements of this conceptual model investigated in clinical studies in contrast to those in the epidemiological studies referenced above. As shown in Figure 2-2, the clinical studies document relationships between directly measured controlled short-term CO exposures and specific levels of an internal dose metric, COHb, which elicited specific myocardial ischemia-related responses in CAD patients. These studies inform our interpretation of the associations we observed in the epidemiological studies. The epidemiological studies reported associations between CO levels measured at fixed-site monitors and emergency department visits and/or hospital admissions for IHD and other cardiovascular disease-related outcomes that are plausibly related to the effects on physiological indicators of myocardial ischemia (e.g., ST-segment changes) demonstrated in the controlled human exposure studies, providing coherence between the two sets of findings (ISA, p. 5-48).

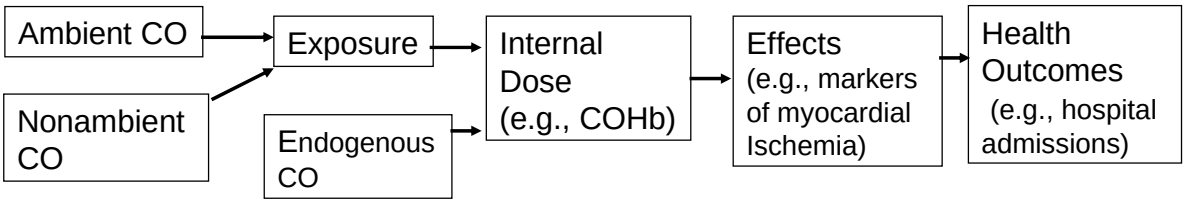

Figure 2-1. A conceptual model of CO source-to-health outcome pathway.

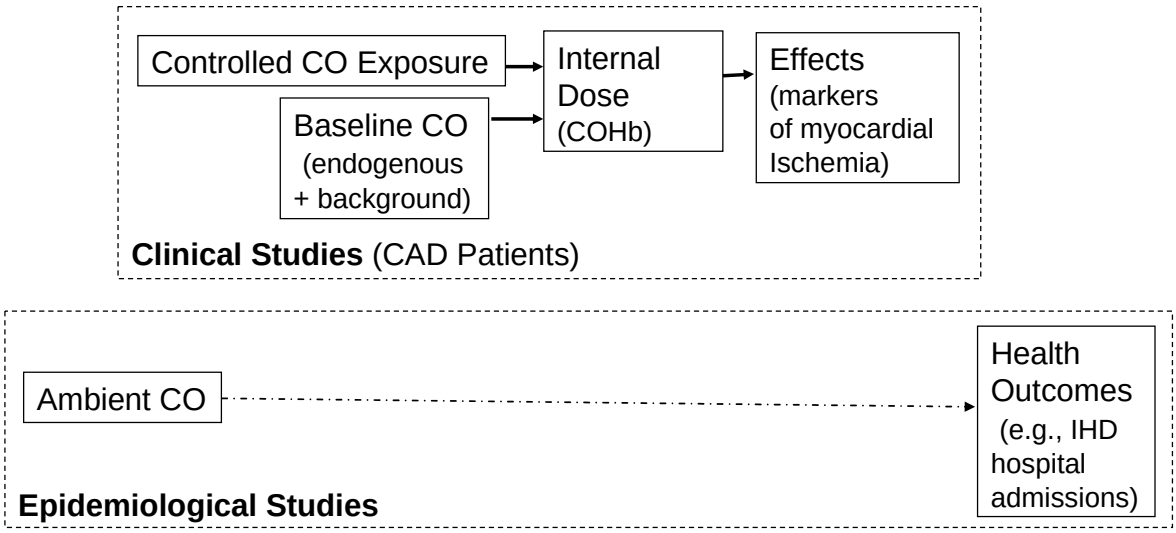

Figure 2-2. Components of CO source-to-outcome conceptual model measured in controlled human exposure and epidemiological studies discussed in this document.

We recognize, however, several gaps between the two lines of evidence (clinical and epidemiological) which complicate their integration, particularly with regard to ambient exposures and ambient concentrations associated with health effects. These gaps limit our ability to integrate the evidence from these epidemiological studies with our knowledge of CO-related effects based on the clinical evidence. Most particularly we lack information on the actual CO exposures and associated internal COHb levels for the epidemiological study populations, including the relative contributions from ambient and nonambient CO. These gaps in our understanding of the role of nonambient CO exposures and their contribution to COHb levels complicate our ability to discern the ambient concentrations that may be eliciting health effects. Moreover, it is also unknown how the unmeasured exposures in epidemiological study populations relate to the exposure concentrations of the clinical studies (e.g., 1 hour at approximately 50-200 ppm), which were substantially higher than current commonly occurring ambient concentrations (ISA, section 2.7).[23,24] We are also limited in our understanding of the

[23] As recognized in the ISA in consideration of the epidemiological studies, the known role of CO in limiting oxygen (O_2) availability lends biological plausibility to ischemia-related health outcomes following CO exposures, although "it is not clear whether the small changes in COHb associated with ambient CO exposures results in substantially reduced O_2 delivery to tissues" (ISA, p. 5-48).

[24] Experimental studies have not been conducted at COHb levels that might be elicited in response to exposures to lower CO concentrations such as those within the ranges of current maximum ambient concentrations

specific relationship between the changes in indicators of myocardial ischemia observed in the controlled human exposure studies and the hospital admissions assessed in the epidemiological studies. Together, these gaps complicate our ability to draw conclusions regarding the levels of CO in ambient air associated with health effects that are based on an integration of the epidemiological evidence into the full body of evidence for CO.

In considering these epidemiological studies in the case of CO, we recognize that in contrast to other traffic-related pollutants, CO presents specific challenges that complicate the quantitative interpretation with regard to ambient concentrations that might be eliciting the reported health outcomes. In particular, a major challenge relates to the difficulty in determining the extent to which ambient CO is independently associated with cardiovascular effects or if CO at ambient levels is acting as a surrogate for the effects of another traffic-related pollutant or mixture of pollutants (ISA, section 5.2.3). As noted in the ISA, in interpreting the epidemiological evidence for cardiovascular morbidity "[i]t is difficult to determine from this group of studies the extent to which CO is independently associated with CVD outcomes or whether CO is a marker for the effects of another traffic-related pollutant or mix of pollutants. As recognized in the ISA, "[o]n-road vehicle exhaust emissions are a nearly ubiquitous source of combustion pollutant mixtures that include CO and can be an important contributor to CO in near-road locations" (ISA, p. 5-40 – 5-41). We also note CASAC's recognition of the potential for co-pollutants to serve as confounders to be "particularly problematic for CO" and the need to give consideration to the possibility of CO serving as a surrogate for a mixture of fossil-fuel-combustion-related pollutants (Brain and Samet, 2010a).

While recognizing the complications regarding the use of the epidemiological studies in drawing conclusions about ambient CO levels associated with health effects, we have nonetheless considered for our purposes in addressing the question posed here the epidemiological studies in U.S. locations that investigated ischemia-related and CHF outcomes presented in Table 2-3. Among the epidemiological evidence, the studies of hospital admissions for CHD (i.e., IHD and MI) were considered to provide the strongest and most consistent weight of evidence for short-term effects of CO (ISA, Figure 5-2, pp. 5-24 to 5-26), with the U.S. studies for the broader category of all CVD hospital admissions also contributing to the ISA's causality determination (ISA, Figure 5-5, pp. 5-33 to 5-36). In considering the studies in Table 2-3, we recognize that CHF is a chronic condition for which there are multiple causes and for which the evidence regarding the role of CO is less clear than it is for IHD, MI and all CVD

(ISA, section 2.7) or closer to personal exposure concentrations more commonly expected currently (e.g., ISA, Figure 3-45).

outcomes (which are inclusive of IHD), for which more direct conceptual linkages can be drawn to the myocardial ischemia effects of the CAD clinical studies.

As recognized above, this set of studies provides epidemiological evidence for a positive association between cardiovascular outcomes and ambient CO concentrations. With regard to our overarching question as to the adequacy of the protection afforded by the current standards, however, the studies in which CO concentrations exceeded the current NAAQS during some portion of the study period are, accordingly, less informative. While the full set of these epidemiological studies, including those reporting associations with ambient CO concentrations under conditions when the current standards were not met, provide support to the previous evidence regarding cardiovascular effects of CO, it is the studies involving air quality conditions in which the current standards were met that are most informative to the overarching question of adequacy. Accordingly, in considering these epidemiological studies, we have considered the air quality conditions during the periods of study as described below.

The set of studies listed in Table 2-3 encompasses a range of air quality conditions extending from those that did not meet the current 8-hour CO standard down to those that did.[25] As mentioned above, the study areas for the IHD and CVD outcomes (inclusive of IHD) include Atlanta, urban areas of California and a group of 126 urban counties[26] and the multi-year time periods for this set of studies together span the period from 1988 through 2005 (Table 2-3 below). During the time periods of study, however, only the Atlanta studies (Metzger et al., 2004; Tolbert et al., 2007) did not include years in which ambient CO concentrations exceeded the 8-hour standard (Table 2-3). The CO concentration with which an association with hospital admissions was observed in the Atlanta studies was the average of daily maximum 1-hour concentrations on the day of and two days prior to hospital admission. Based on data reported to AQS for the general areas studied, ambient CO concentrations during a portion of the time periods studied in the other three of these analyses exceeded the level of the current 8-hour NAAQS (Bell et al., 2007; Linn et al., 2000; Mann et al., 2002), with the second highest non-overlapping 8-hour average CO concentration in a year (i.e., the design value for the 8-hour standard) ranging up to 24.3 ppm, as compared to the 8-hour standard of 9 ppm.[27] In the Atlanta

[25] While the 1-hour CO standard was met during the full study period for all of these studies, the 8-hour standard was not (Table 2-3). As in past reviews, the 8-hour standard continues to be the controlling standard.

[26] The 126 counties span some 40 states, including the District of Columbia.

[27] Staff also note that the national-scale study of 126 urban counties by Bell et al. (2009) included a subset analysis that evaluated associations for all cardiovascular outcomes, adjusted for NO_2, after restricting the dataset to days with a 1-hour daily maximum CO concentration less than 35 ppm, which is the level of the 1-hour standard although its form is the 2nd maximum in a year. This subset analysis found a statistically significant effect estimate identical to the estimated risk with the full data set. Further subset analyses by Bell et al. (2009) using data below cutoff values of 1-10 ppm all found positive, statistically significant associations (with adjustment for NO_2),

study area, the design value for the 8-hour standard ranged from 2.6 up to 5.3 ppm over the combined 12 years of study.

The study areas for which statistically significant positive associations with CHF were observed include the area of California described for Linn et al (2000) above, as well as two additional areas (Table 2-3; ISA, pp. 5-31 to 5-33; Table 2-1 above). The additional study areas were Denver, Colorado, and Allegheny County, Pennsylvania (Koken et al., 2003; Wellenius et al., 2005). While the current 8-hour standard was not met in the Denver or California study areas during the full period of those studies,[28] the current 8-hour standard was just met in the Allegheny County study area, with design values ranging from 3.8 ppm up to 8.8 ppm. This study was focused on 24-hour average CO concentrations, having analyzed the relationship between hospital admissions for CHF hospital admissions and estimates of daily county-wide ambient CO concentrations (Wellenius et al., 2005).[29] We additionally note other positive, though not statistically significant, associations were observed for CHF-related hospitalizations in the Atlanta and Baltimore studies with the average 1- or 8-hour daily maximum CO concentrations over the most recent 3 or 4 days, respectively.[30]

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although confidence intervals increased at the lower cutoff values and the proportion of values near and below the limit of detection grew larger.

[28] In the study focused in Denver, a statistically significant association is observed with CO concentrations 3 days prior to the health outcome (Koken et al., 2003)

[29] Data from three monitors were used to derive a daily estimate for the study area described by the authors as generally representative of the study area as opposed to being reflective of local conditions near any specific monitor (Zanobetti et al., 2000). The algorithm used was described as accounting for monitor-specific measurements and variances (Wellenius et al., 2005).

[30] As noted in the ISA, associations with same day or previous day CO concentrations are considered consistent with the mode of action described for CO (ISA, p. 5-40).

Table 2-3. Air quality information for geographical areas of key U.S. epidemiological studies.

Study Information								Ambient Air Quality[A]			
Study Area	Study Time Period	Study Reference	Health Outcome[B]	CO Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported CO Concentrations,[C] in terms of study metric (ppm)		Design Values for Current NAAQS, across study years (ppm)		99th percentile 1-hour daily maximum (ppm)	99th percentile 8-hour daily maximum (ppm)
						Mean/ median*	Range	1-hour *Standard=35*	8-hour *Standard=9*		
20-county Atlanta area	1993-2000	Metzger et al., 2004	IHD CHF[NS]	1-hour daily maximum, average for most recent 3 days	Single central residential monitor	1.8	0.5 – 3.4 (10th - 90th percentile)	4.8 -16.3	3.2 - 5.3	4.4 – 7.9	3.0 – 4.9
	1993-2004	Tolbert et al., 2007	CVD	1-hour daily maximum, Average for most recent 3 days	Single central residential monitor	1.6	0.1 – 7.7	4.5 -16.3	2.6 - 5.3	3.6 – 7.9	2.4 – 4.9
South Coast Air Basin, CA	1988-1995	Mann et al., 2002	IHD, IHD+CHF	8-hour daily maximum, average for most recent 4 days	Residence grid centroid interpolated from 3 closest monitors	2.07	0.30 – 11.8	16.5 - 32	11.6 – 23.4	16.1 – 31.0	11.2 – 19.1
	1992-1995	Linn et al., 2000	MI CVD CHF	24-hour average, same day	Average across all monitors in basin	1.5	0.3 – 5.3	16.5 - 25	11.6 -16.4	16.1 – 21.0	11.2-14.4
126 urban counties	1999-2005	Bell et al., 2009	CVD 65+	1-hour daily maximum, same day	Average of monitors in county of residence	1.3*	0.05 – 9.7	14.9-33.5	8.2-24.3	10.4 – 31.8	6.7-18.9
Baltimore, MD	2002 (April-Dec)	Symons et al., 2006	CHF[NS]	8-hour daily maximum, Average for most recent 4 days	Single central residential monitor	0.4	0.0 – 2.3	9.2	3.0	4.7	2.8
Allegheny county, PA	1987-1999	Wellenius et al., 2005	CHF	24-hour average, same day	Single area-wide representation of 3 monitors	1.03*	0.42 – 2.04 (5th - 95th percentile)	5.4-19.4	3.8-8.8	4.3 – 15.9	3.3 – 8.0
Denver	1993-1997 (Jul-Aug)	Koken et al., 2003	CHF	24-hour average, 3 days prior	Average across all monitors	0.9	0.3 – 1.6	11.2-18.2[A]	6.4-10.4[A]	9.3 – 16.2	5.8 – 10.4

A Air quality information provided here is drawn from monitors reporting to the U.S. Air Quality System, and two monitors reporting to the SouthEastern Aerosol Research and Characterization study database (Appendix B). Design values are CO concentrations for the study area in the statistical form of the standard. Presented is the range of 2nd maximum 1-hour and 8-hour average concentrations at highest monitor reporting to AQS in each study area across years of study. For partial-year studies, the values presented reflect full year of data, inclusive of study months.
B Hospital admissions for ischemic heart disease (IHD), myocardial infarction (MI), cardiovascular disease (CVD; for Bell et al. [2009] only subjects ≥65 years old), congestive heart failure (CHF).
C Ambient CO concentrations reported in the study.
NS Association was not statistically significant.

In summary, although there is no new evidence regarding the effects of short-term controlled CO exposures that result in lower COHb levels, the evidence is much expanded with regard to epidemiological[31] analyses of ambient monitor concentrations, which observed associations between specific and overall cardiovascular-related outcomes and ambient CO measurements. With regard to extending our understanding of effects occurring below levels of CO observed in the clinical studies, however, the epidemiological evidence for CO is somewhat limited. The epidemiological evidence lacks measurements of COHb or personal exposure concentrations that would facilitate integration with the clinical data. Furthermore, the epidemiological evidence base for IHD outcomes or CVD outcomes as a whole includes a number of studies involving conditions in which the current standard was not met. Though these studies are informative to consideration of the relationship of health effects to the full range of ambient CO concentrations, they are less useful to informing our conclusions regarding adequacy of the current standards. The smaller set of studies, under conditions where the current standards were met, is considered to better inform our assessment of the adequacy of the standards or conditions of lower ambient concentrations.

Among the few studies conducted during conditions in which the current standards were always met, the studies reporting statistical significance for IHD or all CVD outcomes are limited to a single study area (i.e. Atlanta). When the analyses reporting significance for association with CHF outcomes are also considered, a second study area is identified (Allegheny County, PA) in which the current standard is met throughout the study period. The analyses for both areas involve the use of central site monitor locations or area-wide average concentrations, which given the significant concentration gradients of CO in urban areas (ISA, section 3.6.8.2), complicates our ability to draw conclusions from them regarding ambient CO concentrations of concern.

Therefore, with regard to this question of CO concentrations, we have primarily focused our consideration of the epidemiological studies on the extent to which this evidence is consistent with and generally supportive of conclusions drawn from the combined consideration of the controlled human exposure evidence with estimates from the exposure and dose assessment (section 2.2.2 below). As in the previous review, we believe the integration of the controlled human exposure evidence with the exposure and dose estimates will be most important to informing conclusions regarding ambient CO concentrations of public health concern. This integration, based on the REA for the current review, is considered with regard to the adequacy of the current standards in section 2.2.2 below.

[31] Few epidemiological studies that had investigated the relationship between CO exposure and ischemic heart disease were available at the time of the last completed review (USEPA, 1991, section 10.3.3).

- **To what extent have important uncertainties identified in the last review been reduced and/or have new uncertainties emerged?**

Since the time of the last review, some important uncertainties have been reduced, some still remain and others associated with newly available evidence have been identified. A range of important uncertainties were identified in a number of areas at the time of the last review (59 FR 38913, USEPA, 1992), including:

- The adverse nature and health significance of the small changes in time to ST-segment depression identified at the lowest COHb levels investigated in the controlled human exposure studies, and the magnitude of risk associated with such changes for specific health outcomes, such as myocardial infarction, or of slight but cumulative myocardial damage, among other possibilities.
- The extent to which COHb measurements made using CO-Ox do not reflect COHb levels in angina patients studied and the potential for as yet unidentified health effects at COHb levels below 2%.
- The potential for short-term peak CO exposures to contribute to CNS effects which might affect individual's performance of complex activities such as driving a car or to contribute to other effects of concern.
- Effects of ambient CO on potentially susceptible populations other than those with cardiovascular disease, including the developing fetus.
- Modeling of COHb formation associated with exposures to ambient CO under different air quality conditions, including those associated with just meeting different NAAQS.

As discussed below, some of these uncertainties have been reduced, while some still remain.

The CO-induced effects considered of concern at the time of the last review were reduced time to exercise-induced angina and ST-segment depression in patients suffering from coronary artery disease as a result of increases in COHb associated with short CO exposures. These effects had been well documented in multiple studies, and it was recognized that the majority of cardiologists at the time believed that recurrent exercise-induced angina was associated with substantial risk of precipitating myocardial infarction, fatal arrhythmia, or slight but cumulative myocardial damage (USEPA, 1992, p. 22; 59 FR 38911; Basan, 1990; 1991 AQCD). As at the time of the last review, although ST-segment depression is a recognized indicator of myocardial ischemia, the exact physiological significance of the observed changes among individuals with CAD is unclear (ISA, p. 48).

In interpreting the study results at the time of the last review, EPA recognized uncertainty in the COHb measurements made using CO-Ox and associated uncertainty in establishing a lowest effects level for CO (USEPA, 1992, p. 31). A then-recent multicenter study (Allred et al., 1989a, 1989b, 1991) was of great importance at that time for several reasons including the large number of subjects used, the rigorous protocol used for subject selection, the use of the most

accurate method to measure blood COHb levels and the finding of a dose-response relationship between COHb levels and the ischemic events evaluated in the study. This study reported changes in post-exercise ST-segment depression and reduced time to onset of exercise-induced angina as a result of increases in COHb from a mean baseline of 0.6% to mean levels of 2% and 3.9% (ISA, section 5.2.4; (Allred et al., 1989a,1989b, 1991). In the current review of the evidence related to cardiovascular effects associated with CO exposure, we place primary emphasis on the findings of Allred et al. (1989a, 1989b, 1991) recognizing the superior quality of the study, both in terms of the rigorous study design as well as the sensitivity of the analytical methods used in determining COHb concentrations (ISA, Section 2.7). No additional clinical studies are available that evaluate responses to lower COHb levels in the cardiovascular-disease population, and uncertainties still remain in determining specific and quantitative relationships between the CO-induced effects in these studies and the increased risk of specific health outcomes. Further, with regard to then-unidentified effects at lower COHb levels, no studies have identified other effects on the CAD population or on other populations at lower exposures (ISA, sections 5.2.2).

The last review recognized a variety of neurobehavioral effects associated with CO exposure, including changes in visual perception, hearing, motor performance and vigilance among other measures of neurobehavioral performance based on a series of studies conducted from the mid 1960's through the early 1990's (1991 AQCD). Since these effects were observed at exposures to CO resulting in COHb levels ranging from 5-20%, and were poorly understood at the time (1991 AQCD), the review focused on cardiovascular effects, which had been observed at COHb levels below 5% and consequently for which a focus would also provide adequate protection against potential adverse neurobehavioral effects. No new human clinical studies have evaluated CNS or behavioral effects of exposure to CO (ISA, section 5.3.1). However, given the drastic reduction in CO ambient concentrations, the occurrence of these effects in response to ambient CO would be expected to be rare within the current population. Thus, our uncertainty with regard to the potential for such effects to be associated with current ambient CO exposures is reduced.

Since the 1994 review, the epidemiologic and toxicological evidence of effects on birth and developmental outcomes has expanded, although the available evidence is still considered limited with regard to effects on preterm birth, birth defects, decreases in birth weight, measures of fetal growth, and infant mortality (ISA, section 5.4). Further, while animal toxicological studies provide support and coherence for those effects, the understanding of the mechanisms underlying reproductive and developmental effects is still lacking (ISA, section 5.4.1). Thus, although the evidence continues to "suggest[s] that critical developmental phases may be characterized by enhanced sensitivity to CO exposure" (ISA, p. 2-11), evidence is lacking for

adverse developmental or reproductive effects at CO exposure concentrations near those associated with current levels of ambient CO.

Numerous improvements have been made over the last decade that have reduced the uncertainties associated with the models used to estimate COHb levels resulting from ambient CO exposures under different air quality conditions, including those associated with just meeting the current CO NAAQS (REA, section 4.3). This progression in exposure model development has led to the model currently used by the Agency (APEX4.3), which has an enhanced capacity to estimate population CO exposures and more accurately predicts COHb levels in persons exposed to CO. Our application of APEX4.3 in this review, using updated data and new algorithms to estimate exposures and doses experienced by individuals, better represents the variability in population exposure and COHb dose levels than the model version used in previous CO assessments. However, while APEX 4.3 is greatly improved when compared with previously used exposure models, its application is still limited with regard to data to inform our understanding of spatial relationships in ambient CO concentrations and within microenvironments of particular interest. Further information regarding model improvements and remaining exposure modeling uncertainties are described in section 2.2.2 below.

The much-expanded epidemiologic database in the current review includes studies that show associations between ambient CO concentrations and increases in emergency room visits and hospitalizations for disease events plausibly linked to the effects observed in the controlled human exposure studies of CAD patients (ISA, section 2.5.1), providing support for our conclusion regarding coronary artery disease as the most important susceptibility characteristic for increased health risk due to CO exposure (ISA, p. 2-10). However, we recognize aspects of this epidemiological evidence that complicate quantitative interpretation of it with regard to ambient concentrations that might be eliciting the reported health outcomes. As an initial matter, we note the substantially fewer studies conducted in areas meeting the current CO standards than is the case for NO_2 and PM (USEPA, 2008c, 2009a), as recognized above. Further, we recognize complicating aspects of the evidence that relate to conclusions regarding CO as the pollutant eliciting the effect reported in the epidemiological studies and to our understanding of the ambient CO and nonambient concentrations to which study subjects demonstrating these outcomes are exposed.

In considering conclusions regarding CO as the pollutant eliciting the effects in these studies, we note the use of two-pollutant regression models, a commonly used statistical method (ISA, section 1.6.3). Although CO associations, in some studies, are slightly attenuated in models that adjusted for other combustion-related pollutants (e.g., $PM_{2.5}$ or NO_2), they generally

remain robust (ISA, Figures 5-6 and 5-7).[32] In considering these two-pollutant model results, however, we recognize the potential for there to be etiologically relevant pollutants that are correlated with CO yet absent from the analysis. Similarly, CASAC commented that "the problem of co-pollutants serving as potential confounders is particularly problematic for CO". They stated that "consideration needs to be given to the possibility that in some situations CO may be a surrogate for exposure to a mix of pollutants generated by fossil fuel combustion" and "a better understanding of the possible role of co-pollutants is relevant to … the interpretation of epidemiologic studies on the health effects of CO" (Brain and Samet, 2010a). This issue is particularly important in the case of CO in light of the uncertainty (referenced below) regarding the biological plausibility of CO-related effects at low ambient concentrations and in light of the sizeable portion of ambient CO measurements that are at or below monitor detection limits. Consequently, the extent to which multi-pollutant regression models effectively disentangle and quantitatively interpret a CO-specific effect distinct from that of other pollutants remains an area of uncertainty.

In considering ambient concentrations that may be triggering health outcomes analyzed in the epidemiological studies, we recognize the uncertainty introduced by exposure error. Exposure error can occur when a surrogate is used for the actual ambient exposure experienced by the study population (e.g., ISA, section 3.6.8). There are two aspects to the epidemiological studies in the specific case of CO, as contrasted with the cases of other pollutants such as NO_2 and PM, that may contribute to exposure error in the CO studies. The first relates to the low concentrations of CO considered in the epidemiological studies and monitor detection limits. The second relates to the use in the epidemiological studies of area-wide or central-site monitor CO concentrations (Table 2-3) in light of information about the gradient in CO concentrations with distance from source locations such as highly-trafficked roadways (e.g., Karner et al., 2010).

Uncertainty in the assessment of exposure to ambient CO concentrations is related to the prevalence of ambient monitor CO concentrations at or below detection limits, which is a greater concern for the more recently available epidemiological studies in which the study areas have much reduced ambient CO concentrations compared with those in the past. For example, the ISA notes that roughly one third of the 1-hour ambient CO measurements reported to AQS for

[32] In interpreting the epidemiological evidence for cardiovascular morbidity the ISA notes that it "is difficult to determine from this group of studies the extent to which CO is independently associated with CVD outcomes or if CO is a marker for the effects of another traffic-related pollutant or mix of pollutants. On-road vehicle exhaust emissions are a nearly ubiquitous source of combustion pollutant mixtures that include CO and can be an important contributor to CO in near-road locations. Although this complicates the efforts to disentangle specific CO-related health effects, the evidence indicates that CO associations generally remain robust in copollutant models and supports a direct effect of short-term ambient CO exposure on CVD morbidity." (ISA, pp. 5-40 to 5-41).

2005-2007 were below the method limit of detection for the monitors analyzed (ISA, p. 3-34). Appendix B additionally indicates a similarly notable proportion of measurements below limit of detection for epidemiological study areas meeting the current standards (e.g., Atlanta, Allegheny County). This complicates our interpretation of specific ambient CO concentrations associated with health effects (ISA, p. 3-91; Brain and Samet, 2010a). In contrast to CO, other combustion-related criteria pollutants such as $PM_{2.5}$ and NO_2 generally occur above levels of detection, providing us with greater confidence in quantitative interpretations of epidemiological studies for those pollutants.

There are also differences in the spatial variability associated with $PM_{2.5}$ and NO_2 concentrations as compared to CO concentrations that add complexity to the estimation of CO exposures in epidemiological studies. In general, $PM_{2.5}$ concentrations tend to be more spatially homogenous across an urban area than CO concentrations. CO concentrations in urban areas are largely driven by mobile sources, while urban $PM_{2.5}$ concentrations substantially reflect contributions from mobile and a variety of stationary sources. The greater spatial homogeneity in $PM_{2.5}$ concentrations is due in part to the transport and dispersion of small particles from the multiple sources (USEPA, 2009a, sections 3.5.1.2 and 3.9.1.3), as well as to contributions from secondarily formed components "produced by the oxidation of precursor gases (e.g., sulfur dioxide and nitrogen oxides) and reactions of acidic products with NH_3 and organic compounds" (USEPA, 2009a, p. 3-185), which likely contribute to spatial homogeneity. Similarly, "because NO_2 in the ambient air is due largely to the atmospheric oxidation of NO emitted from combustion sources (ISA, section 2.2.1), elevated NO_2 concentrations can extend farther away from roadways than the primary pollutants also emitted by on-road mobile sources" (40 FR 6479, February 9, 2010). In contrast to $PM_{2.5}$ and NO_2, CO is comparatively non-reactive, which may contribute to the steeper CO gradient observed near roadways. Therefore, the misclassification of exposure arising from the utilization of central site monitors to measure $PM_{2.5}$ and NO_2 exposures is likely to be smaller than is the case for CO exposures.

An additional complication to a comparison of our consideration of the CO epidemiological evidence to that for other traffic-related criteria pollutants is that, in contrast to the situation for all other criteria pollutants, the epidemiological studies for CO use a different exposure/dose metric from that which has been the focus of the broader health evidence base, and additional information that might be used to bridge this gap is lacking. In the case of CO, the epidemiological studies use air concentration as the exposure/dose metric, while the broader health effects evidence for CO demonstrates and focuses on an internal biomarker of CO exposure (COHb) which has been considered a critical key to CO toxicity. In the case of the only other criteria pollutant for which the health evidence relies on an internal dose metric – lead

- the epidemiological studies also use that metric.[33] For other criteria pollutants, including PM and NO_2, air concentrations are used as the exposure/dose metric in both the epidemiological studies and the other types of health evidence. Thus, there is no comparable aspect in the PM or NO_2 evidence base. The strong evidence describing the role of COHb in CO toxicity is important to consider in interpreting the CO epidemiological studies and contributes to the biological plausibility of the ischemia-related health outcomes that have been associated with ambient CO concentrations. Yet, we do not have information on the COHb levels of epidemiological study subjects that we can evaluate in the context of the COHb levels eliciting health effects in clinical studies. Further, we lack additional information on the CO exposures of the epidemiological study subjects to both ambient and nonambient sources of CO that might be used to estimate their COHb levels and bridge the gap between the two study types. And, as noted in the ISA, "it is not clear whether the small changes in COHb associated with ambient CO exposure results in substantially reduced {oxygen} delivery to tissues," with further investigations needed to investigate potential roles of other mechanisms in CO toxicity (ISA, p. 5-48). Thus, there are uncertainties associated with the epidemiological evidence that "complicate the quantitative interpretation of the epidemiologic findings, particularly regarding the biological plausibility of health effects occurring at COHb levels resulting from exposures to the ambient CO concentrations" assessed in these studies (ISA, p. 2-17).

In summary, some important uncertainties from the last review have been reduced, including those associated with concerns for ambient levels of CO to pose neurobehavioral risks. A variety of uncertainties still remain including the adverse nature and significance of the small changes in time to ST-segment depression identified at the lowest COHb levels investigated, and the magnitude of associated risk of specific health outcomes, as well as the potential for as-yet-unidentified health effects at COHb levels below 2%. Our exposure and dose models have improved giving us increased confidence in their estimates. Additionally, although the evidence base is somewhat expanded with regard to the potential for CO effects on the developing fetus, uncertainties remain in our understanding of the potential influence of low, ambient CO exposures on conditions existing in the fetus and newborn infant and on maternal-fetal relationships. We additionally recognize that the expanded body of epidemiological evidence includes its own set of uncertainties which complicates its interpretation.

[33] Another criteria pollutant for which the evidence is strengthened through the existence of an internal biomarker is lead (Pb). In the case of Pb, in contrast to that of CO, the epidemiological evidence is focused on associations of Pb-related health effects with measurements of Pb in blood, providing a direct linkage between the pollutant, via the internal biomarker of dose, and the health effects.

2.2.2 Exposure/Risk-based Considerations

Our consideration of the scientific evidence in the current review, as at the time of the last review (summarized in section 2.1.1 above), is informed by results from a quantitative analysis of estimated population exposure and resultant COHb levels. As in our consideration of the evidence in section 2.2.1 above, we have organized the discussion that follows here around a set of key questions to assist us in drawing from the assessment of CO exposure and resultant COHb levels for potentially at-risk populations living in two urban areas under current air quality conditions and conditions simulated to just meet the current CO standards.

Prior to addressing the series of questions below, we provide a summary of key aspects of the assessment, including the study areas and air quality scenarios investigated, modeling tools used, at-risk populations simulated, and COHb benchmark levels of interest. We then consider aspects of the questions beginning with the magnitude of COHb levels estimated in the simulated at-risk populations in response to ambient CO exposure, followed by the key uncertainties associated with our assessment of exposure and dose with regard to drawing conclusions as to the adequacy of the protection afforded by the current CO standards. Lastly, we consider the exposure and dose estimates from the quantitative assessment with regard to the extent to which such estimates may be judged to be important from a public health perspective.

In the assessment conducted for this review, described in detail in the REA and summarized here, we have estimated CO exposure and associated COHb levels in simulated populations in two urban study areas in Denver and Los Angeles, in which current ambient CO concentrations are below the current standards. We selected these areas because: (1) areas of both cities have been included in prior CO NAAQS exposure assessments and thus serve as an important connection with past assessments; (2) historically, they have generally had the highest ambient CO concentrations among urban areas in the U.S.; and (3) Denver is at high altitude and represents an important risk scenario due to the potential increased susceptibility to CO exposure associated with high altitudes. In addition, of 10 urban areas across the U.S. selected for detailed air quality analysis in the ISA and having ambient monitors meeting a 75% completeness criterion, the two study area locations were ranked first (Los Angeles) and second (Denver) regarding the percentage of elderly population within 5, 10, and 15 km of monitor locations, and ranked first (Los Angeles) and fifth (Denver) regarding number of 1- and 8-hour daily maximum CO concentration measurements (ISA, section 3.5.1.1).

Estimates were developed for exposures associated with current "as is" conditions (2006 air quality) and also for higher ambient CO concentrations associated with air quality conditions simulated to just meet the current 8-hour standard,[34] as well as for air quality conditions

[34] As discussed elsewhere, the 8-hour standard is the controlling standard for ambient CO concentrations.

simulated to just meet alternative standards (as discussed in section 2.3.3 below). In considering the adequacy of the current standards, it is important to note that over the last few years, the standards have been met throughout the country with few exceptions.[35] Although we consider it unlikely that air concentrations in many urban areas across the U.S. that are currently well below the current standards would increase to just meet the 8-hour standard, we recognize the potential for CO concentrations in some areas currently below the standard to increase to just meet the standard. Accordingly, we have simulated conditions of increased CO concentrations that just meet the current 8-hour standard in the two study areas. In this scenario, we note there is uncertainty associated with simulating this hypothetical profile of higher CO concentrations that just meet the current 8-hour standard.

To investigate the extent to which the relationship between 1-hour and 8-hour average ambient CO concentrations in the REA-simulated air quality datasets reflect those prevalent in the U.S. today, we have considered ratios of 1-hour design values to 8-hour design values for those datasets in light of the distribution of such ratios for U.S. counties with 2009 CO monitoring data (Appendix C). Under air quality conditions for just meeting the current 8-hour standard and those for just meeting alternative standards these ratios for the two study areas fall well within the 2009 national distribution. More specifically, the ratio of the 1-hour design value to the 8-hour design value for the Los Angeles study area corresponds to approximately the 25th percentile of U.S. counties in 2009 and the ratio for the Denver study area corresponds to approximately the 75th percentile of U.S. counties in 2009. Under "as is" conditions the ratios for these two study areas correspond to approximately the 40th percentile of the 2009 national distribution.

The exposure and dose modeling, presented in detail in the REA, relied on EPA's Air Pollutant Exposure model (APEX4.3), which estimates human exposure using a stochastic, event-based microenvironmental approach (REA, chapter 4). This model has a history of application, evaluation, and progressive model development in estimating human exposure and dose for several NAAQS reviews, including CO, ozone (O_3), NO_2, and sulfur dioxide (SO_2). As mentioned above, we have made major changes to the exposure modeling approach used in the last CO review. The prior review relied on estimated population exposure and dose generated from pNEM, a model that 1) employed a cohort-based approach,[36] 2) relied on a limited set of

[35] As described in section 1.3.3 above, in the most recent period analyzed (2007-2008), all areas of the U.S. met both CO NAAQS. In both of the previous periods (2005-2006 and 2006-2007), one area of the country (Jefferson County, Alabama) did not meet the 8-hour standard. Further, one area of the country (Las Vegas, Nevada) is designated in non-attainment with the CO NAAQS although air quality in that area has met the standards in the past three periods.

[36] When using the cohort approach, each cohort is assumed to contain persons with identical exposures during the specified exposure period. Thus, variability in exposure will be attributed to differences in how the

activity pattern data (approximately 3,600 person-days), 3) used four broadly defined categories to estimate breathing rates (i.e., slow-sleeping, slow-awake, medium, and fast), and 4) implemented a geodesic distance range methodology to approximate workplace commutes (Johnson et al., 1992; US EPA, 1992). Each of these approaches used by pNEM, while appropriate given the data available at that time, would tend to limit the ability to accurately model expected variability in the population exposure and dose distributions.

In contrast, APEX4.3 includes new algorithms to 1) simulate longitudinal activity sequences and exposure profiles for individuals, 2) estimate activity-specific minute-by-minute oxygen consumption and breathing rates, 3) address spatial variability in home and work-tract ambient concentrations for commuters, and 4) estimate event-based microenvironmental concentrations. APEX also uses additional data available from recent activity pattern surveys (CHAD[37] now has about 34,000 person-days of data) and uses the most recent US census data to represent population demographics and home-to-workplace commutes. Modeling the prevalence of exposures or dose in a population of interest (such as individuals with coronary heart disease) has also been enhanced in APEX with the addition of output tables presenting statistics for identified subpopulations and age groups (or lifestages). Further, the current model uses the updated census population demographic data and includes options for selecting algorithms used to estimate microenvironmental concentrations. Each of these new model developments (REA, chapter 4) are designed to allow APEX to better represent human behavior, human physiology, and microenvironmental concentrations and to more accurately estimate variability in CO exposures and COHb levels.

APEX probabilistically generates a sample of hypothetical individuals from an actual population database and simulates each individual's movements through time and space (e.g., indoors at home, inside vehicles) to estimate his or her exposure to a pollutant (REA, chapter 4). Population characteristics are taken into account to represent the demographics of each study area. With regard to age and gender demographics for the two simulated at-risk populations, adults with CAD and adults with heart diseases of any type (HD), we augmented the prevalence estimates provided by the National Health Interview Survey with estimates of undiagnosed ischemia (REA, section 5.5.1). The undiagnosed ischemia estimates were based on two

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cohorts are defined, not necessarily reflecting differences in how individuals might be exposed in a population. In the assessment for the review completed in 1994, a total of 420 cohorts were used to estimate population exposure based on selected demographic information (11 groups using age, gender, work status), residential location, work location, and presence of indoor gas stoves (Johnson, et al., 1992; USEPA, 1992).

[37] CHAD is EPA's Comprehensive Human Activity Database which provides input data for APEX model simulations (REA, sections 4.3 and 4.4).

assumptions: (1) there are 3.5 million persons in U.S. with undiagnosed IHD (AHA, 2003) and (2) persons with undiagnosed IHD are distributed within the population in the same manner as persons with diagnosed IHD (REA, section 5.5.1). Based on exposure concentrations, minute-by-minute activity levels, and physiological characteristics of the simulated person (see REA, chapters 4 and 5), APEX estimates the level of COHb in the blood at the end of each hour based on a nonlinear solution to the Coburn-Forster-Kane equation (REA, section 4.4.7).

APEX simulations performed for this review have focused on exposures to ambient CO occurring in eight microenvironments, absent any contribution to microenvironment concentrations from indoor (nonambient) CO sources, although, where present, indoor sources, including gas stoves, attached garages and tobacco smoke, can also be important contributors to total CO exposure (ISA, section 3.6.1). Some assessments performed previously have included modeling simulations both with and without certain indoor sources, and these assessments provide context for the assessment of ambient CO exposure and dose. For example, a 2000 exposure/dose assessment indicated that the impact of such sources can be substantial with regard to the portion of the at-risk population experiencing higher exposures and COHb levels (Johnson et al., 2000). In the last review it was noted that while these indoor sources were shown to contribute to total CO exposure they would not be effectively mitigated by setting more stringent ambient air quality standards (59 FR 38914). While we are limited with regard to information regarding CO emissions from indoor sources today and how they may differ from the time of the 2000 assessment, we note that ambient contributions have notably declined, and indoor source contributions from some sources may also have declined. Thus, we have no firm basis to conclude a different role for indoor sources today with regard to contribution to population CO exposure and COHb levels.

We note that the absence of indoor (nonambient) sources[38] in the model simulations for this review is expected to result in somewhat higher estimates of the contribution of short-duration increases in ambient CO exposure to COHb levels than is likely to be the case in situations where the presence of nonambient sources contributes to higher baseline COHb levels in the exposed individuals. The amount by which the ambient contribution estimates might differ is influenced by the magnitude of nonambient-source exposures and associated baseline COHb levels. One reason for this is that in the presence of indoor sources, baseline COHb levels (i.e., COHb prior to a short-duration exposure event) will be higher for a given population group than COHb levels for that group arising solely from endogenous CO in the absence of any exposure, which is the "baseline" for the REA estimates of ambient contribution to COHb (REA,

[38] Nonambient sources of CO exposure can include gas stoves, space heaters, attached garages and tobacco smoke (ISA, pp. 3-76, 3-83).

appendix B.6). As CO uptake depends in part on the amount of CO already present in the blood (and the blood-air CO concentration gradient), in general, a higher baseline COHb, with all other variables unchanged, will lead to relatively lesser uptake of CO from short-duration exposures (ISA, section 4.3; AQCD, section 5.2). Additionally, as is indicated by the REA estimates, the attainment of a particular dose level is driven largely by short-term (and often high concentration) exposure events. This is because of the relatively rapid uptake of CO into a person's blood, as demonstrated by the pattern in the REA time-series of ambient concentrations, microenvironmental exposures, and COHb levels (REA, Appendix B, Figure B-2). For example the time lag for response of an individual's COHb levels to variable ambient CO (and hence exposure) concentrations may be only a few hours.

As discussed in the previous section (Section 2.2.1), people with cardiovascular disease are the population of primary focus in this review. More specifically, coronary artery disease, also known as coronary heart disease is the "most important susceptibility characteristic for increased risk due to CO exposure" (ISA, p. 2-11). Controlled human exposure studies have provided quantitative COHb dose-response information for this specific population with regard to effects on markers of myocardial ischemia. In identifying COHb levels of interest for at-risk populations simulated in the REA, we have given primary focus to the multi-laboratory study in which COHb was analyzed by the more accurate GC method (Allred et al., 1989a, 1989b, 1991) discussed in section 2.2.1 above. Based on the strength of the evidence and the availability of quantitative information from these controlled human exposure studies, the REA focuses on estimates of the percent of the simulated at-risk populations expected to experience one or more occurrences of daily maximum end-of-hour COHb levels of interest. Further, based on the current evidence with regard to quantitative information of COHb levels and association with specific health effects, the at-risk populations simulated in the quantitative assessment were (1) adults with CHD (also known as ischemic heart disease IHD or CAD), both diagnosed and undiagnosed, and (2) adults with any heart diseases, including undiagnosed ischemia.[39] Evidence characterizing the nature of specific health effects of CO in other populations is limited and does not include specific COHb levels related to health effects in those groups. As a result, the quantitative assessment does not develop separate quantitative dose estimates for populations other than those with CAD or HD.

The REA developed COHb estimates for the simulated at-risk populations with attention to both COHb in absolute terms and in terms of the contribution to absolute levels associated with ambient CO exposures. For the REA results described below, absolute COHb refers to

[39] As described in section1.2 above, this is the same population group that was the focus of the CO NAAQS exposure/dose assessments conducted previously (e.g., USEPA, 1992; Johnson et al., 2000).

estimates of COHb levels resulting from endogenously produced CO and exposure to ambient CO, in the absence of any nonambient sources. The additional REA estimates of ambient CO exposure contribution to COHb levels are calculated by subtracting COHb estimates obtained in the absence of CO exposure - i.e., that due to endogenous CO production alone (see REA, appendix B.6) - from the corresponding end-of-hour absolute COHb estimates for each simulated individual. The maximum end-of-hour ambient contributions across the simulated year are considered, in addition to the maximum absolute end-of hour COHb levels. In considering these two types of COHb estimates, we note that the lack of nonambient sources is likely to have resulted in lower baseline COHb levels (levels of COHb prior to an individual's encounter with elevated ambient exposure concentrations) than if the individuals' exposure history included additional CO sources. These REA "baseline" COHb levels would also be expected to be lower than the initial, pre-exposure, COHb levels of subjects in the controlled exposure studies.[40] As mentioned above, such a lower baseline contributes to a relatively greater COHb response to short-duration high-concentration exposures.

In our consideration of the REA dose estimates with regard to consideration of the adequacy of the current standards, we focus on estimates of the portion of the simulated at-risk populations estimated to experience daily maximum end-of-hour absolute COHb levels above identified benchmark levels, and also consider the population estimates of daily maximum ambient contribution to end-of-hour COHb levels. The benchmark levels, described below, assist in our interpretation of the COHb estimates in light of the evidence for ischemia-related effects in CAD patients in the controlled human exposure studies.

For purposes of considering estimates of absolute COHb, we identified benchmark levels of 1.5%, 2.0%, 2.5% and 3% COHb. This range includes the range of COHb levels identified as levels of concern in the review completed in 1994 (2.0 to 2.9%) and the level given particular focus (2.1%) at that time, as described in section 2.1.1 above (USEPA, 1992; 59 FR 48914). Selection of this range of benchmark levels is based on consideration of the evidence from controlled human clinical studies of CAD patients (discussed in section 2.2.1 above), with the lower end of the range extending below the lowest mean COHb level resulting from controlled exposure to CO in the clinical evidence (e.g., 2.0% post-exercise in Allred et al., 1989b). The extension of this range reflects a number of considerations, including: (1) comments from the CASAC CO panel on the draft Scope and Methods Plan (Brain, 2009); (2) consideration of the

[40] REA estimates of endogenously formed COHb averaged about 0.3% across the simulated populations, with slightly higher levels in the higher altitude Denver study area (REA, pp. B-21 to B-22). Levels in the Denver study population ranged from 0.1 to 1.1 % COHb, with an average of 0.31%, while levels for Los Angeles ranged from 0.1 to 0.7% with an average of 0.27% COHb. Initial, pre-exposure COHb levels in the subjects of the Allred et al. study (1989b), which reflect the subjects pre-study exposure history as well as endogenous CO formation, ranged from 0.2 to 1.1%, averaging about 0.6% COHb.

uncertainties regarding the actual COHb levels experienced in the controlled human exposure studies; (3) that these studies did not include individuals with most severe cardiovascular disease[41]; (4) the lack of studies that have evaluated effects of experimentally controlled short-term CO exposures resulting in mean COHb levels below 2.0-2.4%; and (5) the lack of evidence of a threshold at the increased COHb levels evaluated. We note that CASAC comments on the first draft REA recommended the addition of a benchmark at 1.0% COHb and results are presented for this COHb level in the REA. Given that this level overlaps with the upper part of the range of endogenous levels in healthy individuals as characterized in the ISA (ISA, p. 2-6), and is within the upper part of the range of baseline COHb levels in the study by Allred et al (1989b, Appendix B), however, we considered that it may not be appropriate to place weight on it as a benchmark level and accordingly have not focused on interpreting absolute COHb estimates at and below this level in the discussion below. Additionally we note the REA estimates indicating that, in the absence of CO exposure, approximately 0.5% to 2% of the simulated at-risk populations in the two study areas were estimated to experience a single daily maximum end-of-hour COHb level, arising solely from endogenous CO production, at or above 1% (REA, Appendix B, Figure B-3).

We also consider the evidence from controlled human exposure studies, discussed in section 2.2.1 above, in our interpretation of the REA estimates of maximum ambient exposure contributions to end-of-hour COHb levels (described in sections 4.4.7 and 5.10.3 of the REA). As discussed above, the study by Allred et al (1989a, 1989b, 1991) observed reduced time to exercise-induced angina and ST-segment change in groups of subjects with pre-existing CAD for which controlled CO exposures increased their COHb levels by on average 1.4-1.7% and 3.3-4.0% COHb from initial COHb levels of on average 0.6% COHb (ISA, section 5.2.4; Allred et al., 1989a, 1989b, 1991). The study reported a dose-response relationship in terms of time reduction per 1% increase in COHb concentration based on analysis of the full data set across both exposure groups. For purposes of the discussion in this document, we have presented the percentage of the simulated at-risk populations estimated to experience maximum ambient contribution to end-of-hour COHb levels above and below a range of levels extending from 1.4 to 2.0%. In considering REA results for this metric, we recognize distinctions between the REA "baseline" (arising from prior ambient exposure and endogenous CO production) and the pre-exposure COHb levels in the clinical study (arising from ambient and nonambient exposure history, as well as from endogenous CO production). We also note the impact of "baseline"

[41] Although the CAD patients evaluated in the controlled human exposure study by Allred et al. (1989a, 1989b, 1991) are not necessarily representative of the most sensitive population, the level of disease in these individuals ranged from moderate to severe, with the majority either having a history of myocardial infarction or having ≥ 70% occlusion of one or more of the coronary arteries (ISA, p. 5-43).

COHb levels on COHb levels occurring in response to short ambient CO exposure events such as those simulated in the REA as discussed above.

- **What is the magnitude of at-risk population COHb levels estimated to occur in areas simulated to just meet the current CO standards? What portion of the at-risk population is estimated to experience maximum COHb levels above levels of potential health concern?**

In addressing this question, we consider the population COHb estimates provided by the REA simulations of exposure to ambient CO (REA, section 6.2). As in the last review, we recognize that indoor sources of CO can be important determinants of population exposures to CO and to population distributions of daily maximum COHb levels, and that for some portions of the population, these sources may dominate CO exposures and related maximum COHb levels. We additionally take note of the conclusions drawn in the previous review that the contribution of indoor sources to individual exposures and associated COHb levels cannot be effectively mitigated by ambient air quality standards (e.g., 59 FR 38914) and so focus here on COHb levels resulting from ambient CO exposures. In so doing, however, we also recognize as noted above, that simulations focused solely on exposures associated with ambient CO may overestimate the response of COHb levels to short-duration ambient exposures (the ambient contribution) as pre-exposure baseline COHb levels will necessarily not reflect the contribution of both nonambient and ambient sources.

In considering the REA estimates for current or "as is" air quality conditions and conditions simulated to just meet the current 8-hour standard, we particularly focus on the extent to which the current standards provide protection to the simulated at-risk population from COHb levels of potential concern, by comparing the estimated levels in the population to the benchmarks described above. As described above, the REA presents two sets of COHb estimates: the first set of absolute estimates reflect the impact of ambient CO exposures in the absence of exposure to nonambient CO, but in the presence of endogenous CO production, while the second set are estimates of the portion of absolute COHb estimated to occur in response to the simulated ambient CO exposures, i.e., after subtraction of COHb resulting from endogenous CO production (REA, sections 4.4.7 and 5.10.3). In describing the REA results here, we draw from exposure and dose estimates for both the HD and CHD populations (REA, section 6.2), recognizing that, in terms of percentages of persons exposed and experiencing daily maximum end-of-hour COHb at or above specific levels, the results are similar for the two simulated at-risk populations (HD and CHD). We note that, in terms of absolute numbers of persons, the results differ due to differences in the size of the two populations.

First, we consider the absolute COHb results, with regard to the percentage of simulated populations experiencing at least one day with an end-of hour COHb level above selected

benchmarks (Table 2-4). Another dimension of the analysis, presented in Table 2-5, is the percentage of simulated populations experiencing multiple days in the simulated year with an end-of-hour COHb level above the same benchmarks. These two dimensions of the dose estimates are combined in the metric, person-days, which is presented in Tables 6-15, 6-16, 6-18 and 6-19 of the REA. The metric, person-days, was the focus of exposure/dose considerations in the last review for which a previous version of the exposure/dose model was used (59 FR 38914; USEPA, 1992).[42] The person-days metric is a common cumulative measure of population exposure/dose that simultaneously takes into account both numbers of people affected and numbers of times affected.

Under current air quality conditions, the absolute COHb estimates are appreciably lower than those for conditions of higher ambient CO concentrations in which the current 8-hour standard is just met (Table 2-4). Under "as is" (2006) conditions in the two study areas, no person in the simulated at-risk populations is estimated to experience any days in the year with end-of-hour COHb concentrations at or above 3% COHb, and less than 0.1 % of the simulated at-risk populations are estimated to experience at least one end-of-hour COHb concentration at or above 2% (Table 2-4). Slightly higher percentages of the simulated populations (1-2%) are estimated to experience a single occurrence of daily maximum COHb at or above 1.5%.

Under conditions with higher ambient CO concentrations simulated to just meet the current 8-hour standard, the portion of the simulated at-risk populations estimated to experience daily maximum end-of-hour COHb levels at or above benchmarks is greater in both study areas, with somewhat higher percentages for the Denver study area population (Table 2-4). In both study areas, nonetheless, less than 1% of the simulated at-risk populations is estimated to experience a single day with a maximum end-of hour COHb level at or above 3% (Table 2-4) and no person is estimated to experience more than one such day in a year (Table 2-5). Further, less than 0.1% of simulated populations in either study area is estimated to experience a single day with maximum end-of-hour COHb at or above 4%. A difference between the study areas is more evident for lower benchmarks, with less than 5% of the simulated at-risk population in the Denver study area and less than 1% of the corresponding population in the Los Angeles study area estimated to experience any days with a maximum end-of-hour COHb level at or above 2% (Table 2-4). Appreciably smaller percentages of the simulated at-risk population were estimated to experience more than one day with such levels (Table 2-5). For example, less than 1.5% of the population is estimated to experience more than one day in a year with a maximum COHb level at or above 2.0%, and less than 0.1% are estimated to experience six or more days such

[42] As described above, pNEM, the model used in the last review, employed a cohort-based approach from which person-days were the exposure and dose metrics (USEPA, 1992; Johnson et al., 1992).

days in a year. Additionally, consistent with the findings of the assessment performed for the review completed in 1994, less than 0.1% of person-days for the simulated at-risk populations were estimated to have end-of-hour COHb levels at or above 2% COHb (REA, Tables 6-18 and 6-19).

Table 2-4. Portion of simulated HD populations with at least one daily maximum end-of-hour COHb level (absolute) at or above indicated levels under air quality conditions simulated to just meet the current standard and "as is" conditions.

Daily Maximum End-of-hour COHb (Absolute)	Percentage (%) of Simulated HD Population[A]			
	Just Meeting Current 8-hour Standard		"As is" (2006) Conditions	
	Los Angeles (1-hr DV = 11.8 ppm)	Denver (1-hr DV = 16.2 ppm)	Los Angeles (8-hr DV = 5.6 ppm) (1-hr DV = 8.2 ppm)	Denver (8-hr DV = 3.1 ppm) (1-hr DV = 4.6 ppm)
≥ 4.0%	0	<0.1[B]	0	0
≥ 3.0%	< 0.1[B]	0.3		
≥ 2.5%	< 0.1[B]	0.9		
≥ 2.0%	0.6	4.5	< 0.1[B]	< 0.1[B]
≥ 1.5%	5.0	24.5	1.6	1.2

[A] Drawn from Tables 6-15 through 6-19 of the REA.
[B] < 0.1" is used to represent nonzero estimates below 0.1%.
Abbreviations: hr = hour, DV = Design Value

Table 2-5. Portion of simulated CHD population with multiple days of maximum end-of-hour COHb levels (absolute) at or above the indicated levels under air quality conditions simulated to just meet the current standard and "as is" conditions.

Maximum End-of-hour COHb Level (Absolute)	Percentage (%) of Simulated CHD Population[A]											
	Just Meeting Current 8-hour Standard						"As is" (2006) Conditions					
	Los Angeles (1-hr DV = 11.8 ppm)			Denver (1-hr DV = 16.2 ppm)			Los Angeles (8-hr DV = 5.6 ppm) (1-hr DV = 8.2 ppm)			Denver (8-hr DV = 3.1 ppm) (1-hr DV = 4.6 ppm)		
	≥2 days	≥4 days	≥6 days	≥2 days	≥4 days	≥6 days	≥2 days	≥4 days	≥6 days	≥2 days	≥4 days	≥6 days
≧ 3.0%	0	0	0	0	0	0	0	0	0	0	0	0
≧ 2.5%	< 0.1[B]	0	0	< 0.1[B]	0	0	0	0	0	0	0	0
≧ 2.0%	0.2	< 0.1[B]	< 0.1[B]	1.4	0.2	< 0.1	0	0	0	< 0.1[B]	< 0.1[B]	< 0.1[B]
≧ 1.5%	2.2	0.7	0.5	11.2	5.0	3.3	0.5	0.2	0.1	0.7	0.5	0.4

[A] These estimates are drawn mainly from Figures 6-5 and 6-6 of the REA and represent the percentage of persons experiencing greater than or equal to 2, 4, or 6 days with a maximum end-of-hour COHb (absolute) at or above the selected level.
[B] < 0.1 is used to represent nonzero estimates below 0.1%.

As described above, the REA also presented estimates of the portion of the absolute COHb levels occurring in response to the simulated ambient CO exposures (i.e., that not derived from endogenous CO production). The REA refers to these estimates as the ambient CO contribution to (absolute) COHb. Under conditions just meeting the current 8-hour standard in the two study areas, less than 3 percent of the simulated at-risk populations was estimated to experience a daily maximum ambient CO contribution to end-of-hour COHb level above 2% (Table 2-6), with less than 13 percent of either population experiencing maximum ambient contributions at or above 1.4% COHb. As observed with the absolute COHb estimates under conditions just meeting the standard, the results for the Denver study area included larger percentages of the population above specified levels than those for the Los Angeles study area, reflecting the study area difference in 1-hour peak concentrations (Table 2-6). Although estimates of population percentages for multiple occurrences are not available for the ambient contribution estimates, it is expected that similar to those for absolute COHb, they would be appreciably lower than those shown here for at least one occurrence. Additionally, as mentioned above, somewhat lower ambient contribution estimates might be expected if other (nonambient) CO sources were present in the simulations.

Table 2-6. Portion of simulated CHD population with at least one daily maximum ambient contribution to end-of-hour COHb at or above the indicated levels under air quality conditions simulated to just meet the current standard and "as is" conditions.

Maximum Ambient Contribution to End-of-hour COHb	Percentage (%) of Simulated CHD Population[A]			
	Just Meeting Current 8-hour Standard		"As is" (2006) Conditions	
	Los Angeles (1-hr DV = 11.8 ppm)	Denver (1-hr DV = 16.2 ppm)	Los Angeles (8-hr DV = 5.6 ppm) (1-hr DV = 8.2 ppm)	Denver (8-hr DV = 3.1 ppm) (1-hr DV = 4.6 ppm)
≧ 2.0%	0.5	2.7	0	0
≧ 1.8%	0.8	3.4		
≧ 1.6%	1.3	6.8	0.5	0.2
≧ 1.4%	2.0	12.8		

[A] Drawn from Tables 6-17 and 6-20 of the REA.
Abbreviations: hr = hour, DV = Design Value

In considering the estimates presented here of population occurrences of daily maximum COHb levels for REA simulations under conditions just meeting the current 8-hour standard, we note that an important contributing factor to the higher percentages estimated for the Denver study area population is the occurrence of higher 1-hour peak ambient CO concentrations and consequent higher CO exposures than occur in the corresponding Los Angeles study area simulation (REA, section 6.1.2, Tables 6-7 and 6-10). The difference in the peak 1-hour ambient concentrations is illustrated by the higher 1-hour design values noted for Denver as compared to Los Angeles in Table 2-4 (16.2 ppm versus 11.8 ppm). This difference, particularly at the upper percentiles of the air quality distribution, is likely driving the higher population percentages estimated to experience higher 1-hour and 8-hour exposures in the Denver study area as compared to Los Angeles (REA, Tables 6-7 and 6-10).[43] The situation is largely reversed under "as is" conditions, where the Los Angeles study area has generally higher 1-hour and 8-hour ambient CO concentrations as illustrated by the design values for alternative standard forms in Table 2-7 below (as well as Tables 3-1 to 3-6, 5-14 and 5-16 of the REA), and Los Angeles also has higher percentages of people estimated to be exposed to the higher exposure concentrations (REA, Tables 6-1 and 6-4). Thus, we recognize the impact on daily maximum COHb levels of

[43] Other factors that contribute less to differences in COHb estimates between the two study areas, include altitude, which slightly enhances endogenous CO and COHb formation and can enhance COHb formation induced by CO exposure under resting conditions (ISA, p. 4-19), and design aspects of the study areas with regard to spatial variation in monitor CO concentrations and population density near these monitors (REA, section 7.2.2.1).

1-hour ambient concentrations separate from the impact of 8-hour average concentrations, and take note of this in considering the REA results with regard to the adequacy of the 1-hour standard. Taken together, the REA results indicate occurrences of COHb levels above the benchmarks considered here that are associated with 1-hour ambient concentrations that are not controlled by the current suite of standards.

- **What are the key uncertainties associated with our exposure and dose estimates, including those of particular significance with regard to drawing conclusions as to the adequacy of the protection afforded by the current CO standards?**

In considering the uncertainties associated with the quantitative estimates of exposure and dose from the REA, we relied on an approach intended to identify and compare the relative impact that important sources of uncertainty may have on the estimated potential health effect endpoints (i.e., estimates of the maximum end-of-hour COHb levels in the simulated at-risk population). The generally qualitative approach used (as described in section 7.2 of the REA) was developed using guidelines outlining how to conduct a qualitative uncertainty characterization (WHO, 2008) and applied in the most recent NO_2 (USEPA, 2008b) and SO_2 NAAQS reviews (USEPA, 2009). We employed this approach given the extremely limited data available to inform probabilistic analyses. The qualitative approach used varies from that of WHO (2008) in that a greater focus of the characterization performed was placed on evaluating the direction and the magnitude of the uncertainty; that is, qualitatively rating how the source of uncertainty, in the presence of alternative information, may affect the estimated exposures and health risk results. In addition and consistent with the WHO (2008) guidance, we discuss the uncertainty in the knowledge-base (e.g., the accuracy of the data used, acknowledgement of data gaps) and decisions made where possible (e.g., selection of particular model forms), though qualitative ratings were assigned only to uncertainty regarding the knowledge-base.

In the characterization of uncertainty for the current analysis, we identified and evaluated sixteen separate sources of uncertainty associated with four main components of the assessment:

- Ambient monitor CO concentrations
  - database quality, spatial and temporal representation, zero concentration frequency, and missing data substitution;
- Adjustment of air quality to simulate just meeting the current and potential alternative standards
  - historical data used, proportional approach used;
- APEX inputs and algorithms
  - population database, activity pattern database, longitudinal profile algorithm, meteorological data, microenvironmental algorithm and input data, commuting algorithm, prevalence rates for at-risk populations, physiological factors; and,

- Potential health effects benchmark levels for the simulated at-risk population.

Further, an additional area of uncertainty not directly evaluated is the contribution of ambient CO to COHb levels in the presence of nonambient exposures, although as discussed above, previous assessments indicated that, where present, the contribution of nonambient exposures to COHb can be substantial in comparison to ambient exposures.

By comparing judgments made regarding the magnitude and direction of influence the identified sources have on estimated exposure concentrations and dose levels and the existing uncertainties in the knowledge-base, we identified seven sources (i.e., the spatial and temporal representation of ambient monitoring data, historical data used in representing alternative air quality scenarios, activity pattern database, longitudinal profile algorithm, microenvironmental algorithm and input data, and physiological factors) that remain as the most important uncertainties in this assessment. Taking into consideration improvements in the model algorithms and data since the last review, and having identified and characterized these uncertainties here, we conclude that the estimates associated with the current analysis, at a minimum, better reflect the full distribution of exposures and dose as compared to results from the 1992 analysis. We note, however, potentially greater uncertainty remaining in our characterization of the upper and lower percentiles of the distribution of population exposures and COHb dose levels relative to that of other portions of the respective distribution. When considering the overall quality of the current exposure modeling approach, the algorithms, and input data used, alongside the identified limitations and uncertainties, we conclude that the quantitative assessment provides reasonable estimates of CO exposure and COHb dose for the simulated population the assessment is intended to represent (i.e., the population residing within the urban core of each study area).

We additionally note the impact on the REA dose estimates for ambient CO contribution to COHb of the lack of nonambient sources in the model simulations. This aspect of the assessment design may contribute to higher estimates of the contribution of short-duration ambient CO exposures to total COHb than would result from simulations that include the range of commonly encountered CO sources beyond just those contributing to ambient air CO concentrations. Although the specific quantitative impact of this on estimates of population percentages discussed in this document is unknown, consideration of COHb estimates from the 2000 assessment indicates a potential for the inclusion of nonambient sources to appreciably affect absolute COHb (REA, section 6.3) and accordingly implies the potential, where present, for an impact on overall ambient contribution to a person's COHb level.

- **To what extent are the estimates of at-risk population COHb levels under conditions just meeting the current CO standards important from a public health perspective?**

In considering the public health implications of the quantitative dose estimates, we first consider the effects identified by the evidence to be associated with COHb levels of a magnitude of the daily maximum end-of-hour levels estimated in the REA for conditions just meeting the current suite of standards. For example, as a result of ambient CO exposures occurring under air quality conditions adjusted to just meet the current standard, the REA estimates that 0.6 percent of the Los Angeles and 4.2 percent of the Denver study CHD populations may experience an occurrence of a daily maximum end-of-hour COHb level at or above 2% COHb, the low end of the range of average COHb levels experienced by the lower controlled exposure group in the study by Allred et al. (1989a, 1989b, 1991), while 0.2 and 1.4 percent, respectively, of the populations are estimated to experience more than one such occurrence. Additionally, less than 0.1 percent of the simulated populations in either study area are estimated to experience a COHb level similar to the higher controlled exposure group (4% COHb).

As discussed in section 2.2.1 above, the study by Allred et al., (1989a, 1989b, 1991) indicates that increases in blood COHb in response to 1-hour CO exposures on the order of 50 to 200 ppm (and higher) produce evidence of myocardial ischemia in CAD patients with reproducible exercise-induced angina. At a study group average COHb level of 2%, the statistically significant reduction in the time to exercise-induced markers of myocardial ischemia (angina and ECG ST-segment change) in CAD patients was 4-5% on average (approximately 30 seconds), with larger reductions observed at the 4% COHb level. In discussing public health implications of the observed responses, the study authors noted that the response observed at the higher COHb level (~4%) were similar to that considered clinically significant when evaluating medications to treat angina from coronary artery disease. The independent review panel for the study further noted that frequent encounters in "everyday life" with increased COHb levels on the order of those tested in the study might be expected to limit activity and affect quality of life (Allred et al., 1989b, pp. 38, 92-94; 1991 AQCD, p. 10-35).

In the review completed in 1994, the body of evidence that demonstrated cardiovascular effects in CAD patients exposed to CO was given primary consideration, with the Administrator recognizing the findings of decreased time to onset of ECG change and angina pain as the "health effects of greatest concern, which clearly have been associated with CO exposures at levels observed [at that time] in the ambient air" (59 FR 38913). We additionally note the dose-response relationship observed for COHb resulting from brief, elevated CO exposures in persons with pre-existing CAD, with no evidence of threshold, as discussed in section 2.2.1 above (ISA,

section 5.2.4; Allred et al., 1989a, 1989b, 1991).[44] In the current review of the evidence, the ISA describes the physiological significance of the changes at the lowest tested dose level (~2% COHb, Allred et al 1989b) as unclear, additionally noting that variability in severity of disease among individuals with CAD is likely to influence the critical level of COHb which leads to adverse cardiovascular effects (ISA, p. 2-6). In the 1994 review decision, less significance was ascribed to the effects at the lowest COHb levels assessed in short-term CO exposure studies than effects associated with higher COHb levels (59 FR 38913), with additional weight given to those occurring at dose levels at or above 2.9%, which were described at that time as "effects … of clear concern" (59 FR 38914).

In considering public health implications of the REA estimates, we also consider the size of the at-risk populations simulated. The population with CAD (including undiagnosed cases) is well recognized as susceptible to increased risk of CO-induced health effects (ISA, sections 5.7.1.1 and 5.7.8). The 2007 estimate from the National Health Interview Survey (NHIS) performed by the U.S. Centers for Disease Control of the size of the U.S. population with coronary heart disease, angina pectoris (cardiac chest pain) or who have experienced a heart attack (ISA, Table 5-26) is 13.7 million people (ISA, pp.5-117). Further, there are estimated to be three to four million additional people with silent ischemia or undiagnosed IHD (AHA, 2003). In combination, this represents a large population represented by the REA analyses and for which the COHb benchmarks described above (based on studies of CAD patients) are relevant, that is, more susceptible to ambient CO exposure when compared to the general population (ISA, section 5.7).

In addition to the population with diagnosed and undiagnosed CAD, the REA also simulated ambient CO exposures for the larger HD population, which may also be at increased risk of CO-induced health effects (ISA, section 2.6.1). Within this broader group, implications of CO exposures are more significant for those persons for whom their disease state affects their ability to compensate for the hypoxia-related effects of CO (ISA, section 4.4.4). The NHIS estimates for 2007 indicate there is a total of approximately 25 million people with heart disease of any type (ISA, Table 5-26). Accordingly, while the REA estimates in terms of percentages of

[44] The dose-response relationship was derived as the average of the regressions of the individual study subject data for changes in time to onset of the monitored measures of ischemia across their range of COHb levels (from their baseline COHb to their two higher COHb levels resulting from the two experimental CO exposures). The clean-air exposure, post-exercise (baseline) COHb levels in the individual study subjects ranged from 0.2% to 1.1%, their post-exercise COHb levels for the lower experimental CO exposure ranged from 1.0 to 3.0% and their post-exercise COHb levels for the higher experimental CO exposure ranged from 2.3 to 5.1%. The dose-response analysis indicated decreases of roughly 1.9% in time to exercise-induced angina and 3.9% in time to exercise-induced ST-segment change per 1% increase in COHb concentration (Allred et al., 1989b).

the CAD and HD populations above COHb levels of interest are similar, the estimates in terms of number of individuals are higher for the larger HD population.

Other populations potentially susceptible to the effects of CO include people with chronic obstructive pulmonary disease, diabetes and anemia, as well as older adults and fetuses during critical phases of development (as discussed in section 2.2.1 above). In considering potential impacts on such populations, we recognize that the evidence is limited or lacking with regard to effects of CO at ambient levels, and associated exposures and COHb levels, while providing no indication of susceptibility to ambient CO greater than that of CHD and HD populations.

In summary, while we note the substantial size of the population of individuals with CHD or other heart diseases in the U.S., we recognize that the REA results for conditions just meeting the current standards indicate a very small portion of this population that might be expected to experience more than one occurrence of COHb above 2%, with less than 0.1% of this population expected to experience such a level on as many as six days in a year or a single occurrence as high as 4%, and 0% of the population expected to experience more than one occurrence above 4% COHb. In light of the implications of the health evidence discussed above, the public health significance of these REA results and conclusions regarding the extent to which they are important from a public health perspective depends in part on public health policy judgments about the public health significance of effects at the COHb benchmark levels considered and judgments about the level of public health protection with an adequate margin of safety.

2.2.3 CASAC Advice

In our consideration of the adequacy of the current standards, in addition to the evidence- and risk/exposure-based information discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the ISA, the REA, and the earlier draft of this document, as well as comments from the public on earlier drafts of this document and the REA.[45]

The few public comments received on this review to date that have addressed adequacy of the current standards conveyed the view that the current standards are adequate. In support of this view, these commenters disagreed with the REA estimates of in-vehicle exposure concentrations and argued that little weight should be given to the epidemiological studies.

In their comments on the draft PA, the CASAC CO Panel stated overall agreement with staff's conclusion that the body of evidence and the quantitative exposure and risk assessment provide support for retaining or revising the current 8-hour standard. They additionally,

[45] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meetings held in conjunction with CASAC's review of the earlier draft of this document, of drafts of the REA, and of drafts of the ISA.

however, expressed a "preference" for a lower standard and stated that "[i]f the epidemiological evidence is given additional weight, the conclusion could be drawn that health effects are occurring at levels below the current standard, which would support the tightening of the current standard." Taking this into account, the Panel further advised that "revisions that result in lowering the standard should be considered" (Brain and Samet, 2010b).

2.2.4 Staff Conclusions on Adequacy of the Current Standards

In considering the adequacy of the current standards, staff gives great weight to the long-standing body of evidence for CO, augmented in some aspects since the last review, that has established: the common mechanism of CO health effects as involving binding to reduced iron in heme proteins and the alteration of their functioning; the important role of hypoxia (reduced oxygen availability) induced by increased COHb blood levels in eliciting health effects; the use of COHb as the bioindicator and dose metric for evaluating CO exposure and potential for health effects; and, people with cardiovascular disease as a key population at risk from low CO exposures (ISA; 2000 AQCD; 1991 AQCD). We additionally recognize the expansion of the epidemiological evidence that provides logical coherence with our conclusions regarding cardiovascular disease-related susceptibility (ISA, section 5.2.1).

As at the time of the last review, we give weight to COHb estimates developed from modeling exposures to ambient CO under conditions simulated to just meet the current, controlling, 8-hour standard. We note the different modeling approach in the last review for which the results metric was person-days, and we note similarities between results of the current and past modeling in terms of that metric. In the current modeling, additional metrics for percentage of population with at least one or multiple occurrences of COHb levels of interest are considered. These absolute COHb estimates and the incremental contribution to them from ambient CO exposures are considered in light of the evidence from controlled human exposure studies, while recognizing distinctions between the measurements in those studies and the REA estimates that would be likely to affect the response of COHb levels to elevated ambient CO exposure events. These estimates, which vary between the two study areas, indicate that 0.5 to 4.5% of the simulated at-risk populations may experience at least one daily maximum COHb level of a magnitude (at or above 2% COHb) that has been associated with shortened time to exercise-induced appearance of markers of myocardial ischemia subsequent to 1-hour elevations in CO exposure, and larger portions of the population would experience lower COHb levels, below those which have been specifically addressed in the health effects evidence. In considering the public health significance of such estimates, while recognizing uncertainty in the health significance associated with the smaller effects observed at the lowest studied COHb levels, we note the substantial size of the at-risk population in the U.S. and the existence of other

potentially susceptible populations for which evidence pertaining to effects resulting from CO exposures associated with ambient concentrations is much more limited or lacking. We also take note of the noticeable variation in the REA exposure and dose estimates between the two study areas under conditions simulated to just meet the current 8-hour standard, and its relationship to differences in hourly ambient CO concentrations which fall well below conditions controlled by the 1-hour standard.

The extent to which the current standards are judged to be adequate depends on a variety of factors inclusive of science policy judgments and public health policy judgments. These factors include public health policy judgments concerning the appropriate COHb benchmark levels on which to place weight, as well as judgments on the public health significance of the effects that have been observed at the lowest levels evaluated, particularly with regard to relatively rare occurrences. The factors relevant to judging the adequacy of the standards also include consideration of the uncertainty associated with interpretation of the epidemiological evidence as providing information on ambient CO as distinct from information on the mixture of pollutants associated with traffic, and, given this uncertainty, the weight to place on interpretations of ambient CO concentrations for the few epidemiological studies available for air quality conditions that did not exceed the current standards. And, lastly these factors include the interpretation of, and decisions as to the weight to place on, the results of the exposure assessment for the two areas studied relative to each other and to results from past assessments, recognizing the implementation of an improved modeling approach and new input data, as well as distinctions between the REA simulations and resulting COHb estimates and the response of COHb levels to experimental CO exposure as recorded in the controlled human exposure studies.

We draw conclusions with regard to the adequacy of the current standards from both the evidence and from the exposure and dose assessment, taking into consideration related information, limitations and uncertainties recognized above. We conclude that the combined consideration of the body of evidence and the quantitative exposure and dose estimates provide support for a suite of standards at least as protective as the current suite. Further, we recognize that conclusions regarding the adequacy of the current standards depend in part on public health policy judgments identified above and judgments about the level of public health protection with an adequate margin of safety. We additionally note the influence that hourly ambient CO concentrations well below the current 1-hour standard may have on ambient CO exposures and resultant COHb levels under conditions just meeting the 8-hour standard, as indicated by the REA results. We conclude that the REA results indicate the potential for the current controlling 8-hour standard to allow the occurrence of 1-hour ambient concentrations that contribute to population estimates of daily maximum COHb levels, that depending on public health judgments in the areas identified above, may be considered to call into question the adequacy of the 1-hour

standard and support consideration of revisions of that standard in order to reduce the likelihood of such occurrences in areas just meeting the 8-hour standard. Thus, we conclude that the combined consideration of the evidence and quantitative estimates described above may be viewed as providing support for either retaining or revising the current suite of standards.

Staff's conclusions with regard to elements of revised primary standards for CO that may be appropriate to consider are discussed in the following sections.

2.3 CONSIDERATION OF ALTERNATIVE STANDARDS

To the extent that the information available in this review suggests that revision of the current standards is appropriate to consider, staff has considered whether the available body of evidence supports consideration of options that are different from the current standards, as articulated by the following overarching question:

- **To what extent does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and REA, support consideration of alternatives to the current CO standards to provide increased protection from ambient CO exposures?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative exposure/risk analyses to address this question, we have focused on a series of more specific questions in sections 2.3.1, 2.3.2 and 2.3.3 below. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since the last review which has been critically analyzed and characterized in the 2000 AQCD and more recently in the ISA. Specifically, we consider how the currently available scientific evidence informs decisions regarding the basic elements of the NAAQS: indicator, averaging time, level and form. Considerations with regard to indicator and averaging time are presented in sections 2.3.1 and 2.3.2. Form and level are discussed in section 2.3.3.

2.3.1 Indicator

The indicator for CO is CO as measured by the federal reference methods and equivalent methods. Unlike several other criteria pollutants, there are not multiple compounds or size fractions of CO. Federal reference methods are available that can effectively measure CO, and as discussed in section 1.3.2 above, the available federal reference methods include a range of analytical sensitivities to support quantification of CO at ambient levels. Thus we have not identified any basis for considering an indicator other than CO for the standard.

2.3.2 Averaging Time

In considering potential averaging times alternative to the current 1- and 8-hour averaging times, we consider the following question:

- **Do health effects evidence and air quality/exposure assessments provide support for considering different exposure indices or averaging times?**

The averaging times for the current standards are 1 hour (35 ppm) and 8 hours (9 ppm). These averaging times were first chosen when EPA promulgated the primary NAAQS for CO in 1971 (36 FR 8186) and were retained in subsequent reviews (1980, 45 FR 55066; 1985, 50 FR

37485; and 1994, 59 FR 38906). The 8-hour averaging time was selected primarily based on the rationale that most individuals appear to approach equilibrium blood levels of COHb after approximately 8 hours of continuous CO exposure (USDHEW, 1971; USEPA, 1979; AQCD 2000, section 7.4; ISA, section 4.3.2.2). Another consideration has been that the 8-hour time frame represented a good basis for tracking continuous exposures during any 24-hour period, recognizing that people may be exposed in approximately 8-hour blocks of time (e.g., working or sleeping) (45 FR 55077). The 1-hour averaging time primarily reflected consideration of the potential impact of shorter exposures. Since the establishment of the 1-hour standard, the health effects rationale for that averaging time has been strengthened, with much of the health evidence linked to exposures of 1 hour or somewhat longer, such as the controlled human studies demonstrating aggravation of myocardial ischemia after exposure of individuals with CAD to CO for approximately 1 hour (USEPA, 1984a, 1991; 2000 AQCD; ISA, section 5.2.4). In the last review of the standards, it was recognized that the 1-hour averaging period provides a better indicator of short-term health effects of CO (59 FR 38914). Further, in considering peak CO concentrations during shorter time periods (eg., hundreds of ppm for several minutes), EPA has recognized that attainment of the 1-hour and 8-hour standards would tend to also limit such short-duration peak concentrations, such as those occurring near busy roadways, to levels below those of concern (45 FR 55077).

In considering whether the information available in this review supports consideration for different averaging times for the CO standards, we note that the available information in this review is generally consistent with and supportive of the conclusions reached in the previous reviews to retain the 1- and 8-hour averaging times.

- Controlled human exposure studies, with which epidemiological evidence is coherent, provide the clearest evidence of short-term CO-induced effects and reflect exposures of 1-2 hours (ISA, sections 5.2, 5.2.6.1).
- Most individuals would be expected to achieve steady-state levels of blood COHb after 6-8 hours of continuous exposure to a fixed CO concentration. Therefore, 8 hours may reasonably be considered closely representative of longer continuous exposures (2000 AQCD, section 7.4).
- Epidemiological studies provide evidence based on analyses of 1-, 8-, and 24-hour averaging times (ISA, section 5.2.6).
- The 1-hour and 8-hour averaging times are considered to provide protection from the occurrence of short-duration elevations in exposure concentrations of ambient CO that may be encountered in some environments and from COHb levels that may result from such exposures (2000 AQCD, section 7.4; ISA, section 2.4.1).

Taken together, we conclude that the information available in the current review continues to support the 1- and 8-hour averaging times and does not provide support for consideration of standards with alternative averaging times.

2.3.3 Form and Level

In considering forms and levels for potential alternative standards below, we address the following overarching question:

- **What is the range of alternative levels and forms for the standard that are supported by the health effects evidence and air quality/exposure assessments, and what are the uncertainties and limitations in that health effects evidence and air quality/exposure assessments?**

We focus on general considerations with regard to form in section 2.3.3.1. We then address specific considerations for level in combination with form in section 2.3.3.2, with regard to the 1-hour and 8-hour standards.

2.3.3.1 Alternative Forms

When evaluating potential alternative forms for the CO NAAQS in conjunction with specific levels, staff considers the adequacy of the public health protection provided by the combination of level and form to be the foremost consideration. In addition, we recognize that it is preferable to have a form that is reasonably stable and relatively insulated from the impacts of extreme meteorological events or other rare, transitory impacts on air quality. A standard set with a high degree of instability could have the effect of reducing public health protection because shifts in and out of attainment could disrupt an area's ongoing implementation plans and associated control programs. Since the 1971 promulgation of the CO NAAQS, this full set of factors have been given weight in revisions to the forms of a number of other NAAQS (e.g., O_3, PM and NO_2). Our consideration of these factors in our evaluation of the form for the CO NAAQS has led us to focus on three aspects which together lead to consideration of a percentile-based daily maximum statistic averaged over three years.

First, as noted in the review of the O_3 NAAQS (EPA, 2007e), forms that call for averaging of concentrations over three years can better reflect pollutant-associated health risks than forms based on allowable exceedances. This is because such forms give proportionally greater weight to periods of time when pollutant concentrations are well above the level of the standard than to times during which the concentrations are just above the standard, while a form that allows for one or more exceedances of the standard level would give the same weight to periods of time with concentrations that just exceed the standard as to times when concentrations greatly exceed the standard level. Additionally, averaging concentrations over three years provides greater regulatory stability (e.g., with regard to control programs associated with

attainment status) than a form based on allowing a single exceedance in a year. In considering revision to this aspect of the standard, we note that while the REA simulations each included only a single year, air quality analyses (e.g., REA, section 3.1.5) may be used to inform consideration of the REA findings with regard to potential revision to a form based on a 3-year average statistic, as well as the appropriate level to accompany such a form.

Secondly, a percentile-based statistic for the form provides for the specification of a more consistent level of air quality among areas of the country having differing numbers of measurements during the time period evaluated. For example, the 90th percentile of an area with 200 measurements and one with 100 measurements would, in both cases, represent the concentration above which 10% of the measurements in either location are estimated to occur, while the 3rd highest concentration in the first case would be the concentration above which 1% of the concentrations in the location occur and in the second case would be the concentration above which 2% of the concentrations occur.

Lastly, in conjunction with a percentile-based statistic, we have focused on daily maximum concentrations for each averaging time as compared to the maximum across the complete data review period as is the case with the current standard forms of maximum 1-hour average and maximum non-overlapping 8-hour average in a year. Daily maximum forms, inherently give greater weight to occurrences on separate days than to multiple occurrences on the same day, and being somewhat less extreme statistics than maximums across a year, may provide for greater regulatory stability, thus affording more consistent air quality protection.

In considering specific forms on which to focus the current review, as shown in Table 2-7, we note such forms that have been considered in other NAAQS reviews, including the 98th and 99th percentile forms. These forms, averaged over three years, were considered with regard to a 24-hour standard for PM and 1-hour standards for NO_2 and SO_2 (USEPA, 2005; USEPA, 2008b; USEPA 2009b). We recognize that at a specific location with measurements taken on every day in a year, a 99th percentile form for an 8-hour daily maximum standard would correspond to the fourth highest daily maximum 8-hour average concentration in a year, while a 98th percentile form would correspond approximately to the 7th or 8th highest daily maximum. We have considered forms of a 99th percentile (fourth highest daily maximum) or a 98th percentile, each averaged over three years, as potentially providing appropriate balances between limiting peak CO concentrations and providing a stable regulatory target. In considering percentile-based statistics for the form of potential alternative CO standards, however, we focus on the REA results in section 2.3.3.2 below with regard to a 99th percentile concentration in recognition of the rapid response of COHb to elevations in CO exposure.

When considering results of the REA as they relate to form of the standard presented in section 2.3.3.2 below, we note that a decision on form must be made in conjunction with

selection of a particular standard level. The primary emphasis in such a decision will be on the level of public health protection provided by the combination of form and level. Table 2-7 presents levels for three standard forms (the current form and two alternatives) for potential alternative 8-hour and 1-hour standards that would be just met in the five air quality scenarios simulated in the REA based on the air quality data sets identified for the two study areas and described in the REA. The current form of both the 8-hour and 1-hour standards is a maximum, not to be exceeded more than once per year. In the section below we consider the results of the REA for different combinations of level with the alternate forms described above in addition to the current "exceeded only once per year" form.

Table 2-7. Level and form for potential alternative 8-hour and 1-hour standards that would be just met in the REA air quality scenarios simulated with data for the Denver and Los Angeles study areas.

8-hour Averaging Time			1- hour Averaging Time			
2nd highest maximum[a]	99th percentile daily maximum	98th percentile daily maximum	2nd highest maximum[a]	99th percentile daily maximum	98th percentile daily maximum	
---------- Level (ppm) ----------						Air Quality Scenario Modeled
Denver Study Area[b]						
9.4[c]	7.2	6.0	16.2	13.3	11.5	Current 8-hour standard (9 ppm)
6.5	5.0	4.2	11.2	9.2	8.0	99th percentile daily max 8-hr (5.0 ppm)
5.6	4.3	3.6	9.7	8.0	6.9	99th percentile daily max 1-hr (8.0 ppm)
5.4[c]	4.1	3.5	9.3	7.7	6.6	2nd highest 8-hour average (5 ppm)
3.1	2.8	2.4	4.6	4.5	3.9	*As Is* (2006)
Los Angeles Study Area[d]						
9.4[c]	8.2	7.2	11.8	11.6	9.9	Current 8-hour standard (9 ppm)
6.5	5.7	4.9	8.1	8.0	6.8	99th percentile daily max 1-hr (8.0 ppm)
5.6	5.1	4.8	8.2	7.4	6.7	*As Is* (2006)
5.7	5.0	4.4	7.2	7.1	6.0	99th percentile daily max 8-hr (5.0 ppm)
5.4[c]	4.7	4.1	6.8	6.7	5.7	2nd highest 8-hour average (5 ppm)[b]

Notes:
[a] This is the form of the current standards.
[b] For other than *As is* (2006) scenario, the relationships shown are based on 1995 air quality data proportionally adjusted to just meet the specified air quality scenario.
[c] Note that the current rounding convention for a standard level for which no decimal places are specified allows for concentrations up to the given standard level plus 0.4 ppm.
[d] For other than *As is* (2006) scenario, the relationships shown are based on 1997 air quality data proportionally adjusted to just meet the specified air quality scenario.

2.3.3.2 Alternative Levels

In considering alternative standard levels with regard to the potential to provide greater protection than that afforded by the current suite of standards against CO-related adverse health effects, we have taken into account scientific evidence from both experimental and epidemiologic studies, as well as the uncertainties and limitations in that evidence, and the quantitative estimates of exposure and dose provided by the REA. We note that the scientific evidence and quantitative assessment can provide insights into alternative standard levels only within the context of specific averaging times and forms. Therefore, this section considers the evidence and quantitative analysis as they relate to alternative levels particular to different forms and averaging times.

We believe that the integration of the health effects evidence with the exposure and dose estimates will be particularly important to informing conclusions regarding standard levels and forms considered to provide protection of public health with an adequate margin of safety. With regard to the scientific evidence, as discussed in section 2.2.1 above, we have given principal emphasis to findings of the multi-laboratory controlled human exposure study of individuals with pre-existing CAD (Allred et al., 1989a, 1989b, 1991). In this study, controlled experimental exposures to CO that resulted in a post-exercise study mean COHb level as low as 2.0% relative to room-air exposures that resulted in a mean baseline COHb level of 0.6% (post-exercise) were associated with myocardial ischemia-related effects, including, specifically, reduced time to exercise-induced angina and ST-segment change.[46] Additionally, analysis of the study subject-specific data indicated a dose-response relationship for these responses, with no evidence of a measurable threshold at the controlled exposures evaluated (ISA, section 5.2.4; Allred et al., 1989a, 1989b, 1991).

For our purposes here, we have also considered the epidemiological studies with regard to the extent to which this evidence is consistent with and generally supportive of conclusions drawn from the combined consideration of the controlled human exposure evidence with estimates from the exposure and dose assessment. We take note of the epidemiological studies showing positive associations with IHD, CHD and CHF health outcomes described in section 2.2.1 above, four of which were based in a study area in which the current standard was met, the associations for three of which (studying two different areas) were statistically significant. In our consideration of the evidence, we recognize CASAC advice, both with regard to relative emphasis to give this evidence and with regard to aspects of the epidemiological evidence that

[46] The exposure resulting in an average of 2-2.4% COHb reduced the time to these markers by approximately one half minute from the 8.5- to 9.5-minute period without exposure (Allred, et al., 1989b).

are "particularly problematic for CO" and the weight to be given to the well-conducted clinical studies (Brain and Samet, 2010a, 2010b).

Turning first to the REA results, we consider the absolute COHb estimates for alternative levels in terms of the current form and of a 99th percentile daily maximum form in Table 2-8 below. In considering health risk implications for the simulated at-risk population associated with these estimates, we focus on the percentage of simulated at-risk population in each study area estimated to experience daily maximum end-of-hour absolute COHb levels below three benchmark COHb levels: 3.0%, 2.0% and 1.5%. Among the various benchmarks described in section 2.2.2 above, we have identified these three specific levels to particularly inform our consideration of the REA estimates in light of different aspects of the evidence that suggest differing degrees of health significance for each of them. As discussed in the sections above, the 3.0% benchmark level is within the range of COHb levels identified in the last review as levels that could induce potentially adverse effects for which it was appropriate that standards provide protection against (59 FR 38913). The 2.0% COHb benchmark level represents the lowest COHb study level resulting from CO exposure at which a response was also observed, although smaller and of less clear health significance. In contrast, the 1.5% benchmark provides a point of comparison that falls below the levels for which we have evidence of effects. In considering the REA estimates across these three benchmarks, we also recognize a distinction in public health implications between single and multiple occurrences, particularly with regard to exceedances of the lower COHb benchmark levels.

In considering alternative standard levels, consistent with conclusions reached in section 2.2.4 above, we consider levels for both standards, while focusing predominantly on the 1-hour standard. We note the current role of the 8-hour standard in controlling CO concentrations nationally which has led to our greater focus on that standard in designing the REA simulations, while recognizing the REA results which indicate the influential role of the 1-hour ambient CO concentrations with regard to magnitude of ambient CO exposures, associated daily maximum end-of-hour COHb levels, and the percentage of the population estimated to experience COHb at or above benchmark levels that may be of concern. For example, as shown in Table 2-8 below, the REA results for just meeting the current 8-hour standard indicate that the corresponding 1-hour ambient CO level is lower in the Los Angeles study area, and the percentage of the at-risk population exceeding the 2% COHb benchmark is also lower there than in the Denver study area (Table 2-8, 1st two rows for each form). A much larger difference in population percentages occurs between these two 1-hour levels at the 1.5% COHb benchmark level. More specifically, the percentage of the simulated at-risk population for whom all daily maximum absolute COHb levels experienced are below 1.5% is estimated to be 95.0% in the Los Angeles study area and

only 75.5% in the Denver study area. At lower alternative 1-hour standard levels, the differences in population percentage at all COHb benchmarks are much smaller.

We have also considered the REA estimates for a series of alternative 1-hour standard levels below those associated with just meeting the current 8-hour standard. Based on this information, presented in Table 2-8, we compared REA results for these alternative levels to the results for levels just meeting the current 8-hour standard. For example, in focusing first on estimates for the 3.0% COHb benchmark level, we note that the percentage of the at-risk population for which all daily maximum COHb levels are below 3.0% COHb ranges from 99.7% of the population under conditions that just meet the current 8-hour standard and for a 1-hour level of 16.2 ppm (2^{nd} highest form) to >99.9-100% of the population under all alternative 1-hour levels. For the 2.0% COHb benchmark, the corresponding percentage of population avoiding even one such occurrence ranges from 95.5% for the highest 1-hour level to approximately 99% or more of the population for all alternative levels. Lastly, the percentage of population avoiding even one occurrence of a 1.5% daily maximum COHb (the benchmark level below the COHb levels represented in the evidence) ranges from approximately 75% of the population under conditions that just meet the current 8-hour standard and for a 1-hour level of 16.2 ppm (2^{nd} highest form) to approximately 90-99% of the population for other standard levels.

Table 2-8. Percentage of simulated HD population with daily maximum end-of-hour COHb levels (absolute) below the indicated COHb levels under alternative levels and forms for the 1-hour and 8-hour standards.

Current and Potential Alternative Standards			Daily Maximum End-of-hour COHb Level (Absolute)						
Form	Level		< 3.0 % COHb		< 2.0 % COHb		< 1.5 % COHb		*8-hour Levels (ppm)*
	1-hour (ppm)	8-hour (ppm)	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	
Second Highest Non-overlapping Concentration (Current Form)	16.2+	9.4+		99.7		95.5		75.5	*9.4+*
	11.8+		>99.9		99.4		95.0		
	11.2	5.4–6.5		>99.9		98.9		90.4	*6.5*
	9.7					99.4		93.9	*5.6*
	9.3							94.6	*5.4*
	8.2*		100		> 99.9		98.4		*5.6**
	8.1						98.5		*6.5*
	7.2						99.0		*5.7*
	6.8						99.2		*5.4*
	4.6*	3.1*		100		>99.9		98.8	*3.1**
99th Percentile of Daily Maximum Concentrations	13.3+	7.2-8.2+		99.7		95.5		75.5	*7.2+*
	11.6+		>99.9		99.4		95.0		*8.2+*
	9.2	4.1– 5.7		>99.9		98.9		90.4	*5.0*
	8.0		100		> 99.9		98.5		*5.7*
				>99.9		99.4		93.9	*4.3*
	7.7							94.6	*4.1*
	7.4*		100		>99.9		98.4		*5.1**
	7.1						99.0		*5.0*
	6.7						99.2		*4.7*
	4.5*	2.8*		100		>99.9		98.8	*2.8**

+ Plus marks indicate simulations based on air quality conditions just meeting the current 8-hour standard.
* Asterisks indicate simulations based on "as is" (2006) air quality conditions for the two study areas.
Drawn from REA tables 6-15 to 6-19 and 6-21 to 6-22, consistent with Table 2-7above.

In addition to consideration of the absolute COHb estimates from the REA, we have also considered the REA population estimates of daily maximum ambient contribution to end-of-hour (ambient contribution COHb)[47] for the same array of alternative levels and forms (Appendix D). In so doing, we recognize additional complexity in interpretations associated with these results,

[47] As described in section 2.2.2 above, "ambient contribution COHb" equals "absolute COHb" minus COHb resulting from simulations absent any CO exposure. The estimates for "absolute COHb" are based on simulations where CO exposure arises only from ambient CO sources, in the absence of nonambient (indoor) sources. As discussed above, by not considering the contribution of other (nonambient) sources to COHb, the magnitude of the ambient contribution may be overestimated for some persons, affecting these population estimates.

and the lack of information regarding multiple occurrences of specific magnitudes of ambient contribution to COHb. Nonetheless, we observe that all daily maximum ambient contributions to end-of-hour COHb estimates are less than 1.5% for more than 90% of the simulated population under all air quality conditions assessed, and for higher percentages of the population for somewhat lower standard levels. In considering this observation, we recognize that the COHb increase from baseline for the 2% COHb exposure group in the controlled human exposure studies was on the order of 1.5% COHb (Allred et al., 1989b).

Taking into account the differences between the simulations for the two study areas, recognizing considerations regarding absolute COHb benchmarks of 1.5 to 2% and above, the influence of baseline COHb on COHb level response to exposure events, and estimates of numbers of occurrences of COHb levels of interest, and being mindful of support provided by the epidemiological studies, we consider the REA estimates of daily maximum end-of-hour COHb levels with regard to level of protection estimated to be provided by the different alternative levels for the 8-hour and 1-hour standards (Table 2-8). In further considering the weight to place on this information, we are mindful of several key aspects of the evidence (described in more detail in section 2.2.1 above):

- Uncertainty in the public health significance of the effects observed at the lowest exposures in the controlled human exposure studies of CAD patients.
- The lack of evidence for a measurable threshold effect for the effects observed in the human clinical studies and the lack of studies that have evaluated effects of experimentally controlled short-term CO exposures of individuals with CAD that resulted in study mean COHb levels below 2%.
- Uncertainty associated with interpretation of COHb levels estimated in the quantitative assessment to result from simulated CO exposure concentrations much lower than the experimental CO exposure concentrations used in the controlled human studies to increase subject COHb levels to COHb study targets.
- The influence of baseline COHb levels on the impact of short-duration increases in CO exposure concentrations on subsequent COHb levels.
- The lack of studies of COHb levels associated with health effects in other potentially susceptible populations.
- Complications associated with quantitative interpretation of the epidemiological studies for CO which affect estimation of ambient concentrations that may be triggering health outcomes.

We recognize that, just as we concluded in section 2.2.4 above with regard to adequacy of the current suite of standards, the range of alternative standards that may be appropriate to consider differs based on the weight placed on different aspects of the evidence and on different aspects of the quantitative dose estimates, as well as on public health policy decisions regarding

the public health significance of the effects considered, the appropriate COHb benchmark on which to focus in considering maximum end-of-hour COHb levels, and the targeted percentage of the at-risk population.

To the extent that consideration is given to revising the 1-hour standard to provide protection against COHb levels associated with elevated 1-hour ambient CO concentrations, in light of the influence of 1-hour CO levels appreciably below the current 1-hour standard of 35 ppm on population estimates of daily maximum end-of-hour COHb levels, we recognize that the alternative levels appropriate to consider vary depending on the weight placed on each of the factors identified above. For example, to the extent that the REA estimates are judged to indicate that the range of 1-hour concentrations allowed under conditions just meeting the current 8-hour standard may contribute to exposures of concern in areas of the country with relatively higher 1-hour peaks, it may be appropriate to consider a revised level for the 1-hour standard that would achieve more uniform protection. As one option for such a level, alternative 1-hour levels that provide generally equivalent protection to the current 8-hour standard, nationally, with increased protection for areas with relatively higher 1-hour peaks, may be appropriate to consider. Accordingly, we note that the population distribution of daily maximum COHb levels associated with just meeting the current 8-hour standard in the REA simulations are also associated with just meeting a 1-hour level of approximately 12 to 16 ppm, in terms of the current form, and approximately 12 to 13 ppm, in terms of a 99^{th} percentile daily maximum form (Table 2-8). Further, depending on judgments with regard to the weight to be placed on the factors identified above, levels for the 1-hour standard appreciably lower than those associated with conditions just meeting the currently controlling 8-hour standard may be appropriate to consider. For example, focusing on the 2.0% absolute COHb benchmark and considering a target for the at-risk population of approximately 99% for at most a single occurrence of daily maximum COHb at such a level would indicate support for 1-hour standard levels ranging down to approximately 11 or 9 ppm, for the current form or a 99^{th} percentile daily maximum form, respectively. On the other hand, if one were to place weight on the two epidemiological studies finding statistically significant associations in two locations in which the current standard was met (and the ambient CO concentrations in those study locations), and on the REA estimates for this same population target (e.g., 99% having no occurrences) combined with an absolute COHb benchmark as low as 1.5% (a level appreciably below those recognized in the evidence), one might find support for a range extending down to approximately 5 ppm (combined with either form).

2.3.4 CASAC Advice

In our consideration of alternative standards, in addition to the evidence- and risk/exposure-based information discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the ISA, the REA, and the earlier draft of this document, as well as comments from the public on earlier drafts of this document and the REA.[48] As recognized in section 2.3.2 above, no public comments have been received to date in support of revisions to the current standards.

In CASAC's consideration of specific revisions to the standards in their review of the draft Policy Assessment, they suggested that staff describe example policy options such as retaining an 8-hour standard with "consideration given to levels within the range of 3 to 6 ppm, with no more than a single exceedance or revise the form of the standard to 99th percentile with a concentration range of 3-5 ppm." With regard to the 1-hour standard, CASAC suggested the policy option of retaining a 1-hour standard "to provide protection against infrequent acute exposures" and considering "a range of concentrations from 5 ppm to 15 ppm, combined with a 99th percentile or fourth highest daily maximum." The Panel further stated that they did not concur with the option of revoking the 1-hour standard (Brain and Samet, 2010b, p. 13).

2.3.5 Staff Conclusions on Alternative Standards

Staff's consideration of alternative primary CO standards builds on our conclusion from section 2.2.4 above that the body of evidence in combination with the results of the REA support standards at least as protective as the current standards, and, depending on the weight given to different aspects of the evidence and both public health and science policy judgments, it may be interpreted to provide support for alternative, more protective, standards. As an initial matter, we conclude it is appropriate to continue to use measurements of CO in accordance with federal reference methods as the indicator to address effects associated with exposure to ambient CO. We additionally conclude that it is appropriate to continue to retain standards with averaging times of 1 and 8 hours. With regard to form and level for those standards, we conclude that, depending on public health policy judgments regarding the protection of public health with an adequate margin of safety, the information available in this review supports consideration of either retaining the current suite of standards or revising one or both standards.

We reach the conclusion that it is appropriate to consider retaining the current suite of standards without revision based on consideration of the health effects evidence in combination

[48] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meetings held in conjunction with CASAC's review of the earlier draft of this document, of drafts of the REA, and of drafts of the ISA.

with the results of the REA (sections 2.2.1, 2.2.2, 2.3.2 and 2.3.3 above) and what may be considered reasonable judgments on the public health implications of the COHb levels estimated to occur under the current standard, the public health significance of the CO effects being considered, the weight to be given to findings in the epidemiological studies in locations where the current standards are met, and advice from CASAC. Such a conclusion takes into account the long-standing body of evidence that supports our understanding of the role of COHb in eliciting effects in susceptible populations, most specifically the evidence for those with cardiovascular disease, and gives particular weight to findings of controlled exposure studies of CAD patients in which sensitive indicators of myocardial ischemia were associated with COHb levels resulting from short-duration, high-concentration CO exposures. This conclusion also takes into account uncertainties associated with the differing circumstances of ambient air CO exposures from the CO exposures in the controlled human exposure studies, as well as, the unclear public health significance of the size of effects at the lowest studied exposures. As in the last review, this conclusion gives more weight to the significance of the effects observed in these studies at somewhat higher COHb levels. Additionally, this conclusion takes into account judgments in interpreting the public health implications of the REA estimates of COHb associated with ambient exposures based on the application of our current exposure modeling tools, and the size of the at-risk populations estimated to be protected from experiencing daily maximum COHb levels of potential concern by the current standard. Further, this conclusion considers the uncertainties in quantitative interpretations associated with the epidemiological studies to be too great for reliance on information from the few studies where the current standards were met as a basis for selection of alternative standards.

In addition to considering retaining the current suite of standards without revision, we also conclude that it is reasonable to consider revising the 1-hour standard downward to provide protection from infrequent short-duration peak ambient concentrations that may not be adequately provided by the current standards. While the quantitative analyses for this review focused predominantly on the controlling, 8-hour standard, the analyses have indicated the influential role of elevated 1-hour concentrations in contributing to daily maximum COHb levels over benchmark levels. In addition to the REA results, we note the health effects evidence from 1-hour controlled exposures, which indicates the effects in susceptible groups from such short duration exposures. The evidence and REA estimates indicate support for consideration of a range of 1-hour standard levels which would address the potential for the current 8-hour standard, as the controlling standard, to "average away" high short-duration exposures that may contribute to exposures of concern. Consequently, in considering alternative standard levels, we focus here on the 1-hour standard as providing the most direct approach for controlling the likelihood of such occurrences. Additionally, following consideration of alternative 1-hour

standard levels below, we also consider the extent to which it is appropriate, in conjunction with revisions to the 1-hour standard, to consider revising or retaining the current 8-hour standard.

We have identified a range of 1-hour standard levels from 15 to 5 ppm as being an appropriate range for consideration. These levels are in terms of a 99th percentile daily maximum form, averaged over three years, which we consider to provide increased regulatory stability over the current form. Given the objective for the 1-hour standard of providing protection from high ambient CO exposures of duration shorter than 8 hours and perhaps much shorter, we suggest the 99th percentile form for consideration in preference to a 98th percentile form. We additionally take note of CASAC's preference for a revision to the standards to provide greater protection and observe that the range of 1-hour standard levels discussed here is also the range that the CASAC CO Panel suggested was appropriate for consideration.

We have identified the upper part of the range of 1-hour standard levels for consideration (11-15 ppm) based on the objective of providing generally equivalent protection, nationally, to that provided by current 8-hour standard and potentially providing increased protection in some areas, such as those with relatively higher 1-hour peaks that are allowed by the current 8-hour standard. This part of the range is estimated to generally correspond to 1-hour CO levels occurring under conditions just meeting the current 8-hour standard based on current relationships between 1-hour and 8-hour average concentrations at current U.S. monitoring locations (Appendix C). Specifically, we considered the 25th and 75th percentiles of the distribution of ratios of 1-hour 99th percentile daily maximum concentrations averaged over three years to 8-hour second maximum concentration for 2009. As a result, this part of the range is represented by levels of about 11 ppm to 15 ppm, with a 99th percentile daily maximum form, averaged over three years. Selection of a 1-hour standard within this upper part of the range would be expected to allow for a somewhat similar pattern of ambient CO concentrations as the current, controlling 8-hour standard, although with explicit and independent control against shorter-duration peak concentrations which may contribute to daily maximum COHb levels in those exposed. Consideration of 1-hour standard levels in this part of the range would take into account the factors recognized with regard to the option of retaining the current standards. But it would additionally recognize the importance of limiting 1-hour concentrations that are not controlled by the current 8-hour standard but that may contribute to exceedances of relevant COHb benchmark levels.

We have also concluded that, based on the evidence and REA estimates and judgments that may be considered reasonable regarding appropriate population targets for maximum COHb levels induced by ambient CO exposures, it may be appropriate to consider standard levels that provide additional protection than that afforded by the current standards against the occurrence of short-duration peak ambient CO exposures and associated COHb levels. With this policy

objective in mind, we consider it appropriate to consider 1-hour standard levels of 9-10 ppm, which comprise the middle part of the range of 1-hour standard levels suggested for consideration. We conclude that consideration of standard levels in this part of the range would give greater weight to protection of the at-risk population against the small decrements in time-to-exercise-induced markers of myocardial ischemia associated with the lowest COHb levels studied, and provide increased protection for at-risk populations from higher COHb levels. Additionally, this conclusion considers the uncertainties in quantitative interpretations associated with the epidemiological studies to be too great for reliance on information from the few studies where the current standards were met as a basis for selection of alternative standards.

In further considering the evidence, we take note of the expanded epidemiological evidence and suggest that to the extent one places weight on the public health significance of the smaller changes in COHb assessed in the REA, gives greater importance to increasing the portion of at-risk populations estimated to keep COHb levels resulting from ambient CO exposures below 1.5% COHb and concludes that the epidemiological studies in areas meeting the current standards are reasonably interpreted as indicating that ambient CO is responsible for the health outcomes observed, consideration of levels extending down to the lower levels assessed in the REA and the lower part of the range of CO levels in the three epidemiological studies may be appropriate. In light of this, we have identified 1-hour standard levels of 5-8 ppm as comprising the lower part of the range of 1-hour standard levels for consideration.

In considering the relative strength of the evidence supporting each of the 3 parts of the range, we conclude that the upper part of the range is most strongly supported, both with regard to judgments concerning adversity and quantitative interpretation of the epidemiological studies with regard to ambient concentrations that may elicit effects. For the lower parts of the range, support provided by the available information is more limited, especially for the lowest part of the range.

As mentioned above, we also conclude it is appropriate to consider retaining a standard with an 8-hour averaging time. As when it was established, the 8-hour standard continues to provide protection from multiple-hour ambient CO exposures which may contribute to elevated COHb levels and associated effects. In conjunction with consideration of a revised 1-hour standard to a level in the upper part of the range described above, we conclude it is appropriate to consider either retaining the current 8-hour standard or revising the form of the standard to potentially provide greater regulatory stability, with adjustment to level to provide generally equivalent protection as the current 8-hour standard. Based on air quality relationships between the current form and an alternative form of 99th percentile daily maximum 8-hour concentration averaged over three years (Appendix C), we conclude that levels for the revised form that are appropriate to consider are 8 or 9 ppm.

In conjunction with consideration of a revised 1-hour standard to a level in the middle part of the range described above, we conclude it is appropriate to consider revising the 8-hour standard to provide additional protection from 8-hour average concentrations to a level within the range of 5-7 ppm in terms of a 99th percentile daily maximum, averaged over three years. Further, in conjunction with consideration of a revised 1-hour standard to a level in the lower part of the range described above, we have also identified 8-hour standard levels of 3-4 ppm, in terms of a 99th percentile daily maximum, averaged over three years. In both cases, the considerations leading to identification of these 8-hour standard levels are those discussed above with regard to the corresponding parts of the range identified for consideration of alternative 1-hour standard levels.

To provide some perspective on the implications of alternative primary standards (within the range of levels identified above), staff analyzed ambient CO concentrations for 2007-2009 to estimate the percentage of counties, and the populations in those counties that likely would not attain various alternative standards identified above. The analysis, shown in Appendix E was not considered as a basis for the above staff conclusions.

2.4 SUMMARY OF STAFF CONCLUSIONS ON PRIMARY STANDARDS

Staff conclusions on policy options for the primary CO NAAQS that are appropriate for the Administrator's consideration in making decisions on the primary standards for CO, together with supporting conclusions from sections 2.2.4 and 2.3.2 above, are briefly summarized below. In reaching conclusions on alternative standards to provide requisite protection for health effects associated with ambient CO exposures, staff has considered these standards in terms of the basic elements of the NAAQS: indicator, averaging time, form, and level. In drawing these conclusions, we are mindful that the Act requires standards to be set that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety, such that the standards are to be neither more nor less stringent than necessary. Thus, the Act does not require that NAAQS be set at zero-risk levels, but rather at levels that avoid unacceptable risks to public health.

(1) Staff concludes that the combined consideration of the body of evidence and the results from the quantitative exposure and dose assessment provide support for standards at least as protective as the current suite of standards to provide appropriate public health protection for susceptible populations, including most particularly individuals with cardiovascular disease, against effects of CO in exacerbating conditions of reduced oxygen availability to the heart.

(2) Staff additionally concludes that, depending on public health policy judgments regarding the protection of public health with an adequate margin of safety, the information available in this review supports consideration of either retaining the current suite of standards or revising one or both standards.

(3) With regard to the indicator for the CO standards, staff concludes that it is appropriate to continue to use measurements of CO in accordance with federal reference methods as the indicator to address effects associated with exposure to ambient CO.

(4) With regard to averaging times for the CO standards, staff concludes that it is appropriate to continue to retain standards with averaging times of 1 and 8 hours.

(5) With regard to the levels and forms of the standards, staff concludes it is appropriate to consider retaining the current standards, without revision, or revising the current suite of standards, with particular focus on revision of the 1-hour standard to provide protection that is not adequately provided by the current standards from infrequent, short-duration, peak ambient concentrations.

(6) With regard to a revised 1-hour standard, we conclude that it is appropriate to give consideration to a range of levels from 15 to 5 ppm, with a revised level selected in

combination with a revised form of 99th percentile (or fourth highest) daily maximum form, averaged over three years. Staff has identified three parts of this range that differ with regard to science and public health policy judgments, and supporting information.

a. To provide additional protection in areas with relatively higher 1-hour peaks that are allowed by the current 8-hour standard, it is appropriate to consider the upper part of the range, represented by 11-15 ppm. This part of the range would provide generally equivalent protection, nationally, to that provided by the current 8-hour standard. Staff concludes that this part of the range is most strongly supported by the current information.

b. To the extent it is judged appropriate to provide additional protection against COHb levels at or above 2% in susceptible populations, it is appropriate to consider the middle part of the range, represented by 9-10 ppm.

c. To the extent one places greater importance on COHb levels below 2.0% and concludes that the epidemiological evidence for areas meeting the current standards is reasonably interpreted as indicating ambient CO is responsible for the health outcomes observed, it may be appropriate to consider the lower part of the range (approximately 5 – 8 ppm) to potentially provide a greater degree of additional protection.

(7) In conjunction with a revision to the 1-hour standard, we conclude it is appropriate to give consideration to the following options for the 8-hour standard:

a. In conjunction with a revision of the 1-hour standard in the upper part of the range, it is appropriate to consider either retaining the current 8-hour standard or revising the form to provide greater regulatory stability, with adjustment to level to provide generally equivalent protection, nationally, as the current 8-hour standard. For a form of 99th percentile daily maximum, averaged over three years, a level of 8 or 9 ppm may be appropriate to consider with this option.

b. In conjunction with a revision of the 1-hour standard to a level in the middle part of the range, it is also appropriate to consider revising the 8-hour standard to a level from 5-7 ppm (with a 99th percentile daily maximum form) to provide additional protection from maximum COHb levels that may result from 8-hour ambient CO exposures.

c. In conjunction with a revision of the 1-hour standard to a level in the lower part of the range, it is also appropriate to consider revising the 8-hour standard to provide a more precautionary level of protection from 8-hour ambient CO

exposures, giving consideration to levels of approximately 3 or 4 ppm, with a 99^{th} percentile daily maximum form.

2.5 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

Staff believes it is important to highlight key uncertainties associated with establishing ambient standards for CO. Such key uncertainties and recommendations for health-related research, model development, and data gathering are outlined below. In some cases, research in these areas can go beyond aiding standard setting to aiding in the development of more efficient and effective control strategies. We note, however, that a full set of research recommendations to meet standards implementation and strategy development needs is beyond the scope of this discussion. We have identified the following key uncertainties, research questions and data gaps that have been highlighted in this review of the health-based primary standards.

- A critical aspect of our consideration of the evidence and the quantitative dose estimates is our interpretation of the controlled human exposure studies. While additional human exposure studies of this type are unlikely to be undertaken, expansion of our understanding of the implications of several aspects of this evidence base would reduce uncertainty in our interpretation for purposes of risk characterization.
    - Additional information relevant to the likelihood of health effects from short-term increases in CO exposure that result in COHb levels below 2% in individuals with CAD.
    - Additional research to help clarify the physiological significance of the changes in time to sensitive markers of myocardial ischemia observed in controlled human exposure studies of CAD patients at the lowest exposures.
    - Research to reduce uncertainty associated with interpretation of COHb levels estimated in the quantitative assessment to result from simulated CO exposure concentrations much lower than the experimental CO exposure concentrations used in the controlled human studies to increase subject COHb levels to COHb study targets.
- Further characterization of the physiological and environmental influences, including characteristics of exposure, on COHb levels, particularly those most influential to COHb levels in today's population, and the magnitude of those influences, would benefit future exposure and dose modeling
- Existing clinical data correlate CO-induced effects (i.e., cardiovascular ischemia observed at specific COHb levels) to COHb levels resulting from short-term, high CO exposure concentrations. However, there is a lack of other studies evaluating effects of CO, in terms of COHb levels, at lower ambient concentrations and/or longer-term exposure periods.
- Further research to characterize CO effects for other potentially susceptible populations, including populations with preexisting diseases other than CAD that limit oxygen availability, such as anemia and obstructive respiratory diseases; populations at certain lifestages (i.e., older adults and fetuses at critical stages of development); and

populations with elevated baseline COHb due to increased endogenous CO formation, such as individuals with diabetes and visitors to high altitude.

- Our interpretation of the epidemiological evidence for CO would benefit from a better understanding of the possible role of co-pollutants, and current information on CO concentrations, arising from both ambient and nonambient sources, in different microenvironments, including traffic- or roadway-related microenvironments.
- Our future assessments would benefit from research on studies evaluating effects of CO linked to biomarkers other than COHb and research on mechanisms of CO-induced effects other that those associated with limiting oxygen availability at ambient CO levels.

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3 CONSIDERATION OF A SECONDARY STANDARD FOR CARBON MONOXIDE

This chapter focuses on the key policy-relevant issues related to the review of welfare-related effects of CO. Under Section 109(b) of the Clean Air Act, a secondary standard is to be established at a level "requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of the pollutant in ambient air." Section 302(h) of the Act defines effects on welfare in part as "effects on soils, water, crops, vegetation, man-made materials, animals, weather, visibility, and climate."

Specifically, this chapter first summarizes the history of EPA's consideration of secondary standards for CO. It then discusses the evidence currently available for welfare effects to inform decisions in this review as to whether, and if so how, to establish secondary standards for CO based on public welfare considerations. Staff conclusions are based on the assessment and integrative synthesis of the scientific evidence presented in the ISA (USEPA, 2010), building on the evidence described in the 2000 AQCD (USEPA, 2000).

3.1 CONSIDERATION IN PREVIOUS REVIEWS

With the establishment of the first NAAQS for CO in 1971, secondary standards were set identical to the primary standards. CO was not shown to produce detrimental effects on certain higher plants at levels below 100 ppm. The only significant welfare effect identified for CO levels possibly approaching those in ambient air was inhibition of nitrogen fixation by microorganisms in the root nodules of legumes associated with CO levels of 100 ppm for one month (U.S. DHEW, 1970). In the first review of the CO NAAQS, which was completed in 1985, the threshold level for plant effects was recognized to occur well above ambient CO levels, such that vegetation damage as a result of CO in ambient air was concluded to be very unlikely (50 FR 37494). As a result, EPA concluded that the evidence did not support maintaining a secondary standard for CO, as welfare-related effects had not been documented to occur at ambient concentrations (50 FR 37494). Based on that conclusion, EPA revoked the secondary standard. In the most recent review of CO, which was completed in 1994, EPA again concluded there was insufficient evidence of welfare effects occurring at or near ambient levels to support setting a secondary NAAQS (59 FR 38906). That review did not consider climate-related effects.

3.2 CONSIDERATION OF EVIDENCE AVAILABLE IN THE CURRENT REVIEW

To evaluate whether consideration of establishment of a secondary standard for CO is appropriate, we adopted an approach in this review that builds upon the general approach used in the last review and reflects the broader body of evidence and information now available. In developing conclusions in this review below, staff has taken into account the following overarching question:

- **Does the currently available scientific information provide support for considering the establishment of a secondary standard for CO?**

In considering this overarching question, we first note that the extensive literature search performed for the current review did not identify any evidence of ecological effects of CO unrelated to climate-related effects, at or near ambient levels (ISA, section 1.3 and p. 1-3). However, ambient CO has been associated with welfare effects related to climate (ISA, section 3.3). Climate-related effects of CO were considered for the first time in the 2000 AQCD. The greater focus on climate in the current ISA relative to the 2000 AQCD reflects comments from CASAC and increased attention to the role of CO in climate forcing (Brain and Samet, 2009). Based on the current evidence, the ISA concludes that "a causal relationship exists between current atmospheric concentrations of CO and effects on climate" (ISA, section 2.2). Accordingly, the following discussion focuses on climate-related effects of CO in addressing the question posed above.

Recently available information does not alter the current well-established understanding of the role of urban and regional CO in continental and global-scale chemistry, as outlined in the 2000 AQCD. CO is a weak direct contributor to greenhouse warming. The most significant effects on climate result indirectly from CO chemistry, related to the role of CO as the major atmospheric sink for hydroxyl groups (OH). Increased concentrations of CO can lead to increased concentrations of other gases whose loss processes also involve OH chemistry. Some of these gases, such as methane (CH_4) and ozone (O_3), contribute to the greenhouse effect directly while others deplete stratospheric O_3 (ISA, section 3.3 and p. 3-11).

Advances in modeling and measurement have improved our understanding of the relative contribution of CO to climate forcing. CO contributes to climate forcing through both direct radiative forcing (RF) of CO, estimated at 0.024 W/m^2 by Sinha and Toumi (1996), and indirect effects of CO on climate through CH_4, O_3 and carbon dioxide (CO_2) (Forster et al. 2007). The Intergovernmental Panel on Climate Change (IPCC) estimated the combined RF for these indirect effects of CO to be ~0.2 W/m^2 over the period 1750-2005 (Forster et al., 2007), with more than one-half of the forcing attributed to O_3 formation (ISA, section 3.3 and p. 3-13).

CO is classified as a short-lived climate forcing agent, prompting CO emission reductions to be considered as a possible strategy to mitigate effects of global warming. However, it is highly problematic to evaluate the indirect effects of CO on climate due to the spatial and temporal variation in emissions and concentrations of CO and due to the localized chemical interdependencies involving CO, CH_4, and O_3 (ISA section 3.3 and p. 3-12). Most climate model simulations are based on global-scale scenarios and have a high degree of uncertainty associated with short-lived climate forcers such as CO (ISA, section 3.3 and p. 3-16). These models may fail to consider the local variations in climate forcing due to emissions sources and local meteorological patterns (ISA, section 3.3 and p. 3-16). It is possible to compute individual contributions to RF of CO from separate emissions sectors, although uncertainty in these estimates has not been quantified (ISA, section 3.3, p. 3-13 and Figure 3-7).

Uncertainties in the estimates of the indirect RF from CO are related to uncertainties in the chemical interdependencies of CO and trace gases, as described above. Large regional variations in CO concentrations also contribute to the uncertainties in the RF from CO and other trace gases (ISA section 3.3 and p. 3-12). Although measurement of and techniques for assessing climate forcing are improving, estimates of RF still have ~50% uncertainty (ISA, section 3.3, and p. 3-13).

3.3 CASAC ADVICE

In our consideration of a secondary standard, in addition to the evidence discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the ISA, and the earlier draft of this document.[1]

In their comments on the draft Policy Assessment, CASAC did not disagree with staff's initial conclusions that there is insufficient information to support consideration of a secondary standard at this time. CASAC did express the view that there is substantial evidence that CO has adverse effects on climate and recommended that staff summarize information that is currently lacking and would assist in consideration of a secondary standard in the future (Brain and Samet 2010). This recommendation is addressed in section 3.5 below.

[1] Thus far in this review, no public comments have been received regarding the secondary standard.

3.4 STAFF CONCLUSIONS FOR THE CURRENT REVIEW

In considering whether the currently available scientific information indicates the need for a secondary standard for CO, we have reached the following conclusions:

(1) With respect to non-climate welfare effects, including ecological effects and impacts to vegetation, there is no currently available scientific information that supports a CO secondary standard.

(2) With respect to climate-related effects, we note that there is evidence of climate forcing effects associated with CO (ISA, sections 2.2 and 3.3). There are weak direct, and stronger, but highly variable and uncertain indirect continental and regional climate forcing effects from CO. However, the available information provides no basis for estimating how localized changes in the temporal and spatial patterns of ambient CO likely to occur across the US with (or without) a secondary standard would affect local, regional, or nationwide changes in climate. Moreover, more than half of the indirect forcing effect of CO is attributable to O_3 formation, and welfare-related effects of O_3 are more appropriately considered in the context of the review of the O_3 NAAQS, rather than in this CO NAAQS review. For these reasons, it is currently not feasible to conduct an analysis for the purpose of considering a CO secondary standard based on climate considerations. Based on these considerations, staff concludes there is insufficient information at this time to support the consideration of a secondary NAAQS based on CO effects on climate processes.

3.5 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

Staff believes it is important to highlight key uncertainties associated with considering a secondary standard for CO, including those associated with CO effects on climate processes. Research and model refinements that would reduce such key uncertainties are outlined below, although we note that a full set of research recommendations is beyond the scope of this document. In summary, to better inform future considerations of a secondary standard for CO, additional information, such as that described below, is needed.

- More accurate U.S. and global emissions inventories to provide source-specific data on CO contributions to climate precursors.
- Improved representation and characterization of localized chemical reactions between CO, CH_4 and O_3 in global and regional climate models to enable the more accurate prediction of the role of CO in climate and local variations in climate forcing due to emissions sources and local meteorological patterns.
- Monitoring designed specifically to improve characterization of the spatial and temporal heterogeneity of the impact of CO emissions and to improve climate modeling capabilities.

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ATTACHMENT

Clean Air Scientific Advisory Committee Letter
(June 8, 2010)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

June 8, 2010

EPA-CASAC-10-013

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: Review of the *Policy Assessment for the Review of the Carbon Monoxide National Ambient Air Quality Standards (NAAQS): External Review Draft*

Dear Administrator Jackson:

The Clean Air Scientific Advisory Committee (CASAC or Committee) Carbon Monoxide (CO) NAAQS Review Panel met on March 22-23, 2010, to review EPA's *Policy Assessment for the Review of the Carbon Monoxide National Ambient Air Quality Standards (NAAQS): External Review Draft*. The chartered CASAC held a public teleconference on April 19, 2010, to review and approve the report. This letter provides CASAC's overall comments and evaluation. We highlight the most important issues which need to be addressed as the draft Policy Assessment (PA) is revised and finalized.

CASAC expresses appreciation to EPA staff in regard to the draft PA document. We recognize that limited time was available for its development, given the court ordered deadline. In this letter, we offer the main suggestions and concerns identified by the Carbon Monoxide Panel and approved by CASAC. The PA needs to be clearer about how the three main sources of carbon monoxide that contribute to the carbon monoxide dose in the body combine and interact. These three primary sources are endogenous production of carbon monoxide, exposure to indoor sources, and ambient outdoor CO exposure. Ambient CO exposure needs to be considered in the context of these other two sources of the biologically effective dose.

The Panel found that there was too much dependence on the now classic clinical study conducted by Allred et al. (1989) and funded by the Health Effects Institute (HEI). While agreeing that this seminal study provided important evidence, its findings should not be so emphasized as to ignore more contemporary epidemiologic studies, especially those directed at coronary artery disease (CAD) and at cardiovascular disease (CVD) more generally. The epidemiologic studies are important because other cardiovascular conditions affect a large number of people who are at risk from CO exposure. We support the high level of attention to populations at risk, but continue to be concerned that the Agency is underestimating CO exposure among some vulnerable groups, especially persons with low income status. This is one

rationale for placing greater emphasis on the findings of the epidemiologic studies versus the controlled clinical studies. As with other criteria pollutants, the existence of these populations and the extent of their increased susceptibility are essential to promulgating NAAQS that protect the public health. We recommend this greater emphasis of the epidemiologic data across all of the CO documents, beginning with the Integrated Science Assessment and extending through the PA. There needs to be greater balance in treating the various lines of evidence.

The chartered CASAC feels that, in general, an ideal PA should be far shorter and more focused. Staff and the Administrator can turn to the REA and the ISA for more background regarding CO as necessary. The PA could be reduced in length to present a more concise summary of the evidence and how the evidence relates to alternative CO standards. A concise description of how the form of the standard is important would also be useful.

It is important to acknowledge the decreases in ambient CO levels over time; however, this success should not preclude an objective assessment of the potential health consequences of exposures at the current CO NAAQS. While measured concentrations infrequently reach the current NAAQS, evidence indicates that adverse health effects could occur at these levels. For that reason, CASAC expresses its preference for a lower standard.

We understand there will not be a subsequent draft before the release of the final PA. After EPA incorporates our major comments and recommendations, the PA will be adequate for rulemaking. We look forward to the Agency's response and the successful completion of the CO NAAQS review. The CASAC and Panel memberships are listed in Enclosure A. The Panel's responses to EPA's charge questions are presented in Enclosure B. Finally, Enclosure C is a compilation of individual panel member comments.

Sincerely,

/Signed/

Dr. Joseph D. Brain, Chair
CASAC CO Review Panel

/Signed/

Dr. Jonathan M. Samet, Chair
Clean Air Scientific Advisory Committee

Enclosures

NOTICE

This report has been written as part of the activities of the EPA's Clean Air Scientific Advisory Committee (CASAC), a federal advisory committee independently chartered to provide extramural scientific information and advice to the Administrator and other officials of the EPA. CASAC provides balanced, expert assessment of scientific matters related to issues and problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the EPA, nor of other agencies within the Executive Branch of the federal government. In addition, any mention of trade names of commercial products does not constitute a recommendation for use. CASAC reports are posted on the EPA website at http://www.epa.gov/CASAC.

Enclosure A

Rosters of the CASAC CO Panel and CASAC

U.S. Environmental Protection Agency
Clean Air Scientific Advisory Committee
Carbon Monoxide Review Panel

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Enclosure B

CASAC's Consensus Responses to EPA's Charge Questions

1. Does the Panel find the introductory and background material, including that pertaining to previous reviews of the CO standard, the current review and current air quality, to be clearly communicated and appropriately characterized?

Chapter 1 of the PA does a good job providing background information. There is a brief review of the CAA and provisions to establish primary and secondary NAAQS; adequate margins of safety; previous reviews; CO sources in ambient air; the monitoring network; low dose levels; new monitors/NCore network; recent ambient and steady-state decreases in ambient CO; and finally, the "staff's evaluation of policy implications of scientific evidence in the ISA and results of quantitative analyses based on that evidence." The PA focuses on the four basic elements of a NAAQS: indicator, averaging time, form and level. None of these elements have been clearly defined in the PA. The Panel recommends including clear definitions of these four elements, consistent with previous CASAC recommendations in the review of other criteria pollutants.

2. Consistent with the revised NAAQS process which includes development of this draft Policy Assessment (PA) document, considerations with regard to the primary standard for CO have been organized around a set of policy-relevant questions for the review.

a. Does the Panel find the question posed to appropriately reflect the policy relevant questions in the review?

The questions posed raise the major issues, and the information provided in response to these questions provides the essential evidence required for making policy decisions. It is difficult to make a judgment on the adequacy of protection because there is no estimate of the total population exposed to benchmark CO concentrations. Only numbers for test cases in Denver and Los Angeles are provided and additional information is needed on the application of the two case studies' findings to the whole country.

The increase in scientific evidence on the effects of environmental CO since the last evaluation of CO standards, as documented in the ISA, comes primarily from epidemiology based studies. A combined consideration of the findings of epidemiological studies and controlled human exposure studies leads to the conclusion that substantial numbers of persons experience ambient CO concentrations resulting in lower effective CO doses than the doses used in the controlled human exposures. The document does not appear to give the epidemiologic studies sufficient standing relative to the controlled human exposure data, even though they may be more realistic.

One question that was not adequately posed is: what are the confounding effects of non-traffic sources of CO (e.g., indoor air)? Numerous studies have shown that we spend 80-90% of time indoors. For healthy elderly and people with CVD, the time they spend indoors may be even greater. The non-traffic sources of CO are at times substantial and may override the ambient CO

levels in contributing to dose. It is suggested that information from the 2000 criteria document on indoor sources be included.

b. Does the Panel consider the document to provide the appropriate level of detail in addressing these policy-relevant questions?

For the controlled human studies, the Panel found the level of detail appropriate. However, the opposite is true for the epidemiological studies.

3. The discussion of the health effects evidence (e.g., section 2.2.1) draws from the most recent information contained in the final ISA for CO and information from the previous review described in previous Air Quality Criteria Documents.

a. Does the draft PA accurately reflect the currently available health effects evidence for CO as characterized in the final ISA and the extent to which it differs from that available at the time of the last review?
b. Does the Pane find the presentation to be technically sound, clearly communicated and appropriately balanced?

The description of the data considered by the previous EPA reviews is basically sound but too focused on the Allred et al. study. There should be a way to mention key elements of other controlled human studies in this document. The document continues to emphasize the use of %COHb as the optimal dose metric for assessing risk associated with CO exposure and its health consequences. However, the discussion of the epidemiological data should also consider non-hypoxia mechanisms. Increased COHb is important, but may not be the only mechanism for CO health effects.

The last review of CO was halted for several years due to the pending study of the effects of CO at high altitude and extreme cold environments and its subsequent report. The PA should very briefly acknowledge the findings of this report. Without that information, it is difficult to determine to what extent there are changes from the last review that commenced in 1999.

In order to facilitate better understanding of the cardiovascular effects, particularly myocardial ischemia, we suggest adding to the reported values of changes in % time to angina on page 2-11 (top paragraph), including the actual changes in seconds with the confidence intervals (CI). Moreover, regarding time to angina endpoint, are there any long-term consequences on repeated exposures, duration of angina, and frequency of occurrence without CO exposure? EPA should address these questions. If data are not available, the PA should state this to be the case. This information would seem to be important for the more complete understanding of the uncertainties associated with using these data to support the standards.

4. The discussion of the quantitative analysis of exposure and dose (e.g., section 2.2.2) draws from the analyses described in the second draft Risk and Exposure Assessment (REA).

a. Does this discussion accurately reflect the analyses contained in the draft REA?

The Panel largely agreed that the discussion in the PA accurately reflects the analyses contained in the second draft REA. We continue to be concerned with whether increased emphasis could be placed on the increment that ambient CO contributes to COHb or whether the emphasis should be on the final resulting %COHb concentration itself. We have a related interest in modeling indoor source contribution to COHb to better understand the total COHB concentrations.

Panel members offered mixed opinion regarding the decision by the EPA not to pursue the 1% COHb benchmark as suggested by the Panel. The staff correctly pointed out that "this level overlaps with the upper part of the range of endogenous levels." One Panel member supported the agency's decision, since this complies with the EPA's task "to establish standards that are neither more nor less stringent than necessary for these purposes", i.e., public health. However, other members considered that a more advanced modeling approach could focus on the increment that ambient CO contributes to %COHb, rather than the final resulting COHb concentration itself. The incremental CO analysis would provide a clear context of the full range of benchmarks for policy analysis. Further, if adverse effects are clearly observed in controlled human exposure studies with a small sample size associated with an increase in the percent COHb of 2%, then it is prudent to consider standards that would use a benchmark of ambient CO-attributable COHb increases as low as 1%. This benchmark would lead to a wider range for a margin of safety, given that a no observable adverse effect level for CO effects among susceptible populations has not been demonstrated.

b. Does the Panel find the presentation to be technically sound, clearly communicated and appropriately balanced?

Most Panel members agreed that the presentation was technically sound and appropriately balanced. However, most of the Panel was concerned that the presentation unnecessarily diminished the value of epidemiological studies in establishing the underpinnings (if not the details) of the quantitative relationship. Despite the fact that the PA may need to be based on a risk assessment drawn primarily from one particularly informative controlled human exposure study (i.e., the multi-center investigation described in Allred, et al.), there would be value in highlighting the supporting role of other studies, in particular the body of epidemiological evidence.

The %COHb module of the APEX model, although the most important, also has weaknesses, given that some physiologic data and the range of values for many variables that enter into the model are not transparent. Despite these limitations, however, there seems to be sufficient information for some variables that can be used to refine the estimates generated (e.g., Hb concentrations stratified by race-ethnicity as should be available from NHANES or other readily accessible sources).

5. Does the document identify and appropriately characterize the important uncertainties associated with the evidence and quantitative analysis of CO exposure and dose, particularly those of particular significance in drawing conclusions as to the adequacy of the current CO standards?

In general, the uncertainties are dealt with appropriately with one exception. Under the pretext of evaluating the uncertainty regarding ST segment changes, the current review suggests that the uncertainty is now greater than in 1991 (p. 2-32). The Allred et al. study used EKG changes in the ST segment to substantiate that the subjective measure of angina was indeed due to ischemia. These two indicators, one subjective and one objective, were very highly correlated and not independent. Therefore, separate analyses of the two indicators should be avoided.

The most thorough clinical studies remain those of Allred et al., Kleinman et al., and Sheps et al. While the effects in these groups are clear, and together these subjects may be "the best characterized population," it is not clear that they represent the "most susceptible population." First, these experiments have not been repeated in the past 20 years, and second, other potential susceptible groups have not been exposed to such controlled clinical conditions. Additionally, the epidemiologic data on cardiovascular (heart) disease, including congestive heart failure (CHF), suggest that those groups might be at least as susceptible to CO-related stress as the coronary artery disease group.

The data available in the PA and the ISA on CO and heart failure are instructive. The statement on page 2-14 (lines 16-19) that there are only "…small or no associations between hospital admissions" and stroke is inaccurate (see next paragraph). Of the five studies listed in the footnote at the bottom of that page, four of the five reported increased hospital admissions for CHF. A close look at Figures 5-2, 5-3, and 5-4 in the ISA support the association of CO with CHF and stroke more than for CAD. If all the studies for stroke, CHF and CAD were placed on the same x-axis, it could very well demonstrate the heightened uncertainty in statements of CAD patients being the most susceptible to CO effects.

Another possible uncertainty regards the question of whether CO is a surrogate and whether its effects at low concentrations can be separated from those of co-pollutants (p. 2-34, lines 24-34). There are analytical and methodological challenges in disentangling the effects of CO from those of co-pollutants, although the problem does not exist in the controlled clinical studies of CO alone.

6. This document has integrated health evidence from the final ISA and risk and exposure information from the second draft REA as it relates to reaching conclusions about the adequacy of the current standard and potential alternative standards for consideration.

a. Does the Panel view this integration to be technically sound, clearly communicated, and appropriately characterized?

Although it may be a challenging task, it is important to integrate the evidence from the epidemiological studies with clinical studies (p. 2-25). Some of the conclusions are not well supported. In particular, the estimation of population exposures (p. 2-5, lines 27-34, and p. 2-6, lines 1-8) may underestimate exposures of those in lower socioeconomic status populations because of their higher likelihood of residing in heavily trafficked areas and an increased probability of exposure to secondhand tobacco smoke. Inclusion of population prevalence of low income status and smoking prevalence in the simulated populations might shift the distribution of estimated CO exposures towards higher levels.

The conclusion that the current evidence supports a primary focus on those with cardiovascular disease is justifiably based on observations from clinical studies. However, the best characterized and most extensively studied population does not necessarily coincide with the most highly susceptible population. Since the last review, there are additional studies with positive findings that assess effects on the fetuses. There is also strong toxicological evidence relevant to the association of prenatal CO exposure with adverse pregnancy outcomes, such as premature birth and low birth weight. A stronger commentary on exposure during pregnancy and reproductive outcomes is needed.

b. Does the document appropriately characterize the results of the draft REA, including their significance from a public health perspective?

The conclusion that the current evidence supports a primary focus on individuals with cardiovascular disease is justified by current clinical research. Discussion should be added, however, that the best characterized and most extensively studied population does not necessarily identify the most highly susceptible population. In particular, commentary on the fetus as an at-risk group should be added because of newer data describing the effects of CO on the fetus coupled with toxicological evidence for risks associated with prenatal CO exposure.

If the PA is going to use %COHb as the dose metric, then there has to be a better rationale provided for interpretation of the epidemiological data using this metric.

7. *What are the views of the Panel regarding the staff's discussion of considerations related to the adequacy of the current and potential alternative standards?*

The staff has provided an extensive analysis of the adequacy of the current and potential alternative primary CO standards. The current standards set the levels for 1-hr average and 8-hr average at 35 ppm and 9 ppm, respectively. The form of the standard is that those levels are not to be exceeded more than once per year. In reviewing the recent literature, staff has documented that the "much expanded epidemiological evidence … provides support for previous conclusions regarding cardiovascular disease-related susceptibility and indications of air quality conditions that may be associated with ambient CO-related risk" and concluded that a causal relationship is likely to exist between relevant short term exposures to CO and cardiovascular morbidity.

Staff also concludes that the currently available evidence provides limited but suggestive epidemiologic evidence for CO-induced effects on preterm births, birth defects, and developmental outcomes. Individuals with conditions limiting their ability to deliver oxygen to target tissues represent groups susceptible to the adverse effects of CO, in addition to those with coronary artery disease. Based on the analyses of epidemiological studies presented in the PA, there is consensus in the Panel that the current standards may not protect public health with an adequate margin of safety, and therefore revisions that result in lowering the standards should be considered.

While the epidemiologic studies provide evidence that is coherent with the controlled exposure studies, the Staff determined that four of the studies cited in Table 2.1 included years in which

the ambient CO concentrations exceeded the 8-hr standard. However, Table 2.1 includes three studies of hospitalizations for ischemic heart disease and/or congestive heart failure from Atlanta for which this was not the case (Tolbert 2007, Peel 2007, Metzger 2007). An additional study of CHF (Wellenius, 2005) also did not include data from years in which either the 1-hr or the 8-hr standards were exceeded.

The PA suggests that CHF could have multiple causes, and for that reason it would be problematic to use it as a health effect indicator. The three studies of ischemic heart disease were consistent, but only the Tolbert et al. study had clear statistically significant results. It should be recognized that new controlled exposure studies of some of the sensitive groups (e.g., infants, fetuses, individuals with CHF or MI's) would be nearly impossible to justify ethically. Therefore more reliance needs to be placed on the epidemiologic studies and assessing whether there are causal relationships. Pooling methods, such as quantitative meta-analyses, may also be useful for developing exposure-response relationships. The available studies cover periods during which the current NAAQS was exceeded as well as studies covering lower ranges. This coverage of a wide range of CO concentrations makes possible a relatively robust estimation of exposure-response relationships. The emphasis should be on studies that used a multipollutant model approach to control for potential confounding of CO effects by those of other co-varying pollutants.

While there have been no new controlled human exposures designed to examine effects of CO at COHb levels below 2%, there have been numerous improvements to the exposure and COHb dosimetry models employed to provide exposure and risk estimates. The Staff analysis indicates that some of the uncertainties identified in previous reviews of the standard have been reduced. Based on their overall analysis, they conclude that the body of evidence and the quantitative exposure and dose estimates provide support for a standard at least as protective as the current standards. I.e. the data provide support for retaining or revising the current 8-hr standard.

Overall the Panel agrees with this conclusion. If the epidemiological evidence is given additional weight, the conclusion could be drawn that health effects are occurring at levels below the current standard, which would support the tightening of the current standard. The PA should include an analysis the number of exceedances that would have occurred if the standard had been based on the epidemiological data.

8. *Staff believes that the evidence presented in the final ISA and the exposure and risk information presented in the second draft REA supports a range of policy options for the CO standards.*

The Staff have proposed a range of policy options based on the quantitative risk analyses performed. As a starting point, the Staff indicates that the evidence is consistent with maintaining standards that are at least as protective as the current levels. However, given new evidence, primarily epidemiological, that there are many individuals potentially at risk in addition to those with coronary artery disease (e.g., fetuses, pregnant women, people with congestive heart disease, and people with anemia of various types), there is reason to consider reducing the standard below the current level(s).

The Panel suggests describing example policy options such as:

- 8 hr – retain the 8-hr averaging time with consideration given to levels within the range of 3 to 6 ppm, with no more than a single exceedance or revise the form of the standard to 99th percentile with a concentration range of 3-5 ppm. See also Figure 1 which shows the linear relationship between the 99th percentile and the design value measured for epidemiologic studies summarized in PA Table 2-1.
- 1 hr – retain the current standard to provide protection against infrequent acute exposures. Consider a range of concentrations from 5 ppm to 15 ppm, combined with a 99th percentile or fourth-highest daily maximum. The panel does not concur with revoking the 1 hr standard.

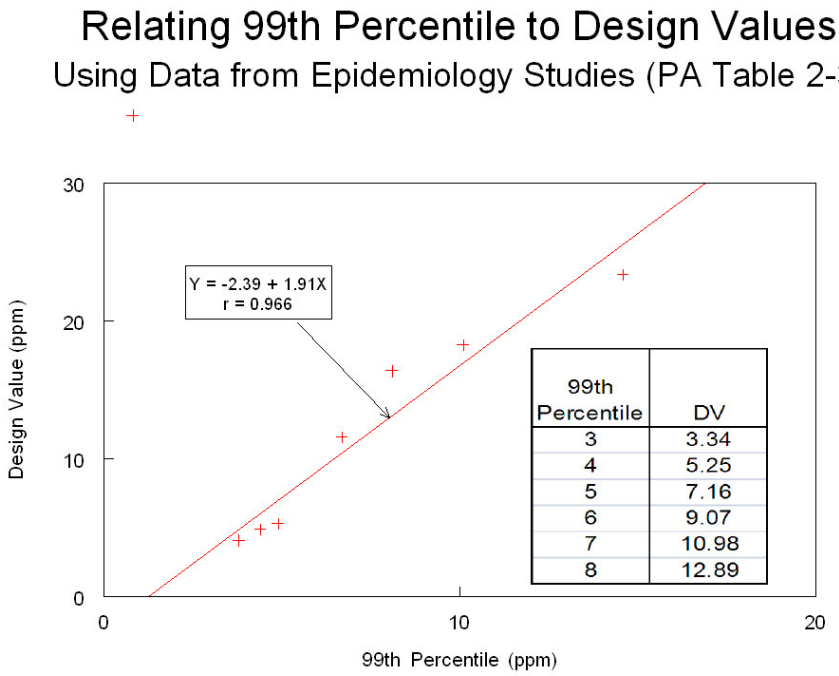

Figure 1

a. *To what extent does the document provide sufficient rationale to justify this range of options?*

The risk models were based on effects in people with coronary artery disease. They were used to estimate the percentages of individuals in LA and Denver that would reach benchmark levels of COHb ranging from <1.5% COHb to <2% COHb. These were summarized in Tables 2-6 and 2-7 in the PA. The overall guidance for the policy was not clearly described and the wide range of options needs better definition. It might be useful to present a table of options with the pros and cons of each respectively. The information is embedded in the RA and PA documents, but the options and their respective advantages or disadvantages need to be more clearly summarized.

The Panel concurs with the staff that the 1-hr standard might provide protection independent of the type of protection provided by the 8-hr standard (page 2-54, line 14); however, the discussion supporting this statement should be more clearly documented.

b. Does the Panel have any recommendations regarding additional considerations which should inform characterization of these options for both the 8-hour and 1-hour standards?

In choosing a more stable form of the standard, such as the 99th percentile, which would allow more days on which the standard can be exceeded in a given year, the level of the standard must be reduced to insure that the degree of health protection is sufficient. EPA should consider conducting an evaluation of the representativeness of the risk analysis to the entire US. Currently, the PA is based on two very different cities. Spatial heterogeneity of CO exposures that increase exposures near major sources, i.e. near and on roadways, should be given more weight since these might drive some of the adverse health effects.

9. What are the Panel's views regarding the level of detail presented in this chapter?

The PA concludes that there is insufficient information at this time to support the consideration of a secondary standard for CO. In general, the level of discussion detail is appropriate; however, some additional detail could be added at the end of chapter 3 on what information is missing in order to make a determination regarding a secondary standard.

10. The discussion of the CO-related welfare effects draws from the most recent information contained in the final ISA for CO.

a. Does the draft PA accurately reflect the currently available evidence as characterized in the final ISA?

The Panel agrees that the Policy Assessment appropriately characterizes the evidence as presented in the ISA.

b. Does this discussion effectively summarize the information on climate-related effects of CO?

Yes, but there should be a clear statement, to match a similar assertion in the ISA, that there is some evidence that CO has effects on climate. It addition, it would be appropriate in the last paragraph of this chapter to summarize what information is missing and thus needed, such as more accurate U.S. and global emissions inventory, monitoring specifically for climate rather than just for standards and exposure, and improvements in localized chemical reactions between CO, CH_4, and O_3 within global models.

11. What are the Panel's views regarding the appropriateness of staff's initial conclusions related to considering a secondary standard for CO?

The PA concludes that there is insufficient information at this time to support consideration of a secondary NAAQS. Nonetheless, there is substantial evidence that CO has adverse effects on climate. It would be appropriate in the last paragraph of this chapter to summarize what information is missing.

Enclosure C

Review Comments from CASAC CO Panel Members on the *Policy Assessment for the Review of the Carbon Monoxide National Ambient Air Quality Standards: External Review Draft*

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4. The discussion of the quantitative analysis of exposure and dose (e.g., section 2.2.2) draws from the analyses described in the second draft Risk and Exposure Assessment (REA).
a. Does this discussion accurately reflect the analyses contained in the draft REA?
b. Does the Panel find the presentation to be technically sound, clearly communicated and appropriately balanced?

The Policy Assessment perpetuates and to a degree magnifies the fundamental misunderstanding of the REA in relation to susceptibility based to narrowly on CAD alone (i.e., past MI or angina) rather than on cardiovascular disease as a group. In both cases this is a misread of the ISA and marks a failure to grasp what the accumulated epidemiological evidence shows. Thus this presentation is unbalanced, in interpreting the ISA through the flawed "lens" of the REA.

6. This document has integrated health evidence from the final ISA and risk and exposure information from the second draft REA as it relates to reaching conclusions about the adequacy of the current standard and potential alternative standards for consideration.
a. Does the Panel view this integration to be technically sound, clearly communicated, and appropriately characterized?
b. Does the document appropriately characterize the results of the draft REA, including their significance from a public health perspective

It is very difficult to decipher the conclusions of Policy Assessment beyond an unequivocal position that what ever is done the current standards should not be *weakened*. I would characterize the conclusion as clearly communicating a sense of not wishing to communicate something definitive, at this point at least. The rationale for not considering how many at risk persons are pushed over a threshold of body burden of COHb because they have baseline exposures form non-ambient sources seems ill-judged and counter-intuitive in terms of public health protection. Perhaps there are parallels in considerations of ambient lead exposure limits?

Dr. Thomas Dahms

Charge Question 2. Consistent with the revised NAAQS process which includes development of this draft Policy Assessment (PA) document, considerations with regard to the primary standard for CO have been organized around a set of policy-relevant questions for the review.

a. Does the Panel find the question posed to appropriately reflect the policy relevant questions in the review?

I believe that the questions posed raise the major issues and the information provided in response to these questions provides the essential data required for making policy decisions. These questions regarding 1. the adequacy of protection by the current standards; 2.does new information alter previous conclusions regarding health effects; 3. should COHb continue to be the dose indicator for CO exposure; 4. the health effects of ambient CO levels; and 5. any reduction in the uncertainties regarding CO.

Regarding the adequacy of protection: it is difficult to make a judgement in this area for two reasons.

1. There is no definition presented of what is considered to be an acceptable risk and 2. The number of persons in the at risk groups exposed to criteria levels of CO is not defined for the country. The only description of numbers exposed is for two cities: Los Angeles and Denver with no guidance provided for extrapolation to the whole country. For example, if the document is to discuss the numbers of persons in the U.S. with CAD, then the reader needs to have some estimate of how many of these persons would reach criteria levels of COHb on an annual basis given the current standards. Therefore it is difficult to judge the effectiveness of the current standards in protecting the population

2. The new information in this area all comes from epidemiological studies that are crucial to the interpretation of the meaning of the controlled human exposures. The adverse health effect of limiting the amount of work a person with CAD can perform with doses of CO near the current standard has been clearly established. However it is not clear that the extent of limitation has any further impact on the health of this at risk group. This concern is implied in the discussion regarding the uncertainty about the significance of ST segment changes on page 2-32. The epidemiological studies are designed to provide one means of determine if low CO doses have measureable impacts on health by correlating CO exposure with hospital based treatment for CV related events. This link between the two types of studies is clear in my mind but I'm not sure that the connection is clearly stated in this document.

3. Carbon monoxide is unique among the regulated air pollutants because it has a clear marker of dose, %COHb. The document indicates that the well established effects of COHb are related to the reduction in oxygen delivery to the tissues. This is in the face of the immerging evidence of effects of the partial pressure of CO, P_{CO}, as a messenger molecule, which could result in various patho-physiological conditions in combination with CO exposure. What is missing from the REA and carried through to the PA is a brief description of the relationship between P_{CO} and %COHb. This could possibly provide some prospective for the reader as to the importance of the physiological tensions of carbon monoxide in

tissues of interest. This would not distract from the current understanding that the dose indicator of %COHb is currently the primary focus for policy assessment.

4. The decreasing ambient levels of CO in the United States makes it ever more difficult to demonstrate health effects of CO based on the concept of sufficient exogenous dose to result in %COHb levels that have been shown to have pathophysiological effects. It would appear that the epidemiological effects of CO occur at such low levels of exposure as to result in very little increases in %COHb. Accepting the premise that the epidemiological results attributed primarily to CO exposure implies that adverse health effects occur at levels of %COHb considerably below those shown to have statistically significant effects in controlled human exposures. For these effects to be consistent with the controlled human exposure data, one would have to accept the statement that the effects of CO are without threshold (page2-11, Line 9; 2-12, L4; 2-15, L24; 2-16, L26; 2-40, L2). Are we to assume that the reason that the epidemiological studies can show significant effects of very low levels of exposure (very small increases in %COHb) is due to the large number of subjects being studied. Or is there another hypothesis regarding how these effects are mediated?

5. The uncertainties related to CO exposure have not been lessened.

b. Does the Panel consider the document to provide the appropriate level of detail in addressing these policy-relevant questions?

Yes but brief verbiage linking concepts as noted above would be helpful in creating transitions between the types of information.

Charge question 3. The discussion of the health effects evidence (e.g., section 2.2.1) draws from the most recent information contained in the final ISA for CO and information from the previous review described in previous Air Quality Criteria Documents.

a. Does the draft PA accurately reflect the currently available health effects evidence for CO as characterized in the final ISA and the extent to which it differs from that available at the time of the last review?

b. Does the Pane find the presentation to be technically sound, clearly communicated and appropriately balanced?

The description of the current state of knowledge includes suggestive information regarding cellular processes that can result in regional increases in endogenous levels of CO that could be altered by exogenous exposure. Given the considerable amount of current research in this area, mention of this data should exist in this document. The last review of CO was halted for several years due to the pending study and report on the effects of CO at altitude and at extreme cold environments. The document should very briefly acknowledge the findings of this report. Without that information in the current document it is difficult to determine how this report should differ from the last review started in 1999.

Charge Question 4. The discussion of the quantitative analysis of exposure and dose (e.g.,section 2.2.2) draws from the analyses described n the second draft Risk and exposure Assessment (REA).

a. Does this discussion accurately reflect the analyses contained in the draft REA?

The discussion focuses on the detail of one multicenter study following brief mention of the supporting studies. I believe that this information could be strengthened by working in the information that the CO exposures in the other studies was very similar with confirming evidence regarding time to angina. This would address the current concern of imbalance in the discussion of the studies in this area.

b. Does the panel find the presentation to be technically sound, clearly communicated and appropriately balanced?

There are some concerns regarding the technical soundness of the descriptions given which do make physiological sense.

i. Inaccuracy: page 2-8, line 26. The statement "This binding to reduced iron…" is very misleading. It has been transferred from the REA description of CO binding to hemoglobin. In particular it comes from the mathematical fiddle noted in Appendix B of the REA on page B-5 which states: "In working with the CFK model it is convenient to express COHb as a percent of $[RHb]_0$." This false concept should not be repeated in the text of the document. The fundamental relationship as described by Haldane clearly indicates that the much higher affinity of hemoglobin for CO vs Oxygen results in CO displacing O2 from oxygenated hemoglobin. The implication that CO binds preferentially to only reduced Hb is incorrect and needs to be corrected.

ii. Page 2-9, line 1. The statement "…or increased cardiac output) is not clear. The preceding sentence is discussing cardiovascular disease in the context of CAD. Therefore the normal compensatory mechanism that exist in healthy individuals is increased myocardial blood flow through vasodilatation, not vasodilatation and increased cardiac output. The current verbiage does not make sense and needs to be changed.

Charge Question 5. Does the document identify and appropriately characterize the important uncertainties associated with the evidence and quantitative analysis of CO exposure and dose, particularly those of particular significance in drawing conclusions as to the adequacy of the current CO standards?

Generally the uncertainties are dealt with appropriately with the exception of the item mentioned below.

The current review on page 2-32 under the guise of evaluating the uncertainty regarding ST segment changes suggests that the uncertainty is now greater than it was in 1991. The policy assessment is based on the adverse health effects of 2% COHb resulting in reducing the amount of work a person with CAD can perform before chest pain develops with is due to myocardial ischemia. The Allred et al study used EKG changes in the ST segment to substantiate that the

subject measure of angina was indeed due to ischemia. These two indicators, one subjective and one objective, were very highly correlated and not independent. Therefore the separation of the two indicators (page 2-32, line 25-28) is a reflection of the reviewers not understanding the study design. (This should have been corrected throughout the ISA, REA and the PA. The statement attributed to the ISA, p.48 –assumed to be 5-48—on page 2-32 needs to have a line reference otherwise it is difficult to locate this conclusion in the ISA.) In fact the ever increasing amount of epidemiological data on the effects of CO probably reduces the uncertainty of the effects of CO exposure in individuals with cardiovascular disease.

Exposure/Risk-based Considerations

Page 2-40 lines 3-10. The rationale for not using the benchmark of 1% COHb is flawed. In the version of the ISA dated January 2010, I cannot find a reference to the range of endogenous levels of %COHb: the source needs to be better documented. There is a list of rates of endogenous product provided in the Appendix but there are multiple studies listed. If one of these studies is the source it should be identified. The rationale for requesting the inclusion of this benchmark was the sense that 'the effects of CO are without threshold (page2-11, Line 9; 2-12, L4; 2-15, L24; 2-16, L26; 2-40, L2).' The %COHb data that is being used is that of Allred et al cited on page 2-11, line 1 as showing %COHb levels for exposure to 0-2 ppm CO as being 0.6%. The benchmark of 1% does not appreciably overlap 0.6% any more than one would expect there to be overlap between 1.5% and 2.0%. What is not stated is that the Apex model may overestimate the range of values resulting from no exposure to exogenous CO.

Without the 1% COHb benchmark how are the epidemiologic studies to be interpreted? Are these effects due to the effects of a pollutant that is not measured but very highly correlated to atmospheric CO? If the Policy Assessment is going to use %COHb as the dose metric, then there has to be a rationale provided for interpretation of the epidemiological data using this metric. If the result is a very high number of individuals with CAD having doses of 1%COHb and very few appearing in the ER or being admitted, this point should be discussed.

Dr. Russell Dickerson

The Policy Assessment in Chapter 3 addresses the issue of a secondary standard.

9. What are the Panel's views regarding the level of detail?

The detail is a little light as indicated below.

10. a. *Does the draft PA accurately reflect the currently available evidence?*

Within the limits of what is written yes.

b. *Does this discussion effectively summarize the information on climate related effects of CO?*

See below.

11. What are the Panel's views regarding the appropriateness of the initial conclusions?

The PA concludes that there is insufficient information at this time to support the consideration of a secondary NAAQS. None-the-less, there is evidence that CO has adverse effects on climate. It would be appropriate in the last paragraph of this chapter to summarize what information is missing. For example, U.S. and global emissions inventories must achieve a certain level of accuracy before a secondary standard is established. Is the level of uncertainty sufficient and if not what would it take? Monitoring was being phased out – should this policy be reconsidered? Representative monitoring to evaluate emissions inventories or models may look different from monitoring to access exposure. The basic question of what form is needed for regulations or standards should be addressed. A concentration-based standard would probably be inappropriate. Emissions standards such as are being considered for CO_2 would be more applicable to the issue of how to control CO emissions. The ISA (Figure 3.8) shows nicely how CO is low hanging fruit with respect to short term (20-year) climate forcing. The PA may be an appropriate forum to provide guidance to how these environmental benefits may be realized.

Dr. Milan Hazucha

The first external draft of the document provides a comprehensive overview of the legislative requirements and approaches to policy decision making process. The draft presents in a succinct way all aspects of the scientific evidence required for a successful policy assessment. The staff has reviewed and discusses key scientific and technical knowledge with clear understanding of health effects associated with CO presence in the ambient air. Various related issues are presented in sufficient detail and clearly communicated.
Asking specific questions throughout the document and answering them in a succinct manner has been very helpful in focusing on the critical aspect of the policy setting.
Answers to charge questions and specific comments:
Introduction and Background for the Policy Assessment (Chapter 1)

1. Does the Panel find the introductory and background material, including that pertaining to previous reviews of the CO standard, the current review and current air quality, to be clearly communicated and appropriately characterized?

I find the introductory and background material pertaining to the previous and current reviews to be clearly communicated and appropriately characterized. All the important factors needed to make an informed judgment are adequately presented and briefly discussed.

Review of the Primary Standard (Chapter 2)

2. Consistent with the revised NAAQS process which includes development of this draft Policy Assessment (PA) document, considerations with regard to the primary standard for CO have been organized around a set of policy-relevant questions for the review.

a. Does the Panel find the questions posed to appropriately reflect the policy-relevant questions in this review?

Qualified yes in all respects. One question that was not posed is about the confounding effects of no-traffic sources of CO, e.g., indoor air. Numerous studies have shown that we spend~80% of time indoors. For healthy elderly and people with CVD the time spend indoors may be even longer. The non-traffic sources of CO are at times substantial and will override the ambient CO levels.

b. Does the Panel consider the document to provide the appropriate level of detail in addressing these policy-relevant questions?

Yes, in all respects. The PA is well written, providing sufficient details, and highlighting important factors/concerns so that the policy relevant questions can be addressed both quantitatively and qualitatively.

3. The discussion of the health effects evidence (e.g., section 2.2.1) draws from the most recent information contained in the final ISA for CO and information from the previous review described in previous Air Quality Criteria Documents.

a. Does the draft PA accurately reflect the currently available health effects evidence for CO as characterized in the final ISA and the extent to which it differs from that available at the time of the last review?

Yes, in all respects. The currently available scientific evidence is evaluated, characterized and presented in a sufficient detail supporting the adequacy of the protection afforded by the current CO standard. The differences with the last review are clearly presented. There are no new human laboratory studies or exposure/risk-based evidence that would alter the conclusions. The evidence from new epidemiologic studies has been presented in a balanced way. The PA correctly points out to limitations in integrating the evidence from laboratory and epidemiologic studies.

Based on the current scientific evidence and practical considerations (e.g. arterial blood draw) venous blood COHb level is the optimal indicator of "CO health."

b. Does the Panel find the presentation to be technically sound, clearly communicated, and appropriately balanced?

Qualified yes. In order to facilitate better understanding of the cardiovascular effects, particularly myocardial ischemia, I suggest to add to the reported values of % time changes to angina on p.2-11, top paragraph, the actual changes in seconds with the confidence intervals (CI) included as well. For example, the reported 4.2% shorter time to angina from a control ~ 9 min interval amounts to 22 sec, with the CI=8.7%. Since Allred et al. studies are considered the key studies, it would be very helpful to comment briefly on the clinical significance of the shortened time. Moreover, regarding time to angina endpoint, are there any long-term consequences on repeated exposures, on the duration of angina, and frequency of occurrence without CO exposure? EPA should address these questions and if we do not have respective data the PA should state so.
Moreover, the first part of the statement in footnote #12 (p. 2-12) commenting on the difficulty determining association of CO with CVD and as a marker for traffic-related pollutants should, because of its importance, be moved from the footnote to the body of respective paragraph. Recently published HEI Special Report #17 (Jan. 2010) entitled:"Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects" discusses CO as a marker for another traffic-related pollutants such as PM and NO_2 and not as a major health hazard.
The review of the epidemiologic evidence (p.2-14) accurately reflects the difficulties to establish causal relationship between CO and reported effects. Similarly, well reasoned section (p. 2-25) points to difficulties integrating laboratory/clinical findings and epidemiologic observations.

4. The discussion of the quantitative analysis of exposure and dose (e.g., section 2.2.2) draws from the analyses described in the second draft Risk and Exposure Assessment (REA).

a. Does this discussion accurately reflect the analyses contained in the draft REA?

Qualified yes. The COHb module of the APEX model though the most important is also the weakest, since we do not have sufficient physiologic data or the range of values for many variables that enter into the model. However, despite this limitation there seems to be sufficient information for some variables that can be used to tune the estimates, e.g. Hb concentration for whites and blacks.

As far 1% COHb benchmark suggested by the Panel, the staff correctly pointed out that "this level overlaps with the upper part of the range of endogenous levels" and decided not to focus on dose estimates (p.2-40). I support this approach since this complies with the EPA's task "to establish standards that are neither more nor less stringent than necessary for these purposes", .i.e. public health.

b. Does the Panel find the presentation to be technically sound, clearly communicated and appropriately balanced?

Yes, in all respects. Again, because of the importance of the statement, the first sentence of the footnote #25 on the difficulty to determine association between CO and CVD in interpreting epidemiological evidence should be moved to the body of a respective paragraph.

5. Does the document identify and appropriately characterize the important uncertainties associated with the evidence and quantitative analysis of CO exposure and dose, particularly those of particular significance in drawing conclusions as to the adequacy of the current CO standards?

Yes, in all respects; The key uncertainties associated with exposure and dose estimates should, besides traffic, list other sources of CO, such as indoor air, smoking, occupational exposures, to name the main ones (p.2-42, l.31). A succinct discussion of how these sources can override the protection afforded by the current CO standard would be helpful.

6. This document has integrated health evidence from the final ISA and risk and exposure information from the second draft REA as it relates to reaching conclusions about the adequacy of the current standard and potential alternative standards for consideration.

a. Does the Panel view this integration to be technically sound, clearly communicated, and appropriately characterized?

Yes, in all respects

b. Does the document appropriately characterize the results of the draft REA, including their significance from a public health perspective?

Yes, in all respects

7. What are the views of the Panel regarding the staff's discussion of considerations related to the adequacy of the current and potential alternative standards?

I find the initial staff conclusion "for either retaining or revising the current 8-hour standard" (p. 2-46) based on the available estimates of exposure ambivalent. Does this mean that EPA is undecided or that the evidence is split 50/50? It is true, as subsequently stated, that a variety of factors will be considered in judging the adequacy of the current standard. But such adequacy should be based primarily on the evidence from laboratory/clinical studies and not on policy and other considerations. The evidence from the epidemiology studies, as commented on in several previous sections of this document, is difficult to evaluate and integrate with clinical evidence (p. 2-25).
The CO concentrations reported in epidemiology studies will produce COHb levels within a normal range. From reading interpretation of these studies in the latest EPA PM ISA the dominant effects in these studies are due to PM. Since we do not have any measurements of COHb level or other adverse effects that can be specifically associated with CO the studies provide no proof beyond statistics that there is a causal relationship. CO is primarily known for its anti-inflammatory effects. However, CO is highly correlated with PM and other pollutants, therefore, it is very likely that CO acts as a surrogate for PM and other pollutants. Thus based strictly on scientific evidence, I agree with the staff interpretation of epidemiology studies and their leaning towards retaining the current 8-hour standard.
The section 2.3 of the discussion of the averaging time, the form and level of alternative standard and potential alternative levels is succinct and well reasoned. What is not clear what form might the alternative standard have?

8. Staff believes that the evidence presented in the final ISA and the exposure and risk information presented in the second draft REA supports a range of policy options for the CO standards.

a. To what extent does the document provide sufficient rationale to justify this range of options?

Yes, the staff provides sufficient rationale for discussion of the range of options, particularly the policy options.

b. Does the Panel have any recommendations regarding additional considerations which should inform characterization of these options for both the 8-hour and 1-hour standards?

There should be a greater emphasis on the evidence based on laboratory/clinical studies.

<u>Consideration of a Secondary Standard (Chapter 3)</u>

9. What are the Panel's views regarding the level of detail presented in this chapter?

The level of detail presented in this chapter is sufficient.

10. The discussion of the CO-related welfare effects draws from the most recent information contained in the final ISA for CO.

a. Does the draft PA accurately reflect the currently available evidence as characterized in the final ISA?

Yes, in all respects

b. Does this discussion effectively summarize the information on climate-related effects of CO?

Yes, in all respects

11. What are the Panel's views regarding the appropriateness of staff's initial conclusions related to considering a secondary standard for CO?

Fully agree with staff conclusions.

Dr. Michael Kleinman

7. What are the views of the Panel regarding the staff's discussion of considerations related to the adequacy of the current and potential alternative standards?

The staff has provided an extensive analysis of the adequacy of the current and potential alternative primary CO standards. The current standards include a 1-hr average and an 8-hr average standard of 35 ppm and 9 ppm, respectively. The form of the standard is that those levels are not to be exceeded more than once per year. In reviewing the recent literature staff has documented that the "much expanded epidemiological evidence … provides support for previous conclusions regarding cardiovascular disease –related susceptibility and indications of air quality conditions that may be associated with ambient CO-related risk" and concluded that a causal relationship is likely to exist between relevant short term exposures to CO and cardiovascular morbidity. Staff also conclude that the currently available evidence provides limited but suggestive epidemiologic evidence for CO-induced effects on pre-term births, birth defects, developmental outcomes and that individuals with conditions limiting their ability to deliver oxygen to target tissues represent groups susceptible to the adverse effects of CO, in addition to those with coronary artery disease. Based on the analyses of epidemiological studies presented in the PA there is a consensus in the panel that the current standards may not adequately protect public health with a reasonable margin of safety and therefore revisions that result in reducing the standards should be considered.

While the epidemiologic studies provide evidence of coherence with the controlled exposure studies, the Staff determined that four of the studies cited in Table 2.1 included years in which the ambient CO concentrations exceeded the 8 hr standard. However Table 2.1 includes 3 studies of hospitalizations for ischemic heart disease and/or congestive heart failure (CGF) from Atlanta for which this was not the case (Tolbert 2007, Peel 2007, Metzger 2007) and one additional study of CGF (Wellenius, 2005) which did not include data from years in which the either the 1 hr or the 8 hr standards were exceeded. The PA suggests that CHF could have multiple causes and for that reason it would be problematic to use as a health effect indicator. The 3 IHD studies were consistent but only the Tolbert study had clearly statistically significant results. It should be recognized new controlled exposure studies of some of the sensitive groups (e.g. infants, fetuses, individuals with CHF or MI's) would be nearly impossible to justify ethically. Therefore more reliance needs to be placed on the epidemiologic studies and uncovering causal relationships may require methods such as meta-analyses to develop exposure-response curves. For this purpose the fact that some studies included periods in which the current standard was exceeded becomes less important because there are also studies at lower levels so that CR relationships can be interpolated (as opposed to extrapolated). The emphasis should be on studies that used a multipollutant model approach to control for potential confounding of CO effects by other co-varying pollutants.

While there have been no new controlled human exposures that were designed to examine effects of CO at COHb levels below 2%, there have been numerous improvements to the exposure and COHb dosimetry models employed to provide exposure and risk estimates. The Staff analysis

indicates that some of the uncertainties identified in previous reviews of the standard have been reduced and that based on their overall analysis conclude that the body of evidence and the quantitative exposure and dose estimates provide support for a standard at least as protective as the current standards, i.e. the data provide support for retaining or revising the current 8-hr standard. Overall the panel agrees with this conclusion, at the bare minimum. If the epidemiological evidence is given additional weight, than one might conclude that health effects are accruing at levels below the current standard and therefore the evidence might be leaning in the direction of revising the current standard. An issue is that some of the epidemiological studies were under conditions in which the current standard was exceeded at least in some part. More complete details of the degree to which the standard was exceeded should be summarized in the PA document, i.e. some studies covered as many as 7 years; would it have been excluded for as little as 1 exceedence in 7 years?

8. Staff believes that the evidence presented in the final ISA and the exposure and risk information presented in the second draft REA supports a range of policy options for the CO standards.

The Staff have proposed a range of policy options based on the quantitative risk analysis performed. As a starting point the Staff indicates that the evidence is consistent with maintaining standards that are at least as protective as the current levels. However, given the new evidence, primarily epidemiologic, that there are many individuals potentially at risk in addition to those with coronary artery disease (e.g. fetuses, pregnant women, people with congestive heart disease, people with anemia of various types) there is reason to consider reducing the standard below the current level(s).

The panel suggests example policy options such as:

8 hr – retain the 8h r averaging time with consideration given to levels within the range of 3 to 6 ppm, with no more than 1 exceedance or revise the form of the standard to 99th percentile with a concentration range of 3-5. Note see Figure 1 which shows the linear relationship between the 99th percentile and the design value measured for epidemiologic studies summarized in PA Table 2-1 that showed significant IHD hospitalizations.

1 hr – retain the current standard to provide protection against infrequent acute exposures. Consider a range of concentrations from 5 ppm to 15 ppm, combined with a 99th percentile or fourth-highest daily maximum. The panel does not concur with revoking the 1 hr standard..

a. To what extent does the document provide sufficient rationale to justify this range of options?

The risk models were based on coronary artery disease effects and were used to estimate the percents of individuals in LA and Denver that would reach benchmark levels of COHb ranging from <1.5% COHb to <2% COHb. These were summarized in Tables 2-6 and 2-7 in the PA document. The overall guidance for the policy was not very clearly described and the wide range of options needs better definition. It might be useful to present the options in a table with the pros and cons laid out. The information is embedded in the RA and PA documents but the options and their respective advantages or disadvantages need to be more clearly summarized.

The panel concurs with the staff that the 1 hr standard might provide protection independent of the type of protection provided by the 8 hr standard (2-54; L 14), however the discussion supporting this statement should be more clearly documented.

b. Does the Panel have any recommendations regarding additional considerations which should inform characterization of these options for both the 8-hour and 1-hour standards?

i. In choosing a more stable form of the standard, such as the 99th percentile, which would allow more days on which the standard can be exceeded in a given year, the level of the standard must be reduced to insure that the degree of health protection is sufficient.

ii. A summary of the options and their pros or cons would be more helpful.

iii. An evaluation of how representative the risk analysis which is based on 2 very different cities is with regard to the entire US.

i. Spatial heterogeneity of CO exposures that increase exposures near major sources i.e. near and on roadways should be given some weight since these might drive a lot of the adverse health effects.

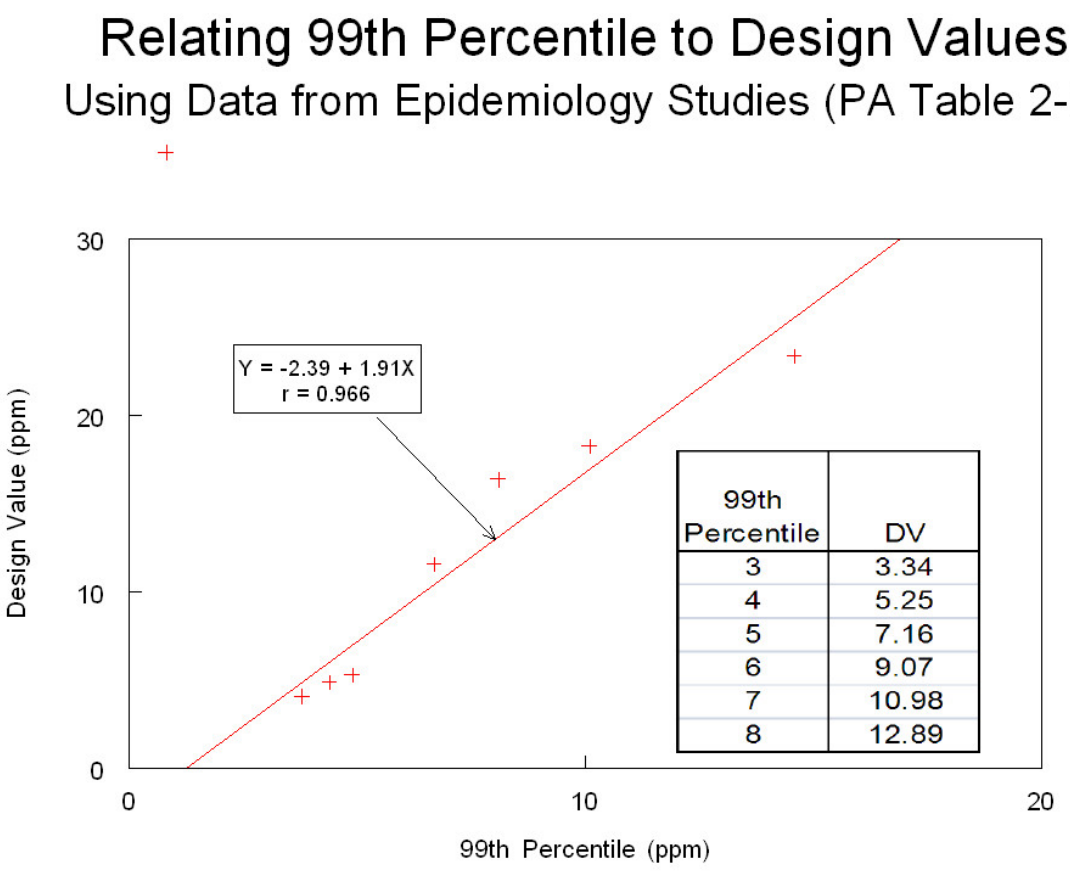

Figure 1

Dr. Francine Laden

2. Consistent with the revised NAAQS process which includes development of this draft Policy Assessment (PA) document, considerations with regard to the primary standard for CO have been organized around a set of policy-relevant questions for the review.
a. Does the Panel find the questions posed to appropriately reflect the policy relevant questions in this review?

Yes – the questions appropriately reflect the policy relevant questions.

b. Does the Panel consider the document to provide the appropriate level of detail in addressing these policy-relevant questions?

Yes – the level of detail is appropriate.

3. The discussion of the health effects evidence (e.g., section 2.2.1) draws from the most recent information contained in the final ISA for CO and information from the previous review described in previous Air Quality Criteria Documents.
a. Does the draft PA accurately reflect the currently available health effects evidence for CO as characterized in the final ISA and the extent to which it differs from that available at the time of the last review?

Yes – the draft PA accurately reflects the currently available health effects evidence for CO. One minor point: On page 2-9, it is stated that "it was concluded that there is not likely to be a causal relationship between relevant long-term CO exposures and mortality." Is EPA confident of this conclusion, or is there not sufficient data to address this relationship?

b. Does the Panel find the presentation to be technically sound, clearly communicated, and appropriately balanced?

Yes – the presentation is technically sound, clearly communicated and appropriately balanced.

Dr. Arthur Penn

1. Does the Panel find the introductory and background material, including that pertaining to previous reviews of the CO standard, the current review and current air quality, to be clearly communicated and appropriately characterized?

Chapter 1 of the PA does a good job, in a limited # of pages, of providing intro/background for the PA. There is a brief review of the CAA and establishment of NAAQS (1^o, 2^o); adequate margins of safety; previous reviews; CO sources in ambient air; the monitoring network; low dose levels; new monitors/NCore network; recent ambient and steady-state decreases in ambient CO; and finally, the "staff's evaluation of policy implications of scientific evidence in the ISA and results of quantitative analyses based on that evidence".

There is one item on p. 1-1 that could benefit from some clarification and possible change of location. Lines 22-25 on that page emphasize that the focus of the PA is on the 4 basic elements of NAAQS: indicator, averaging time, form and level. None of these items is explicitly defined in the first 46 pages of the PA. "Indicator" & "averaging time" both on p. 2-47 are clearly defined. "Level" is not defined explicitly, but its meaning is implicit in Tables 2-6 & 2-7. "Form" (pp. 2-48 & 2-49) is never defined clearly. "Concentration-based form", apparently an area of focus, also is not defined. Lines 15-23 on p. 2-49 suggest that "form" = percentile. Is that correct? Is it ever anything else? If it = percentile, why not say so?

If everything in the PA is based on these 4 elements, perhaps they should be defined on p.1.

5. Does the document identify and appropriately characterize the important uncertainties associated with the evidence and quantitative analysis of CO exposure and dose, particularly those of particular significance in drawing conclusions as to the adequacy of the current CO standards?

2 major uncertainties are listed on pp. 2-26 &2-27. 3 others are listed on pp. 2-4 & 2-5; + 5 on p. 2-53.

There are a couple of other conclusions of the PA that have raised questions for me. Whether they rise to the level of uncertainty depends on how other CASAC CO panelists respond.
p.2-18: The most thorough clinical studies remain those of Allred-Kleinman-Sheps. While the effects in these similar subject groups are clear, and together these subjects may be "the best characterized population" it is not clear that they represent the "most susceptible population". Since a) these experiments have not been repeated in the past 20 years and b) no other groups have been exposed to such controlled clinical conditions, it's difficult to conclude that this is the "most susceptible population". Additionally, the epidemiologic data on congestive heart failure and stroke patients, while minimized in the PA write-up, suggest that those groups might be at least as susceptible to CO-related stress as the coronary heart disease group.

The data available in the PA and the ISA on CO/heart failure are instructive. The statements in the PA, p 2-14, lines 16-19, that there are only "…small or no associations between hospital admissions" and stroke are not accurate (see next paragraph). This tone continues on p. 2-27, lines 8-10, where the document states that "…we did not include studies of associations with CHF… for which the evidence is less clear". Unless I've misread the data, of the 5 studies listed in the footnote at the bottom of that page, 4/5 reported increased hospital admissions for CHF. A close look at Figures 5-2, 5-3 & 5-4 in the ISA supports the CO association with CHF and stroke more than for CHD. In those 3 figures the range of relative risk (RR) values on the x-axis varies widely. In Figure 5-2 the range is from 1.0-1.4, so small changes in RR appear to be larger than they are. On the other hand, the wider ranges of RR values for CHF (1.0-2.20) and for stroke (1.0-4.5) make larger RR values in those figures appear smaller than they really are. In Figure 5-2 (CHD) 27/31 values have a RR< 1.05 and only 4/31 with values between 1.10 &1.18. In Figure 5-3 (stroke) at least 6 studies reported a RR of at least 1.25 and one was as high as 2.8. In Figure 5-4 (CHF), 4/10 studies had RR between 1.2-1.75. If all the studies for stroke, CHF and CHD were placed on the same x-axis, uncertainty could well be heightened about CHD patients being the most susceptible to CO effects. In addition, the mean ambient CO levels (24 hr) reported in 2 of the studies with large increases in RR were ~0.8 ppm, i.e., even lower than the 1 ppm value recommended by the CASAC CO panel at its Nov. 2009 meeting as worthy of attention.

Another possible uncertainty regards the question (PA-p. 2-34, lines 24-34) of whether CO is a surrogate and whether its effects at low concentrations can be untangled from those of co-pollutants. While there may be administrative reasons for focusing on these distinctions, the science justification is not clear. Both CO and organic particles in ambient air are largely products of incomplete combustion (PICs). In real-world (and in most laboratory) situations it is essentially impossible to generate, and therefore to breathe, organic particle PICs without volatiles, including CO. So, disentangling CO effects from those of co-pollutants (not a problem in the Allred-Kleinman-Sheps controlled clinical studies) is not only difficult, but likely also artificial.

Dr. Beate Ritz

7. What are the views of the Panel regarding the staff's discussion of considerations related to the adequacy of the current and potential alternative standards?

In reviewing the recent literature EPA staff has concluded that a causal relationship is likely to exist between relevant short term exposures to CO and cardiovascular morbidity based mainly on the coherence between the results from controlled human chamber studies and the more recent epidemiologic literature. However, the PA makes an argument that epidemiologic studies of IHD and CVD are including some areas with CO concentrations that exceeded the 8-hour standards but also cited and commented on 3 studies from Atlanta for which this was not the case (Tolbert 2007, Peel 2007, Metzger 2007) and stated that 2 of the three studies reported non-statistically significant results.

For the Atlanta studies, first this statement is incorrect, i.e. all 3 studies from Atlanta reported significant results for CVDs (I checked the original papers and this is also not correct according to the ISA table on page C-25), and second the effect estimate sizes are all very comparable (in all three studies) and this is more important than statistically significance testing. Nevertheless, since the 3 Atlanta studies do not use mutually exclusive data and the Tolbert study is the most comprehensive one with regard to the time frame and # of hospitals covered, this largest study can be considered the most informative of the three. Concerning the studies covering areas that exceeded the current standards during the study period, it seems not completely justified to disregard them because of this fact when assessing whether or not to use alternate standards, unless these studies can be shown to be less valid in principle or show some kind of threshold effect rather than a dose response and are very different in the estimated effect sizes reported. Thus, altogether Page 2- 27-28 provide an example of a general tendency of the PA to mis-interpretate and mis-represent epidemiologic study results that is even more evident when it comes to interpreting results for other types of health outcomes.

This is very obvious on page 2-33 in the text addressing the available evidence for CO-induced effects on pre-term births, birth defects, developmental outcomes; the PA states that "the epidemiologic evidence ….has somewhat expanded, although the available evidence is still considered limited with regard to effects .." This, is a misrepresentation of the large expansion of data on these outcomes in the epidemiologic literature in past decade. The category of limited evidence is not attributable to little or conflicting epidemiologic evidence but rather to the lack or impossibility of human chamber studies and valid animal models for many of these outcomes and a general tendency of the EPA staff to not attribute causality solely on the basis of epidemiologic evidence alone.

The EPA staff indicates that some of the uncertainties identified in previous reviews of the standard have been reduced and they provide support for a standard at least as protective as the current standards, i.e. the data provide support for retaining or revising the current 8-hr standard. In fact if the epidemiological evidence was not down-weighted or outright ignored as much as it

currently is in this PA, enough evidence has accrued at levels below the current standard to revise them downwards in the interest of public health in general (not just for CVD outcomes).

8. Staff believes that the evidence presented in the final ISA and the exposure and risk information presented in the second draft REA supports a range of policy options for the CO standards.

a. To what extent does the document provide sufficient rationale to justify this range of options?

Yes, the sufficient rationale for discussion of the range of options is provided

b. Does the Panel have any recommendations regarding additional considerations which should inform characterization of these options for both the 8-hour and 1-hour standards?

Spatial heterogeneity of CO exposures that increase exposures near major sources i.e. near and on roadways should be given some weight since these might drive a lot of the adverse health effects.

Dr. Anne Sweeney

CQ. 7. The discussion of considerations related to the adequacy of the current and potential alternative standards was comprehensive and clearly established the context for the ensuing discussions. However, some of the conclusions reached were not well-supported, including:

a. The Estimation of Population Exposures (Page 2-5, lines 27-34, and page 2-6, lines 1-8). The contribution of ambient air CO levels to indoor CO levels would be especially relevant among lower socioeconomic status populations. Given environmental justice concerns rendering lower income individuals more likely to reside in heavily trafficked areas, as well as lower income resulting in lack of air conditioning and extended periods of time with windows opened allowing influx of ambient air, and an increased probability of exposure to tobacco smoke, it seems critical to examine the contribution of indoor CO exposures in the modeling. Inclusion of population prevalence of low income status and smoking prevalence (based on income status) in the simulated populations would greatly enhance the ability to estimate CO exposures.

b. Regarding Evidence-based Considerations (2.2.1): The conclusion that the current evidence supports a primary focus on cardiovascular disease (CVD) is justifiably based on the research examining formation of COHb and related CVDs as the most extensively studied adverse health effect supporting an association with CO. It is stated on Page 2-18, lines 15-18 that "*.. the population with pre-existing cardiovascular disease associated with limitation in oxygen availability continues to be the est characterized population at risk of adverse CO-induced effects*..". However, the best characterized and most extensively studied population does not necessarily identify the most highly susceptible population. The expansion of studies with positive findings evaluating effects on fetuses since the previous review, supported by strong toxicological evidence for the finding of prenatal CO exposure and adverse pregnancy outcomes, warrants more attention to this subpopulation. As stated on Page 2-16, lines 12-18: "*With regard to potential effects of CO on birth outcomes and developmental effects, the currently available evidence includes limited but suggestive epidemiologic evidence for a CO-induced effect on preterm birth, birth defects, decrease in birth weight, other measures of fetal growth, and infant mortality (ISA, section 5.4.3). The available animal toxicological studies provide some support and coherence for these birth and developmental outcomes, although a clear understanding of the mechanisms underlying potential reproductive and developmental effects is still lacking (ISA, section 2.5.3).*" This reviewer agrees that the number of human studies in these areas is limited, however, the strength of the evidence to date supports an association of greater concern than the current evaluation bestows.

CQ. 8.

a. Overall, the range of options recommended by the staff support at minimum the continuation of the current CO standards and possibly a lowering of those standards to provide increased public health protection (Page 2-56, lines 23-27). This position is well-supported chiefly by the review of the effects of ambient CO exposure at levels at or below the current standards and the effects on CVD endpoints.

b. Again, the additive or multiplicative effects of ambient and indoor CO exposures need to be given more consideration. In assessing averaging time (section 2.3.2). the 8-hour averaging time was selected in part because *".. this time-frame represented a good basis for tracking continuous exposures during any 24-hour period, recognizing that most people may be exposed in approximately 8-hour blocks of time (e.g., working or sleeping)."* The comments regarding indoor CO exposures especially among lower income populations are relevant here as well.

Dr. Stephen Thom

1. Background/introduction is clear and appropriate.

2. Chapter 2.1 - the approach taken to review primary standards for CO is well organized.

Section 2.2 discusses the adequacy of the current standard by listing key questions. The format involves reiterating much of the rationale listed in the REA, sometimes stating the same evidence used in conclusions multiple times (*e.g.* the Allred, *et al.* findings – page 2-10 lines 4 – 26; page 2-22, lines 17 – 31; page 2-23, lines 7 – 13; page 2-32, line 36 – 37; page 2-33, line 1 – 5). This seems quite redundant.

Of greater concern, there are instances where questions are posed but not answered. Therefore, this reviewer feels that some sections are poorly communicated. For example, section 2-2 poses the question: "Does the currently available scientific evidence and exposure/risk-based information, as reflected in the ISA and draft REA, support or call into question the adequacy of the protection afforded by the current CO standards?" I cannot find any place in the document where the question is answered. Instead section 2-2 is broken down into other questions in sections 2.2.1 and 2.2.2, some of which are answered and some are not.

3. In section 2.2.1 on page 2-8, line 9 the question "Does the current evidence alter our conclusions from the previous review regarding the health effects associated with exposure to CO" is answered (page 2-16, line 23-27). On page 2-16 the question, "Does the current evidence continue to support a focus on COHb ... or does the current evidence provide support for ... alternate dose indicators ..." is answered (page 2-17, line 29-31). On page 2-18, line 1 the question "Does the current evidence alter our understanding of populations that are particularly susceptible to CO exposures?" is answered (page 2-21, line 17 – 20). Of note, there is also a second question posed on line 2-19 that is redundant with that posed on 2-18. The question on page 2-22, line 1, "Does the current evidence alter our conclusions from the previous review regarding the levels of CO in ambient air associated with health effects?" is not answered. The staff reiterates much of the uncertainty with the current state of CO pathophysiology but never offers a conclusion. Moreover, there are parts of this section that are unnecessarily convoluted (*e.g.* the paragraph on page 2-27, lines 14 – 22). The question posed on page 2-31, line 29, "To what extent have important uncertainties identified in the last review been reduced and/or have new uncertainties emerged?" is answered (page 2-35, line 12-19).

4. In section 2.2.2 the end of the first paragraph has the sentence: "These questions are intended to inform consideration of the following overarching question.", but no question stated. On page 2-40 two questions read, "What is the magnitude of ... COHb levels estimated to occur in areas [that] just meet the current CO standards" and "What proportion of the .... population experience maximum COHb levels above levels of potential health concern?" The answers to these questions are, for the most part, outlined in table 2-5 but there is no written summary. The question on page 2-42, "What are the key uncertainties associated with our exposure and dose estimates ... ?" This question is clearly answered in the ensuing paragraph. The question on page 2-43,"To what extent are the estimates of at-risk population COHb levels ....important from a

public health perspective?" is not answered. Instead, the staff state that the answer depends on public health policy (page 2-44, line 26). This is common sense and does not draw upon the scientific data outlined in the ISA.

To conclude, the section 2.2 starts with a question: "Does the currently available scientific evidence and exposure/risk-based information, as reflected in the ISA and draft REA, support or call into question the adequacy of the protection afforded by the current CO standards?". This is clearly important but it remains unanswered in the current policy assessment.

5. Section 2.2.3 is said to offer conclusions on the adequacy of the current standard. The first two paragraphs clearly outline the rational taken by the staff and why they give weight to the 8-hour standard (versus the 1-hour standard). The first three sentences of the third paragraph state what appear to be truisms and in the fourth sentence the "conclusion" is that the eight hour standard should be either retained or revised. Hence, there is no conclusion.

6. Section 2.3, considerations of alternative standards, is organized by posing a series of questions. The first question (page 2-46) is," To what extent does .... information ... support consideration of alternatives to the current CO standards ... ?" is broken down into sub-headings and more questions. Section 2.3.1 states the indicator for carbon monoxide is carbon monoxide (not sure this is really necessary). Alternatively, you fail to mention the issues outlined in ISA chapter 3. Might it be appropriate to mention that CO is an O_3 precursor and there is a localized chemical interdependency of the CO-CH_4-NO_x system, although these alternative products are not used in estimating local CO production? Section 2.3.2 is said to consider alternatives to the current averaging times of 1- and 8-hour exposures. A question (page 2-47) is then posed, "Do health effects ... assessments provide support for considering different exposure ... times?". It seems to me the answer is stated on page 2-24, line 4 (... retain the 1- and 8- hour averaging times) but then the staff back away from this in later sections. A new question is posed on page 2-48, "What is the range of alternative levels and forms for the standard ... ?" The ensuing paragraphs and sections discuss use of a 99^{th} percentile concentration-based form and the 'exceeded only once per year' form. Much of the discussion in the REA is recapitulated in the following pages and the 'conclusions', summarized in section 2.3.4, are that the standards could be either revised or retained. Hence, the document offers no conclusion. A minor comment on the tables 2-6 and 2-7 is uncertainty over the term 'level' in the second columns. I assume, but am unsure that 'level' refers to ppm of CO.

7. I think discussion of current and potential alternative standards is adequate. I have one last comment pertaining to the uncertainties sections of the staff analysis. This relates to the APEX modeling. The discussion in the REA document includes information that most fixed monitors have a 1 ppm CO lower detectable limit so the modelers added 0.5 ppm CO to all measured values to remove zeros and negative numbers thought to be related to monitor drift. It seems to me that this severely weakens estimates of the at-risk population and threshold COHb levels and thus contradicts consideration of changes from the current standards. However, I defer to other Review Panel members with modeling expertise on whether my concerns are valid.

8. I do not think the options listed by the staff are helpful. They merely state what was obvious before starting the entire review process – that is, the guidelines can be left as they are or they could be changed.

9. Section 3 pertaining to consideration of a secondary standard for CO concludes, I think justifiably, that the science does not support establishing a secondary standard. I think the level of detail presented is adequate.

Appendix A

Summary of 2003 NRC Report Recommendations

Summary of NRC Report[1] Recommendations

This report was requested to investigate the characteristics of CO in areas of the country with meteorological and topographical characteristics that exacerbate pollution and to address potential approaches to predicting, assessing, and managing episodes of high CO concentrations in such areas. A summarized list of the report recommendations is provided here.

Vulnerability to Future Violations

- Planning for worst-case combinations of emissions and meteorology, particularly in areas of high population growth and/or high meteorological variability and investigation of how large-scale and local meteorological/climatological phenomena can affect the susceptibility of a location to CO buildup in ambient air.

Health Effects:

- Emphasis of ambient CO issues through enhanced public-awareness campaigns. Further study of CO effects on the fetus, on CO effects separate from those of copollutants, and on the automobile exhaust mixture is encouraged.

Management and Control of CO:

- Federal/state assistance to communities with special CO problems and periodic reassessment of programs implemented to reduce CO emissions. Assessment of the relationship of CO to other pollutants, such as $PM_{2.5}$ and air toxics.
- Additional emissions testing of cold-start conditions.
- Targeting of high-emitting vehicles for repair or removal from the fleet and evaluation of the effectiveness of programs to control high-emitting vehicles.
- Review of the oxygenated fuels programs and implementation of cost-effective reductions to help areas in attaining/maintaining compliance with the NAAQS.
- Inclusion of public-health education components in local emissions-reduction programs and regular evaluation of their effectiveness.

CO Assessment

- Use of CO to represent distribution of other mobile-source pollutants, such as in hot spots identification, improvement of model representation of relationships between transportation activity and emissions, and approximation of concentrations of some motor-vehicle-related pollutants.[2]
- Use of several tools to better characterize CO spatial distribution, in particular, to better understand the upper end of the distribution of ambient exposures.
- Support from federal/other sources to continue monitoring operations in areas where CO concentrations are well below the standard.
- Continued model development in concert with improved monitoring.

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[1] Summarized from *Managing Carbon Monoxide Pollution in Meteorological and Topographical Problem Areas*. National Research Council of the National Academies of Science. April 2003. The report should be consulted for complete recommendations in context of the NRC analysis.

[2] The committee noted that CO is most useful as an indicator in microscale settings and less reliable in representing regional distributions of these pollutants.

Appendix B

Air Quality Data from Ambient Monitors Reporting CO Measurements in Geographical Areas of Key Epidemiological Studies During Periods of Study

Air quality information provided here is drawn from monitors reporting to the U.S. Air Quality System (AQS)[1], and for the Atlanta study, two monitors reporting to the SouthEastern Aerosol Research and Characterization study (SEARCH)[2] database. Available meta-data from AQS and SEARCH were used to create the monitor description tables for each monitor. Annual design values for each monitor are CO concentrations for the study area in the statistical form of the standards (i.e., second maximum, non-overlapping 8-hour average in a year and second maximum 1-hour average in a year). Annual statistics for each monitor were computed for each study period and area from available 1-hour data and summarized 8-hour daily maximum data. For the 126-county Bell et al (2009) study, the highest annual statistic reported by any monitor in the county was used.

[1] http://www.epa.gov/ttn/airs/airsaqs/

[2] The Southeastern Aerosol Research and Characterization (SEARCH) network data are publically available at the SEARCH website (http://www.atmospheric-research.com/studies/SEARCH/index.html). This information includes meterological, gaseous, and particle (filter and continuous) measurements from 1998 to the present.

Atlanta - January 1st 1993 to August 31st 2000
(Study area and time period for Metzger et al., 2004)

Monitor Description

Site ID	Street Address	Latitude	Longitude	Time Period	Tangent Road Number	Traffic Count	Tangent Road Name	Type Road	Year of Traffic Count
130891002	495 N Indian Crk Dr	33.789617	-84.235833	93-00	1	500	Unknown	Local St/Hy	-
131210099	4434 Roswell Rd	33.87647278	-84.3802857	8/4/94-00	1	44000	Roswell Rd	Maj St/Hy	1992
Search database	Jefferson St	33.776	-84.413	8/1/98-8/31/00	-	-	-	-	-

Atlanta - January 1st 1993 to August 31st 2000
(Study area and time period for Metzger et al., 2004)

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1993-2000 (current standard is 35 ppm)

SITE	1993	1994	1995	1996	1997	1998	1999	2000
130891002	6.5	7.1	6.3	5.5	5.4	4.8	6.3	4.6
131210099		6.6	16.3	6.6	5.8	5.0	5.1	4.3
search database						5.6	7.7	4.8
Maxperyear	6.5	7.1	16.3	6.6	5.8	5.6	7.7	4.8

* (of all 8760 possible 1-hr values in each year)

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1993-2000 (current standard is 9 ppm)

SITE	1993	1994	1995	1996	1997	1998	1999	2000
130891002	4.9	5.3	4.5	3.7	4.3	4.1	4.1	3.2
131210099		4.3	5.2	3.8	3.7	3.1	3.0	2.6
Maxperyear	4.9	5.3	5.2	3.8	4.3	4.1	4.1	3.2

Atlanta - January 1st 1993 to August 31st 2000
(Study area and time period for Metzger et al., 2004)

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	6.1	6.3	5.8	5.3	5.0	4.8	5.4	4.1		
131210099		6.6	7.9	6.1	5.4	4.9	4.9	3.8		
search database						5.6	7.2	4.4		
Maxperyear	6.1	6.6	7.9	6.1	5.4	5.6	7.2	4.4	7.9	***Total Max***
Minperyear	6.1	6.3	5.8	5.3	5.0	4.8	4.9	3.8	3.8	***Total Min***

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	1.7	1.6	1.6	1.5	1.3	1.5	1.2	1.1		
131210099		2.8	2.6	2.1	1.9	1.8	1.7	1.6		
search database						0.9	1.0	0.8		
Maxperyear	1.7	2.8	2.6	2.1	1.9	1.8	1.7	1.6	2.8	***Total Max***
Minperyear	1.7	1.6	1.6	1.5	1.3	0.9	1.0	0.8	0.8	***Total Min***

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	4.4	4.9	4.3	3.6	4.2	3.9	3.8	3.0		
131210099		4.3	4.9	3.5	3.1	2.8	3.0	2.4		
Maxperyear	4.4	4.9	4.9	3.6	4.2	3.9	3.8	3.0	4.9	***Total Max***
Minperyear	4.4	4.3	4.3	3.5	3.1	2.8	3.0	2.4	2.4	***Total Min***

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	1.2	1.2	1.1	1.1	1.0	1.2	0.9	0.7		
131210099		2.0	2.0	1.6	1.4	1.3	1.3	1.2		
Maxperyear	1.2	2.0	2.0	1.6	1.4	1.3	1.3	1.2	2.0	***Total Max***
Minperyear	1.2	1.2	1.1	1.1	1.0	1.2	0.9	0.7	0.7	***Total Min***

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	2.4	2.7	2.4	2.0	2.3	2.4	2.0	1.5		
131210099		3.3	3.2	2.7	2.4	2.1	2.3	1.8		
search database						2.0	2.6	1.5		
Maxperyear	2.4	3.3	3.2	2.7	2.4	2.4	2.6	1.8	3.3	***Total Max***
Minperyear	2.4	2.7	2.4	2.0	2.3	2.0	2.0	1.5	1.5	***Total Min***

24-hour - 50th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000		
130891002	0.7	0.7	0.6	0.6	0.7	0.8	0.5	0.4		
131210099		1.3	1.4	1.1	0.9	0.9	0.8	0.8		
search database						0.4	0.4	0.3		
Maxperyear	0.7	1.3	1.4	1.1	0.9	0.9	0.8	0.8	1.4	***Total Max***
Minperyear	0.7	0.7	0.6	0.6	0.7	0.4	0.4	0.3	0.3	***Total Min***

Atlanta-- January 1st 1993 through December 31st, 2004
(Study area and time period for Tolbert et al., 2007)

Monitor Description

Site ID	Street Address	Latitude	Longitude	Time Period	Tangent Road Number	Traffic Count	Tangent Road Name	Type Road	Year of Traffic Count
130891002	495 N Indian Crk Dr	33.789617	-84.235833	93-6/30/03	1	500	Unknown	Local St/Hy	-
131210099	4434 Roswell Rd	33.87647278	-84.3802857	8/4/94-04	1	44000	Roswell Rd	Maj St/Hy	1992
132230003	CRAWFORD ROAD	33.92855	-85.04548	7/16/02-04	1	6	Crawford Rd	Local St/Hy	1995
130890002	2390-B Wildcat Road, Decatur GA	33.68808	-84.29018	5/19/03-04	1	9250	Clifton Springs Rd/Wildcat Rd	Local St/Hy	2007
Search database	Jefferson St	33.776	-84.413	8/1/98-04	-	-	-	-	-

Atlanta-- January 1st 1993 through December 31st, 2004
(Study area and time period for Tolbert et al., 2007)

Percentage of Measurements below method MDL

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
130891002	33	38	41	46	34	21	52	64	38	45	64	
131210099		16	10	20	24	19	25	26	31	37	25	42
132230003										96	0	14
130890002											3	11

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1993-2004 (current standard is 35 ppm)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
130891002	6.5	7.1	6.3	5.5	5.4	4.8	6.3	4.6	5.3	4.6	2.9	
131210099		6.6	16.3	6.6	5.8	5.0	5.1	4.3	4.9	3.7	3.5	4.6
132230003										0.7	0.9	0.9
130890002											3.0	3.7
search database						5.6	7.7	5.7	5.9	5.9	4.5	4.4
Maxperyear	6.5	7.1	16.3	6.6	5.8	5.6	7.7	5.7	5.9	5.9	4.5	4.6

* (of all possible 8760 1-hr values in each year)

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1993-2004 (current standard is 9 ppm)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
130891002	4.9	5.3	4.5	3.7	4.3	4.1	4.1	3.2	4.1	3.6	2.0	
131210099		4.3	5.2	3.8	3.7	3.1	3.0	2.6	3.0	2.3	2.5	2.5
132230003										0.6	0.8	0.5
130890002											2.6	2.6
Maxperyear	4.9	5.3	5.2	3.8	4.3	4.1	4.1	3.2	4.1	3.6	2.6	2.6

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	6.1	6.3	5.8	5.3	5.0	4.8	5.4	4.1	4.3	3.6	2.6			
131210099		6.6	7.9	6.1	5.4	4.9	4.9	3.8	4.2	3.5	3.3	2.9		
132230003										0.7	0.9	0.7		
130890002											2.7	2.7		
search database						5.6	7.2	5.0	5.0	4.9	3.6	3.6		
Maxperyear	6.1	6.6	7.9	6.1	5.4	5.6	7.2	5.0	5.0	4.9	3.6	3.6	7.9	***Total Max***
Minperyear	6.1	6.3	5.8	5.3	5.0	4.8	4.9	3.8	4.2	0.7	0.9	0.7	0.7	***Total Min***

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	1.7	1.6	1.6	1.5	1.3	1.5	1.2	1.1	1.3	0.9	0.8			
131210099		2.8	2.6	2.1	1.9	1.8	1.7	1.6	1.4	1.2	1.2	1.0		
132230003										0.3	0.4	0.3		
130890002											1.0	0.9		
search database						0.9	1.0	0.9	0.8	0.7	0.7	0.7		
Maxperyear	1.7	2.8	2.6	2.1	1.9	1.8	1.7	1.6	1.4	1.2	1.2	1.0	2.8	***Total Max***
Minperyear	1.7	1.6	1.6	1.5	1.3	0.9	1.0	0.9	0.8	0.3	0.4	0.3	0.3	***Total Min***

Atlanta-- January 1st 1993 through December 31st, 2004
(Study area and time period for Tolbert et al., 2007)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	4.4	4.9	4.3	3.6	4.2	3.9	3.8	3.0	2.8	2.5	2.0			
131210099		4.3	4.9	3.5	3.1	2.8	3.0	2.4	2.3	2.2	2.1	2.0		
132230003										0.6	0.7	0.5		
130890002											2.5	2.4		
Maxperyear	4.4	4.9	4.9	3.6	4.2	3.9	3.8	3.0	2.8	2.5	2.5	2.4	4.9	***Total Max***
Minperyear	4.4	4.3	4.3	3.5	3.1	2.8	3.0	2.4	2.3	0.6	0.7	0.5	0.5	***Total Min***

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	1.2	1.2	1.1	1.1	1.0	1.2	0.9	0.7	0.9	0.8	0.6			
131210099		2.0	2.0	1.6	1.4	1.3	1.3	1.2	1.0	0.9	1.0	0.8		
132230003										0.3	0.4	0.3		
130890002											0.8	0.7		
Maxperyear	1.2	2.0	2.0	1.6	1.4	1.3	1.3	1.2	1.0	0.9	1.0	0.8	2.0	***Total Max***
Minperyear	1.2	1.2	1.1	1.1	1.0	1.2	0.9	0.7	0.9	0.3	0.4	0.3	0.3	***Total Min***

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	2.4	2.7	2.4	2.0	2.3	2.4	2.0	1.5	1.9	1.5	1.2			
131210099		3.3	3.2	2.7	2.4	2.1	2.3	1.8	1.8	1.6	1.7	1.5		
132230003										0.4	0.7	0.5		
130890002											1.6	1.5		
search database						2.0	2.6	2.3	1.8	1.6	1.5	1.4		
Maxperyear	2.4	3.3	3.2	2.7	2.4	2.4	2.6	2.3	1.9	1.6	1.7	1.5	3.3	***Total Max***
Minperyear	2.4	2.7	2.4	2.0	2.3	2.0	2.0	1.5	1.8	0.4	0.7	0.5	0.4	***Total Min***

24-hour - 50th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
130891002	0.7	0.7	0.6	0.6	0.7	0.8	0.5	0.4	0.6	0.5	0.4			
131210099		1.3	1.4	1.1	0.9	0.9	0.8	0.8	0.7	0.6	0.7	0.5		
132230003										0.3	0.3	0.3		
130890002											0.6	0.5		
search database						0.4	0.4	0.4	0.3	0.4	0.3	0.3		
Maxperyear	0.7	1.3	1.4	1.1	0.9	0.9	0.8	0.8	0.7	0.6	0.7	0.5	1.4	***Total Max***
Minperyear	0.7	0.7	0.6	0.6	0.7	0.4	0.4	0.4	0.3	0.3	0.3	0.3	0.3	***Total Min***

Denver-- January 1st 1993 through December 31st, 1997
(Study area and time period for Koken et al., 2003)

Monitor Description

Site ID	Street Address	Latitude	Longitude	Time Period	Tangent Road Number	Traffic Count	Tangent Road Name	Type Road	Year of Traffic Count
080310020	935 Colorado Blvd	39.731389	-104.940833	11/01/94-3/31/95	1	50000	Colorado Blvd	Maj St/Hy	1992
080310013	14th Ave and Albion St	39.738578	-104.939925	93-97	1	57000	Colorado Blvd	Maj St/Hy	-
080310018	1300 Blaje St - Blake side	39.748168	-105.002604	11/18/93-3/16/94	1	500	Blake St	Maj St/Hy	1992
080310014	2325 Irving St	39.751761	-105.030681	93-97	1	5000	Unknown	Thru St/Hy	-
080310019	1300 Blake St	39.748163	-105.002564	11/18/93-97	1	500	Speer Pkwy	Maj St/Hy	1993
080310002	2105 Broadway	39.751184	-104.987625	93-97	1	17200	Broadway	Maj St/Hy	1995

Denver-- January 1st 1993 through December 31st, 1997
(Study area and time period for Koken et al., 2003)

Percentage of Measurements below method MDL

SITE	1993	1994	1995	1996	1997
080310020		6	8		
080310013	30	22	12	18	19
080310018	7	5			
080310014	43	33	22	29	31
080310019	5	8	8	7	10
080310002	16	11	6	8	9

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1993-2004 (current standard is 35

SITE	1993	1994	1995	1996	1997
080310020		11.7	10.3		
080310013	14.9	12.2	13.6	9.4	10.6
080310018	15.3	11.6			
080310014	12.1	10.9	9.9	8.2	8.4
080310019	16.1	13.4	14.0	12.5	11.2
080310002	18.2	17.1	16.4	16.7	10.0
Maxperyear	18.2	17.1	16.4	16.7	11.2

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1993-

SITE	1993	1994	1995	1996	1997
080310020		6.8	6.0		
080310013	7.8	7.6	6.2	5.2	4.7
080310018	7.7	6.5			
080310014	8.2	7.3	5.9	5.7	6.2
080310019	7.7	8.2	7.1	7.0	6.4
080310002	10.4	8.2	9.5	7.3	5.5
Maxperyear	10.4	8.2	9.5	7.3	6.4

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997		
080310020		12.8	11.9				
080310013	12.8	11.3	10.1	9.0	7.9		
080310018	16.2	12.2					
080310014	9.8	9.6	8.1	7.3	7.7		
080310019	16.2	13.4	14.0	9.9	9.3		
080310002	13.5	13.8	13.1	12.8	8.8		
Maxperyear	16.2	13.8	14.0	12.8	9.3	16.2	***Total Max***
Minperyear	9.8	9.6	8.1	7.3	7.7	7.3	***Total Min***

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1993	1994	1995	1996	1997		
080310020		4.9	3.9				
080310013	2.7	2.9	2.8	2.5	2.4		
080310018	6.3	5.3					
080310014	2.2	2.5	2.3	2.2	2.0		
080310019	7.5	5.6	4.3	3.6	3.0		
080310002	3.2	3.1	2.9	2.7	2.6		
Maxperyear	7.5	5.6	4.3	3.6	3.0	7.5	***Total Max***
Minperyear	2.2	2.5	2.3	2.2	2.0	2.0	***Total Min***

Denver-- January 1st 1993 through December 31st, 1997
(Study area and time period for Koken et al., 2003)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997		
080310020		7.6	7.4				
080310013	6.5	6.7	5.3	5.1	4.7		
080310018	10.4	7.8					
080310014	6.9	6.2	5.7	5.4	5.6		
080310019	10.4	8.6	7.1	7.0	5.8		
080310002	8.7	8.2	7.3	7.2	5.1		
Maxperyear	10.4	8.6	7.4	7.2	5.8	10.4	***Total Max***
Minperyear	6.5	6.2	5.3	5.1	4.7	4.7	***Total Min***

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1993	1994	1995	1996	1997		
080310020		3.4	2.5				
080310013	1.7	1.8	1.9	1.7	1.6		
080310018	4.9	3.3					
080310014	1.2	1.5	1.5	1.4	1.3		
080310019	5.1	3.8	3.0	2.2	2.0		
080310002	2.0	1.9	2.0	1.8	1.7		
Maxperyear	5.1	3.8	3.0	2.2	2.0	5.1	***Total Max***
Minperyear	1.2	1.5	1.5	1.4	1.3	1.2	***Total Min***

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997		
080310020		4.9	5.0				
080310013	3.6	3.2	3.0	3.0	2.8		
080310018	6.0	4.7					
080310014	4.0	4.0	3.1	2.9	3.2		
080310019	5.5	5.2	4.5	3.6	3.8		
080310002	4.8	4.4	3.9	3.6	3.9		
Maxperyear	6.0	5.2	5.0	3.6	3.9	6.0	***Total Max***
Minperyear	3.6	3.2	3.0	2.9	2.8	2.8	***Total Min***

24-hour -50th percentile (of all 365 possible daily 24-hour averages)

SITE	1993	1994	1995	1996	1997		
080310020		2.1	1.6				
080310013	0.9	1.1	1.1	1.0	1.0		
080310018	2.6	2.0					
080310014	0.7	0.8	0.9	0.8	0.8		
080310019	2.7	2.1	1.7	1.4	1.3		
080310002	1.3	1.3	1.4	1.2	1.2		
Maxperyear	2.7	2.1	1.7	1.4	1.3	2.7	***Total Max***
Minperyear	0.7	0.8	0.9	0.8	0.8	0.7	***Total Min***

Pennsylvania-- January 1st 1987 through November 30th, 1999
(Study area and time period for Wellenius et al., 2005)

Monitor Description

Site ID	Street Address	Latitude	Longitude	Time Period	Tangent Road Number	Traffic Count	Tangent Road Name	Type Road	Year of Traffic Count
420030003	Harper Rd Evergreen Park Monroeville	40.45	-79.771111	3/16/87-4/30/93	-	-	-	-	-
420030010	Carnegie Scienct Center - 1 Allecheney Rd	40.4455765	-80.0161549	11/25/97-99	1	1000	Allegheny Rd	Maj St/Hy	1997
420030026	Oakland 4 416 Semple St	40.436667	-79.954722	87-10/26/97	1	2000	Unknown	Maj St/Hy	-
420030038	Forbes Ave and Grant St Pgh. PA 15219	40.438889	-79.997222	87-99	1	15000	Unknown	Maj St/Hy	-
420030052	Gateway Center Subway Entrance on Roof	40.441389	-80.003333	3/16/87-99	-	-	-	-	-

Allegheny County, PA-- January 1st 1987 through November 30th, 1999
(Study area and time period for Wellenius et al., 2005)

Percentage of Measurements below method MDL

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
420030003	9	43	32	42	45	55	61						
420030010											55	54	68
420030026	4	8	4	18	18	16	28	25	38	48	58		
420030038	3	7	11	11	9	13	4	9	8	12	17	8	17
420030052	0	7	10	9	4	6	12	31	25	23	22	25	39

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1993-2004 (current standard is 35 ppm)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
420030003	4.5	6.7	5.1	5.3	3.6	5.4	3.3						
420030010											4.2	3.5	3.3
420030026	18.0	12.6	9.0	11.0	6.3	9.0	6.1	7.3	6.2	7.0	4.6		
420030038	19.4	10.7	11.2	10.5	10.2	8.9	7.6	10.2	8.3	6.8	5.4	7.0	6.0
420030052	15.9	8.3	9.4	9.2	7.9	9.0	6.4	8.2	5.3	5.7	4.2	3.8	4.3
Maxperyear	19.4	12.6	11.2	11.0	10.2	9.0	7.6	10.2	8.3	7.0	5.4	7.0	6.0

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1993-2004 (current standard is 9 ppm)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999
420030003	3.1	2.8	2.8	2.9	2.2	2.6	1.3						
420030010											3.2	2.7	2.5
420030026	7.9	7.5	6.1	6.9	4.3	6.3	4.7	4.2	4.2	3.5	2.4		
420030038	8.8	6.6	7.8	8.1	5.3	6.9	5.4	7.0	5.9	4.3	3.8	3.8	3.9
420030052	7.9	6.1	6.6	6.0	6.0	5.6	4.7	4.5	3.7	3.9	2.9	3.1	2.6
Maxperyear	8.8	7.5	7.8	8.1	6.0	6.9	5.4	7.0	5.9	4.3	3.8	3.8	3.9

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	4.5	5.3	4.4	4.0	3.4	4.3	3.3								
420030010											5.0	3.0	2.9		
420030026	10.9	11.1	11.2	8.4	8.0	5.5	6.5	6.0	6.0	5.7	5.9	3.7			
420030038	12.7	9.5	10.4	9.9	8.0	8.3	7.5	8.4	7.2	5.8	5.0	4.3	5.6		
420030052	15.9	8.0	8.7	7.6	6.5	6.0	5.6	5.5	5.1	4.8	3.7	3.4	3.9		
Maxperyear	15.9	11.1	11.2	9.9	8.0	8.3	7.5	8.4	7.2	5.8	5.9	4.3	5.6	15.9	***Total Max***
Minperyear	4.5	5.3	4.4	4.0	3.4	4.3	3.3	5.5	5.1	4.8	3.7	3.0	2.9	2.9	***Total Min***

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	1.6	1.2	1.1	1.2	1.0	0.8	0.7								
420030010											1.0	1.0	0.9		
420030026	2.0	2.0	2.0	1.9	1.5	1.5	1.2	1.4	1.2	1.0	0.9				
420030038	4.2	3.1	3.0	2.8	2.7	2.6	2.7	2.4	2.3	2.1	1.8	1.9	1.7		
420030052	3.1	2.5	2.5	2.4	1.9	1.8	1.7	1.8	1.4	1.4	1.3	1.2	1.1		
Maxperyear	4.2	3.1	3.0	2.8	2.7	2.6	2.7	2.4	2.3	2.1	1.8	1.9	1.7	4.2	***Total Max***
Minperyear	1.6	1.2	1.1	1.2	1.0	0.8	0.7	1.4	1.2	1.0	0.9	1.0	0.9	0.7	***Total Min***

Allegheny County, PA-- January 1st 1987 through November 30th, 1999
(Study area and time period for Wellenius et al., 2005)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	3.1	2.6	2.4	2.6	2.0	2.0	1.3								
420030010											3.5	2.6	2.2		
420030026	7.1	6.0	5.8	6.1	3.9	4.4	4.3	4.2	3.9	3.4	2.3				
420030038	8.0	6.1	7.0	5.7	5.2	6.9	5.2	6.9	5.4	4.2	3.7	3.3	3.5		
420030052	7.9	5.1	5.6	5.5	4.4	5.5	4.3	4.2	3.7	3.8	2.7	3.0	2.6		
Maxperyear	8.0	6.1	7.0	6.1	5.2	6.9	5.2	6.9	5.4	4.2	3.7	3.3	3.5	8.0	***Total Max***
Minperyear	3.1	2.6	2.4	2.6	2.0	2.0	1.3	4.2	3.7	3.4	2.3	2.6	2.2	1.3	***Total Min***

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	1.3	0.8	0.9	0.8	0.8	0.6	0.5								
420030010											0.8	0.8	0.6		
420030026	1.5	1.5	1.5	1.4	1.2	1.2	1.0	1.0	0.9	0.8	0.7				
420030038	3.1	2.3	2.2	2.1	2.1	1.9	2.0	1.9	1.7	1.7	1.3	1.5	1.3		
420030052	2.3	1.8	1.8	1.8	1.5	1.4	1.3	1.2	1.1	1.0	1.0	1.0	0.8		
Maxperyear	3.1	2.3	2.2	2.1	2.1	1.9	2.0	1.9	1.7	1.7	1.3	1.5	1.3	3.1	***Total Max***
Minperyear	1.3	0.8	0.9	0.8	0.8	0.6	0.5	1.0	0.9	0.8	0.7	0.8	0.6	0.5	***Total Min***

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	1.9	1.7	1.7	1.9	1.3	1.4	0.9								
420030010											2.8	1.5	1.5		
420030026	4.1	4.2	3.6	3.3	2.4	2.5	2.6	2.6	2.4	2.0	1.3				
420030038	5.5	3.3	4.9	4.2	3.7	4.0	4.0	4.0	3.4	2.8	2.5	2.5	2.3		
420030052	5.6	3.0	4.3	3.7	3.4	2.9	3.4	3.3	2.5	2.5	1.9	2.1	1.9		
Maxperyear	5.6	4.2	4.9	4.2	3.7	4.0	4.0	4.0	3.4	2.8	2.5	2.5	2.3	5.6	***Total Max***
Minperyear	1.9	1.7	1.7	1.9	1.3	1.4	0.9	2.6	2.4	2.0	1.3	1.5	1.5	0.9	***Total Min***

24-hour -50th percentile (of all 365 possible daily

SITE	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999		
420030003	0.9	0.6	0.6	0.6	0.5	0.4	0.4								
420030010											0.4	0.5	0.4		
420030026	1.1	1.1	1.1	1.0	0.9	0.8	0.7	0.7	0.6	0.5	0.4				
420030038	2.1	1.5	1.4	1.4	1.4	1.2	1.4	1.3	1.2	1.1	0.9	1.1	0.9		
420030052	1.7	1.3	1.3	1.3	1.1	1.0	0.9	0.8	0.7	0.8	0.7	0.7	0.6		
Maxperyear	2.1	1.5	1.4	1.4	1.4	1.2	1.4	1.3	1.2	1.1	0.9	1.1	0.9	2.1	***Total Max***
Minperyear	0.9	0.6	0.6	0.6	0.5	0.4	0.4	0.7	0.6	0.5	0.4	0.5	0.4	0.4	***Total Min***

Baltimore-- April 1st 2002 through December 31st, 2002
(Study area and time period for Symons et al., 2006)

Monitor Description

Site ID	Street Address	Latitude	Longitude	Time Period	Tangent Road Number	Traffic Count	Tangent Road Name	Type Road	Year of Traffic Count
240053001	600 Dorsey Avenue	39.310833	-76.474444	2002	1	500	Woodward	Local St/Hy	1993
245100040	Oldtown Fire Station, 1100 Hillen Street	39.298056	-76.604722	2002	1	15300	Hillen St	Thru St/Hy	1990

Baltimore-- April 1st 2002 through December 31st, 2002
(Study area and time period for Symons et al., 2006)

Percentage of Measurements below method MDL

Baltimore	2002	
240053001	61	county
245100040	43	city

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1993-2004 (current standard is 35 ppm)

Baltimore	2002	
240053001	9.2	county
245100040	4.4	city

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1993-2004 (current standard is 9 ppm)

Baltimore	2002	
240053001	2.6	county
245100040	3	city

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

Baltimore	2002	
240053001	4.7	county
245100040	3.9	city

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

Baltimore	2002	
240053001	0.9	county
245100040	0.9	city

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

Baltimore	2002	
240053001	2.4	county
245100040	2.8	city

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

Baltimore	2002	
240053001	0.60	county
245100040	0.70	city

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

Baltimore	2002	
240053001	1.46	county
245100040	1.86	city

24-hour -50th percentile (of all 365 possible daily 24-hour averages)

Baltimore	2002	
240053001	0.4	county
245100040	0.5	city

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1992-1995 (current standard is 35 ppm)

	California	1992	1993	1994	1995
Los Angeles	060370002	6.0	6.0	6.4	7.3
	060370113	8.0	8.0	7.9	7.4
	060371002	13.0	11.0	12.1	12.5
	060371103	11.0	9.0	10.7	9.2
	060371201	12.0	10.0	12.8	11.8
	060371301	25.0	20.0	20.8	16.5
	060371601	11.0	8.0	9.9	9.3
	060371701	12.0	8.0	9.8	7.7
	060372005	11.0	10.0	12.3	11.4
	060374002	10.0	9.0	11.5	8.1
	060375001	18.0	14.0	13.9	11.1
	060376002	8.0	7.0	7.5	6.5
	060379002	9.0	8.0	9.0	6.8
Orange	060590001	15.0	15.0	12.0	9.8
	060591003	11.0	9.0	9.5	7.5
	060592001	9.0	7.0	7.8	6.0
	060595001	18.0	14.0	16.1	11.5
Riverside	060650006	5.0	2.0		
	060651003	10.0	9.0	11.0	9.0
	060655001	4.0	4.0	3.5	3.1
	060658001	6.0	8.0	7.6	6.7
San Bernadino	060710001	6.0	4.0	3.5	3.1
	060710006		2.0	2.0	
	060710012	3.0	2.0	1.8	1.7
	060710014		4.0	3.7	3.1
	060710017			5.5	3.3
	060714001	5.0	4.0	3.4	3.0
	060719004	7.0	7.0	7.5	7.4
Maxperyear		25.0	20.0	20.8	16.5

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1992-1995 (current standard is 9 ppm)

	California	1992	1993	1994	1995
Los Angeles	060370002	4.3	4.0	4.4	6.2
	060370113	5.7	4.6	5.8	5.6
	060371002	9.8	8.1	10.2	11.0
	060371103	8.0	6.7	8.4	7.9
	060371201	8.1	8.0	9.9	9.4
	060371301	16.4	13.8	16.0	11.6
	060371601	7.7	6.3	8.4	7.6
	060371701	6.9	5.1	6.3	6.0
	060372005	7.1	6.3	7.7	8.6
	060374002	7.3	6.9	7.6	6.2
	060375001	11.3	9.6	11.3	8.7
	060376002	3.7	3.8	3.8	3.8
	060379002	5.3	5.3	5.1	4.5
Orange	060590001	8.6	6.6	8.1	7.3
	060591003	8.3	6.7	7.8	5.3
	060592001	5.0	3.9	5.4	3.9
	060595001	8.0	6.0	8.0	6.4
Riverside	060650006	3.6	1.8		
	060651003	6.0	5.8	6.0	5.8
	060655001	2.0	1.8	1.7	1.5
	060658001	4.6	5.3	5.6	5.2
San Bernadino	060710001	3.3	2.6	2.1	2.1
	060710006		2.0	1.9	
	060710012	1.3	1.3	1.4	1.3
	060710014		2.6	2.0	2.4
	060710017			2.6	1.8
	060714001	3.1	3.0	2.1	2.0
	060719004	5.1	5.0	5.5	5.9
Maxperyear		16.4	13.8	16.0	11.6

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

	California	1992	1993	1994	1995		
Los Angeles	060370002	6.0	6.0	5.5	6.9		
	060370113	7.0	7.0	7.6	6.5		
	060371002	12.0	10.0	11.4	11.6		
	060371103	10.0	8.0	10.0	9.0		
	060371201	12.0	10.0	11.0	10.4		
	060371301	21.0	19.0	18.0	16.1		
	060371601	10.0	8.0	9.8	9.2		
	060371701	10.0	8.0	8.8	7.3		
	060372005	10.0	9.0	10.9	10.1		
	060374002	9.0	9.0	9.0	7.4		
	060375001	15.0	13.0	12.8	11.0		
	060376002	7.0	7.0	6.9	6.3		
	060379002	8.0	7.0	7.6	6.1		
Orange	060590001	12.0	9.0	11.0	9.0		
	060591003	10.0	8.0	9.1	7.3		
	060592001	7.0	7.0	7.1	5.4		
	060595001	16.0	13.0	14.5	10.7		
Riverside	060650006	5.0	2.0				
	060651003	10.0	9.0	10.0	8.7		
	060655001	4.0	3.0	2.9	2.9		
	060658001	6.0	7.0	6.5	6.3		
San Bernadino	060710001	5.0	3.0	3.5	2.9		
	060710006		2.0	2.1			
	060710012	2.0	2.0	1.7	1.5		
	060710014		4.0	3.7	3.1		
	060710017			2.8	3.3		
	060714001	4.0	3.0	2.9	2.9		
	060719004	7.0	6.0	6.6	7.1		
Maxperyear		21.0	19.0	18.0	16.1	21.0	***Total Max***
Minperyear		2.0	2.0	1.7	1.5	1.5	***Total Min***

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

	California	1992	1993	1994	1995		
Los Angeles	060370002	2.0	2.0	2.1	1.9		
	060370113	3.0	2.0	2.5	2.0		
	060371002	3.0	3.0	3.3	3.2		
	060371103	4.0	3.0	3.8	3.1		
	060371201	3.0	3.0	3.1	1.9		
	060371301	4.0	4.0	4.4	3.8		
	060371601	3.0	3.0	3.1	2.7		
	060371701	3.0	3.0	3.4	2.8		
	060372005	3.0	3.0	2.8	2.7		
	060374002	2.0	2.0	2.3	1.7		
	060375001	3.0	3.0	3.2	2.6		
	060376002	2.0	2.0	2.6	2.1		
	060379002	2.0	2.0	2.0	1.7		
Orange	060590001	2.0	2.0	2.3	2.0		
	060591003	2.0	1.0	1.6	1.1		
	060592001	2.0	2.0	1.6	1.4		
	060595001	3.0	3.0	2.9	2.4		
Riverside	060650006	1.0	1.0				
	060651003	3.0	3.0	4.0	3.0		
	060655001	1.0	1.0	0.9	0.9		
	060658001	2.0	2.0	2.7	2.3		
San Bernadino	060710001	1.0	1.0	0.7	0.9		
	060710006		1.0	0.9			
	060710012	1.0	1.0	0.7	0.5		
	060710014		1.0	1.2	0.9		
	060710017			0.9	1.3		
	060714001	2.0	2.0	1.4	1.2		
	060719004	2.0	2.0	2.0	2.0		
Maxperyear		4.0	4.0	4.4	3.8	4.4	***Total Max***
Minperyear		1.0	1.0	0.7	0.5	0.5	***Total Min***

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

	California	1992	1993	1994	1995		
Los Angeles	060370002	4.0	3.9	4.1	5.3		
	060370113	5.3	4.6	5.3	4.7		
	060371002	9.4	7.5	9.9	9.5		
	060371103	7.9	6.3	8.0	7.6		
	060371201	7.8	7.6	9.5	8.8		
	060371301	14.4	13.0	14.2	11.2		
	060371601	7.4	6.0	8.0	7.0		
	060371701	6.4	5.0	6.3	5.5		
	060372005	7.0	5.9	7.5	8.4		
	060374002	6.9	6.7	6.8	5.8		
	060375001	10.9	9.1	9.8	8.5		
	060376002	3.3	3.6	3.7	3.8		
	060379002	5.0	5.1	5.0	4.5		
Orange	060590001	8.3	6.0	7.7	6.5		
	060591003	8.1	6.4	6.7	4.8		
	060592001	4.3	3.9	4.8	3.8		
	060595001	7.3	6.0	7.5	6.3		
Riverside	060650006	3.5	1.8				
	060651003	5.8	5.4	5.9	5.6		
	060655001	2.0	1.5	1.6	1.4		
	060658001	4.5	4.9	5.4	4.9		
San Bernadino	060710001	3.0	2.6	2.1	2.0		
	060710006		2.0	1.9			
	060710012	1.1	1.3	1.4	1.3		
	060710014		2.3	1.9	2.4		
	060710017			2.3	2.0		
	060714001	3.1	2.3	2.0	1.9		
	060719004	4.5	4.9	5.2	5.7		
Maxperyear		14.4	13.0	14.2	11.2	14.4	***Total Max***
Minperyear		1.1	1.3	1.4	1.3	1.1	***Total Min***

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

	California	1992	1993	1994	1995		
Los Angeles	060370002	1.6	1.4	1.6	1.5		
	060370113	1.6	1.7	1.7	1.2		
	060371002	2.6	2.4	2.6	2.7		
	060371103	2.9	2.4	2.8	2.4		
	060371201	2.0	2.0	2.3	1.2		
	060371301	2.9	2.7	3.0	2.8		
	060371601	2.1	2.0	2.2	2.0		
	060371701	2.6	2.4	2.5	1.9		
	060372005	2.1	2.0	2.2	2.2		
	060374002	1.7	1.7	1.8	1.3		
	060375001	2.0	1.8	1.8	1.6		
	060376002	1.6	1.5	1.8	1.4		
	060379002	1.4	1.3	1.2	1.1		
Orange	060590001	1.7	1.6	1.6	1.5		
	060591003	1.2	1.0	0.9	0.7		
	060592001	1.5	1.3	1.1	1.0		
	060595001	2.0	1.9	2.0	1.7		
Riverside	060650006	1.0	1.0				
	060651003	2.6	2.4	2.6	2.3		
	060655001	1.0	1.0	0.7	0.7		
	060658001	1.6	1.4	1.8	1.6		
San Bernadino	060710001	1.0	1.0	0.5	0.7		
	060710006		0.9	0.7			
	060710012	1.0	0.9	0.6	0.4		
	060710014		1.0	0.8	0.6		
	060710017			0.7	1.0		
	060714001	1.9	1.4	1.0	0.9		
	060719004	1.4	1.5	1.6	1.5		
Maxperyear		2.9	2.7	3.0	2.8	3.0	***Total Max***
Minperyear		1.0	0.9	0.5	0.4	0.4	***Total Min***

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

	California	1992	1993	1994	1995		
Los Angeles	060370002	3.0	3.0	2.9	4.1		
	060370113	3.1	2.9	3.3	3.3		
	060371002	5.9	4.9	6.6	6.8		
	060371103	5.0	4.3	5.4	5.4		
	060371201	4.6	5.0	5.5	5.7		
	060371301	9.6	8.0	8.9	7.6		
	060371601	4.6	4.0	5.3	5.2		
	060371701	4.2	3.5	4.2	4.2		
	060372005	3.9	3.5	4.7	4.9		
	060374002	4.8	4.1	4.7	3.7		
	060375001	5.9	5.2	5.8	4.9		
	060376002	2.3	2.3	2.5	2.7		
	060379002	3.1	2.7	2.6	2.4		
Orange	060590001	4.4	3.7	4.8	4.7		
	060591003	4.8	3.4	4.0	3.4		
	060592001	3.0	2.7	3.5	2.4		
	060595001	5.0	3.8	5.4	4.5		
Riverside	060650006	2.6	1.2				
	060651003	4.0	4.0	4.1	4.2		
	060655001	1.4	1.2	1.0	1.0		
	060658001	2.7	2.9	3.6	3.5		
San Bernadino	060710001	2.1	1.9	1.2	1.2		
	060710006		1.7	1.8			
	060710012	1.0	1.1	1.1	1.0		
	060710014		1.7	1.5	2.1		
	060710017			1.8	1.5		
	060714001	2.8	2.0	1.5	1.4		
	060719004	2.8	3.0	3.3	3.8		
Maxperyear		9.6	8.0	8.9	7.6	9.6	***Total Max***
Minperyear		1.0	1.1	1.0	1.0	1.0	***Total Min***

California-- January 1st 1992 through December 31st, 1995
(Study area and time period for Linn et al., 2000)

24-hour -50th percentile (of all 365 possible daily 24-hour averages)

	California	1992	1993	1994	1995		
Los Angeles	060370002	1.2	1.0	1.2	1.1		
	060370113	1.0	1.0	0.9	0.7		
	060371002	1.7	1.7	1.8	1.9		
	060371103	1.7	1.6	1.9	1.6		
	060371201	1.4	1.3	1.5	0.8		
	060371301	1.9	1.9	2.2	2.0		
	060371601	1.5	1.3	1.5	1.5		
	060371701	1.7	1.7	1.8	1.2		
	060372005	1.5	1.5	1.5	1.6		
	060374002	1.2	1.2	1.2	0.9		
	060375001	1.1	0.9	0.9	1.0		
	060376002	1.1	1.0	1.2	1.0		
	060379002	1.0	0.9	0.8	0.7		
Orange	060590001	1.2	1.1	1.1	1.1		
	060591003	0.8	0.7	0.5	0.4		
	060592001	1.1	1.0	0.7	0.6		
	060595001	1.3	1.3	1.3	1.2		
Riverside	060650006	0.9	0.6				
	060651003	1.8	1.7	1.9	1.6		
	060655001	0.6	0.6	0.4	0.4		
	060658001	1.0	0.9	1.1	0.9		
San Bernadino	060710001	1.0	0.6	0.3	0.4		
	060710006		0.5	0.6			
	060710012	0.5	0.5	0.3	0.3		
	060710014		0.8	0.5	0.4		
	060710017			0.5	0.7		
	060714001	1.3	1.1	0.7	0.6		
	060719004	1.0	1.1	1.2	1.2		
Maxperyear		1.9	1.9	2.2	2.0	2.2	***Total Max***
Minperyear		0.5	0.5	0.3	0.3	0.3	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1988-1995 (current standard is 35 ppm)

	California	1988	1989	1990	1991	1992	1993	1994	1995
Los Angeles	060370002	8.0	7.0	7.0	7.0	6.0	6.0	6.4	7.3
	060370113	12.0	12.0	11.0	9.0	8.0	8.0	7.9	7.4
	060371002	14.0	19.0	15.0	12.0	13.0	11.0	12.1	12.5
	060371103	15.0	14.0	13.0	11.0	11.0	9.0	10.7	9.2
	060371201	15.0	16.0	18.0	16.0	12.0	10.0	12.8	11.8
	060371301	32.0	28.0	23.0	29.0	25.0	20.0	20.8	16.5
	060371601		12.0	12.0	11.0	11.0	8.0	9.9	9.3
	060371701		11.0	11.0	10.0	12.0	8.0	9.8	7.7
	060372005		13.0	15.0	13.0	11.0	10.0	12.3	11.4
	060374002	12.0	12.0	10.0	12.0	10.0	9.0	11.5	8.1
	060375001	21.0	22.0	17.0	15.0	18.0	14.0	13.9	11.1
	060376002		9.0	10.0	8.0	8.0	7.0	7.5	6.5
	060379002			10.0	10.0	9.0	8.0	9.0	6.8
Orange	060590001	16.0	17.0	15.0	13.0	15.0	15.0	12.0	9.8
	060591002	16.0	13.0						
	060591003			13.0	10.0	11.0	9.0	9.5	7.5
	060592001	9.0	8.0	8.0	8.0	9.0	7.0	7.8	6.0
	060595001	20.0	23.0	18.0	16.0	18.0	14.0	16.1	11.5
Riverside	060650006				5.0	5.0	2.0		
	060651003	11.0	13.0	15.0	12.0	10.0	9.0	11.0	9.0
	060655001		6.0	5.0	4.0	4.0	4.0	3.5	3.1
	060658001	9.0	12.0	9.0	8.0	6.0	8.0	7.6	6.7
San Bernadino	060710001	9.0	6.0	5.0	10.0	6.0	4.0	3.5	3.1
	060710006						2.0	2.0	
	060710012	8.0	3.0	8.0	2.0	3.0	2.0	1.8	1.7
	060710014						4.0	3.7	3.1
	060710017							5.5	3.3
	060711004	8.0	7.0	8.0	7.0				
	060712002	8.0	7.0	6.0	5.0				
	060714001	6.0	6.0	7.0	5.0	5.0	4.0	3.4	3.0
	060719004	9.0	10.0	9.0	8.0	7.0	7.0	7.5	7.4
Maxperyear		32.0	28.0	23.0	29.0	25.0	20.0	20.8	16.5

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1988-1995 (current standard is 9 ppm)

	California	1988	1989	1990	1991	1992	1993	1994	1995
Los Angeles	060370002	5.4	5.4	5.0	5.3	4.3	4.0	4.4	6.2
	060370113	6.7	7.8	6.6	6.0	5.7	4.6	5.8	5.6
	060371002	11.7	11.6	11.6	10.6	9.8	8.1	10.2	11.0
	060371103	10.4	9.4	8.8	8.7	8.0	6.7	8.4	7.9
	060371201	11.9	12.0	13.4	12.3	8.1	8.0	9.9	9.4
	060371301	23.4	18.3	15.9	15.7	16.4	13.8	16.0	11.6
	060371601		9.3	8.4	8.9	7.7	6.3	8.4	7.6
	060371701		7.1	6.4	6.8	6.9	5.1	6.3	6.0
	060372005		8.4	8.7	9.4	7.1	6.3	7.7	8.6
	060374002	9.4	9.6	8.9	7.8	7.3	6.9	7.6	6.2
	060375001	15.6	14.4	12.1	11.1	11.3	9.6	11.3	8.7
	060376002		4.9	4.5	4.8	3.7	3.8	3.8	3.8
	060379002			6.9	7.0	5.3	5.3	5.1	4.5
Orange	060590001	10.4	10.9	8.7	8.6	8.6	6.6	8.1	7.3
	060591002	10.0	9.0						
	060591003			9.9	7.1	8.3	6.7	7.8	5.3
	060592001	4.9	4.8	5.1	4.5	5.0	3.9	5.4	3.9
	060595001	9.8	10.3	9.4	7.6	8.0	6.0	8.0	6.4
Riverside	060650006				2.9	3.6	1.8		
	060651003	7.1	8.1	7.3	6.8	6.0	5.8	6.0	5.8
	060655001		2.4	2.0	2.4	2.0	1.8	1.7	1.5
	060658001	5.9	7.9	5.9	6.1	4.6	5.3	5.6	5.2
San Bernadino	060710001	3.3	3.6	3.8	8.1	3.3	2.6	2.1	2.1
	060710006						2.0	1.9	
	060710012	3.7	2.0	3.0	2.0	1.3	1.3	1.4	1.3
	060710014						2.6	2.0	2.4
	060710017							2.6	1.8
	060711004	4.9	5.3	5.8	4.3				
	060712002	4.6	5.6	4.4	4.1				
	060714001	3.6	3.4	3.1	3.7	3.1	3.0	2.1	2.0
	060719004	7.0	8.0	6.0	6.9	5.1	5.0	5.5	5.9
Maxperyear		23.4	18.3	15.9	15.7	16.4	13.8	16.0	11.6

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	7.0	7.0	6.0	7.0	6.0	6.0	5.5	6.9		
	060370113	11.0	10.0	8.0	8.0	7.0	7.0	7.6	6.5		
	060371002	14.0	18.0	14.0	12.0	12.0	10.0	11.4	11.6		
	060371103	13.0	13.0	10.0	10.0	10.0	8.0	10.0	9.0		
	060371201	13.0	14.0	14.0	13.0	12.0	10.0	11.0	10.4		
	060371301	31.0	25.0	22.0	21.0	21.0	19.0	18.0	16.1		
	060371601		12.0	11.0	10.0	10.0	8.0	9.8	9.2		
	060371701		11.0	10.0	10.0	10.0	8.0	8.8	7.3		
	060372005		12.0	10.0	11.0	10.0	9.0	10.9	10.1		
	060374002	11.0	12.0	10.0	10.0	9.0	9.0	9.0	7.4		
	060375001	21.0	19.0	15.0	14.0	15.0	13.0	12.8	11.0		
	060376002		9.0	9.0	8.0	7.0	7.0	6.9	6.3		
	060379002			9.0	9.0	8.0	7.0	7.6	6.1		
Orange	060590001	15.0	14.0	11.0	12.0	12.0	9.0	11.0	9.0		
	060591002	12.0	13.0								
	060591003			11.0	10.0	10.0	8.0	9.1	7.3		
	060592001	8.0	8.0	8.0	7.0	7.0	7.0	7.1	5.4		
	060595001	19.0	22.0	18.0	15.0	16.0	13.0	14.5	10.7		
Riverside	060650006				5.0	5.0	2.0				
	060651003	11.0	12.0	13.0	11.0	10.0	9.0	10.0	8.7		
	060655001		4.0	4.0	4.0	4.0	3.0	2.9	2.9		
	060658001	9.0	9.0	7.0	8.0	6.0	7.0	6.5	6.3		
San Bernadino	060710001	7.0	5.0	5.0	9.0	5.0	3.0	3.5	2.9		
	060710006						2.0	2.1			
	060710012	4.0	2.0	5.0	2.0	2.0	2.0	1.7	1.5		
	060710014						4.0	3.7	3.1		
	060710017							2.8	3.3		
	060711004	7.0	7.0	7.0	7.0						
	060712002	6.0	6.0	6.0	5.0						
	060714001	5.0	5.0	5.0	5.0	4.0	3.0	2.9	2.9		
	060719004	9.0	9.0	8.0	7.0	7.0	6.0	6.6	7.1		
Maxperyear		31.0	25.0	22.0	21.0	21.0	19.0	18.0	16.1	31.0	***Total Max***
Minperyear		4.0	2.0	4.0	2.0	2.0	2.0	1.7	1.5	1.5	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

1-hour - 50th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	2.0	2.0	2.0	2.0	2.0	2.0	2.1	1.9		
	060370113	3.0	3.0	3.0	2.0	3.0	2.0	2.5	2.0		
	060371002	4.0	4.0	4.0	3.0	3.0	3.0	3.3	3.2		
	060371103	4.0	4.0	4.0	4.0	4.0	3.0	3.8	3.1		
	060371201	3.0	3.0	3.0	3.0	3.0	3.0	3.1	1.9		
	060371301	5.0	5.0	5.0	4.0	4.0	4.0	4.4	3.8		
	060371601		3.0	3.0	3.0	3.0	3.0	3.1	2.7		
	060371701		4.0	4.0	4.0	3.0	3.0	3.4	2.8		
	060372005		3.0	3.0	3.0	3.0	3.0	2.8	2.7		
	060374002	2.0	2.0	3.0	2.0	2.0	2.0	2.3	1.7		
	060375001	3.0	3.0	4.0	3.0	3.0	3.0	3.2	2.6		
	060376002		3.0	2.0	2.0	2.0	2.0	2.6	2.1		
	060379002			2.0	2.0	2.0	2.0	2.0	1.7		
Orange	060590001	3.0	2.0	3.0	2.0	2.0	2.0	2.3	2.0		
	060591002	2.0	4.0								
	060591003			1.0	1.0	2.0	1.0	1.6	1.1		
	060592001	2.0	2.0	2.0	2.0	2.0	2.0	1.6	1.4		
	060595001	4.0	4.0	3.0	3.0	3.0	3.0	2.9	2.4		
Riverside	060650006				3.0	1.0	1.0				
	060651003	4.0	4.0	4.0	3.0	3.0	3.0	4.0	3.0		
	060655001		1.0	1.0	1.0	1.0	1.0	0.9	0.9		
	060658001	2.0	3.0	3.0	2.0	2.0	2.0	2.7	2.3		
San Bernadino	060710001	2.0	1.0	2.0	2.0	1.0	1.0	0.7	0.9		
	060710006						1.0	0.9			
	060710012	1.0	1.0	1.0	1.0	1.0	1.0	0.7	0.5		
	060710014						1.0	1.2	0.9		
	060710017							0.9	1.3		
	060711004	3.0	3.0	3.0	3.0						
	060712002	2.0	2.0	2.0	2.0						
	060714001	2.0	2.0	2.0	2.0	2.0	2.0	1.4	1.2		
	060719004	3.0	3.0	3.0	2.0	2.0	2.0	2.0	2.0		
Maxperyear		5.0	5.0	5.0	4.0	4.0	4.0	4.4	3.8	5.0	***Total Max***
Minperyear		1.0	1.0	1.0	1.0	1.0	1.0	0.7	0.5	0.5	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	4.5	4.9	4.4	5.1	4.0	3.9	4.1	5.3		
	060370113	6.6	7.3	6.0	5.6	5.3	4.6	5.3	4.7		
	060371002	10.9	11.3	11.3	10.4	9.4	7.5	9.9	9.5		
	060371103	9.8	9.4	8.1	8.6	7.9	6.3	8.0	7.6		
	060371201	10.7	11.6	12.3	11.9	7.8	7.6	9.5	8.8		
	060371301	19.1	18.0	15.3	13.6	14.4	13.0	14.2	11.2		
	060371601		8.9	8.1	8.3	7.4	6.0	8.0	7.0		
	060371701		7.1	6.1	6.8	6.4	5.0	6.3	5.5		
	060372005		8.1	7.6	8.6	7.0	5.9	7.5	8.4		
	060374002	8.9	8.7	8.5	7.1	6.9	6.7	6.8	5.8		
	060375001	13.8	12.4	11.7	10.3	10.9	9.1	9.8	8.5		
	060376002		4.5	4.3	4.8	3.3	3.6	3.7	3.8		
	060379002			6.6	6.5	5.0	5.1	5.0	4.5		
Orange	060590001	9.7	10.7	8.6	8.3	8.3	6.0	7.7	6.5		
	060591002	8.9	9.7								
	060591003			9.4	7.0	8.1	6.4	6.7	4.8		
	060592001	4.4	4.5	4.1	4.5	4.3	3.9	4.8	3.8		
	060595001	9.3	10.0	8.6	7.4	7.3	6.0	7.5	6.3		
Riverside	060650006				2.9	3.5	1.8				
	060651003	6.1	7.8	6.3	6.4	5.8	5.4	5.9	5.6		
	060655001		2.1	1.9	1.9	2.0	1.5	1.6	1.4		
	060658001	5.8	7.1	5.3	5.1	4.5	4.9	5.4	4.9		
San Bernadino	060710001	3.3	3.5	3.7	7.3	3.0	2.6	2.1	2.0		
	060710006						2.0	1.9			
	060710012	2.9	2.0	2.0	2.0	1.1	1.3	1.4	1.3		
	060710014						2.3	1.9	2.4		
	060710017							2.3	2.0		
	060711004	4.8	5.0	4.0	4.4						
	060712002	4.5	4.9	4.1	4.1						
	060714001	3.6	3.0	3.1	3.7	3.1	2.3	2.0	1.9		
	060719004	7.0	7.4	5.8	6.5	4.5	4.9	5.2	5.7		
Maxperyear		19.1	18.0	15.3	13.6	14.4	13.0	14.2	11.2	19.1	***Total Max***
Minperyear		2.9	2.0	1.9	1.9	1.1	1.3	1.4	1.3	1.1	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

8-hour -50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	1.4	1.8	1.5	1.6	1.6	1.4	1.6	1.5		
	060370113	2.0	1.8	1.9	1.6	1.6	1.7	1.7	1.2		
	060371002	3.0	3.1	2.6	2.4	2.6	2.4	2.6	2.7		
	060371103	3.0	2.9	2.6	2.6	2.9	2.4	2.8	2.4		
	060371201	2.1	1.9	2.4	2.1	2.0	2.0	2.3	1.2		
	060371301	3.3	3.0	2.9	2.6	2.9	2.7	3.0	2.8		
	060371601		2.1	2.1	2.0	2.1	2.0	2.2	2.0		
	060371701		3.0	2.8	2.7	2.6	2.4	2.5	1.9		
	060372005		2.6	2.3	2.0	2.1	2.0	2.2	2.2		
	060374002	1.6	1.3	2.1	1.7	1.7	1.7	1.8	1.3		
	060375001	1.6	1.6	2.0	1.6	2.0	1.8	1.8	1.6		
	060376002		1.7	1.6	1.5	1.6	1.5	1.8	1.4		
	060379002			1.4	1.3	1.4	1.3	1.2	1.1		
Orange	060590001	1.9	1.6	2.0	1.7	1.7	1.6	1.6	1.5		
	060591002	1.1	2.9								
	060591003			0.8	1.0	1.2	1.0	0.9	0.7		
	060592001	1.4	1.1	1.4	1.4	1.5	1.3	1.1	1.0		
	060595001	2.7	2.6	2.1	2.0	2.0	1.9	2.0	1.7		
Riverside	060650006				1.9	1.0	1.0				
	060651003	2.9	2.5	2.6	2.3	2.6	2.4	2.6	2.3		
	060655001		0.9	0.9	1.0	1.0	1.0	0.7	0.7		
	060658001	1.7	1.9	1.7	1.4	1.6	1.4	1.8	1.6		
San Bernadino	060710001	1.1	1.1	1.7	1.6	1.0	1.0	0.5	0.7		
	060710006						0.9	0.7			
	060710012	0.8	1.0	1.0	1.0	1.0	0.9	0.6	0.4		
	060710014						1.0	0.8	0.6		
	060710017							0.7	1.0		
	060711004	2.0	2.0	2.0	1.6						
	060712002	1.6	1.7	1.7	1.3						
	060714001	1.7	1.7	1.7	1.9	1.9	1.4	1.0	0.9		
	060719004	2.3	2.3	2.0	1.8	1.4	1.5	1.6	1.5		
Maxperyear		3.3	3.1	2.9	2.7	2.9	2.7	3.0	2.8	3.3	***Total Max***
Minperyear		0.8	0.9	0.8	1.0	1.0	0.9	0.5	0.4	0.4	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

24-hour - 99th percentile (of all 365 possible daily 24-hour averages)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	3.3	3.4	3.4	3.7	3.0	3.0	2.9	4.1		
	060370113	4.0	4.0	3.9	4.0	3.1	2.9	3.3	3.3		
	060371002	6.6	7.8	6.7	6.6	5.9	4.9	6.6	6.8		
	060371103	6.0	6.0	5.5	5.9	5.0	4.3	5.4	5.4		
	060371201	6.0	6.9	6.6	6.2	4.6	5.0	5.5	5.7		
	060371301	10.9	9.6	8.7	8.7	9.6	8.0	8.9	7.6		
	060371601		6.1	5.1	5.7	4.6	4.0	5.3	5.2		
	060371701		5.0	4.7	4.7	4.2	3.5	4.2	4.2		
	060372005		4.9	4.8	4.6	3.9	3.5	4.7	4.9		
	060374002	6.0	6.1	5.4	4.7	4.8	4.1	4.7	3.7		
	060375001	8.2	6.9	6.1	5.5	5.9	5.2	5.8	4.9		
	060376002		3.0	2.7	3.0	2.3	2.3	2.5	2.7		
	060379002			3.6	3.3	3.1	2.7	2.6	2.4		
Orange	060590001	6.2	6.4	4.5	5.0	4.4	3.7	4.8	4.7		
	060591002		6.2	6.9							
	060591003			5.2	4.7	4.8	3.4	4.0	3.4		
	060592001	3.2	3.1	3.0	3.0	3.0	2.7	3.5	2.4		
	060595001	6.2	6.5	5.2	5.0	5.0	3.8	5.4	4.5		
Riverside	060650006				2.1	2.6	1.2				
	060651003	4.6	5.7	4.5	4.3	4.0	4.0	4.1	4.2		
	060655001		1.4	1.2	1.4	1.4	1.2	1.0	1.0		
	060658001	3.3	4.5	3.7	3.3	2.7	2.9	3.6	3.5		
San Bernadino	060710001	2.1	2.4	3.1	5.3	2.1	1.9	1.2	1.2		
	060710006						1.7	1.8			
	060710012	1.2	1.7	1.3	2.0	1.0	1.1	1.1	1.0		
	060710014						1.7	1.5	2.1		
	060710017							1.8	1.5		
	060711004	3.3	3.3	3.0	2.7						
	060712002	3.0	3.2	2.8	2.7						
	060714001	3.2	2.5	3.0	3.0	2.8	2.0	1.5	1.4		
	060719004	4.3	4.5	3.6	3.6	2.8	3.0	3.3	3.8		
Maxperyear		10.9	9.6	8.7	8.7	9.6	8.0	8.9	7.6	10.9	***Total Max***
Minperyear		1.2	1.4	1.2	1.4	1.0	1.1	1.0	1.0	1.0	***Total Min***

California-- January 1st 1988 through December 31st, 1995
(Study area and time period for Mann et al., 2002)

24-hour -50th percentile (of all 365 possible daily 24-hour averages)

	California	1988	1989	1990	1991	1992	1993	1994	1995		
Los Angeles	060370002	1.1	1.2	1.1	1.1	1.2	1.0	1.2	1.1		
	060370113	1.3	1.0	1.2	1.0	1.0	1.0	0.9	0.7		
	060371002	2.1	2.2	1.8	1.7	1.7	1.7	1.8	1.9		
	060371103	1.8	1.7	1.6	1.8	1.7	1.6	1.9	1.6		
	060371201	1.2	1.1	1.5	1.3	1.4	1.3	1.5	0.8		
	060371301	2.3	2.3	1.9	1.8	1.9	1.9	2.2	2.0		
	060371601		1.3	1.4	1.5	1.5	1.3	1.5	1.5		
	060371701		1.9	1.9	1.7	1.7	1.7	1.8	1.2		
	060372005		2.0	1.6	1.4	1.5	1.5	1.5	1.6		
	060374002	1.0	0.8	1.6	1.2	1.2	1.2	1.2	0.9		
	060375001	0.9	0.7	1.0	0.8	1.1	0.9	0.9	1.0		
	060376002		1.2	1.1	1.0	1.1	1.0	1.2	1.0		
	060379002			1.0	0.9	1.0	0.9	0.8	0.7		
Orange	060590001	1.2	1.1	1.3	1.2	1.2	1.1	1.1	1.1		
	060591002	0.7	1.5								
	060591003			0.4	0.5	0.8	0.7	0.5	0.4		
	060592001	0.9	0.8	1.0	1.0	1.1	1.0	0.7	0.6		
	060595001	2.0	2.0	1.5	1.3	1.3	1.3	1.3	1.2		
Riverside	060650006				1.2	0.9	0.6				
	060651003	2.0	1.9	1.8	1.5	1.8	1.7	1.9	1.6		
	060655001		0.5	0.5	0.5	0.6	0.6	0.4	0.4		
	060658001	1.1	1.1	1.0	1.0	1.0	0.9	1.1	0.9		
San Bernadino	060710001	1.0	1.0	1.2	1.1	1.0	0.6	0.3	0.4		
	060710006						0.5	0.6			
	060710012	0.4	0.6	0.6	1.0	0.5	0.5	0.3	0.3		
	060710014						0.8	0.5	0.4		
	060710017							0.5	0.7		
	060711004	1.4	1.3	1.3	1.1						
	060712002	1.1	1.2	1.1	1.0						
	060714001	1.2	1.3	1.2	1.3	1.3	1.1	0.7	0.6		
	060719004	1.7	1.6	1.3	1.2	1.0	1.1	1.2	1.2		
Maxperyear		2.3	2.3	1.9	1.8	1.9	1.9	2.2	2.0	2.3	***Total Max***
Minperyear		0.4	0.5	0.4	0.5	0.5	0.5	0.3	0.3	0.3	***Total Min***

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

Design values for current standards

1-hour - 2nd maximum (ppm) per monitor, per year for 1999-2005
(current standard is 35 ppm)

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005
Alabama	01	*Jefferson*	073	32.3	23.6	33.5	17.7	9.1	15.0	20.9
Arizona	04	*Maricopa*	013	13.5	10.5	9.6	7.9	7.3	7.5	7.0
Arizona	04	*Pima*	019	7.8	7.5	5.7	5.1	9.6	4.7	3.6
Arkansas	05	*Pulaski*	119	6.0	4.5	4.0	6.1	3.7	2.8	2.6
California	06	*Alameda*	001	6.1	5.4	5.2	7.6	6.0	3.5	3.3
California	06	*Contra Costa*	013	6.2	4.2	4.2	5.9	2.9	3.7	3.1
California	06	*Fresno*	019	10.0	8.2	6.2	6.0	5.0	3.9	4.0
California	06	*Kern*	029	5.5	8.9	5.8	4.4	4.2	3.3	3.2
California	06	*Los Angeles*	037	16.0	13.2	11.7	13.6	10.4	8.7	7.2
California	06	*Riverside*	065	7.1	7.5	5.7	6.0	4.4	3.9	3.7
California	06	*Sacramento*	067	7.3	7.2	6.7	7.5	4.7	7.1	6.6
California	06	*San Bernardino*	071	7.4	4.7	4.1	4.0	5.0	3.8	3.3
California	06	*San Diego*	073	9.2	9.2	8.4	8.0	8.3	6.1	7.4
California	06	*San Francisco*	075	7.8	4.9	4.7	4.9	5.1	3.4	3.8
California	06	*San Joaquin*	077	8.9	8.0	7.0	5.2	4.9	3.5	4.2
California	06	*San Mateo*	081	7.1	8.9	6.9	5.5	5.1	4.3	4.4
California	06	*Santa Clara*	085	8.8	8.6	7.4	5.9	5.3	3.9	4.0
California	06	*Stanislaus*	099	10.9	7.8	7.0	5.1	4.6	4.4	3.5
Colorado	08	*Adams*	001	6.0	4.3	5.8	4.4	5.2	4.0	3.3
Colorado	08	*Denver*	031	12.1	12.8	9.3	7.5	14.9	8.7	4.3
Connecticut	09	*Fairfield*	001	5.1	6.0	4.7	4.4	4.0	4.0	3.4
Connecticut	09	*Hartford*	003	11.9	15.2	7.8	9.5	10.1	9.8	9.9
Connecticut	09	*New Haven*	009	4.2	4.3	3.5	3.4	2.7	2.8	2.6
Delaware	10	*New Castle*	003	4.0	3.5	3.7	3.1	3.4	3.4	4.3
District Of	11	*District of*	001	7.3	6.6	6.3	7.5	8.3	4.0	3.8
Florida	12	*Broward*	011	7.9	7.5	5.9	6.8	5.3	4.2	4.6
Florida	12	*Duval*	031	6.3	6.0	5.3	3.9	3.8	3.4	3.2
Florida	12	*Hillsborough*	057	8.5	5.4	5.1	5.3	5.7	4.4	4.0
Florida	12	*Miami-Dade*	086	7.1	6.2	7.3	5.1	5.8	9.1	4.1
Florida	12	*Orange*	095	5.2	7.5	3.8	4.4	3.2	2.7	7.8
Florida	12	*Palm Beach*	099	5.2	4.9	3.2	3.8	4.2	3.7	3.4
Florida	12	*Pinellas*	103	4.5	5.3	4.0	3.7	3.6	2.8	2.5
Georgia	13	*DeKalb*	*089*	6.3	4.6	5.3	4.6	3.0	3.7	3.2
Georgia	13	*Fulton*	*121*	5.1	4.3	4.9	3.7	3.5	4.6	2.6
Hawaii	15	*Honolulu*	*003*	3.8	3.8	4.1	3.1	3.0	2.8	2.7
Idaho	16	*Ada*	001	9.1	7.6	5.3	5.2	4.7	3.9	4.6
Illinois	17	*Cook*	031	6.4	6.2	5.0	4.9	4.4	4.8	3.5
Illinois	17	*Winnebago*	201	6.5	4.8	4.9	3.3	3.3	3.2	3.2
Indiana	18	*Allen*	003	5.5	5.8	4.4	4.8	4.3	4.4	2.8
Indiana	18	*Marion*	097	4.8	5.7	3.8	14.2	3.8	4.0	4.2
Iowa	19	*Polk*	153	6.5	16.5	9.0	4.1	3.7	3.8	2.8
Kansas	20	*Sedgwick*	173	8.6	6.8	6.3	6.3	4.5	4.8	9.7
Kentucky	21	*Jefferson*	111	9.1	10.7	5.1	9.9	5.6	4.5	4.0
Louisiana	22	*East Baton*	033	6.8	6.2	6.5	5.8	4.3	3.8	3.1
Louisiana	22	*Orleans*	071	6.1	5.7	5.4	5.6	6.5	5.2	2.7

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(Study area and time period for Bell et al., 2009)

Maryland	24	*Baltimore (City)*	510	10.2	4.6	4.4	4.4	3.8	4.8	9.2
Massachusetts	25	*Hampden*	013	7.9	5.4	4.9	5.5	3.9	4.7	3.3
Massachusetts	25	*Middlesex*	017	8.4	5.3	4.2	3.6	3.8	2.5	2.6
Massachusetts	25	*Suffolk*	025	7.0	3.4	4.9	3.7	4.0	2.8	3.6
Massachusetts	25	*Worcester*	027	6.8	9.0	5.9	4.5	3.6	3.9	3.3
Michigan	26	*Kent*	081	4.1	4.2	4.3	4.7	3.2	2.8	2.6
Michigan	26	*Macomb*	099	4.0	5.1	4.0	3.7	3.5	3.3	3.6
Michigan	26	*Oakland*	125	4.8	4.7	4.5	3.4	3.7	4.0	3.4
Michigan	26	*Wayne*	163	6.0	7.7	5.4	4.6	4.5	3.9	3.4
Minnesota	27	*Dakota*	037	1.2	1.7	1.5	1.3	1.2	1.0	0.7
Minnesota	27	*Hennepin*	053	4.3	5.0	5.6	6.1	4.7	3.2	3.3
Minnesota	27	*Ramsey*	123	7.6	6.6	11.1	5.8	6.1	5.3	5.6
Minnesota	27	*Saint Louis*	137	4.6	3.8	4.1	3.2	2.8	2.9	4.2
Mississippi	28	*Hinds*	049	8.2	4.4	5.3	4.9	4.9	4.2	3.4
Missouri	29	*Jackson*	095	6.7	6.7	10.1	10.2	3.5	3.4	3.6
Missouri	29	*Saint Louis*	189	5.0	4.3	4.3	4.5	3.3	2.3	1.7
Missouri	29	*St. Louis City*	510	6.1	5.6	8.1	8.1	4.5	4.3	4.7
Nevada	32	*Clark*	003	10.2	8.5	8.7	7.5	6.9	6.5	6.3
Nevada	32	*Washoe*	031	9.5	7.2	7.1	6.7	6.1	5.8	4.4
New Hampshire	33	*Hillsborough*	011	12.0	8.0	6.5	5.9	7.5	4.8	6.1
New Jersey	34	*Bergen*	003	5.7	4.8	4.5	4.2	5.2	4.1	3.4
New Jersey	34	*Camden*	007	5.5	5.3	5.6	3.8	2.9	3.7	3.9
New Jersey	34	*Hudson*	017	10.0	8.5	5.1	4.4	4.0	4.1	4.8
New Jersey	34	*Middlesex*	023	4.5	4.7	4.8	3.7	3.7	3.5	3.0
New Jersey	34	*Morris*	027	5.9	5.1	5.6	4.9	3.6	3.1	2.8
New Jersey	34	*Union*	039	8.9	7.2	6.1	6.4	6.0	5.4	4.6
New Mexico	35	*Bernalillo*	001	7.5	7.0	9.5	6.0	9.6	5.0	4.3
New York	36	*Albany*	001	2.5	1.8	1.9	2.2	2.9	1.7	1.9
New York	36	*Bronx*	005	5.7	6.0	4.9	3.7	3.4	2.8	3.5
New York	36	*Erie*	029	3.9	4.7	3.2	4.0	3.0	2.6	2.5
New York	36	*Kings*	047	7.3	6.2	5.7	4.9	3.5	3.6	4.1
New York	36	*Monroe*	055	5.5	4.2	4.3	5.7	2.8	2.8	2.5
New York	36	*New York*	061	7.1	6.0	6.5	4.5	4.0	3.6	2.2
New York	36	*Niagara*	063	4.2	3.8	2.3	2.2	2.8	2.5	5.7
New York	36	*Onondaga*	067	5.6	3.9	3.8	3.4	3.2	3.0	2.8
New York	36	*Queens*	081	3.4	6.3	3.8	3.5	11.2	3.1	2.4
New York	36	*Suffolk*	103		3.4	3.4	3.7	2.8	2.9	2.3
North Carolina	37	*Forsyth*	067	4.8	5.1	6.1	5.7	5.5	4.4	3.1
North Carolina	37	*Mecklenburg*	119	7.1	10.8	8.8	5.7	4.6	3.5	2.9
North Carolina	37	*Wake*	183	6.6	6.8	5.4	4.6	4.3	3.4	3.4
Ohio	39	*Cuyahoga*	035	6.8	11.1	5.5	8.0	5.5	12.8	5.5
Ohio	39	*Franklin*	049	8.5	4.9	4.7	3.6	4.8	4.4	4.2
Ohio	39	*Hamilton*	061	4.9	4.9	4.5	5.7	3.0	5.8	3.2
Ohio	39	*Lake*	085	2.5	2.7	2.4	2.7	2.9	1.7	2.2
Ohio	39	*Montgomery*	113	4.1	5.0	4.8	3.4	3.7	4.1	2.8
Ohio	39	*Stark*	151	4.5	3.3	4.0	3.8	5.4	3.6	3.8
Ohio	39	*Summit*	153	4.7	5.6	5.2	3.6	5.1	3.5	3.1
Oklahoma	40	*Oklahoma*	109	6.5	5.5	5.6	5.0	4.6	3.4	3.2
Oklahoma	40	*Tulsa*	143	5.6	6.7	7.5	4.9	4.2	2.6	3.2
Oregon	41	*Lane*	039	7.8	6.6	5.5	5.6	5.4	6.8	4.9

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(Study area and time period for Bell et al., 2009)

Oregon	41	*Multnomah*	051	10.4	8.4	6.4	5.4	5.2	8.6	4.5
Pennsylvania	42	*Allegheny*	003	6.0	5.5	4.2	5.7	5.6	3.5	3.1
Pennsylvania	42	*Berks*	011	4.6	3.8	3.8	4.1	3.2	2.7	2.4
Pennsylvania	42	*Dauphin*	043	4.9	3.5	4.4	3.6	3.0	2.3	2.0
Pennsylvania	42	*Erie*	049	10.6	11.9	7.2	7.5	7.6	1.8	3.1
Pennsylvania	42	*Lackawanna*	069	3.5	4.4	2.9	2.7	2.4	2.9	2.6
Pennsylvania	42	*Lancaster*	071	3.1	3.0	2.9	3.0	2.7	3.2	2.5
Pennsylvania	42	*Northampton*	095	4.4	5.5	3.1	2.3	2.3	2.4	2.5
Pennsylvania	42	*Philadelphia*	101	7.4	6.3	7.7	5.0	5.4	5.1	12.1
Pennsylvania	42	*York*	133	5.3	3.7	3.8	4.3	2.6	2.8	2.5
Rhode Island	44	*Providence*	007	7.0	5.4	9.0	3.9	3.7	3.5	7.9
South Carolina	45	*Charleston*	019	6.4	4.4	5.1	3.5	4.6	3.1	2.1
South Carolina	45	*Greenville*	045	6.9	5.3	4.7	4.2	4.0	3.4	3.6
South Carolina	45	*Richland*	079	4.2	4.6	4.6	3.4	2.9	3.0	2.5
Tennessee	47	*Davidson*	037	7.6	6.7	7.1	6.2	4.9	5.0	4.1
Tennessee	47	*Shelby*	157	7.6	5.8	6.6	4.9	4.9	8.0	3.8
Texas	48	*Bexar*	029	6.3	4.6	6.0	4.5	4.6	3.1	3.7
Texas	48	*Cameron*	061	4.4	3.3	2.7	3.7	3.9	3.9	3.4
Texas	48	*Dallas*	113	4.3	3.5	3.6	3.3	3.0	2.9	4.1
Texas	48	*El Paso*	141	14.4	17.0	14.3	13.1	11.7	11.3	11.5
Texas	48	*Harris*	201	6.3	5.7	5.7	4.4	5.4	3.6	3.8
Texas	48	Tarrant	439	4.8	4.3	4.3	4.0	3.5	2.9	7.5
Texas	48	Travis	453	2.1	1.8	1.7	1.6	2.2	0.9	1.0
Utah	49	Salt Lake	035	9.7	10.0	7.1	6.2	8.2	5.4	5.5
Virginia	51	*Fairfax*	059	7.0	5.6	4.6	3.3	3.3	3.6	2.5
Virginia	51	*Norfolk City*	710	9.2	6.9	7.2	6.6	4.6	3.8	2.1
Washington	53	*Clark*	011	12.9	8.4	8.0	7.2	7.1	6.3	6.9
Washington	53	*King*	033	15.6	8.6	8.8	7.0	8.7	4.7	5.9
Washington	53	*Pierce*	053	11.0	7.7	8.4	9.3	10.1	6.4	6.1
Washington	53	*Spokane*	063	9.3	8.9	7.6	8.6	8.2	7.8	7.4
Wisconsin	55	*Milwaukee*	079	3.5	3.5	4.2	4.2	5.0	4.9	9.5
Maxperyear				32.3	23.6	33.5	17.7	14.9	15.0	20.9

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

8-hour - 2nd maximum non-overlapping 8-hr average (ppm) per monitor, per year for1999-2005 (current standard is 9 ppm)

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005
Alabama	01	Jefferson	073	19.8	16.3	24.3	11.7	4.5	8.2	8.8
Arizona	04	Maricopa	013	8.1	7.2	6.6	5.5	5.5	5.1	4.8
Arizona	04	Pima	019	3.8	4.7	2.9	2.6	2.7	2.5	2.4
Arkansas	05	Pulaski	119	4.0	2.9	2.2	5.2	2.2	1.8	2.1
California	06	Alameda	001	4.5	3.0	3.9	4.9	4.3	2.4	2.3
California	06	Contra Costa	013	3.3	2.6	2.4	2.5	1.8	1.8	1.7
California	06	Fresno	019	7.6	5.9	4.5	4.3	3.6	2.8	2.8
California	06	Kern	029	4.1	5.2	3.4	2.5	2.2	2.4	2.0
California	06	Los Angeles	037	11.1	9.9	7.2	8.5	7.2	6.1	5.6
California	06	Riverside	065	4.1	4.2	3.6	3.5	3.6	2.8	2.3
California	06	Sacramento	067	6.2	6.2	5.0	4.2	3.8	3.2	3.7
California	06	San Bernardino	071	4.0	3.7	3.2	3.1	3.9	2.9	2.2
California	06	San Diego	073	4.9	4.7	4.7	3.8	4.1	3.9	4.4
California	06	San Francisco	075	4.5	2.8	3.1	2.4	3.2	2.6	2.5
California	06	San Joaquin	077	6.0	6.3	3.6	3.2	3.0	2.3	2.5
California	06	San Mateo	081	3.7	3.6	3.5	2.6	2.3	2.0	2.2
California	06	Santa Clara	085	6.2	6.9	5.0	4.1	3.7	2.9	3.0
California	06	Stanislaus	099	6.3	5.6	4.8	3.7	3.3	2.9	2.5
Colorado	08	Adams	001	3.6	2.9	3.3	2.6	3.0	2.8	2.2
Colorado	08	Denver	031	5.2	5.4	4.1	3.7	4.5	4.1	2.5
Connecticut	09	Fairfield	001	3.8	3.0	3.1	3.2	2.6	2.2	2.5
Connecticut	09	Hartford	003	5.5	7.3	4.5	5.1	5.2	5.7	4.4
Connecticut	09	New Haven	009	3.1	2.6	2.5	2.3	1.9	2.0	1.9
Delaware	10	New Castle	003	3.0	2.6	2.5	2.2	2.4	2.1	2.0
District Of	11	District of	001	5.6	5.0	4.7	4.5	4.0	3.4	3.2
Florida	12	Broward	011	5.4	3.7	4.2	4.0	3.7	3.4	3.2
Florida	12	Duval	031	4.1	3.8	3.4	2.6	2.4	2.3	2.2
Florida	12	Hillsborough	057	4.7	3.1	3.0	3.8	3.3	2.5	3.0
Florida	12	Miami-Dade	086	4.0	3.4	4.2	3.4	3.6	5.3	2.5
Florida	12	Orange	095	3.3	2.6	2.1	2.5	2.0	1.8	2.6
Florida	12	Palm Beach	099	3.1	2.9	2.2	2.3	1.8	2.1	2.2
Florida	12	Pinellas	103	2.6	2.3	2.4	2.3	1.9	1.7	1.6
Georgia	13	DeKalb	089	4.1	3.2	4.1	3.6	2.6	2.6	2.3
Georgia	13	Fulton	121	3.0	2.6	3.0	2.3	2.5	2.5	1.8
Hawaii	15	Honolulu	003	2.1	2.0	1.9	1.8	2.2	2.0	1.6
Idaho	16	Ada	001	4.6	3.1	3.2	3.1	3.2	2.4	2.2
Illinois	17	Cook	031	4.9	4.3	3.8	3.7	3.4	3.2	2.5
Illinois	17	Winnebago	201	3.8	2.9	2.9	2.4	2.4	2.7	2.3
Indiana	18	Allen	003	3.3	3.9	2.6	3.3	2.6	2.6	2.1
Indiana	18	Marion	097	2.6	3.8	2.5	5.0	2.7	2.2	2.4
Iowa	19	Polk	153	3.5	4.9	4.6	2.7	2.3	2.5	2.1
Kansas	20	Sedgwick	173	4.6	6.2	4.1	3.7	3.1	3.3	4.9
Kentucky	21	Jefferson	111	4.8	4.1	3.9	4.8	3.3	3.3	2.6
Louisiana	22	East Baton	033	4.5	3.6	4.8	3.7	3.1	2.7	2.1
Louisiana	22	Orleans	071	3.3	4.0	3.6	3.6	3.5	3.4	1.7
Maryland	24	Baltimore (City)	510	5.6	3.4	3.3	3.0	2.5	2.9	2.7

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Massachusetts	25	Hampden	013	5.6	4.0	3.0	3.6	3.0	3.1	2.6
Massachusetts	25	Middlesex	017	4.2	3.2	2.7	2.4	2.7	1.6	1.8
Massachusetts	25	Suffolk	025	4.2	2.4	2.8	2.2	2.4	1.5	2.3
Massachusetts	25	Worcester	027	3.3	3.3	2.6	2.9	1.7	1.9	2.3
Michigan	26	Kent	081	3.5	2.6	3.1	2.8	2.0	1.8	1.9
Michigan	26	Macomb	099	2.5	1.4	2.7	2.3	2.2	2.3	2.0
Michigan	26	Oakland	125	2.8	2.6	3.0	2.1	2.5	2.3	2.1
Michigan	26	Wayne	163	4.4	4.5	3.4	3.7	3.0	2.4	2.0
Minnesota	27	Dakota	037	1.0	1.5	1.0	0.9	0.8	0.8	0.4
Minnesota	27	Hennepin	053	2.9	2.7	2.9	3.1	2.3	1.8	1.8
Minnesota	27	Ramsey	123	5.2	5.1	5.1	3.9	4.3	3.2	3.0
Minnesota	27	Saint Louis	137	2.9	2.1	2.5	2.1	1.7	1.5	1.6
Mississippi	28	Hinds	049	5.0	3.2	4.2	3.0	3.3	2.5	2.6
Missouri	29	Jackson	095	3.6	5.2	3.6	3.3	1.8	2.2	2.4
Missouri	29	Saint Louis	189	2.7	3.0	2.6	2.5	2.1	1.6	1.3
Missouri	29	St. Louis City	510	4.1	3.8	4.5	6.9	3.0	2.8	3.0
Nevada	32	Clark	003	8.2	7.1	6.5	5.8	5.3	5.1	5.2
Nevada	32	Washoe	031	7.1	5.2	5.2	4.4	3.8	3.9	3.2
New Hampshire	33	Hillsborough	011	5.3	4.1	4.0	3.7	4.0	2.8	3.2
New Jersey	34	Bergen	003	4.4	3.4	3.1	3.4	2.4	2.7	2.8
New Jersey	34	Camden	007	3.7	4.3	3.9	2.1	1.8	2.9	3.0
New Jersey	34	Hudson	017	6.1	4.8	3.0	2.8	2.9	3.2	2.8
New Jersey	34	Middlesex	023	3.3	3.3	3.3	2.6	1.9	2.1	1.9
New Jersey	34	Morris	027	4.1	3.1	3.3	2.4	2.3	1.9	1.8
New Jersey	34	Union	039	6.6	4.7	4.8	4.4	3.3	3.3	3.4
New Mexico	35	Bernalillo	001	4.6	3.8	4.2	3.9	3.5	3.5	3.1
New York	36	Albany	001	1.4	1.1	1.2	1.5	1.5	1.1	1.4
New York	36	Bronx	005	4.0	3.5	2.7	2.4	2.1	2.0	2.2
New York	36	Erie	029	2.2	2.0	1.9	1.8	2.4	1.4	1.6
New York	36	Kings	047	4.9	4.3	3.5	3.4	2.6	2.5	2.2
New York	36	Monroe	055	2.6	2.6	1.8	2.2	2.1	1.6	2.0
New York	36	New York	061	4.7	4.2	3.2	3.1	3.0	2.4	1.5
New York	36	Niagara	063	2.5	1.6	1.6	1.7	2.0	1.4	1.8
New York	36	Onondaga	067	3.1	2.4	2.2	2.1	1.8	1.4	1.9
New York	36	Queens	081	2.5	3.3	3.0	2.5	2.6	2.0	1.6
New York	36	Suffolk	103		2.8	2.2	1.9	1.6	1.6	1.5
North Carolina	37	Forsyth	067	3.6	3.7	4.0	4.1	3.7	3.2	2.5
North Carolina	37	Mecklenburg	119	4.3	4.7	4.3	3.2	3.0	3.1	2.3
North Carolina	37	Wake	183	4.9	5.2	3.5	3.1	2.9	2.7	2.4
Ohio	39	Cuyahoga	035	3.9	7.9	3.5	2.2	3.4	5.4	2.9
Ohio	39	Franklin	049	2.5	2.8	2.6	2.5	3.0	2.6	2.0
Ohio	39	Hamilton	061	2.8	2.4	2.9	2.6	1.9	2.8	1.8
Ohio	39	Lake	085	1.4	1.4	1.4	2.4	2.7	1.3	1.3
Ohio	39	Montgomery	113	2.8	3.1	2.6	1.8	2.5	2.6	1.8
Ohio	39	Stark	151	2.3	2.6	2.6	2.8	2.2	2.6	2.7
Ohio	39	Summit	153	3.1	2.6	2.7	2.1	2.5	2.2	1.7
Oklahoma	40	Oklahoma	109	4.3	4.2	4.0	3.0	2.8	2.2	2.1
Oklahoma	40	Tulsa	143	3.5	3.7	4.1	3.0	2.5	1.7	1.4
Oregon	41	Lane	039	5.0	4.3	4.1	4.2	3.4	3.4	2.7
Oregon	41	Multnomah	051	6.2	4.4	4.1	4.5	4.0	3.9	3.1

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Pennsylvania	42	Allegheny	003	3.9	3.2	3.4	2.7	2.3	2.3	2.2
Pennsylvania	42	Berks	011	2.8	2.1	2.2	2.2	2.0	1.8	1.9
Pennsylvania	42	Dauphin	043	4.3	2.1	2.8	2.3	2.0	1.3	1.3
Pennsylvania	42	Erie	049	5.6	6.0	4.4	4.5	3.4	1.3	1.4
Pennsylvania	42	Lackawanna	069	1.7	2.1	1.8	1.6	1.5	1.8	1.5
Pennsylvania	42	Lancaster	071	2.1	1.9	2.2	2.2	1.7	1.6	1.5
Pennsylvania	42	Northampton	095	3.0	2.4	2.4	1.8	1.4	1.7	1.9
Pennsylvania	42	Philadelphia	101	4.9	4.0	4.7	2.9	3.9	3.4	3.4
Pennsylvania	42	York	133	2.4	1.8	2.2	2.2	1.7	1.8	1.4
Rhode Island	44	Providence	007	3.9	3.5	3.8	2.7	2.3	2.5	2.5
South Carolina	45	Charleston	019	4.0	2.7	3.0	2.8	2.7	2.1	0.6
South Carolina	45	Greenville	045	4.8	3.7	3.4	3.3	3.1	2.5	2.4
South Carolina	45	Richland	079	3.7	3.6	3.3	2.8	2.2	2.3	2.2
Tennessee	47	Davidson	037	5.4	5.6	5.8	5.1	3.7	3.8	3.3
Tennessee	47	Shelby	157	5.2	4.4	4.8	3.6	3.3	4.0	2.8
Texas	48	Bexar	029	4.2	2.7	2.7	2.6	2.7	1.9	2.7
Texas	48	Cameron	061	2.6	1.6	1.5	1.9	1.9	2.1	1.7
Texas	48	Dallas	113	3.2	2.2	2.4	2.1	2.0	1.8	2.6
Texas	48	El Paso	141	8.2	9.2	8.8	6.8	6.4	6.4	5.4
Texas	48	Harris	201	4.1	4.2	4.4	3.3	4.2	2.9	2.6
Texas	48	Tarrant	439	2.6	2.2	2.3	2.1	1.6	1.7	2.6
Texas	48	Travis	453	1.2	1.1	0.9	0.9	0.9	0.6	0.6
Utah	49	Salt Lake	035	5.7	5.2	4.1	3.7	4.3	3.7	3.6
Virginia	51	Fairfax	059	3.2	3.5	3.0	2.3	2.7	2.3	1.9
Virginia	51	Norfolk City	710	4.6	4.1	4.2	3.7	2.9	3.0	1.1
Washington	53	Clark	011	6.7	6.2	4.7	5.7	4.5	4.8	4.6
Washington	53	King	033	6.1	6.3	6.5	5.0	5.5	3.3	3.8
Washington	53	Pierce	053	6.6	5.5	5.0	4.5	5.3	4.0	3.9
Washington	53	Spokane	063	5.7	5.6	5.2	5.2	4.6	4.0	5.1
Wisconsin	55	Milwaukee	079	2.2	2.4	2.7	3.1	2.6	3.0	4.5
Maxperyear				19.8	16.3	24.3	11.7	7.2	8.2	8.8

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Statistics for Other Metrics

1-hour - 99th percentile daily maximum (of all 365 possible daily maximum 1-hour values)

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	26.4	18.5	31.8	15.9	7.9	14.1	16.1	
Arizona	04	Maricopa	013	13.5	9.8	9.0	7.7	6.9	6.4	6.4	
Arizona	04	Pima	019	7.2	7.0	5.5	4.9	5.4	4.4	3.2	
Arkansas	05	Pulaski	119	5.4	4.3	3.8	4.9	3.5	2.7	2.5	
California	06	Alameda	001	5.5	4.7	5.8	6.2	6.0	2.7	2.9	
California	06	Contra Costa	013	5.3	4.0	4.2	4.6	2.5	2.8	2.7	
California	06	Fresno	019	9.0	8.2	5.5	5.6	5.0	3.6	3.7	
California	06	Kern	029	5.2	6.8	5.3	3.7	3.1	2.9	3.0	
California	06	Los Angeles	037	14.5	12.5	10.6	10.4	9.1	8.0	7.1	
California	06	Riverside	065	6.8	6.5	5.7	5.6	4.3	3.6	3.4	
California	06	Sacramento	067	7.0	6.5	5.3	4.8	4.4	6.1	6.2	
California	06	San Bernardino	071	4.9	4.6	3.9	3.7	4.3	3.4	2.9	
California	06	San Diego	073	9.1	9.1	7.9	7.7	6.9	6.0	5.8	
California	06	San Francisco	075	5.8	3.7	4.2	3.7	4.2	3.2	3.6	
California	06	San Joaquin	077	8.4	7.6	5.3	5.0	4.2	3.1	3.4	
California	06	San Mateo	081	7.0	6.6	6.8	5.4	4.9	4.0	4.3	
California	06	Santa Clara	085	8.4	7.2	7.5	5.7	4.4	3.8	3.8	
California	06	Stanislaus	099	7.5	6.5	6.0	4.9	4.4	3.9	3.3	
Colorado	08	Adams	001	5.3	4.2	4.7	3.7	3.6	3.4	3.0	
Colorado	08	Denver	031	8.8	8.7	7.2	6.9	6.6	5.9	3.8	
Connecticut	09	Fairfield	001	4.5	5.2	4.2	4.2	3.3	2.6	3.2	
Connecticut	09	Hartford	003	10.4	11.0	7.5	9.4	9.8	7.8	7.1	
Connecticut	09	New Haven	009	3.4	3.3	3.1	3.0	2.5	2.4	2.6	
Delaware	10	New Castle	003	4.0	3.1	3.4	2.8	2.9	3.0	2.6	
District Of Columbia	11	District of Columbia	001	6.7	6.0	4.7	5.1	5.4	3.7	3.5	
Florida	12	Broward	011	6.6	4.7	5.7	5.4	4.7	4.1	3.2	
Florida	12	Duval	031	4.6	4.1	4.7	3.3	3.5	3.3	2.8	
Florida	12	Hillsborough	057	5.1	4.8	4.1	4.8	4.4	3.8	3.8	
Florida	12	Miami-Dade	086	6.7	5.2	5.8	4.4	4.9	6.0	4.1	
Florida	12	Orange	095	4.8	4.2	3.5	3.5	2.8	2.1	2.5	
Florida	12	Palm Beach	099	5.0	4.4	2.8	3.1	3.4	3.3	2.9	
Florida	12	Pinellas	103	3.8	3.8	3.0	3.2	2.8	2.5	2.2	
Georgia	13	DeKalb	089	5.4	4.1	4.3	3.6	2.7	2.7	2.6	
Georgia	13	Fulton	121	4.9	3.8	4.2	3.5	3.3	2.9	2.4	
Hawaii	15	Honolulu	003	3.4	3.4	3.0	2.8	2.8	2.7	2.3	
Idaho	16	Ada	001	7.8	5.7	4.8	5.0	4.6	3.7	3.5	
Illinois	17	Cook	031	5.9	4.9	4.2	4.3	4.0	3.7	2.9	
Illinois	17	Winnebago	201	5.6	4.4	3.5	3.2	3.1	2.9	3.0	
Indiana	18	Allen	003	4.5	4.6	4.1	4.4	3.7	3.2	2.5	
Indiana	18	Marion	097	4.5	4.4	3.7	10.1	3.4	3.0	3.1	
Iowa	19	Polk	153	6.5	5.8	5.2	3.9	3.2	3.4	2.8	
Kansas	20	Sedgwick	173	6.4	6.5	4.7	4.6	4.3	4.3	3.6	
Kentucky	21	Jefferson	111	9.1	5.5	4.4	7.0	4.5	3.8	3.7	
Louisiana	22	East Baton Rouge	033	6.5	5.2	4.8	4.2	3.6	3.5	2.6	
Louisiana	22	Orleans	071	5.3	4.7	4.3	4.5	3.4	4.5	2.4	

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(Study area and time period for Bell et al., 2009)

Maryland	24	Baltimore (City)	510	14.9	3.8	3.6	3.9	3.1	2.8	9.2	
Massachusetts	25	Hampden	013	5.1	5.1	4.0	4.3	4.0	3.5	3.0	
Massachusetts	25	Middlesex	017	7.3	4.3	3.9	2.8	3.1	2.4	2.3	
Massachusetts	25	Suffolk	025	5.9	3.0	3.5	3.4	2.9	2.0	2.6	
Massachusetts	25	Worcester	027	4.5	4.1	4.5	3.8	3.1	2.4	2.9	
Michigan	26	Kent	081	3.8	3.8	4.0	3.4	2.8	2.5	2.3	
Michigan	26	Macomb	099	3.5	2.4	2.6	3.3	3.1	2.6	2.6	
Michigan	26	Oakland	125	4.0	4.1	3.7	2.8	3.4	3.0	2.5	
Michigan	26	Wayne	163	5.3	5.8	4.9	3.6	3.8	3.7	2.9	
Minnesota	27	Dakota	037	1.2	1.5	1.3	1.0	0.9	0.8	0.6	
Minnesota	27	Hennepin	053	4.1	4.6	4.0	4.0	3.7	2.6	2.9	
Minnesota	27	Ramsey	123	6.8	6.4	8.0	4.8	4.9	5.0	4.1	
Minnesota	27	Saint Louis	137	3.8	3.4	2.5	2.7	2.4	2.6	2.6	
Mississippi	28	Hinds	049	5.9	4.1	4.8	3.8	3.3	3.3	2.9	
Missouri	29	Jackson	095	4.8	5.0	7.9	7.5	4.5	2.6	3.1	
Missouri	29	Saint Louis	189	4.1	4.1	3.9	3.8	2.6	2.2	1.5	
Missouri	29	St. Louis City	510	4.9	5.1	5.6	8.1	3.8	4.1	3.3	
Nevada	32	Clark	003	10.0	8.5	7.9	6.6	6.3	6.0	6.0	
Nevada	32	Washoe	031	8.6	7.0	6.8	6.3	5.6	5.5	4.0	
New Hampshire	33	Hillsborough	011	9.0	6.7	6.0	5.1	4.5	3.4	4.6	
New Jersey	34	Bergen	003	4.9	4.3	4.0	3.9	3.1	3.1	2.7	
New Jersey	34	Camden	007	4.6	4.6	4.5	3.2	2.8	2.7	3.5	
New Jersey	34	Hudson	017	9.1	8.1	4.2	4.2	4.0	3.4	3.6	
New Jersey	34	Middlesex	023	4.4	4.0	4.1	3.5	2.8	2.9	2.7	
New Jersey	34	Morris	027	5.6	4.5	4.4	3.8	3.4	2.5	2.4	
New Jersey	34	Union	039	7.9	6.7	6.0	5.8	5.0	4.5	4.1	
New Mexico	35	Bernalillo	001	7.3	5.8	7.7	5.4	4.4	4.3	3.9	
New York	36	Albany	001	1.7	1.6	1.7	1.7	1.5	1.6	1.2	
New York	36	Bronx	005	4.8	3.5	3.9	3.4	2.9	2.2	2.5	
New York	36	Erie	029	3.3	3.3	2.7	2.7	2.8	2.1	2.1	
New York	36	Kings	047	5.8	5.6	4.8	4.5	3.4	3.5	3.4	
New York	36	Monroe	055	4.7	3.3	3.5	2.8	2.5	2.8	2.2	
New York	36	New York	061	5.3	5.5	4.5	3.9	3.2	2.6	2.0	
New York	36	Niagara	063	2.5	2.6	2.1	1.9	2.6	2.2	2.2	
New York	36	Onondaga	067	5.0	3.6	2.7	3.0	2.6	2.5	2.4	
New York	36	Queens	081	3.3	3.4	3.8	2.8	3.2	2.5	2.1	
New York	36	Suffolk	103		3.4	2.9	2.6	2.5	2.1	2.1	
North Carolina	37	Forsyth	067	4.6	5.1	5.6	5.5	4.4	3.7	3.0	
North Carolina	37	Mecklenburg	119	6.2	7.7	6.7	5.1	3.7	3.3	2.7	
North Carolina	37	Wake	183	6.2	6.3	6.2	4.6	4.3	3.4	3.4	
Ohio	39	Cuyahoga	035	5.5	5.5	5.3	4.0	4.7	11.6	5.0	
Ohio	39	Franklin	049	3.7	4.6	3.4	3.0	3.8	2.7	2.6	
Ohio	39	Hamilton	061	3.6	3.9	3.1	3.3	2.8	4.9	2.4	
Ohio	39	Lake	085	2.0	2.1	2.0	2.5	2.7	1.6	2.0	
Ohio	39	Montgomery	113	4.0	4.6	3.2	2.6	3.4	3.7	2.3	
Ohio	39	Stark	151	3.2	3.2	2.8	3.0	2.8	3.2	3.3	
Ohio	39	Summit	153	4.1	4.7	4.2	3.3	3.7	3.2	2.6	
Oklahoma	40	Oklahoma	109	5.0	5.1	4.7	3.9	3.4	2.6	2.9	
Oklahoma	40	Tulsa	143	5.0	5.2	6.3	4.4	3.6	2.1	1.8	
Oregon	41	Lane	039	6.5	6.0	5.4	5.1	4.9	4.5	4.8	

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Oregon	41	Multnomah	051	10.4	6.0	6.3	5.4	5.2	5.1	4.5		
Pennsylvania	42	Allegheny	003	5.6	4.4	4.0	3.9	3.3	2.8	2.5		
Pennsylvania	42	Berks	011	3.8	3.4	3.2	3.2	2.2	2.4	2.3		
Pennsylvania	42	Dauphin	043	3.8	3.1	3.5	3.0	2.2	2.1	1.7		
Pennsylvania	42	Erie	049	9.1	10.1	6.7	6.8	6.3	1.7	2.3		
Pennsylvania	42	Lackawanna	069	2.7	3.1	2.8	2.6	2.3	2.6	2.2		
Pennsylvania	42	Lancaster	071	3.0	2.3	2.6	2.8	2.2	2.4	2.2		
Pennsylvania	42	Northampton	095	3.7	3.3	2.7	2.3	1.9	2.0	2.1		
Pennsylvania	42	Philadelphia	101	6.4	4.7	5.9	4.0	4.7	4.6	3.7		
Pennsylvania	42	York	133	4.1	3.2	3.2	2.8	2.5	2.6	2.4		
Rhode Island	44	Providence	007	6.5	4.5	5.2	3.5	3.3	3.2	3.0		
South Carolina	45	Charleston	019	5.0	3.9	3.9	3.5	3.0	2.3	2.3		
South Carolina	45	Greenville	045	5.5	4.1	4.5	4.0	3.9	3.3	2.7		
South Carolina	45	Richland	079	4.2	4.2	3.8	3.3	2.7	2.6	2.4		
Tennessee	47	Davidson	037	7.1	6.6	6.6	5.3	4.7	4.2	3.8		
Tennessee	47	Shelby	157	6.6	5.5	5.5	4.7	4.1	4.6	3.6		
Texas	48	Bexar	029	5.3	3.7	4.4	3.5	3.5	3.1	3.6		
Texas	48	Cameron	061	4.2	2.5	2.2	3.5	2.8	2.5	2.4		
Texas	48	Dallas	113	3.8	3.0	3.4	3.2	2.5	2.3	2.7		
Texas	48	El Paso	141	14.2	15.6	12.7	12.4	10.4	10.0	10.3		
Texas	48	Harris	201	6.0	4.7	7.1	4.2	4.1	3.5	3.0		
Texas	48	Tarrant	439	4.5	3.1	3.9	3.3	3.0	2.3	6.3		
Texas	48	Travis	453	1.7	1.5	1.4	1.4	1.2	0.9	0.9		
Utah	49	Salt Lake	035	9.7	10.0	6.5	6.2	8.2	5.4	5.5		
Virginia	51	Fairfax	059	4.5	4.5	4.0	2.9	3.0	2.6	2.5		
Virginia	51	Norfolk City	710	5.3	5.8	5.7	5.1	4.3	3.7	2.2		
Washington	53	Clark	011	8.8	7.8	7.1	6.6	6.5	5.6	5.9		
Washington	53	King	033	7.8	7.8	6.6	6.3	6.9	4.4	5.0		
Washington	53	Pierce	053	9.7	6.7	5.9	5.3	6.6	4.9	4.9		
Washington	53	Spokane	063	7.8	8.3	6.8	7.1	7.3	5.6	6.2		
Wisconsin	55	Milwaukee	079	3.0	3.3	3.6	3.2	3.3	4.2	7.7		
Maxperyear				26.4	18.5	31.8	15.9	10.4	14.1	16.1	31.8	***Total Max***
Minperyear				1.2	1.5	1.3	1.0	0.9	0.8	0.6	0.6	***Total Min***

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

1-hour - 50th percentile daily maximum (of all 365 possible daily

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	2.3	2.4	2.4	1.9	1.3	1.1	1.5	
Arizona	04	Maricopa	013	5.9	4.4	3.6	4.5	3.2	2.6	2.1	
Arizona	04	Pima	019	3.5	3.2	2.5	2.2	1.8	1.7	1.5	
Arkansas	05	Pulaski	119	2.0	1.8	1.3	0.9	0.8	0.6	0.7	
California	06	Alameda	001	1.2	1.1	2.0	1.0	1.9	0.7	0.7	
California	06	Contra Costa	013	1.0	1.0	0.8	1.2	0.9	0.9	0.6	
California	06	Fresno	019	2.8	1.6	1.0	0.9	1.0	0.7	0.7	
California	06	Kern	029	1.4	1.4	1.5	1.3	1.1	1.1	1.0	
California	06	Los Angeles	037	3.1	2.7	2.2	2.5	2.0	2.4	1.6	
California	06	Riverside	065	2.4	2.2	2.0	1.7	1.4	1.3	1.2	
California	06	Sacramento	067	1.3	1.3	1.5	1.1	1.4	1.0	1.1	
California	06	San Bernardino	071	1.7	1.3	1.4	1.4	1.4	1.2	1.2	
California	06	San Diego	073	4.7	1.9	3.4	1.6	1.6	1.6	1.8	
California	06	San Francisco	075	1.4	1.3	1.3	1.2	1.2	1.1	1.0	
California	06	San Joaquin	077	1.0	1.0	1.0	1.0	1.0	0.7	0.7	
California	06	San Mateo	081	1.4	1.5	1.3	1.2	1.1	1.0	1.0	
California	06	Santa Clara	085	1.6	1.4	1.8	3.5	1.1	1.0	0.9	
California	06	Stanislaus	099	1.0	0.8	0.7	0.7	0.7	0.6	0.6	
Colorado	08	Adams	001	1.6	1.3	1.4	1.2	1.1	1.1	1.0	
Colorado	08	Denver	031	2.7	2.3	2.1	1.7	1.5	1.3	1.3	
Connecticut	09	Fairfield	001	1.8	1.6	1.5	1.5	1.4	1.1	1.2	
Connecticut	09	Hartford	003	2.3	2.3	2.8	2.9	2.8	2.3	2.2	
Connecticut	09	New Haven	009	1.1	1.1	1.0	1.0	1.0	0.8	1.2	
Delaware	10	New Castle	003	1.0	1.0	0.8	0.7	0.7	0.7	0.7	
District Of Columbia	11	District of Columbia	001	1.7	1.7	1.7	1.6	1.4	1.2	1.2	
Florida	12	Broward	011	1.8	1.5	1.3	1.3	1.3	1.6	1.1	
Florida	12	Duval	031	1.5	1.3	1.4	1.4	1.4	1.2	0.7	
Florida	12	Hillsborough	057	2.6	2.3	2.2	1.9	1.9	1.9	1.6	
Florida	12	Miami-Dade	086	1.6	1.4	1.2	1.3	2.1	1.9	0.9	
Florida	12	Orange	095	1.8	1.4	1.3	1.4	1.3	1.0	0.8	
Florida	12	Palm Beach	099	1.7	1.4	0.3	0.5	1.4	1.1	1.0	
Florida	12	Pinellas	103	1.7	1.6	1.4	1.3	1.3	1.2	1.0	
Georgia	13	DeKalb	089	1.2	1.1	1.3	0.9	1.0	0.9	1.0	
Georgia	13	Fulton	121	1.7	1.6	1.4	1.2	1.2	1.0	1.0	
Hawaii	15	Honolulu	003	1.3	1.2	1.1	1.6	1.2	1.2	0.9	
Idaho	16	Ada	001	2.4	2.2	2.1	1.9	1.8	1.6	1.4	
Illinois	17	Cook	031	3.1	2.6	2.4	2.4	2.0	2.1	1.4	
Illinois	17	Winnebago	201	1.9	1.7	1.6	1.5	1.1	0.9	1.0	
Indiana	18	Allen	003	1.5	1.3	1.2	1.3	1.2	1.2	1.0	
Indiana	18	Marion	097	1.9	1.7	1.7	1.4	1.3	1.3	1.2	
Iowa	19	Polk	153	1.5	1.1	1.3	1.4	1.2	1.1	0.7	
Kansas	20	Sedgwick	173	1.3	1.6	1.2	1.2	0.8	0.8	1.1	
Kentucky	21	Jefferson	111	2.4	1.4	1.4	1.5	1.4	1.3	1.1	
Louisiana	22	East Baton Rouge	033	1.3	1.1	1.1	1.0	0.9	0.8	0.6	
Louisiana	22	Orleans	071	1.6	1.5	1.4	1.1	1.4	1.3	1.2	
Maryland	24	Baltimore (City)	510	2.5	0.9	1.0	0.9	0.9	0.9	0.8	
Massachusetts	25	Hampden	013	1.7	1.7	1.7	1.5	1.7	0.7	0.6	

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(Study area and time period for Bell et al., 2009)

Massachusetts	25	Middlesex	017	1.2	1.0	0.8	0.7	0.7	0.5	0.5	
Massachusetts	25	Suffolk	025	1.5	1.2	1.0	1.2	0.9	0.6	0.5	
Massachusetts	25	Worcester	027	1.1	0.9	1.0	0.9	0.9	0.7	0.8	
Michigan	26	Kent	081	0.9	0.8	0.6	0.6	0.6	0.6	0.6	
Michigan	26	Macomb	099	0.7	0.3	0.3	0.3	0.6	0.6	0.7	
Michigan	26	Oakland	125	0.7	0.6	0.6	0.3	0.7	0.6	0.5	
Michigan	26	Wayne	163	1.4	1.3	0.9	0.8	0.8	0.8	0.7	
Minnesota	27	Dakota	037	0.5	0.3	0.3	0.3	0.3	0.3	0.3	
Minnesota	27	Hennepin	053	1.5	1.4	1.1	1.1	0.8	0.6	0.6	
Minnesota	27	Ramsey	123	2.4	2.0	1.9	1.5	1.3	1.4	1.2	
Minnesota	27	Saint Louis	137	1.1	0.9	0.8	0.9	0.8	0.7	0.7	
Mississippi	28	Hinds	049	0.5	0.6	0.7	0.6	0.7	0.6	0.6	
Missouri	29	Jackson	095	1.6	1.6	1.1	1.2	1.2	0.7	0.8	
Missouri	29	Saint Louis	189	1.3	1.0	1.1	1.0	0.9	0.8	0.7	
Missouri	29	St. Louis City	510	1.4	1.2	0.9	1.0	1.0	0.8	0.7	
Nevada	32	Clark	003	3.4	3.5	2.7	3.2	2.3	2.1	2.0	
Nevada	32	Washoe	031	2.8	2.8	2.4	2.5	2.1	1.9	1.6	
New Hampshire	33	Hillsborough	011	2.0	1.1	1.2	0.9	1.2	1.2	1.0	
New Jersey	34	Bergen	003	2.4	2.0	2.1	1.7	1.4	1.2	1.1	
New Jersey	34	Camden	007	0.8	0.8	0.9	0.8	0.8	0.7	0.6	
New Jersey	34	Hudson	017	3.4	3.1	1.9	1.9	1.9	1.8	1.7	
New Jersey	34	Middlesex	023	1.9	1.5	1.4	1.5	1.1	0.9	1.0	
New Jersey	34	Morris	027	2.0	1.8	1.5	1.3	1.1	1.0	0.9	
New Jersey	34	Union	039	2.5	2.3	1.9	1.7	1.8	1.5	1.4	
New Mexico	35	Bernalillo	001	2.1	1.9	1.9	1.8	1.3	1.2	1.2	
New York	36	Albany	001	0.5	0.6	0.5	0.6	0.7	0.6	0.5	
New York	36	Bronx	005	1.0	1.0	1.0	0.9	1.0	0.9	0.8	
New York	36	Erie	029	1.0	0.9	0.8	0.7	0.8	0.6	0.6	
New York	36	Kings	047	2.5	2.9	1.9	1.7	1.8	1.6	1.4	
New York	36	Monroe	055	1.0	0.9	0.8	0.8	0.8	0.7	0.6	
New York	36	New York	061	2.8	2.4	2.0	1.5	1.5	1.2	0.9	
New York	36	Niagara	063	0.6	0.5	0.5	0.5	0.5	0.3	0.3	
New York	36	Onondaga	067	0.9	1.0	0.9	0.9	1.0	0.8	0.8	
New York	36	Queens	081	1.0	0.9	1.0	0.9	0.9	0.8	0.8	
New York	36	Suffolk	103		0.5	0.3	0.3	0.3	0.3	0.3	
North Carolina	37	Forsyth	067	1.7	1.7	1.6	1.4	1.4	1.2	1.2	
North Carolina	37	Mecklenburg	119	1.8	1.5	1.2	1.3	0.8	0.8	0.8	
North Carolina	37	Wake	183	2.1	2.1	2.4	1.9	1.8	1.7	1.4	
Ohio	39	Cuyahoga	035	2.0	1.8	1.8	1.3	1.4	1.3	1.3	
Ohio	39	Franklin	049	1.1	1.0	0.9	1.1	1.2	1.0	0.8	
Ohio	39	Hamilton	061	1.4	1.3	1.2	1.1	1.0	0.9	0.8	
Ohio	39	Lake	085	0.8	0.8	0.8	1.0	1.5	0.7	0.7	
Ohio	39	Montgomery	113	1.0	1.1	1.0	0.9	0.8	1.0	0.6	
Ohio	39	Stark	151	1.0	0.7	0.9	0.9	0.3	0.7	1.0	
Ohio	39	Summit	153	1.5	1.5	1.5	1.3	1.2	1.0	0.9	
Oklahoma	40	Oklahoma	109	1.6	1.4	1.2	1.1	1.1	0.7	0.6	
Oklahoma	40	Tulsa	143	1.4	1.2	1.2	1.3	1.3	0.7	0.8	
Oregon	41	Lane	039	2.1	2.1	1.9	1.8	1.5	1.5	1.4	
Oregon	41	Multnomah	051	3.1	2.9	2.4	2.3	2.2	1.9	1.9	
Pennsylvania	42	Allegheny	003	1.7	1.6	1.3	0.8	0.8	0.9	0.8	

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(Study area and time period for Bell et al., 2009)

Pennsylvania	42	Berks	011	0.8	0.7	0.9	0.8	0.7	0.7	0.8		
Pennsylvania	42	Dauphin	043	0.8	0.8	0.7	0.8	0.7	0.7	0.6		
Pennsylvania	42	Erie	049	1.2	1.4	1.0	0.8	0.8	0.5	0.7		
Pennsylvania	42	Lackawanna	069	0.3	0.8	0.9	0.3	0.3	0.5	0.5		
Pennsylvania	42	Lancaster	071	0.5	0.5	0.7	0.8	0.6	0.6	0.5		
Pennsylvania	42	Northampton	095	0.6	0.7	0.5	0.5	0.3	0.5	0.3		
Pennsylvania	42	Philadelphia	101	1.8	1.6	1.5	1.3	1.4	1.2	1.1		
Pennsylvania	42	York	133	0.6	0.7	0.7	0.6	0.5	0.7	0.3		
Rhode Island	44	Providence	007	1.9	1.7	1.5	1.4	1.2	1.1	1.0		
South Carolina	45	Charleston	019	1.2	1.0	1.0	0.8	0.8	0.6	1.0		
South Carolina	45	Greenville	045	1.3	1.1	1.0	0.9	0.8	0.8	0.5		
South Carolina	45	Richland	079	1.7	1.5	1.3	1.2	1.1	0.9	0.9		
Tennessee	47	Davidson	037	2.4	1.8	1.7	1.5	1.6	1.4	1.4		
Tennessee	47	Shelby	157	2.0	1.9	1.3	1.3	1.1	1.0	0.8		
Texas	48	Bexar	029	1.4	0.9	0.9	0.8	0.9	0.8	0.8		
Texas	48	Cameron	061	0.7	0.7	0.6	0.6	0.7	0.5	0.6		
Texas	48	Dallas	113	0.6	0.7	0.6	0.6	0.6	0.6	0.6		
Texas	48	El Paso	141	4.2	2.5	2.0	1.7	1.6	2.3	1.5		
Texas	48	Harris	201	1.4	1.0	1.3	1.0	1.1	0.8	0.8		
Texas	48	Tarrant	439	1.2	0.8	0.8	0.6	0.6	0.6	0.6		
Texas	48	Travis	453	0.5	0.5	0.5	0.5	0.3	0.3	0.3		
Utah	49	Salt Lake	035	3.5	2.9	2.4	2.5	2.1	2.1	1.9		
Virginia	51	Fairfax	059	1.2	1.1	1.2	0.8	0.9	0.8	0.9		
Virginia	51	Norfolk City	710	1.3	0.9	0.9	0.8	0.7	0.5	1.0		
Washington	53	Clark	011	2.2	2.2	2.7	1.8	1.6	1.5	1.5		
Washington	53	King	033	3.0	2.8	2.6	2.1	2.1	1.8	1.7		
Washington	53	Pierce	053	1.8	2.1	1.8	1.5	1.4	1.4	1.3		
Washington	53	Spokane	063	3.5	3.2	2.9	2.8	2.7	2.3	2.1		
Wisconsin	55	Milwaukee	079	1.0	0.7	0.9	0.9	0.8	0.8	0.7		
Maxperyear				5.9	4.4	3.6	4.5	3.2	2.6	2.2	5.9	***Total Max***
Minperyear				0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	***Total Min***

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

8-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	15.8	14.5	18.9	10.7	3.9	7.7	8.5	
Arizona	04	Maricopa	013	10.0	7.0	5.9	5.5	5.2	4.3	4.6	
Arizona	04	Pima	019	3.5	4.1	2.8	3.3	2.8	2.4	2.3	
Arkansas	05	Pulaski	119	3.6	2.7	2.2	4.9	2.2	1.6	1.6	
California	06	Alameda	001	4.3	2.9	3.4	4.4	4.3	2.3	1.9	
California	06	Contra Costa	013	3.0	2.4	2.4	2.4	1.7	1.7	1.6	
California	06	Fresno	019	6.9	5.9	4.1	4.0	3.4	2.7	2.7	
California	06	Kern	029	3.7	5.1	3.1	2.3	2.2	2.2	2.0	
California	06	Los Angeles	037	10.7	9.4	6.8	7.9	6.7	5.3	4.7	
California	06	Riverside	065	3.7	4.2	3.5	3.3	2.9	2.6	2.2	
California	06	Sacramento	067	6.1	5.6	4.3	4.1	3.7	3.2	3.6	
California	06	San Bernardino	071	3.7	3.6	3.0	3.0	3.3	2.5	2.1	
California	06	San Diego	073	5.1	4.6	4.5	3.6	4.3	3.6	4.4	
California	06	San Francisco	075	4.0	2.6	2.8	2.2	2.3	2.2	2.3	
California	06	San Joaquin	077	5.4	5.5	3.4	2.9	3.0	2.2	2.3	
California	06	San Mateo	081	3.6	3.3	3.1	2.6	2.2	1.9	2.1	
California	06	Santa Clara	085	5.6	5.1	4.7	4.5	3.4	2.8	2.9	
California	06	Stanislaus	099	6.1	5.1	4.3	3.4	3.2	2.7	2.5	
Colorado	08	Adams	001	3.4	2.8	3.1	2.5	2.8	2.8	2.0	
Colorado	08	Denver	031	4.7	4.6	4.1	3.6	4.0	3.9	2.3	
Connecticut	09	Fairfield	001	3.7	2.9	2.9	2.9	2.4	2.2	2.4	
Connecticut	09	Hartford	003	4.8	5.6	4.1	5.1	5.0	4.7	3.8	
Connecticut	09	New Haven	009	3.1	2.6	2.3	2.1	1.9	1.9	1.9	
Delaware	10	New Castle	003	2.8	2.5	2.4	2.0	2.2	2.0	1.9	
District Of Columbia	11	District of Columbia	001	5.1	4.2	4.0	3.9	4.0	3.0	3.1	
Florida	12	Broward	011	4.5	3.5	3.6	3.4	3.1	2.7	2.7	
Florida	12	Duval	031	3.2	3.0	2.7	2.5	2.4	2.2	1.9	
Florida	12	Hillsborough	057	3.3	3.1	2.8	3.2	3.1	2.4	2.8	
Florida	12	Miami-Dade	086	3.6	2.9	3.8	3.0	3.5	4.7	2.0	
Florida	12	Orange	095	2.7	2.4	2.1	2.4	1.9	1.6	1.7	
Florida	12	Palm Beach	099	2.5	2.7	1.9	2.0	1.8	2.1	2.1	
Florida	12	Pinellas	103	2.5	2.2	2.2	2.2	1.6	1.6	1.5	
Georgia	13	DeKalb	089	3.8	3.0	2.8	2.5	2.5	2.4	2.0	
Georgia	13	Fulton	121	3.0	2.4	2.3	2.2	2.1	2.0	1.7	
Hawaii	15	Honolulu	003	2.1	2.0	1.6	2.1	1.9	1.9	1.5	
Idaho	16	Ada	001	4.4	2.9	2.8	3.0	3.1	2.3	2.1	
Illinois	17	Cook	031	4.7	4.3	3.6	3.6	3.3	3.1	2.3	
Illinois	17	Winnebago	201	3.5	2.8	2.4	2.2	2.1	2.1	1.9	
Indiana	18	Allen	003	3.3	3.3	2.6	3.1	2.2	2.4	2.0	
Indiana	18	Marion	097	2.6	3.8	2.1	4.4	2.3	1.9	2.0	
Iowa	19	Polk	153	3.5	3.6	2.9	2.7	2.2	2.3	2.0	
Kansas	20	Sedgwick	173	4.0	6.2	3.6	3.2	2.5	2.8	2.8	
Kentucky	21	Jefferson	111	4.8	3.4	3.6	3.9	3.0	3.1	2.5	
Louisiana	22	East Baton Rouge	033	4.2	3.4	3.6	2.8	2.4	2.3	1.7	
Louisiana	22	Orleans	071	3.0	3.4	3.2	3.3	2.6	3.3	1.7	

126County-- January 1st 1999 through December 31st, 2005

(Study area and time period for Bell et al., 2009)

Maryland	24	Baltimore (City)	510	8.0	3.0	2.9	2.8	2.4	2.5	2.1	
Massachusetts	25	Hampden	013	4.0	3.8	2.8	3.4	2.8	2.5	2.5	
Massachusetts	25	Middlesex	017	3.7	3.0	2.6	2.2	2.6	1.6	1.5	
Massachusetts	25	Suffolk	025	3.8	2.2	2.8	2.0	2.3	1.4	2.1	
Massachusetts	25	Worcester	027	2.7	2.4	2.5	2.9	1.7	1.8	2.1	
Michigan	26	Kent	081	3.1	2.3	2.6	2.6	2.0	1.6	1.6	
Michigan	26	Macomb	099	2.0	0.9	1.9	2.1	2.0	1.9	1.9	
Michigan	26	Oakland	125	2.6	2.6	3.0	1.9	2.3	2.1	1.4	
Michigan	26	Wayne	163	3.8	3.2	2.9	2.6	2.6	2.3	1.8	
Minnesota	27	Dakota	037	1.0	1.3	1.0	0.7	0.7	0.6	0.4	
Minnesota	27	Hennepin	053	2.8	2.5	2.4	2.8	1.9	1.6	1.5	
Minnesota	27	Ramsey	123	4.8	4.5	4.7	3.6	3.3	2.7	2.4	
Minnesota	27	Saint Louis	137	2.4	2.1	1.6	1.8	1.6	1.5	1.5	
Mississippi	28	Hinds	049	4.8	2.7	4.0	2.7	2.3	2.3	2.2	
Missouri	29	Jackson	095	3.3	2.9	3.4	3.2	2.1	2.1	2.2	
Missouri	29	Saint Louis	189	2.6	2.8	2.6	2.2	2.0	1.6	1.2	
Missouri	29	St. Louis City	510	3.7	3.8	4.1	7.7	2.5	2.8	2.2	
Nevada	32	Clark	003	7.6	6.2	6.2	5.8	5.2	5.0	5.1	
Nevada	32	Washoe	031	6.4	4.9	4.9	4.2	3.8	3.6	3.0	
New Hampshire	33	Hillsborough	011	4.9	4.0	3.9	3.7	3.9	2.8	3.2	
New Jersey	34	Bergen	003	4.0	3.3	2.9	3.3	2.4	2.6	2.1	
New Jersey	34	Camden	007	3.1	3.6	2.8	2.0	1.7	2.3	2.5	
New Jersey	34	Hudson	017	5.8	5.2	2.9	2.7	2.8	2.7	2.6	
New Jersey	34	Middlesex	023	3.4	3.1	2.8	2.6	1.8	2.1	1.9	
New Jersey	34	Morris	027	3.9	3.0	3.1	2.3	2.1	1.7	1.6	
New Jersey	34	Union	039	6.2	4.3	4.4	3.6	3.2	2.9	3.1	
New Mexico	35	Bernalillo	001	4.6	3.7	4.0	3.8	3.4	3.3	2.9	
New York	36	Albany	001	1.3	1.1	1.1	1.2	1.3	1.0	0.9	
New York	36	Bronx	005	3.6	3.3	2.6	1.9	2.0	1.8	1.7	
New York	36	Erie	029	2.1	1.8	1.8	1.6	1.8	1.3	1.5	
New York	36	Kings	047	4.7	4.0	3.2	3.3	2.5	2.4	2.2	
New York	36	Monroe	055	2.5	2.4	1.8	1.9	1.9	1.6	1.8	
New York	36	New York	061	4.4	3.8	3.1	2.9	2.6	2.1	1.5	
New York	36	Niagara	063	1.4	1.4	1.3	1.5	1.4	1.3	1.4	
New York	36	Onondaga	067	3.0	2.3	2.0	1.8	1.7	1.4	1.8	
New York	36	Queens	081	2.4	3.2	3.0	2.3	2.3	1.7	1.6	
New York	36	Suffolk	103		2.2	2.1	1.8	1.6	1.6	1.4	
North Carolina	37	Forsyth	067	3.4	3.6	4.0	3.9	3.4	2.9	2.4	
North Carolina	37	Mecklenburg	119	3.8	4.3	4.2	3.0	2.7	2.5	2.0	
North Carolina	37	Wake	183	4.7	5.2	3.7	3.1	2.9	2.7	2.4	
Ohio	39	Cuyahoga	035	3.6	6.6	3.0	2.1	2.6	4.7	2.8	
Ohio	39	Franklin	049	2.2	2.6	2.4	1.9	2.3	1.9	1.8	
Ohio	39	Hamilton	061	2.7	2.3	2.4	2.4	1.8	2.2	1.7	
Ohio	39	Lake	085	1.4	1.4	1.4	2.1	2.6	1.2	1.3	
Ohio	39	Montgomery	113	2.6	2.4	2.0	1.7	2.1	3.2	1.7	
Ohio	39	Stark	151	2.1	2.4	1.8	2.5	1.9	2.5	2.4	
Ohio	39	Summit	153	2.5	2.4	2.3	2.0	2.1	1.9	1.6	
Oklahoma	40	Oklahoma	109	4.0	3.8	3.2	2.8	2.6	2.0	2.0	
Oklahoma	40	Tulsa	143	3.3	3.3	4.0	2.9	3.0	1.6	1.3	
Oregon	41	Lane	039	4.5	4.2	4.0	4.1	3.3	3.4	2.7	

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Oregon	41	Multnomah	051	6.2	5.2	4.1	4.5	4.0	4.1	3.1		
Pennsylvania	42	Allegheny	003	3.5	3.2	2.6	2.6	2.2	2.3	2.0		
Pennsylvania	42	Berks	011	2.5	1.8	1.9	1.9	1.9	1.7	1.7		
Pennsylvania	42	Dauphin	043	2.5	2.1	2.6	2.0	1.5	1.3	1.3		
Pennsylvania	42	Erie	049	5.3	5.2	4.1	3.8	3.2	1.2	1.4		
Pennsylvania	42	Lackawanna	069	1.7	2.0	1.7	1.4	1.4	1.6	1.5		
Pennsylvania	42	Lancaster	071	2.0	1.8	1.8	2.0	1.6	1.5	1.3		
Pennsylvania	42	Northampton	095	2.5	2.3	1.9	1.6	1.3	1.6	1.8		
Pennsylvania	42	Philadelphia	101	4.0	3.4	3.9	2.5	3.8	3.1	3.3		
Pennsylvania	42	York	133	2.2	1.8	1.9	2.1	1.5	1.6	1.2		
Rhode Island	44	Providence	007	3.7	3.3	3.1	2.6	2.2	2.2	2.3		
South Carolina	45	Charleston	019	3.2	2.6	2.6	2.5	2.3	1.7	1.6		
South Carolina	45	Greenville	045	4.1	3.4	3.0	3.1	2.8	2.4	2.1		
South Carolina	45	Richland	079	3.3	3.2	3.0	2.5	2.1	2.1	1.9		
Tennessee	47	Davidson	037	5.3	5.0	5.6	4.4	3.6	3.6	3.1		
Tennessee	47	Shelby	157	4.7	4.2	4.5	3.5	3.2	3.0	2.5		
Texas	48	Bexar	029	3.5	2.6	2.5	2.4	2.3	1.6	2.1		
Texas	48	Cameron	061	2.5	1.5	1.2	1.8	1.7	1.2	1.3		
Texas	48	Dallas	113	2.4	2.0	2.2	2.0	1.8	1.8	2.2		
Texas	48	El Paso	141	8.8	8.8	8.0	6.2	5.9	5.4	5.1		
Texas	48	Harris	201	3.5	3.3	3.6	2.9	2.9	2.5	2.5		
Texas	48	Tarrant	439	2.6	2.1	2.3	2.0	1.5	1.5	2.5		
Texas	48	Travis	453	1.1	1.0	0.9	0.9	0.7	0.5	0.6		
Utah	49	Salt Lake	035	5.7	5.2	4.5	3.7	4.3	3.5	3.4		
Virginia	51	Fairfax	059	3.0	3.4	2.9	2.0	2.5	2.0	1.8		
Virginia	51	Norfolk City	710	3.9	3.8	3.9	3.7	2.4	2.7	1.3		
Washington	53	Clark	011	6.0	6.1	4.2	5.3	4.4	4.2	4.5		
Washington	53	King	033	6.1	6.1	6.2	4.6	5.8	3.3	3.8		
Washington	53	Pierce	053	6.4	5.1	4.4	4.3	4.8	3.3	3.9		
Washington	53	Spokane	063	5.3	5.5	4.5	4.9	4.4	3.9	4.5		
Wisconsin	55	Milwaukee	079	2.1	2.3	1.8	2.4	2.0	2.1	3.8		
Maxperyear				15.8	14.5	18.9	10.7	6.7	7.7	8.5	18.9	***Total Max***
Minperyear				1.0	0.9	0.9	0.7	0.7	0.5	0.4	0.4	***Total Min***

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8-hour - 50th percentile daily maximum (of all 365 possible daily 8-hour maximum averages)

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	1.7	1.7	1.6	1.5	0.8	0.8	0.9	
Arizona	04	Maricopa	013	4.2	2.8	2.4	3.1	2.0	1.5	1.3	
Arizona	04	Pima	019	2.0	1.8	1.6	1.4	1.2	1.2	1.1	
Arkansas	05	Pulaski	119	1.7	1.5	1.1	0.6	0.6	0.5	0.5	
California	06	Alameda	001	0.9	0.9	1.2	0.7	1.1	0.5	0.6	
California	06	Contra Costa	013	0.7	0.8	0.6	0.9	0.6	0.8	0.4	
California	06	Fresno	019	2.2	1.0	0.7	0.7	1.0	0.5	0.5	
California	06	Kern	029	0.9	1.0	1.1	0.9	0.8	0.7	0.7	
California	06	Los Angeles	037	2.2	2.0	1.6	1.6	1.4	1.6	1.1	
California	06	Riverside	065	1.7	1.6	1.4	1.2	1.0	1.0	0.9	
California	06	Sacramento	067	1.0	0.9	1.2	0.9	1.1	0.9	0.9	
California	06	San Bernardino	071	1.2	1.0	1.0	1.1	1.2	0.9	0.9	
California	06	San Diego	073	3.2	1.3	2.3	1.1	1.2	1.2	1.5	
California	06	San Francisco	075	1.2	1.0	1.1	0.9	0.9	0.9	0.8	
California	06	San Joaquin	077	0.7	0.7	0.6	0.6	0.7	0.6	0.5	
California	06	San Mateo	081	0.9	1.0	0.8	0.8	0.7	0.7	0.6	
California	06	Santa Clara	085	1.1	1.1	1.0	2.3	0.8	0.7	0.7	
California	06	Stanislaus	099	0.7	0.6	0.5	0.5	0.5	0.4	0.4	
Colorado	08	Adams	001	1.0	0.8	0.9	0.7	0.7	0.8	0.7	
Colorado	08	Denver	031	1.8	1.6	1.4	1.1	1.0	0.9	0.9	
Connecticut	09	Fairfield	001	1.4	1.2	1.2	1.2	1.1	0.9	0.9	
Connecticut	09	Hartford	003	1.8	1.7	2.2	2.4	2.2	2.0	1.8	
Connecticut	09	New Haven	009	0.9	0.9	0.8	0.8	0.8	0.7	1.1	
Delaware	10	New Castle	003	0.7	0.8	0.6	0.6	0.5	0.5	0.6	
District Of Columbia	11	District of Columbia	001	1.4	1.4	1.3	1.3	1.2	1.1	1.1	
Florida	12	Broward	011	1.2	1.1	0.9	0.8	0.9	1.3	0.9	
Florida	12	Duval	031	0.9	0.8	1.0	1.0	1.0	0.8	0.5	
Florida	12	Hillsborough	057	1.8	1.6	1.5	1.3	1.3	1.4	1.3	
Florida	12	Miami-Dade	086	1.2	1.1	1.0	1.0	1.7	1.6	0.8	
Florida	12	Orange	095	1.4	1.0	0.9	1.0	1.0	0.7	0.6	
Florida	12	Palm Beach	099	0.8	0.6	0.3	0.3	0.6	0.8	0.9	
Florida	12	Pinellas	103	1.2	1.2	1.0	0.9	1.0	0.9	0.8	
Georgia	13	DeKalb	089	0.9	0.7	0.9	0.8	0.8	0.7	0.7	
Georgia	13	Fulton	121	1.3	1.2	1.0	0.9	1.0	0.8	0.8	
Hawaii	15	Honolulu	003	1.0	0.9	0.9	1.3	1.0	1.0	0.7	
Idaho	16	Ada	001	1.6	1.5	1.5	1.4	1.3	1.2	1.1	
Illinois	17	Cook	031	2.5	2.1	2.0	2.0	1.7	1.7	1.1	
Illinois	17	Winnebago	201	1.5	1.3	1.2	1.2	0.9	0.7	0.8	
Indiana	18	Allen	003	1.1	1.0	0.9	1.0	1.0	1.1	0.9	
Indiana	18	Marion	097	1.4	1.3	1.2	1.1	1.0	0.9	0.9	
Iowa	19	Polk	153	1.2	0.9	1.2	1.2	1.0	1.0	0.6	
Kansas	20	Sedgwick	173	0.9	1.2	1.0	0.9	0.5	0.5	0.9	
Kentucky	21	Jefferson	111	1.8	1.0	1.1	1.3	1.2	1.1	0.9	
Louisiana	22	East Baton Rouge	033	1.1	0.9	1.0	0.9	0.8	0.7	0.5	
Louisiana	22	Orleans	071	1.1	1.1	1.0	0.8	1.1	1.0	1.0	
Maryland	24	Baltimore (City)	510	1.6	0.7	0.7	0.7	0.7	0.7	0.6	

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Massachusetts	25	Hampden	013	1.3	1.2	1.2	1.2	1.0	0.6	0.4	
Massachusetts	25	Middlesex	017	0.9	0.7	0.6	0.5	0.5	0.3	0.3	
Massachusetts	25	Suffolk	025	1.2	0.9	0.8	1.0	0.8	0.4	0.4	
Massachusetts	25	Worcester	027	0.8	0.7	0.8	0.7	0.7	0.5	0.7	
Michigan	26	Kent	081	0.6	0.6	0.4	0.4	0.4	0.4	0.4	
Michigan	26	Macomb	099	0.4	0.3	0.3	0.3	0.4	0.4	0.5	
Michigan	26	Oakland	125	0.4	0.4	0.4	0.3	0.5	0.4	0.3	
Michigan	26	Wayne	163	0.9	0.9	0.5	0.6	0.5	0.6	0.5	
Minnesota	27	Dakota	037	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
Minnesota	27	Hennepin	053	1.1	1.1	0.8	0.9	0.5	0.4	0.4	
Minnesota	27	Ramsey	123	1.8	1.6	1.4	1.1	0.9	0.9	0.8	
Minnesota	27	Saint Louis	137	0.8	0.6	0.5	0.7	0.6	0.4	0.5	
Mississippi	28	Hinds	049	0.4	0.4	0.4	0.4	0.5	0.4	0.4	
Missouri	29	Jackson	095	1.2	1.1	0.8	0.8	0.8	0.6	0.7	
Missouri	29	Saint Louis	189	1.0	0.8	0.8	0.8	0.8	0.7	0.6	
Missouri	29	St. Louis City	510	1.2	0.9	0.8	0.8	0.8	0.6	0.4	
Nevada	32	Clark	003	2.0	2.7	1.6	2.3	1.4	1.3	1.2	
Nevada	32	Washoe	031	1.5	1.5	1.5	1.2	1.2	1.0	0.7	
New Hampshire	33	Hillsborough	011	1.1	1.0	0.8	0.5	0.9	1.0	0.8	
New Jersey	34	Bergen	003	2.0	1.7	1.8	1.4	1.1	1.0	0.9	
New Jersey	34	Camden	007	0.6	0.7	0.7	0.6	0.7	0.6	0.5	
New Jersey	34	Hudson	017	2.4	2.2	1.5	1.5	1.5	1.5	1.4	
New Jersey	34	Middlesex	023	1.4	1.1	1.1	1.2	0.8	0.7	0.8	
New Jersey	34	Morris	027	1.5	1.4	1.2	1.0	0.9	0.8	0.7	
New Jersey	34	Union	039	1.9	1.8	1.5	1.3	1.3	1.2	1.1	
New Mexico	35	Bernalillo	001	1.3	1.3	1.4	1.2	0.8	0.7	0.8	
New York	36	Albany	001	0.3	0.4	0.4	0.4	0.6	0.5	0.3	
New York	36	Bronx	005	0.8	0.8	0.8	0.8	0.9	0.7	0.7	
New York	36	Erie	029	0.8	0.7	0.6	0.6	0.6	0.5	0.4	
New York	36	Kings	047	2.0	2.2	1.5	1.4	1.4	1.2	1.0	
New York	36	Monroe	055	0.8	0.7	0.6	0.6	0.7	0.7	0.5	
New York	36	New York	061	2.3	2.0	1.7	1.3	1.3	1.1	0.8	
New York	36	Niagara	063	0.4	0.4	0.3	0.3	0.4	0.3	0.3	
New York	36	Onondaga	067	0.6	0.8	0.6	0.6	0.8	0.6	0.6	
New York	36	Queens	081	0.9	0.7	0.7	0.7	0.8	0.7	0.6	
New York	36	Suffolk	103		0.3	0.3	0.3	0.3	0.3	0.3	
North Carolina	37	Forsyth	067	1.3	1.1	1.1	1.1	1.1	0.9	1.0	
North Carolina	37	Mecklenburg	119	1.3	1.1	0.9	1.1	0.6	0.5	0.7	
North Carolina	37	Wake	183	1.4	1.5	1.9	1.6	1.5	1.4	1.1	
Ohio	39	Cuyahoga	035	1.5	1.3	1.2	1.0	1.0	0.9	0.9	
Ohio	39	Franklin	049	0.8	0.7	0.7	0.6	0.7	0.6	0.6	
Ohio	39	Hamilton	061	1.0	1.0	0.9	0.8	0.7	0.7	0.6	
Ohio	39	Lake	085	0.6	0.7	0.7	0.8	1.3	0.7	0.5	
Ohio	39	Montgomery	113	0.7	0.8	0.8	0.7	0.6	0.8	0.5	
Ohio	39	Stark	151	0.8	0.5	0.7	0.6	0.3	0.6	0.9	
Ohio	39	Summit	153	1.0	1.1	1.1	0.9	0.8	0.7	0.6	
Oklahoma	40	Oklahoma	109	1.4	1.1	1.0	0.9	0.9	0.6	0.4	
Oklahoma	40	Tulsa	143	1.1	0.9	1.1	1.1	1.1	0.5	0.7	
Oregon	41	Lane	039	1.6	1.6	1.5	1.4	1.2	1.1	1.1	
Oregon	41	Multnomah	051	2.4	2.1	1.9	1.8	1.7	1.6	1.5	

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Pennsylvania	42	Allegheny	003	1.3	1.3	1.0	0.5	0.6	0.6	0.6		
Pennsylvania	42	Berks	011	0.5	0.5	0.7	0.6	0.4	0.5	0.5		
Pennsylvania	42	Dauphin	043	0.5	0.5	0.4	0.5	0.6	0.5	0.5		
Pennsylvania	42	Erie	049	0.8	0.9	0.8	0.6	0.6	0.4	0.5		
Pennsylvania	42	Lackawanna	069	0.3	0.5	0.6	0.3	0.3	0.3	0.3		
Pennsylvania	42	Lancaster	071	0.3	0.3	0.4	0.6	0.4	0.4	0.3		
Pennsylvania	42	Northampton	095	0.4	0.4	0.3	0.3	0.3	0.3	0.3		
Pennsylvania	42	Philadelphia	101	1.4	1.2	1.2	1.0	1.1	1.0	1.0		
Pennsylvania	42	York	133	0.4	0.4	0.4	0.4	0.4	0.5	0.3		
Rhode Island	44	Providence	007	1.4	1.3	1.1	1.0	0.9	0.8	0.8		
South Carolina	45	Charleston	019	0.8	0.7	0.6	0.5	0.5	0.3	0.6		
South Carolina	45	Greenville	045	0.8	0.8	0.7	0.6	0.6	0.5	0.3		
South Carolina	45	Richland	079	1.1	1.1	1.0	0.9	0.8	0.7	0.6		
Tennessee	47	Davidson	037	1.8	1.2	1.3	1.2	1.3	1.1	1.1		
Tennessee	47	Shelby	157	1.4	1.4	0.9	1.0	0.9	0.8	0.5		
Texas	48	Bexar	029	1.1	0.7	0.7	0.6	0.7	0.6	0.7		
Texas	48	Cameron	061	0.4	0.5	0.4	0.4	0.5	0.3	0.3		
Texas	48	Dallas	113	0.4	0.4	0.4	0.4	0.5	0.4	0.4		
Texas	48	El Paso	141	3.3	1.6	1.3	1.1	1.1	1.6	1.0		
Texas	48	Harris	201	1.0	0.7	1.0	0.7	0.8	0.6	0.5		
Texas	48	Tarrant	439	1.0	0.7	0.6	0.5	0.5	0.4	0.4		
Texas	48	Travis	453	0.3	0.3	0.3	0.3	0.3	0.3	0.3		
Utah	49	Salt Lake	035	2.4	1.9	1.7	1.6	1.4	1.4	1.2		
Virginia	51	Fairfax	059	1.0	0.9	1.1	0.6	0.8	0.7	0.8		
Virginia	51	Norfolk City	710	1.0	0.7	0.7	0.6	0.5	0.3	0.8		
Washington	53	Clark	011	1.7	1.7	2.0	1.3	1.2	1.1	1.0		
Washington	53	King	033	2.4	2.1	2.0	1.7	1.8	1.4	1.4		
Washington	53	Pierce	053	1.3	1.6	1.2	1.1	1.1	1.1	1.0		
Washington	53	Spokane	063	2.7	2.4	2.3	2.1	2.0	1.7	1.6		
Wisconsin	55	Milwaukee	079	0.8	0.5	0.9	0.7	0.6	0.7	0.5		
Maxperyear				4.2	2.8	2.4	3.1	2.2	2.0	1.8	4.2	***Total Max***
Minperyear				0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	***Total Min***

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

24-hour - 99th percentile daily maximum (of all 365 possible daily 8-hour

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	9.3	6.4	11.0	4.0	2.2	3.7	5.0	
Arizona	04	Maricopa	013	5.6	4.1	3.6	4.2	3.5	3.1	3.3	
Arizona	04	Pima	019	2.1	2.4	1.9	1.7	1.7	1.3	1.3	
Arkansas	05	Pulaski	119	2.7	1.8	1.6	4.0	1.3	0.9	1.0	
California	06	Alameda	001	2.9	2.1	2.3	2.6	2.3	1.3	1.4	
California	06	Contra Costa	013	1.9	1.7	1.6	1.4	1.1	1.0	1.0	
California	06	Fresno	019	3.7	3.3	2.4	2.0	1.9	1.6	1.5	
California	06	Kern	029	2.5	3.8	2.1	1.5	1.5	1.3	1.3	
California	06	Los Angeles	037	6.8	5.9	4.5	4.4	4.2	3.6	2.9	
California	06	Riverside	065	2.8	3.1	2.6	2.3	2.1	1.9	1.7	
California	06	Sacramento	067	3.7	3.7	2.2	2.1	2.6	2.0	2.2	
California	06	San Bernardino	071	2.4	2.4	2.1	1.9	2.1	1.7	1.4	
California	06	San Diego	073	3.3	3.5	2.9	2.5	2.6	2.3	3.9	
California	06	San Francisco	075	2.1	1.6	1.8	1.5	1.5	1.3	1.2	
California	06	San Joaquin	077	3.1	2.8	1.7	1.6	1.5	1.2	1.3	
California	06	San Mateo	081	2.6	2.2	2.1	1.8	1.5	1.2	1.4	
California	06	Santa Clara	085	3.7	3.6	3.4	3.3	2.2	1.9	2.0	
California	06	Stanislaus	099	2.8	2.2	2.1	1.8	1.5	1.4	1.6	
Colorado	08	Adams	001	2.1	1.9	1.9	1.4	1.5	1.7	1.3	
Colorado	08	Denver	031	2.9	3.2	2.6	1.7	2.3	1.8	1.8	
Connecticut	09	Fairfield	001	2.3	2.0	2.2	1.9	1.7	1.5	1.6	
Connecticut	09	Hartford	003	2.9	3.1	2.8	2.9	2.7	2.5	2.5	
Connecticut	09	New Haven	009	1.6	1.6	1.6	1.5	1.3	1.3	1.3	
Delaware	10	New Castle	003	1.7	1.5	1.5	1.2	1.2	1.1	1.3	
District Of Columbia	11	District of Columbia	001	3.1	2.7	2.7	2.2	2.6	1.7	2.2	
Florida	12	Broward	011	2.1	2.0	2.3	1.7	2.4	2.1	1.8	
Florida	12	Duval	031	2.1	2.1	1.4	1.7	1.5	1.4	1.8	
Florida	12	Hillsborough	057	2.4	2.1	1.8	2.5	2.1	1.9	2.2	
Florida	12	Miami-Dade	086	2.0	1.8	1.9	1.7	2.4	2.5	1.3	
Florida	12	Orange	095	2.1	1.7	1.5	1.6	1.4	1.2	1.3	
Florida	12	Palm Beach	099	1.4	1.3	0.9	0.9	1.1	1.2	1.3	
Florida	12	Pinellas	103	1.5	1.5	1.3	1.5	1.3	1.1	1.1	
Georgia	13	DeKalb	089	2.0	1.5	1.9	1.5	1.6	1.4	1.2	
Georgia	13	Fulton	121	2.3	1.8	1.8	1.6	1.7	1.5	1.2	
Hawaii	15	Honolulu	003	1.6	1.4	1.2	1.7	1.4	1.5	1.2	
Idaho	16	Ada	001	2.8	2.1	2.0	2.4	2.4	1.7	1.6	
Illinois	17	Cook	031	3.4	3.0	3.1	2.7	2.5	2.6	1.6	
Illinois	17	Winnebago	201	2.0	1.9	1.7	1.6	1.2	1.0	1.3	
Indiana	18	Allen	003	1.8	2.7	1.6	1.7	1.6	1.4	1.2	
Indiana	18	Marion	097	1.5	3.2	1.4	2.1	1.3	1.2	1.3	
Iowa	19	Polk	153	2.0	1.7	1.7	1.6	1.6	1.3	1.4	
Kansas	20	Sedgwick	173	3.0	5.8	1.6	1.3	1.3	1.2	1.6	
Kentucky	21	Jefferson	111	3.6	2.1	2.7	2.8	2.4	2.0	1.6	
Louisiana	22	East Baton Rouge	033	2.2	1.6	1.8	2.0	1.3	1.3	0.9	
Louisiana	22	Orleans	071	1.6	1.6	1.8	1.6	1.6	2.3	1.3	
Maryland	24	Baltimore (City)	510	3.9	1.7	1.8	1.9	1.6	1.4	1.4	
Massachusetts	25	Hampden	013	2.7	2.0	2.0	2.1	1.7	1.3	1.4	

126County-- January 1st 1999 through December 31st, 2005

(Study area and time period for Bell et al., 2009)

Massachusetts	25	Middlesex	017	2.0	1.4	1.6	1.3	1.5	1.0	1.0	
Massachusetts	25	Suffolk	025	2.1	1.5	1.6	1.5	1.4	0.9	1.3	
Massachusetts	25	Worcester	027	1.8	1.5	1.6	1.5	1.1	0.9	1.1	
Michigan	26	Kent	081	1.5	1.2	1.9	1.4	1.0	0.9	1.0	
Michigan	26	Macomb	099	1.0	0.6	1.4	1.0	1.1	1.5	1.3	
Michigan	26	Oakland	125	2.4	1.5	1.9	1.4	1.2	1.1	0.9	
Michigan	26	Wayne	163	2.2	1.6	1.5	2.5	1.2	1.3	1.0	
Minnesota	27	Dakota	037	0.7	1.1	0.8	0.5	0.6	0.5	0.3	
Minnesota	27	Hennepin	053	1.6	1.7	1.2	1.3	0.9	0.9	0.7	
Minnesota	27	Ramsey	123	2.8	2.4	2.5	1.7	1.5	1.3	1.5	
Minnesota	27	Saint Louis	137	1.3	1.2	0.9	1.2	1.0	0.8	0.9	
Mississippi	28	Hinds	049	2.2	1.6	1.7	1.2	1.2	1.2	1.1	
Missouri	29	Jackson	095	2.3	2.1	1.8	1.3	1.2	1.4	1.6	
Missouri	29	Saint Louis	189	1.6	1.4	1.9	1.3	1.2	1.1	1.1	
Missouri	29	St. Louis City	510	2.0	2.1	1.8	5.2	1.5	1.2	1.2	
Nevada	32	Clark	003	4.9	4.0	3.8	3.3	3.2	3.0	3.1	
Nevada	32	Washoe	031	4.1	3.5	3.4	2.6	2.3	2.2	2.1	
New Hampshire	33	Hillsborough	011	2.5	2.3	2.3	1.9	2.1	1.6	1.7	
New Jersey	34	Bergen	003	2.7	2.3	2.0	1.9	1.6	1.5	1.5	
New Jersey	34	Camden	007	1.5	1.6	1.7	1.4	1.2	1.2	1.3	
New Jersey	34	Hudson	017	3.7	3.0	2.0	1.9	2.1	2.2	1.8	
New Jersey	34	Middlesex	023	2.9	1.8	2.0	1.6	1.3	1.3	1.4	
New Jersey	34	Morris	027	2.5	1.9	2.0	1.4	1.3	1.2	1.2	
New Jersey	34	Union	039	3.3	2.8	2.8	2.2	2.1	2.0	2.1	
New Mexico	35	Bernalillo	001	2.8	2.3	2.6	2.5	1.8	1.9	1.8	
New York	36	Albany	001	1.1	0.9	0.8	1.0	1.1	0.7	0.7	
New York	36	Bronx	005	1.7	1.6	1.5	1.3	1.4	1.2	1.1	
New York	36	Erie	029	1.2	1.2	0.9	1.1	1.2	0.8	1.0	
New York	36	Kings	047	3.1	2.8	2.3	2.0	2.0	1.7	1.6	
New York	36	Monroe	055	1.4	1.2	1.0	1.0	1.0	1.1	1.2	
New York	36	New York	061	2.7	2.9	2.2	1.9	1.8	1.5	1.0	
New York	36	Niagara	063	0.8	1.0	0.8	1.0	0.8	0.6	0.8	
New York	36	Onondaga	067	1.7	1.4	1.3	1.1	1.1	0.9	1.1	
New York	36	Queens	081	1.5	1.4	1.6	1.3	1.4	1.4	1.1	
New York	36	Suffolk	103		1.0	1.2	0.9	0.7	0.6	0.6	
North Carolina	37	Forsyth	067	2.1	2.0	1.9	1.9	1.8	1.8	1.5	
North Carolina	37	Mecklenburg	119	2.5	2.2	2.3	1.7	1.5	1.5	1.3	
North Carolina	37	Wake	183	2.6	3.0	2.3	2.3	2.4	2.0	1.9	
Ohio	39	Cuyahoga	035	2.1	2.4	1.8	1.3	1.6	2.4	1.6	
Ohio	39	Franklin	049	1.2	1.4	1.3	1.1	1.2	1.2	1.0	
Ohio	39	Hamilton	061	1.9	1.6	1.5	1.2	1.2	1.1	1.2	
Ohio	39	Lake	085	1.2	1.0	1.0	2.0	2.5	0.9	1.0	
Ohio	39	Montgomery	113	1.2	1.6	1.3	1.1	1.3	1.8	1.2	
Ohio	39	Stark	151	1.3	1.6	1.3	2.2	1.5	1.9	1.8	
Ohio	39	Summit	153	1.5	1.5	1.6	1.3	1.3	1.2	1.1	
Oklahoma	40	Oklahoma	109	1.8	1.7	1.8	1.3	1.2	1.0	1.1	
Oklahoma	40	Tulsa	143	2.0	2.0	2.2	2.1	1.7	1.0	0.9	
Oregon	41	Lane	039	2.9	2.3	2.3	2.4	1.9	1.9	1.6	
Oregon	41	Multnomah	051	3.6	2.7	2.6	2.8	2.4	2.3	2.1	
Pennsylvania	42	Allegheny	003	2.3	2.3	1.8	1.5	1.4	1.2	1.4	

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(Study area and time period for Bell et al., 2009)

Pennsylvania	42	Berks	011	1.4	1.2	1.2	1.2	1.2	1.1	1.2		
Pennsylvania	42	Dauphin	043	1.1	1.4	1.5	1.1	1.1	0.9	1.0		
Pennsylvania	42	Erie	049	1.9	1.9	1.7	1.6	1.2	0.7	1.2		
Pennsylvania	42	Lackawanna	069	0.8	1.2	1.2	1.0	0.8	1.0	1.1		
Pennsylvania	42	Lancaster	071	1.3	1.3	1.5	1.3	1.0	0.9	0.9		
Pennsylvania	42	Northampton	095	1.6	1.3	1.3	0.9	0.7	1.2	1.2		
Pennsylvania	42	Philadelphia	101	4.0	1.8	1.9	1.6	1.9	1.6	1.6		
Pennsylvania	42	York	133	1.4	1.0	1.2	1.0	0.9	1.1	0.8		
Rhode Island	44	Providence	007	2.3	2.1	1.9	1.7	1.5	1.4	1.3		
South Carolina	45	Charleston	019	1.7	1.2	1.1	1.1	0.9	0.8	0.8		
South Carolina	45	Greenville	045	2.2	1.5	1.5	1.6	1.5	1.2	1.4		
South Carolina	45	Richland	079	2.0	2.0	1.8	1.5	1.2	1.1	1.2		
Tennessee	47	Davidson	037	3.1	2.4	3.0	2.1	2.3	2.0	1.8		
Tennessee	47	Shelby	157	2.2	2.5	2.3	1.7	1.6	1.6	1.3		
Texas	48	Bexar	029	2.0	1.3	1.4	1.1	1.3	0.9	1.2		
Texas	48	Cameron	061	1.7	1.0	0.7	0.8	0.8	0.7	0.9		
Texas	48	Dallas	113	1.4	1.1	1.1	1.0	0.9	0.9	1.3		
Texas	48	El Paso	141	3.8	3.9	3.4	2.8	2.4	2.3	2.2		
Texas	48	Harris	201	2.1	1.8	2.3	1.5	1.9	1.4	1.1		
Texas	48	Tarrant	439	1.7	0.9	1.2	0.8	0.8	0.8	1.2		
Texas	48	Travis	453	0.8	0.5	0.6	0.5	0.5	0.4	0.3		
Utah	49	Salt Lake	035	3.7	3.7	2.9	2.5	2.9	2.1	2.4		
Virginia	51	Fairfax	059	1.8	2.3	1.8	1.4	1.4	1.2	1.2		
Virginia	51	Norfolk City	710	1.8	1.8	1.7	1.7	1.2	1.1	0.9		
Washington	53	Clark	011	3.5	3.5	2.8	3.0	2.7	2.8	2.6		
Washington	53	King	033	4.0	3.4	3.4	2.8	3.6	2.1	2.0		
Washington	53	Pierce	053	3.9	3.4	2.8	2.7	2.8	2.5	2.3		
Washington	53	Spokane	063	3.5	3.1	2.8	3.2	2.7	2.3	2.7		
Wisconsin	55	Milwaukee	079	1.4	1.3	1.3	1.1	1.3	1.2	1.5		
Maxperyear				9.3	6.4	11.0	5.2	4.2	3.7	5.0	11.0	***Total Max***
Minperyear				0.7	0.5	0.6	0.5	0.5	0.4	0.3	0.3	***Total Min***

126County-- January 1st 1999 through December 31st, 2005
(Study area and time period for Bell et al., 2009)

24-hour - 50th percentile daily maximum (of all 365 possible daily 8-hour

STATENAME	fips	COUNTYNAME	fips	1999	2000	2001	2002	2003	2004	2005	
Alabama	01	Jefferson	073	1.1	1.3	1.1	1.1	0.5	0.4	0.6	
Arizona	04	Maricopa	013	2.7	1.8	1.5	1.9	1.2	0.9	0.8	
Arizona	04	Pima	019	1.1	1.0	0.9	0.8	0.7	0.7	0.7	
Arkansas	05	Pulaski	119	1.3	1.2	0.8	0.4	0.4	0.3	0.3	
California	06	Alameda	001	0.7	0.7	0.6	0.6	0.6	0.3	0.5	
California	06	Contra Costa	013	0.5	0.5	0.4	0.5	0.5	0.6	0.3	
California	06	Fresno	019	1.0	0.5	0.4	0.5	0.8	0.3	0.3	
California	06	Kern	029	0.7	0.7	0.8	0.5	0.6	0.5	0.5	
California	06	Los Angeles	037	1.5	1.4	1.2	1.1	1.1	0.9	0.8	
California	06	Riverside	065	1.2	1.2	1.1	0.9	0.8	0.7	0.7	
California	06	Sacramento	067	0.6	0.5	0.7	0.6	0.8	0.6	0.7	
California	06	San Bernardino	071	0.9	0.8	0.7	0.9	0.9	0.6	0.6	
California	06	San Diego	073	2.1	0.9	1.3	0.8	0.9	1.0	1.2	
California	06	San Francisco	075	0.9	0.8	0.8	0.7	0.7	0.7	0.6	
California	06	San Joaquin	077	0.5	0.4	0.4	0.4	0.5	0.4	0.3	
California	06	San Mateo	081	0.6	0.7	0.6	0.5	0.5	0.5	0.4	
California	06	Santa Clara	085	0.8	0.7	0.9	1.4	0.5	0.5	0.6	
California	06	Stanislaus	099	0.5	0.4	0.3	0.3	0.4	0.3	0.3	
Colorado	08	Adams	001	0.6	0.5	0.5	0.4	0.4	0.5	0.5	
Colorado	08	Denver	031	1.1	1.0	1.0	0.7	0.7	0.6	0.6	
Connecticut	09	Fairfield	001	0.9	0.9	0.9	0.9	0.9	0.7	0.7	
Connecticut	09	Hartford	003	1.2	1.1	1.5	1.5	1.4	1.3	1.2	
Connecticut	09	New Haven	009	0.6	0.6	0.6	0.6	0.6	0.5	0.8	
Delaware	10	New Castle	003	0.4	0.6	0.4	0.4	0.3	0.3	0.4	
District Of Columbia	11	District of Columbia	001	1.0	1.1	1.0	1.1	1.0	0.9	0.9	
Florida	12	Broward	011	0.9	0.8	0.6	0.7	0.6	1.1	0.7	
Florida	12	Duval	031	0.5	0.5	0.6	0.7	0.6	0.6	0.4	
Florida	12	Hillsborough	057	1.3	1.2	1.1	1.0	0.9	1.1	1.0	
Florida	12	Miami-Dade	086	0.9	0.9	0.8	0.8	1.4	1.1	0.6	
Florida	12	Orange	095	1.0	0.7	0.6	0.7	0.8	0.5	0.5	
Florida	12	Palm Beach	099	0.4	0.4	0.3	0.3	0.4	0.7	0.7	
Florida	12	Pinellas	103	0.8	0.6	0.6	0.6	0.7	0.6	0.5	
Georgia	13	DeKalb	089	0.5	0.4	0.6	0.5	0.6	0.5	0.5	
Georgia	13	Fulton	121	0.8	0.8	0.7	0.6	0.7	0.5	0.5	
Hawaii	15	Honolulu	003	0.9	0.8	0.8	1.0	0.8	0.8	0.6	
Idaho	16	Ada	001	1.1	1.0	1.0	1.0	0.9	0.9	0.8	
Illinois	17	Cook	031	1.7	1.5	1.6	1.6	1.2	1.3	0.7	
Illinois	17	Winnebago	201	1.0	0.9	0.8	0.8	0.5	0.4	0.5	
Indiana	18	Allen	003	0.7	0.6	0.5	0.6	0.6	0.8	0.6	
Indiana	18	Marion	097	0.9	0.8	0.8	0.7	0.6	0.6	0.6	
Iowa	19	Polk	153	0.9	0.8	0.9	1.0	0.9	0.8	0.4	
Kansas	20	Sedgwick	173	0.5	0.8	0.9	0.6	0.3	0.3	0.7	
Kentucky	21	Jefferson	111	1.2	0.6	0.7	0.6	0.9	0.9	0.7	
Louisiana	22	East Baton Rouge	033	0.9	0.7	0.8	0.7	0.6	0.5	0.4	
Louisiana	22	Orleans	071	0.8	0.8	0.8	0.6	0.9	0.8	0.8	
Maryland	24	Baltimore (City)	510	1.0	0.5	0.5	0.5	0.5	0.5	0.4	
Massachusetts	25	Hampden	013	0.8	0.8	0.8	0.9	0.6	0.4	0.3	

126County-- January 1st 1999 through December 31st, 2005

(Study area and time period for Bell et al., 2009)

Massachusetts	25	Middlesex	017	0.6	0.4	0.4	0.3	0.3	0.3	0.3	
Massachusetts	25	Suffolk	025	0.9	0.6	0.5	0.8	0.6	0.3	0.3	
Massachusetts	25	Worcester	027	0.6	0.5	0.5	0.5	0.5	0.4	0.5	
Michigan	26	Kent	081	0.4	0.4	0.3	0.3	0.3	0.3	0.3	
Michigan	26	Macomb	099	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
Michigan	26	Oakland	125	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
Michigan	26	Wayne	163	0.6	0.5	0.3	0.4	0.3	0.4	0.3	
Minnesota	27	Dakota	037	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
Minnesota	27	Hennepin	053	0.7	0.7	0.5	0.6	0.3	0.3	0.3	
Minnesota	27	Ramsey	123	1.1	1.0	0.8	0.6	0.5	0.5	0.4	
Minnesota	27	Saint Louis	137	0.4	0.4	0.3	0.4	0.4	0.3	0.3	
Mississippi	28	Hinds	049	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
Missouri	29	Jackson	095	0.8	0.8	0.5	0.5	0.5	0.4	0.5	
Missouri	29	Saint Louis	189	0.7	0.6	0.5	0.6	0.6	0.6	0.4	
Missouri	29	St. Louis City	510	0.9	0.7	0.7	0.7	0.6	0.4	0.3	
Nevada	32	Clark	003	1.4	1.2	1.0	1.2	0.7	0.9	0.9	
Nevada	32	Washoe	031	1.1	0.8	0.9	0.7	0.7	0.6	0.4	
New Hampshire	33	Hillsborough	011	0.8	0.6	0.5	0.3	0.6	0.7	0.6	
New Jersey	34	Bergen	003	1.4	1.2	1.3	1.0	0.9	0.7	0.6	
New Jersey	34	Camden	007	0.4	0.5	0.5	0.5	0.6	0.5	0.4	
New Jersey	34	Hudson	017	1.6	1.5	1.0	1.0	1.0	1.0	1.0	
New Jersey	34	Middlesex	023	1.0	0.8	0.8	0.8	0.6	0.5	0.6	
New Jersey	34	Morris	027	1.0	0.9	0.8	0.7	0.7	0.6	0.5	
New Jersey	34	Union	039	1.2	1.1	0.9	0.9	0.9	0.8	0.7	
New Mexico	35	Bernalillo	001	1.0	0.9	1.0	0.8	0.6	0.4	0.5	
New York	36	Albany	001	0.3	0.3	0.3	0.3	0.4	0.3	0.3	
New York	36	Bronx	005	0.6	0.6	0.6	0.6	0.7	0.6	0.6	
New York	36	Erie	029	0.5	0.5	0.4	0.4	0.4	0.3	0.3	
New York	36	Kings	047	1.4	1.7	1.1	1.0	1.1	1.0	0.8	
New York	36	Monroe	055	0.6	0.5	0.4	0.5	0.5	0.5	0.4	
New York	36	New York	061	1.7	1.5	1.3	1.0	1.0	0.9	0.6	
New York	36	Niagara	063	0.3	0.3	0.3	0.3	0.3	0.3	0.3	
New York	36	Onondaga	067	0.4	0.5	0.4	0.4	0.6	0.4	0.4	
New York	36	Queens	081	0.6	0.5	0.5	0.5	0.6	0.5	0.5	
New York	36	Suffolk	103		0.3	0.3	0.3	0.3	0.3	0.3	
North Carolina	37	Forsyth	067	0.8	0.8	0.7	0.8	0.7	0.6	0.7	
North Carolina	37	Mecklenburg	119	1.0	0.8	0.6	0.8	0.5	0.4	0.5	
North Carolina	37	Wake	183	0.9	0.9	1.0	0.9	1.0	1.0	0.8	
Ohio	39	Cuyahoga	035	1.0	0.8	0.8	0.6	0.7	0.6	0.6	
Ohio	39	Franklin	049	0.5	0.4	0.4	0.4	0.5	0.4	0.4	
Ohio	39	Hamilton	061	0.7	0.7	0.7	0.5	0.5	0.4	0.4	
Ohio	39	Lake	085	0.4	0.4	0.4	0.6	1.1	0.4	0.3	
Ohio	39	Montgomery	113	0.5	0.6	0.5	0.4	0.4	0.7	0.3	
Ohio	39	Stark	151	0.6	0.3	0.6	0.4	0.3	0.4	0.7	
Ohio	39	Summit	153	0.7	0.7	0.8	0.6	0.5	0.4	0.4	
Oklahoma	40	Oklahoma	109	0.9	0.8	0.7	0.6	0.6	0.4	0.3	
Oklahoma	40	Tulsa	143	0.8	0.7	0.9	0.8	0.8	0.3	0.5	
Oregon	41	Lane	039	1.0	1.0	1.0	0.9	0.8	0.7	0.7	
Oregon	41	Multnomah	051	1.5	1.4	1.2	1.1	1.0	0.9	0.9	
Pennsylvania	42	Allegheny	003	0.9	0.9	0.7	0.3	0.4	0.4	0.4	

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(Study area and time period for Bell et al., 2009)

Pennsylvania	42	Berks	011	0.3	0.3	0.5	0.4	0.3	0.4	0.4		
Pennsylvania	42	Dauphin	043	0.3	0.3	0.3	0.4	0.4	0.3	0.3		
Pennsylvania	42	Erie	049	0.5	0.5	0.5	0.4	0.4	0.3	0.4		
Pennsylvania	42	Lackawanna	069	0.3	0.3	0.4	0.3	0.3	0.3	0.3		
Pennsylvania	42	Lancaster	071	0.3	0.3	0.3	0.4	0.3	0.3	0.3		
Pennsylvania	42	Northampton	095	0.3	0.3	0.3	0.3	0.3	0.3	0.3		
Pennsylvania	42	Philadelphia	101	1.0	0.8	0.8	0.7	0.8	0.7	0.7		
Pennsylvania	42	York	133	0.3	0.3	0.3	0.3	0.3	0.4	0.3		
Rhode Island	44	Providence	007	1.0	1.0	0.8	0.7	0.6	0.6	0.6		
South Carolina	45	Charleston	019	0.5	0.4	0.4	0.3	0.4	0.3	0.4		
South Carolina	45	Greenville	045	0.5	0.4	0.4	0.4	0.4	0.3	0.3		
South Carolina	45	Richland	079	0.7	0.7	0.6	0.5	0.5	0.5	0.4		
Tennessee	47	Davidson	037	1.3	0.9	0.8	0.8	1.0	0.9	0.9		
Tennessee	47	Shelby	157	0.8	0.9	0.6	0.6	0.5	0.5	0.3		
Texas	48	Bexar	029	0.8	0.4	0.5	0.4	0.5	0.4	0.5		
Texas	48	Cameron	061	0.3	0.3	0.3	0.3	0.4	0.3	0.3		
Texas	48	Dallas	113	0.3	0.3	0.3	0.3	0.3	0.3	0.3		
Texas	48	El Paso	141	1.4	0.8	0.7	0.6	0.6	0.8	0.7		
Texas	48	Harris	201	0.8	0.5	0.8	0.5	0.6	0.4	0.4		
Texas	48	Tarrant	439	0.7	0.5	0.4	0.3	0.3	0.3	0.3		
Texas	48	Travis	453	0.3	0.3	0.3	0.3	0.3	0.3	0.3		
Utah	49	Salt Lake	035	1.3	1.2	1.0	0.9	0.9	1.0	0.8		
Virginia	51	Fairfax	059	0.8	0.8	0.9	0.5	0.6	0.5	0.6		
Virginia	51	Norfolk City	710	0.7	0.4	0.4	0.4	0.3	0.3	0.4		
Washington	53	Clark	011	1.1	1.2	1.4	0.9	0.8	0.8	0.8		
Washington	53	King	033	1.6	1.5	1.5	1.2	1.3	1.0	1.0		
Washington	53	Pierce	053	0.7	1.0	0.8	0.7	0.8	0.7	0.7		
Washington	53	Spokane	063	1.7	1.6	1.5	1.5	1.3	1.1	1.1		
Wisconsin	55	Milwaukee	079	0.6	0.3	0.4	0.5	0.4	0.4	0.3		
Maxperyear				2.7	1.8	1.6	1.9	1.4	1.3	1.2	2.7	***Total Max***
Minperyear				0.3	0.3	0.3	0.3	0.3	0.3	0.3	0.3	***Total Min***

Appendix C

Relationships (Ratios) Between 1-hour and 8-hour CO Concentration Metrics For Counties with CO Monitors (2007-2009)

In calculating the different metrics analyzed here, different completeness requirements were followed for the current CO standard CO design values and for the metrics involving averaging over three years.

For the current CO standard running 8-hour and 1-hour metrics, there are no completeness requirements other than that each running 8-hour period is required to have 75% (or 6) of the 8 hours in the period. For the current standards, the 2nd maximum running 8-hour period and the 2nd maximum 1-hour value for the entire year, regardless of how incomplete the site data was, is compared to the standard.

With regard to data completeness for the alternative metrics, past EPA practice for other NAAQS pollutants was followed by requiring that in general at least 75% of the monitoring data that should have resulted from following the planned monitoring schedule in a period must be available for the key air quality statistic from that period to be considered valid. The alternative CO metrics presented here are the daily maximum 1-hour and 8-hour concentrations in three successive years. It is important that sampling within a day encompass the period when concentrations are likely to be highest and that all seasons of the year are well represented. Hence, in judging data completeness for derivation of these metrics, the 75% requirement is applied at the daily and quarterly levels. Also, because CO has been shown to have seasonal variability, 3 of the 4 quarters are required. And because it is a 3-year average, all 3 years are required. For the metrics derived here, the largest value is identified from two calculation procedures. Procedure 1 uses the proposed completeness requirements above and procedure 2 relaxes the daily requirement of having 75% of the hours in a day but still requires the quarterly and yearly completeness.

Because of the differing completeness requirements, when comparing the alternative daily maximum metrics to the current running 8-hour and 1-hour metrics, different sites are complete for different metrics. To avoid having ratios of 0 or a division by 0 error, sites that had a design value for the current 8-hour standard were only used if they had 3 years worth of design values on the site level comparison tables and the 2nd maximum 8-hour value for 2009 was used as the design value when comparing at the county level. After the alternative design values were calculated at each site, the county level metrics were created by taking the maximum value among all the sites in the county.

STATE	COUNTY	Annual 99th percentile daily maximum 1-hour concentration, averaged over 3 years (ppm) (2007-2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
UT	WEBER CO	12.2	2.9	4.21
WV	BROOKE CO	2.5	0.8	3.17
VA	NORFOLK	2.1	0.8	2.67
CO	DENVER CO	5.4	2.2	2.45
OH	HAMILTON CO	2.7	1.1	2.42
IN	LAKE CO	5.0	2.2	2.26
TN	BLOUNT CO	0.7	0.3	2.24
PR	SAN JUAN	5.3	2.5	2.12
AL	JEFFERSON CO	14.2	6.7	2.12
IA	POLK CO	1.9	0.9	2.08
NV	DOUGLAS CO	5.3	2.6	2.05
CA	SAN MATEO CO	3.5	1.7	2.04
WV	HANCOCK CO	2.2	1.1	2.03
AZ	PIMA CO	2.4	1.2	2.00
MT	GALLATIN CO	4.6	2.3	2.00
TX	CAMERON CO	1.6	0.8	1.96
TX	JEFFERSON CO	1.0	0.5	1.95
CT	HARTFORD CO	4.8	2.5	1.92
LA	EAST BATON ROUGE PAR	2.1	1.1	1.87
CA	ORANGE CO	4.5	2.4	1.86
TX	WEBB CO	3.0	1.6	1.85
CO	BOULDER CO	2.9	1.6	1.83
PA	WASHINGTON CO	1.3	0.7	1.81
TX	HARRIS CO	3.6	2	1.78
SC	CHARLESTON CO	0.5	0.3	1.78
NJ	CAMDEN CO	0.7	0.4	1.75
UT	SALT LAKE CO	3.9	2.3	1.68
CA	NAPA CO	2.3	1.4	1.67
CO	EL PASO CO	3.2	1.9	1.67
MO	ST LOUIS	2.8	1.7	1.67
MT	CASCADE CO	2.7	1.6	1.67
VT	RUTLAND CO	2.5	1.5	1.67
CA	MARIN CO	1.8	1.1	1.67
CO	LARIMER CO	3.0	1.8	1.67
CO	WELD CO	3.5	2.1	1.67
MO	GREENE CO	2.2	1.3	1.67
TX	BEXAR CO	3.0	1.8	1.67
ME	AROOSTOOK CO	0.8	0.5	1.65
CA	MONTEREY CO	1.5	0.9	1.63
IL	SANGAMON CO	1.9	1.2	1.61
TX	MC LENNAN CO	0.5	0.3	1.61
KS	SEDGWICK CO	2.7	1.7	1.61
NJ	MORRIS CO	1.6	1	1.60
FL	PINELLAS CO	1.9	1.2	1.58
CA	KERN CO	2.4	1.5	1.58
PA	ALLEGHENY CO	2.4	1.5	1.58
DE	NEW CASTLE CO	2.1	1.3	1.58
MT	YELLOWSTONE CO	2.8	1.8	1.57

STATE	COUNTY	Annual 99th percentile daily maximum 1-hour concentration, averaged over 3 years (ppm) (2007-2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
OH	LAKE CO	1.6	1	1.57
NY	SCHENECTADY CO	1.9	1.2	1.56
CA	SONOMA CO	1.8	1.2	1.53
WI	DODGE CO	0.5	0.3	1.51
TX	TRAVIS CO	0.6	0.4	1.50
NV	WASHOE CO	3.8	2.6	1.47
OR	LANE CO	2.3	1.6	1.46
OH	STARK CO	2.0	1.4	1.45
CA	RIVERSIDE CO	2.6	1.8	1.44
OH	FRANKLIN CO	2.2	1.5	1.44
PA	DAUPHIN CO	1.3	0.9	1.44
IN	MARION CO	3.6	2.5	1.43
CA	HUMBOLDT CO	1.6	1.1	1.42
GA	PAULDING CO	0.7	0.5	1.42
OR	JACKSON CO	3.4	2.4	1.42
OK	TULSA CO	1.8	1.3	1.41
CO	LA PLATA CO	1.3	0.9	1.41
PA	YORK CO	2.0	1.4	1.40
CA	STANISLAUS CO	2.8	2	1.40
TX	TARRANT CO	1.5	1.1	1.39
TN	DAVIDSON CO	2.4	1.7	1.39
MD	BALTIMORE CO	2.6	1.9	1.39
CA	SANTA BARBARA CO	1.8	1.3	1.38
SC	GREENVILLE CO	1.8	1.3	1.38
AZ	MARICOPA CO	4.6	3.3	1.38
CA	SAN JOAQUIN CO	2.8	2	1.38
TX	EL PASO CO	5.5	4	1.38
MO	ST LOUIS CO	1.0	0.7	1.38
CT	LITCHFIELD CO	1.0	0.7	1.38
CA	SAN DIEGO CO	4.1	3	1.38
KS	WYANDOTTE CO	2.2	1.6	1.38
NC	ROWAN CO	1.1	0.8	1.38
CO	ADAMS CO	2.6	1.9	1.37
FL	BROWARD CO	2.6	1.9	1.37
CA	FRESNO CO	2.7	2	1.37
TN	SULLIVAN CO	1.4	1	1.37
NY	ERIE CO	1.5	1.1	1.36
NY	ONONDAGA CO	1.6	1.2	1.36
NJ	MONMOUTH CO	1.9	1.4	1.36
MD	BALTIMORE	2.4	1.8	1.35
WA	SPOKANE CO	3.7	2.8	1.33
AR	PULASKI CO	2.0	1.5	1.33
CA	SAN BERNARDINO CO	2.1	1.6	1.33
IL	ST CLAIR CO	2.1	1.6	1.33
UT	UTAH CO	3.3	2.5	1.32
KY	JEFFERSON CO	2.8	2.1	1.32
CA	BUTTE CO	2.6	2	1.32
CA	IMPERIAL CO	7.2	5.5	1.32

STATE	COUNTY	Annual 99th percentile daily maximum 1-hour concentration, averaged over 3 years (ppm) (2007-2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
NV	CLARK CO	4.1	3.1	1.31
CA	CONTRA COSTA CO	1.4	1.1	1.30
FL	MIAMI-DADE CO	2.9	2.2	1.30
PA	ERIE CO	1.4	1.1	1.30
MT	FLATHEAD CO	3.4	2.6	1.29
NC	FORSYTH CO	2.2	1.7	1.29
NY	ALBANY CO	1.0	0.8	1.29
NJ	BURLINGTON CO	1.8	1.4	1.29
NY	SUFFOLK CO	1.7	1.3	1.28
NC	MECKLENBURG CO	2.2	1.7	1.27
CA	SANTA CLARA CO	2.9	2.3	1.27
OR	MULTNOMAH CO	2.9	2.3	1.27
MN	ST LOUIS CO	1.9	1.5	1.27
MA	SUFFOLK CO	1.5	1.2	1.26
CA	SOLANO CO	2.8	2.2	1.26
HI	HONOLULU CO	1.5	1.2	1.25
IL	PEORIA CO	2.5	2	1.25
NJ	MIDDLESEX CO	1.7	1.4	1.24
VA	ARLINGTON CO	1.6	1.3	1.23
GA	FULTON CO	1.8	1.5	1.22
MN	STEARNS CO	2.0	1.7	1.20
NY	BRONX CO	2.3	1.9	1.19
OH	CUYAHOGA CO	7.9	6.6	1.19
OH	MONTGOMERY CO	1.9	1.6	1.19
NJ	BERGEN CO	2.0	1.7	1.18
VA	ROANOKE	2.0	1.7	1.18
NH	HILLSBOROUGH CO	2.3	2	1.17
PA	LAWRENCE CO	1.2	1	1.17
NY	QUEENS CO	1.9	1.6	1.16
MN	RAMSEY CO	2.3	2	1.15
IA	SCOTT CO	1.0	0.9	1.15
NE	LANCASTER CO	3.2	2.8	1.14
NM	BERNALILLO CO	2.7	2.4	1.14
PA	PHILADELPHIA CO	2.3	2	1.13
RI	PROVIDENCE CO	1.5	1.3	1.13
FL	DUVAL CO	1.8	1.6	1.13
PA	CAMBRIA CO	1.6	1.4	1.12
VA	ALEXANDRIA	1.6	1.4	1.12
CA	SAN FRANCISCO CO	2.1	1.9	1.11
CA	SACRAMENTO CO	3.1	2.8	1.10
NY	NIAGARA CO	1.2	1.1	1.09
WY	TETON CO	0.9	0.8	1.08
WA	KING CO	2.8	2.6	1.08
MA	HAMPDEN CO	1.9	1.8	1.07
IL	COOK CO	2.5	2.3	1.07
IL	WINNEBAGO CO	2.0	1.9	1.07
PA	NORTHAMPTON CO	1.8	1.7	1.06
MN	HENNEPIN CO	2.0	1.9	1.05

STATE	COUNTY	**Annual 99th percentile daily maximum 1-hour concentration, averaged over 3 years** (ppm) (2007-2009)	**2nd highest, nonoverlapping, 8-hour average concentration in year** (ppm) (2009)	**ratio**
CO	MESA CO	2.3	2.2	1.05
MD	PRINCE GEORGES CO	0.9	0.9	1.04
TN	SHELBY CO	2.1	2	1.03
OH	SUMMIT CO	2.1	2.1	1.02
MA	WORCESTER CO	1.9	1.9	1.00
NY	MONROE CO	1.3	1.3	1.00
VT	CHITTENDEN CO	1.6	1.6	1.00
IN	ALLEN CO	2.5	2.5	0.99
PA	BUCKS CO	2.3	2.3	0.99
CA	ALAMEDA CO	1.9	1.9	0.98
MA	MIDDLESEX CO	1.6	1.6	0.98
FL	ORANGE CO	1.2	1.2	0.97
VA	RICHMOND	1.4	1.5	0.93
ME	HANCOCK CO	0.3	0.3	0.92
DC	WASHINGTON	3.4	3.8	0.89
FL	HILLSBOROUGH CO	0.8	1	0.81
CA	LOS ANGELES CO	3.3	4.5	0.73
CA	SANTA CRUZ CO	2.6	3.8	0.69
MN	DAKOTA CO	0.9	1.5	0.61
CT	FAIRFIELD CO	1.0	1.8	0.56
NJ	HUDSON CO	2.3	8.2	0.28
min		0.28	0.30	0.28
p5		0.81	0.50	0.94
p10		1.02	0.80	1.01
p25		1.57	1.20	1.17
p50		2.13	1.60	1.37
mean		2.44	1.77	1.42
p75		2.77	2.00	1.61
p90		3.79	2.60	1.94
p95		4.93	3.26	2.11
max		14.20	8.20	4.21

STAT	COUNTY	2nd highest 1-hour CO concentration in year (ppm) (2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
UT	WEBER CO	16.2	2.9	5.59
MT	FLATHEAD CO	12.8	2.6	4.92
PR	SAN JUAN	9.4	2.5	3.76
IN	LAKE CO	7.5	2.2	3.41
CO	DENVER CO	6.8	2.2	3.09
WI	DODGE CO	0.921	0.3	3.07
MD	BALTIMORE CO	5.8	1.9	3.05
NJ	MONMOUTH CO	4.2	1.4	3.00
NV	DOUGLAS CO	7.6	2.6	2.92
ID	ADA CO	9.5	3.3	2.88
MO	ST LOUIS	4.7	1.7	2.76
OH	SUMMIT CO	5.8	2.1	2.76
MT	GALLATIN CO	6.2	2.3	2.70
PA	BERKS CO	3.2	1.2	2.67
OH	CUYAHOGA CO	17.3	6.6	2.62
CA	SANTA BARBARA CO	3.4	1.3	2.62
PA	ADAMS CO	1	0.4	2.50
OK	TULSA CO	3.2	1.3	2.46
MT	YELLOWSTONE CO	4.3	1.8	2.39
CA	SANTA CRUZ CO	9	3.8	2.37
OK	CHEROKEE CO	1.4	0.6	2.33
WY	TETON CO	1.8	0.8	2.25
PA	LACKAWANNA CO	1.7	0.8	2.13
TX	CAMERON CO	1.7	0.8	2.13
RI	PROVIDENCE CO	2.7	1.3	2.08
VT	RUTLAND CO	3.1	1.5	2.07
ND	CASS CO	1.003	0.5	2.01
CA	SAN FRANCISCO CO	3.8	1.9	2.00
CO	BOULDER CO	3.2	1.6	2.00
IN	HENDRICKS CO	1.4	0.7	2.00
NJ	CAMDEN CO	0.8	0.4	2.00
NJ	MORRIS CO	2	1	2.00
OH	MEDINA CO	1.6	0.8	2.00
SC	CHARLESTON CO	0.6	0.3	2.00
TX	MC LENNAN CO	0.6	0.3	2.00
MA	SUFFOLK CO	2.366	1.2	1.97
CA	SAN MATEO CO	3.3	1.7	1.94
CA	IMPERIAL CO	10.5	5.5	1.91
CO	EL PASO CO	3.6	1.9	1.89
AZ	PIMA CO	2.2	1.2	1.83
MN	STEARNS CO	3.1	1.7	1.82
MT	CASCADE CO	2.9	1.6	1.81
TX	JEFFERSON CO	0.906	0.5	1.81
LA	EAST BATON ROUGE PAR	1.99	1.1	1.81
AL	JEFFERSON CO	12.1	6.7	1.81
CT	HARTFORD CO	4.5	2.5	1.80
GA	FULTON CO	2.7	1.5	1.80
OR	MULTNOMAH CO	4.1	2.3	1.78

STAT	COUNTY	2nd highest 1-hour CO concentration in year (ppm) (2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
OK	ADAIR CO	0.702	0.4	1.76
IA	POLK CO	1.576	0.9	1.75
CA	ORANGE CO	4.2	2.4	1.75
FL	PINELLAS CO	2.1	1.2	1.75
TX	TRAVIS CO	0.7	0.4	1.75
VA	NEWPORT NEWS	1.4	0.8	1.75
CA	ALAMEDA CO	3.3	1.9	1.74
CA	MARIN CO	1.9	1.1	1.73
OH	HAMILTON CO	1.9	1.1	1.73
NY	QUEENS CO	2.76	1.6	1.73
TX	BEXAR CO	3.1	1.8	1.72
PA	WASHINGTON CO	1.2	0.7	1.71
PA	YORK CO	2.4	1.4	1.71
CO	WELD CO	3.6	2.1	1.71
MO	GREENE CO	2.2	1.3	1.69
TX	WEBB CO	2.7	1.6	1.69
CA	SONOMA CO	2	1.2	1.67
IL	SANGAMON CO	2	1.2	1.67
OK	OKLAHOMA CO	2	1.2	1.67
CA	MONTEREY CO	1.5	0.9	1.67
CO	LARIMER CO	3	1.8	1.67
PA	PHILADELPHIA CO	3.3	2	1.65
KS	SEDGWICK CO	2.8	1.7	1.65
CA	NAPA CO	2.3	1.4	1.64
NJ	MIDDLESEX CO	2.3	1.4	1.64
PA	CAMBRIA CO	2.3	1.4	1.64
CA	CONTRA COSTA CO	1.8	1.1	1.64
CA	HUMBOLDT CO	1.8	1.1	1.64
PA	ERIE CO	1.8	1.1	1.64
WV	HANCOCK CO	1.8	1.1	1.64
NC	ROWAN CO	1.3	0.8	1.63
VA	HAMPTON	1.3	0.8	1.63
VA	NORFOLK	1.3	0.8	1.63
ME	AROOSTOOK CO	0.808	0.5	1.62
DE	NEW CASTLE CO	2.1	1.3	1.62
NV	WASHOE CO	4.2	2.6	1.62
NY	SUFFOLK CO	2.1	1.3	1.62
UT	SALT LAKE CO	3.7	2.3	1.61
WA	SPOKANE CO	4.5	2.8	1.61
IL	PEORIA CO	3.2	2	1.60
IN	MARION CO	4	2.5	1.60
NH	HILLSBOROUGH CO	3.2	2	1.60
TN	SULLIVAN CO	1.6	1	1.60
MN	ST LOUIS CO	2.4	1.5	1.60
TX	DALLAS CO	2.377	1.5	1.58
FL	ORANGE CO	1.9	1.2	1.58
MI	WAYNE CO	1.9	1.2	1.58
NY	ONONDAGA CO	1.9	1.2	1.58

STAT	COUNTY	2nd highest 1-hour CO concentration in year (ppm) (2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
NJ	BURLINGTON CO	2.2	1.4	1.57
IA	LINN CO	1.882	1.2	1.57
UT	UTAH CO	3.9	2.5	1.56
CO	LA PLATA CO	1.4	0.9	1.56
MD	BALTIMORE	2.8	1.8	1.56
TX	EL PASO CO	6.2	4	1.55
TX	HARRIS CO	3.1	2	1.55
WA	KING CO	4	2.6	1.54
PA	ALLEGHENY CO	2.3	1.5	1.53
NV	CLARK CO	4.7	3.1	1.52
AZ	MARICOPA CO	5	3.3	1.52
CT	FAIRFIELD CO	2.7	1.8	1.50
NM	BERNALILLO CO	3.6	2.4	1.50
FL	BROWARD CO	2.8	1.9	1.47
NY	BRONX CO	2.8	1.9	1.47
CA	KERN CO	2.2	1.5	1.47
OH	FRANKLIN CO	2.2	1.5	1.47
TX	TARRANT CO	1.6	1.1	1.45
AK	FAIRBANKS NORTH STAR BOROUGH	4.5	3.1	1.45
CA	FRESNO CO	2.9	2	1.45
CA	SAN JOAQUIN CO	2.9	2	1.45
CA	RIVERSIDE CO	2.6	1.8	1.44
CA	SAN BERNARDINO CO	2.3	1.6	1.44
KS	WYANDOTTE CO	2.3	1.6	1.44
NC	WAKE CO	2.3	1.6	1.44
OH	MONTGOMERY CO	2.3	1.6	1.44
CA	SAN DIEGO CO	4.3	3	1.43
NJ	UNION CO	3	2.1	1.43
IL	WINNEBAGO CO	2.7	1.9	1.42
NY	SCHENECTADY CO	1.7	1.2	1.42
OR	JACKSON CO	3.4	2.4	1.42
IA	SCOTT CO	1.271	0.9	1.41
VA	ROANOKE	2.4	1.7	1.41
ME	CUMBERLAND CO	2.1	1.5	1.40
VA	RICHMOND	2.1	1.5	1.40
CA	BUTTE CO	2.8	2	1.40
NE	LANCASTER CO	3.9	2.8	1.39
VT	CHITTENDEN CO	2.2	1.6	1.38
CO	ADAMS CO	2.6	1.9	1.37
NY	ERIE CO	1.5	1.1	1.36
NC	FORSYTH CO	2.3	1.7	1.35
CA	STANISLAUS CO	2.7	2	1.35
CT	NEW HAVEN CO	1.89	1.4	1.35
TN	SHELBY CO	2.7	2	1.35
IL	COOK CO	3.1	2.3	1.35
HI	HONOLULU CO	1.6	1.2	1.33
NC	DURHAM CO	0.8	0.6	1.33
PR	BAYAMON	3.2	2.4	1.33

STAT	COUNTY	2nd highest 1-hour CO concentration in year (ppm) (2009)	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	ratio
IN	VANDERBURGH CO	2.4	1.8	1.33
KY	JEFFERSON CO	2.8	2.1	1.33
MA	ESSEX CO	0.791	0.6	1.32
IL	ST CLAIR CO	2.1	1.6	1.31
MI	KENT CO	2.1	1.6	1.31
OR	LANE CO	2.1	1.6	1.31
AK	ANCHORAGE BOROUGH	7.6	5.8	1.31
VA	ARLINGTON CO	1.7	1.3	1.31
VA	FAIRFAX CO	1.7	1.3	1.31
NJ	ESSEX CO	2.6	2	1.30
CA	SANTA CLARA CO	2.98	2.3	1.30
PA	NORTHAMPTON CO	2.2	1.7	1.29
TN	DAVIDSON CO	2.2	1.7	1.29
MD	GARRETT CO	0.387	0.3	1.29
MO	ST LOUIS CO	0.9	0.7	1.29
OH	STARK CO	1.8	1.4	1.29
NY	NEW YORK CO	2.3	1.8	1.28
CA	SOLANO CO	2.8	2.2	1.27
NY	NIAGARA CO	1.4	1.1	1.27
CT	LITCHFIELD CO	0.89	0.7	1.27
AR	PULASKI CO	1.9	1.5	1.27
MA	WORCESTER CO	2.4	1.9	1.26
MN	HENNEPIN CO	2.4	1.9	1.26
NE	DOUGLAS CO	2.9	2.3	1.26
FL	DUVAL CO	2	1.6	1.25
MN	RAMSEY CO	2.5	2	1.25
NY	ALBANY CO	1	0.8	1.25
WV	BROOKE CO	1	0.8	1.25
MD	PRINCE GEORGES CO	1.123	0.9	1.25
MT	MISSOULA CO	3.1	2.5	1.24
NC	MECKLENBURG CO	2.1	1.7	1.24
NJ	BERGEN CO	2.1	1.7	1.24
NJ	HUDSON CO	10.1	8.2	1.23
SC	GREENVILLE CO	1.6	1.3	1.23
FL	MIAMI-DADE CO	2.7	2.2	1.23
CA	LOS ANGELES CO	5.5	4.5	1.22
MA	HAMPDEN CO	2.2	1.8	1.22
PA	DAUPHIN CO	1.1	0.9	1.22
VA	ALEXANDRIA	1.7	1.4	1.21
IN	ALLEN CO	3	2.5	1.20
OH	LAKE CO	1.2	1	1.20
PA	LAWRENCE CO	1.2	1	1.20
CA	SACRAMENTO CO	3.2	2.8	1.14
TN	BLOUNT CO	0.34	0.3	1.13
MA	MIDDLESEX CO	1.8	1.6	1.13
DC	WASHINGTON	4.2	3.8	1.11
OH	ATHENS CO	1.3	1.2	1.08
FL	HILLSBOROUGH CO	1.082	1	1.08

STAT	COUNTY	**2nd highest 1-hour CO concentration in year** (ppm) (2009)	**2nd highest, nonoverlapping, 8-hour average concentration in year** (ppm) (2009)	**ratio**
NY	MONROE CO	1.4	1.3	1.08
CO	MESA CO	2.3	2.2	1.05
PA	BUCKS CO	2.4	2.3	1.04
WA	CLALLAM CO	0.311	0.3	1.04
GA	PAULDING CO	0.502	0.5	1.00
MN	DAKOTA CO	1.5	1.5	1.00
NY	STEUBEN CO	0.29	0.3	0.97
ME	HANCOCK CO	0.288	0.3	0.96
KY	EDMONSON CO	0.25	0.3	0.83
OK	KAY CO	0.25	0.3	0.83
SD	UNION CO	0.25	0.3	0.83
WY	UINTA CO	0.25	0.3	0.83
min		0.25	0.30	0.83
p5		0.60	0.30	1.05
p10		0.91	0.60	1.20
p25		1.60	1.10	1.31
p50		2.30	1.55	1.55
mean		2.86	1.68	1.65
p75		3.20	2.00	1.75
p90		4.64	2.60	2.31
p95		7.59	3.27	2.76
max		17.30	8.20	5.59

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
NJ	HUDSON CO	8.2	1.7	4.92
CA	SANTA CRUZ CO	3.8	1.4	2.65
WY	TETON CO	0.8	0.4	2.18
MN	HENNEPIN CO	1.9	1.1	1.78
CA	LOS ANGELES CO	4.5	2.5	1.78
MN	DAKOTA CO	1.5	0.9	1.67
OH	SUMMIT CO	2.1	1.3	1.58
OH	CUYAHOGA CO	6.6	4.3	1.55
FL	HILLSBOROUGH CO	1	0.7	1.50
PA	BUCKS CO	2.3	1.6	1.47
CO	MESA CO	2.2	1.5	1.47
ID	ADA CO	3.3	2.3	1.41
MI	KENT CO	1.6	1.1	1.41
VA	RICHMOND	1.5	1.1	1.41
MA	WORCESTER CO	1.9	1.4	1.36
VT	CHITTENDEN CO	1.6	1.2	1.33
FL	ORANGE CO	1.2	0.9	1.33
PA	BERKS CO	1.2	0.9	1.33
TN	SHELBY CO	2	1.5	1.30
MT	FLATHEAD CO	2.6	2.0	1.30
NY	SUFFOLK CO	1.3	1.0	1.30
RI	PROVIDENCE CO	1.3	1.0	1.30
DC	WASHINGTON	3.8	2.9	1.30
IA	SCOTT CO	0.9	0.7	1.29
IN	VANDERBURGH CO	1.8	1.4	1.29
HI	HONOLULU CO	1.2	0.9	1.29
ME	HANCOCK CO	0.3	0.2	1.29
OK	CHEROKEE CO	0.6	0.5	1.29
NJ	MIDDLESEX CO	1.4	1.1	1.27
PA	YORK CO	1.4	1.1	1.27
UT	UTAH CO	2.5	2.0	1.27
NY	NIAGARA CO	1.1	0.9	1.27
CA	SAN FRANCISCO CO	1.9	1.5	1.27
FL	DUVAL CO	1.6	1.3	1.26
MA	MIDDLESEX CO	1.6	1.3	1.26
IN	ALLEN CO	2.5	2.0	1.25
IL	WINNEBAGO CO	1.9	1.5	1.24
IL	COOK CO	2.3	1.9	1.23
NY	QUEENS CO	1.6	1.3	1.23
FL	MIAMI-DADE CO	2.2	1.8	1.22
GA	FULTON CO	1.5	1.2	1.22
MN	STEARNS CO	1.7	1.4	1.21

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
MA	HAMPDEN CO	1.8	1.5	1.20
PA	PHILADELPHIA CO	2	1.7	1.20
VA	ROANOKE	1.7	1.4	1.19
MN	ST LOUIS CO	1.5	1.3	1.18
NE	LANCASTER CO	2.8	2.4	1.18
MD	PRINCE GEORGES CO	0.9	0.8	1.17
TX	EL PASO CO	4	3.4	1.17
ME	AROOSTOOK CO	0.5	0.4	1.15
PA	LAWRENCE CO	1	0.9	1.15
WA	SPOKANE CO	2.8	2.4	1.15
NY	MONROE CO	1.3	1.1	1.15
NC	ROWAN CO	0.8	0.7	1.14
NM	BERNALILLO CO	2.4	2.1	1.14
CA	ALAMEDA CO	1.9	1.7	1.14
NJ	BURLINGTON CO	1.4	1.2	1.14
NH	HILLSBOROUGH CO	2	1.8	1.13
CA	SACRAMENTO CO	2.8	2.5	1.12
OH	MONTGOMERY CO	1.6	1.4	1.12
VA	ARLINGTON CO	1.3	1.2	1.11
PA	NORTHAMPTON CO	1.7	1.5	1.11
VA	ALEXANDRIA	1.4	1.3	1.11
NY	ERIE CO	1.1	1.0	1.10
PA	ERIE CO	1.1	1.0	1.10
WA	KING CO	2.6	2.4	1.10
AR	PULASKI CO	1.5	1.4	1.10
TX	DALLAS CO	1.5	1.4	1.10
NY	BRONX CO	1.9	1.7	1.10
CA	IMPERIAL CO	5.5	5.0	1.09
FL	PINELLAS CO	1.2	1.1	1.09
MA	SUFFOLK CO	1.2	1.1	1.09
PA	ADAMS CO	0.4	0.4	1.09
TX	TRAVIS CO	0.4	0.4	1.09
NJ	UNION CO	2.1	1.9	1.09
NJ	BERGEN CO	1.7	1.6	1.09
CA	MONTEREY CO	0.9	0.8	1.08
TX	BEXAR CO	1.8	1.7	1.08
NE	DOUGLAS CO	2.3	2.1	1.08
PA	CAMBRIA CO	1.4	1.3	1.08
AZ	MARICOPA CO	3.3	3.1	1.08
MD	BALTIMORE CO	1.9	1.8	1.08
PA	ALLEGHENY CO	1.5	1.4	1.07
MT	CASCADE CO	1.6	1.5	1.07

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
CA	CONTRA COSTA CO	1.1	1.0	1.06
TX	TARRANT CO	1.1	1.0	1.06
NC	MECKLENBURG CO	1.7	1.6	1.06
MT	GALLATIN CO	2.3	2.2	1.06
CA	SANTA BARBARA CO	1.3	1.2	1.05
CA	SAN JOAQUIN CO	2	1.9	1.05
MN	RAMSEY CO	2	1.9	1.05
CT	NEW HAVEN CO	1.4	1.3	1.05
AK	ANCHORAGE BOROUGH	5.8	5.5	1.05
CA	SANTA CLARA CO	2.3	2.2	1.05
IL	ST CLAIR CO	1.6	1.5	1.04
CT	FAIRFIELD CO	1.8	1.7	1.04
CA	BUTTE CO	2	1.9	1.03
CA	SOLANO CO	2.2	2.1	1.03
MI	WAYNE CO	1.2	1.2	1.03
NJ	MONMOUTH CO	1.4	1.4	1.02
CA	SAN BERNARDINO CO	1.6	1.6	1.02
CA	RIVERSIDE CO	1.8	1.8	1.02
CO	EL PASO CO	1.9	1.9	1.02
CA	FRESNO CO	2	2.0	1.02
IL	PEORIA CO	2	2.0	1.02
KY	JEFFERSON CO	2.1	2.1	1.02
CA	ORANGE CO	2.4	2.4	1.01
IN	MARION CO	2.5	2.5	1.01
CO	WELD CO	2.1	2.1	1.00
FL	BROWARD CO	1.9	1.9	1.00
MD	GARRETT CO	0.3	0.3	1.00
MT	YELLOWSTONE CO	1.8	1.8	1.00
NV	DOUGLAS CO	2.6	2.6	1.00
NV	WASHOE CO	2.6	2.6	1.00
NY	ONONDAGA CO	1.2	1.2	1.00
OH	FRANKLIN CO	1.5	1.5	1.00
OK	KAY CO	0.3	0.3	1.00
SC	CHARLESTON CO	0.3	0.3	1.00
SC	GREENVILLE CO	1.3	1.3	1.00
TX	CAMERON CO	0.8	0.8	1.00
UT	SALT LAKE CO	2.3	2.3	1.00
VT	RUTLAND CO	1.5	1.5	1.00
CA	SAN DIEGO CO	3	3.0	0.99
OR	JACKSON CO	2.4	2.4	0.99
CA	STANISLAUS CO	2	2.0	0.98
CO	ADAMS CO	1.9	1.9	0.98

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
MD	BALTIMORE	1.8	1.8	0.98
NC	FORSYTH CO	1.7	1.7	0.98
KS	WYANDOTTE CO	1.6	1.6	0.98
DE	NEW CASTLE CO	1.3	1.3	0.98
IL	SANGAMON CO	1.2	1.2	0.97
OR	MULTNOMAH CO	2.3	2.4	0.97
PA	DAUPHIN CO	0.9	0.9	0.96
NY	ALBANY CO	0.8	0.8	0.96
MO	ST LOUIS CO	0.7	0.7	0.95
PR	SAN JUAN	2.5	2.6	0.95
AK	FAIRBANKS NORTH STAR BOROUGH	3.1	3.3	0.95
CA	SONOMA CO	1.2	1.3	0.95
CA	SAN MATEO CO	1.7	1.8	0.94
TN	DAVIDSON CO	1.7	1.8	0.94
CA	HUMBOLDT CO	1.1	1.2	0.94
CA	MARIN CO	1.1	1.2	0.94
NV	CLARK CO	3.1	3.3	0.94
CA	KERN CO	1.5	1.6	0.94
NJ	MORRIS CO	1	1.1	0.94
TN	SULLIVAN CO	1	1.1	0.94
VA	FAIRFAX CO	1.3	1.4	0.93
AL	JEFFERSON CO	6.7	7.2	0.93
NJ	CAMDEN CO	0.4	0.4	0.92
OR	LANE CO	1.6	1.7	0.92
IN	LAKE CO	2.2	2.4	0.92
CT	HARTFORD CO	2.5	2.7	0.91
CT	LITCHFIELD CO	0.7	0.8	0.91
CO	LA PLATA CO	0.9	1.0	0.90
CA	NAPA CO	1.4	1.6	0.89
MT	MISSOULA CO	2.5	2.8	0.89
MO	GREENE CO	1.3	1.5	0.89
OK	TULSA CO	1.3	1.5	0.89
TX	WEBB CO	1.6	1.8	0.87
MO	ST LOUIS	1.7	2.0	0.86
CO	BOULDER CO	1.6	1.9	0.86
NC	DURHAM CO	0.6	0.7	0.86
IA	POLK CO	0.9	1.1	0.84
CO	DENVER CO	2.2	2.6	0.84
TX	JEFFERSON CO	0.5	0.6	0.83
PA	LACKAWANNA CO	0.8	1.0	0.83
VA	NORFOLK	0.8	1.0	0.83
OH	STARK CO	1.4	1.7	0.82

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
KS	SEDGWICK CO	1.7	2.1	0.82
TX	MC LENNAN CO	0.3	0.4	0.82
NY	SCHENECTADY CO	1.2	1.5	0.82
WI	DODGE CO	0.3	0.4	0.82
TX	HARRIS CO	2	2.5	0.81
NC	WAKE CO	1.6	2.0	0.80
AZ	PIMA CO	1.2	1.5	0.80
CO	LARIMER CO	1.8	2.3	0.79
GA	PAULDING CO	0.5	0.6	0.79
VA	HAMPTON	0.8	1.0	0.77
LA	EAST BATON ROUGE PAR	1.1	1.4	0.77
WV	HANCOCK CO	1.1	1.5	0.75
IN	HENDRICKS CO	0.7	0.9	0.75
OH	LAKE CO	1	1.3	0.75
PA	WASHINGTON CO	0.7	1.0	0.70
OH	HAMILTON CO	1.1	1.7	0.63
UT	WEBER CO	2.9	4.6	0.63
TN	BLOUNT CO	0.3	0.5	0.60
WV	BROOKE CO	0.8	1.6	0.50

STATE	COUNTY	2nd highest, nonoverlapping, 8-hour average concentration in year (ppm) (2009)	Annual 99th percentile daily maximum 8-hour average concentration, averaged over 3 years (ppm) (2007-2009)	ratio
min		0.30	0.23	0.50
p5		0.43	0.43	0.78
p10		0.76	0.72	0.82
p25		1.10	1.07	0.94
p50		1.60	1.50	1.05
mean		1.75	1.61	1.10
p75		2.05	1.95	1.18
p90		2.60	2.48	1.30
p95		3.30	3.00	1.47
max		8.20	7.23	4.92

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Appendix D

Additional REA Estimates from Simulations for Alternative Levels and Forms for the 1-hour and 8-hour Standards.

Table D-1. Percentage of simulated HD population with daily maximum end-of-hour COHb levels (absolute) below the indicated COHb levels under alternative levels and forms for the 1-hour and 8-hour standards.

Current and Potential Alternative Standards			Daily Maximum End-of-hour COHb Level (Absolute)										
Form	Level		< 3.0 % COHb		< 2.5 % COHb		< 2.0 % COHb		< 1.75 % COHb		< 1.5 % COHb		*8-hour Levels (ppm)*
	1-hour (ppm)	8-hour (ppm)	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	
Second Highest Non-overlapping Concentration (Current Form)	16.2+	9.4+		99.7		99.1		95.5		88.9		75.5	*9.4+*
	11.8+		>99.9		>99.9		99.4		98.3		95.0		
	11.2	5.4–6.5		>99.9		99.8		98.9		96.8		90.4	*6.5*
	9.7					99.9		99.4		98.0		93.9	*5.6*
	9.3									98.4		94.6	*5.4*
	8.2*		100		100		> 99.9		99.5		98.4		*5.6**
	8.1										98.5		*6.5*
	7.2								99.7		99.0		*5.7*
	6.8								99.8		99.2		*5.4*
	4.6*	3.1*		100		100		>99.9		99.7		98.8	*3.1**
99th Percentile of Daily Maximum Concentrations	13.3+	7.2-8.2+		99.7		99.1		95.5		88.9		75.5	*7.2+*
	11.6+		>99.9		>99.9		99.4		98.3		95.0		*8.2+*
	9.2	4.1– 5.7		>99.9		99.8		98.9		96.8		90.4	*5.0*
	8.0		100		100		> 99.9		99.5		98.5		*5.7*
				>99.9		99.9		99.4		98.0		93.9	*4.3*
	7.7									98.4		94.6	*4.1*
	7.4*		100		100		>99.9		99.5		98.4		*5.1**
	7.1								99.7		99.0		*5.0*
	6.7								99.8		99.2		*4.7*
	4.5*	2.8*		100		100		>99.9		99.7		98.8	*2.8**

+ Plus marks indicate simulations based on air quality conditions just meeting the current 8-hour standard.
* Asterisks indicate simulations based on "as is" (2006) air quality conditions for the two study areas.
Drawn from REA tables 6-15 to 6-19 and 6-21 to 6-22, consistent with Table 2-7above.

Table D-2. Percentage of simulated CHD population with maximum ambient contributions to end-of-hour COHb levels below the indicated COHb levels under alternative levels and forms for the 1-hour and 8-hour standards.

Current and Potential Alternative Standards			Maximum Ambient Contribution to End-of-hour COHb Level								8-hour Level (ppm)
Form	Level		< 2.0 % COHb		< 1.8 % COHb		< 1.6 % COHb		< 1.4 % COHb		
	1-hour (ppm)	8-hour (ppm)	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	Los Angeles	Denver	
Second Highest Non-overlapping Concentration (Current Form)	16.2+	9.4		97.3		96.6		93.2		87.2	*9.4*
	11.8+		99.5		99.2		98.7		98.0		
	11.2	5.6-6.5		99.8		99.1		97.3		96.3	*6.5*
	8.2*		100*		99.7*		99.5*		99.5*		*5.6*
	7.2		99.7		99.7		99.7		99.5		*5.7*
	4.6*	3.1		100*		100*		99.8*		99.8*	*3.1*
99th Percentile of Daily Maximum Concentrations	13.3+	7.2		97.3		96.6		93.2		87.2	*7.2*
	11.6+	8.2	99.5		99.2		98.7		98.0		*8.2*
	9.2	5.0-5.1		99.8		99.1		97.3		96.3	*5.0*
	7.4*		100*		99.7*		99.5*		99.5*		*5.1*
	7.1		99.7		99.7		99.7		99.5		*5.0*
	4.5*	2.8		100*		100*		99.8*		99.8*	*2.8*

+ Plus marks indicate simulations based on air quality conditions just meeting the current 8-hour standard.
* Asterisks indicate simulations based on "as is" (2006) air quality conditions for the two study areas.
Drawn from REA tables 6-17, 6-20 and 6-23, consistent withTable 2-7 in chapter 2 of this document above.

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Appendix E

Predicted Percentage of Counties with a Monitor Not Likely to Meet Alternative Standards and Associated Percentage Population

Data Analysis

Hourly data for CO were pulled from EPA's Air Quality System in July 2010. Daily 1-hour maximums were calculated from the hourly data. Past EPA practice for other NAAQS pollutants was followed by requiring that in general at least 75% of the monitoring data that should have resulted from following the planned monitoring schedule in a period must be available for the key air quality statistic from that period to be considered valid. The key air quality statistics are the daily maximum 1-hour and 8-hour concentrations in three successive years. The 75% requirement was applied at the daily and quarterly levels. Also, because CO has been shown to have seasonal variability, 3 of the 4 quarters were required. And because it is a 3-year average, all 3 years were required. For the alternative design values, the largest value from two calculation procedures is what was compared to the potential alternative standards. Procedure 1 uses the completeness requirements above and procedure 2 relaxes the daily requirement of having 75% of the hours in a day but still requires the quarterly and yearly completeness. After the alternative design values in were calculated at each site, the county level design values were created by taking the maximum design value of all the sites in the county. The regions are the same as those used for similar analyses in recent NAAQS reviews (e.g., lead, particulate matter).

Appendix E. Predicted percentage of counties with a monitor not likely to meet alternative atandards (based on 2007-2009 data) and associated percentage population.

	Total Counties (population in millions)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern California	Outside Regions
Number of Counties with monitors ➔	165 (115)	41	26	30	10	9	38	9	2
Alternative Levels for a form of 99th percentile daily 1-hour maximum, averaged over 3 years ⬇	**Of counties with a monitor, the percentage not likely to meet stated alternative standard (population - percentage of population in counties with monitors)**								
15.0	0	0	0	0	0	0	0	0	0
14.0	.6 (.7)	0	4	0	0	0	0	0	0
13.0	.6 (.7)	0	4	0	0	0	0	0	0
12.0	1.2 (.9)	0	4	0	0	0	3	0	0
11.0	1.2 (.9)	0	4	0	0	0	3	0	0
10.0	1.2 (.9)	0	4	0	0	0	3	0	0
9.0	1.2 (.9)	0	4	0	0	0	3	0	0
8.0	1.2 (.9)	0	4	0	0	0	3	0	0
7.0	2.4 (2.4)	0	4	3	0	11	3	0	0
6.0	2.4 (2.4)	0	4	3	0	11	3	0	0
5.0	4.8 (4.1)	0	4	3	0	22	8	0	50
Number of Counties with monitors ➔	187 (125)	48	31	35	11	9	40	9	4
Alternative Levels for a form of 99th percentile daily 8-hour maximum, averaged over 3 years ⬇	**Of counties with a monitor, the percentage not likely to meet stated alternative standard (population - percentage of population in counties with monitors)**								
9.0	0	0	0	0	0	0	0	0	0
8.0	0	0	0	0	0	0	0	0	0
7.0	0.5 (.7)	0	3	0	0	0	0	0	0
6.0	0.5 (.7)	0	3	0	0	0	0	0	0
5.0	1.1 (.9)	0	3	0	0	0	0	0	25
4.0	2.7 (2.7)	0	3	3	0	11	3	0	25
3.0	4.8 (7.9)	0	3	3	0	44	3	0	50

** "Outside Regions" includes Alaska, Hawaii, Puerto Rico, and the Virgin Islands.

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