



Risk and Exposure Assessment to Support the Review of the NO₂ Primary National Ambient Air Quality Standard: First Draft

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Risk and Exposure Assessment to Support the Review of the NO₂ Primary National Ambient Air Quality Standard: First Draft

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Disclaimer

This draft document has been prepared by staff from the Ambient Standards Group, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. Any opinions, findings, conclusions, or recommendations are those of the authors and do not necessarily reflect the views of the EPA. This document is being circulated to obtain review and comment from the Clean Air Scientific Advisory Committee (CASAC) and the general public. Comments on this draft document should be addressed to Scott Jenkins, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C504-06, Research Triangle Park, North Carolina 27711 (email: Jenkins.scott@epa.gov).

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1. INTRODUCTION

1.1 OVERVIEW

The U.S. Environmental Protection Agency (EPA) is conducting a review of the national ambient air quality standards (NAAQS) for nitrogen dioxide (NO₂). Sections 108 and 109 of the Clean Air Act (The Act) govern the establishment and periodic review of the air quality criteria and the NAAQS. These standards are established for pollutants that 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. The NAAQS are based on air quality criteria, which reflect the latest scientific knowledge useful in indicating the kind and extent of identifiable effects on public health or welfare that may be expected from the presence of the pollutant in ambient air. The EPA Administrator promulgates and periodically reviews primary (health-based) and secondary (welfare-based) NAAQS for such pollutants. Based on periodic reviews of the air quality criteria and standards, the Administrator makes revisions in the criteria and standards and promulgates any new standards as may be appropriate. The Act also requires that an independent scientific review committee advise the Administrator as part of this NAAQS review process, a function now performed by the Clean Air Scientific Advisory Committee (CASAC).

The Agency has recently made a number of changes to the process for reviewing the NAAQS (described at <http://www.epa.gov/ttn/naaqs/>). In making these changes, the Agency consulted with CASAC. This new process, which is being applied to the current review of the NO₂ NAAQS, contains four major components. Each of these components, as they relate to the review of the NO₂ primary NAAQS, is described below.

The first of these components is an integrated review plan. This plan presents the schedule for the review, the process for conducting the review, and the key policy-relevant science issues that guide the review. The integrated review plan for this review of the NO₂ primary NAAQS is presented in the *Integrated Review Plan for the Primary National Ambient*

1 *Air Quality Standard for Nitrogen Dioxide* (EPA, 2007a). The policy-relevant questions
2 identified in this document to guide the review are:

- 3 • Has new information altered the scientific support for the occurrence of health effects
4 following short- and/or long-term exposure to levels of NO_x found in the ambient air?
- 5 • What do recent studies focused on the near-roadway environment tell us about health
6 effects of NO_x?
- 7 • At what levels of NO_x exposure do health effects of concern occur?
- 8 • Has new information altered conclusions from previous reviews regarding the plausibility
9 of adverse health effects caused by NO_x exposure?
- 10 • To what extent have important uncertainties identified in the last review been reduced
11 and/or have new uncertainties emerged?
- 12 • What are the air quality relationships between short-term and long-term exposures
13 to NO_x?

14 Additional questions will become relevant if the evidence suggests that revision of the current
15 standard might be appropriate. These questions are:

- 16 • Is there evidence for the occurrence of adverse health effects at levels of NO_x lower than
17 those observed previously? If so, at what levels and what are the important uncertainties
18 associated with that evidence?
- 19 • Do exposure estimates suggest that exposures of concern for NO_x-induced health effects
20 will occur with current ambient levels of NO₂ or with levels that just meet current, or
21 potential alternative, standards? If so, are these exposures of sufficient magnitude such
22 that the health effects might reasonably be judged to be important from a public health
23 perspective? What are the important uncertainties associated with these exposure
24 estimates?
- 25 • Do the evidence, the air quality assessment, and the risk/exposure assessment provide
26 support for considering different standard indicators or averaging times?
- 27 • What range of levels is supported by the evidence, the air quality assessment, and the
28 risk/exposure assessments? What are the uncertainties and limitations in the evidence
29 and the assessments?

- 1 • What is the range of forms supported by the evidence, the air quality assessment, and the
2 exposure/risk assessments? What are the uncertainties and limitations in the evidence
3 and the assessments?

4 The second component of the review process is a science assessment. A concise
5 synthesis of the most policy-relevant science has been compiled into a draft Integrated Science
6 Assessment (draft ISA). The draft ISA is supported by a series of annexes that contain more
7 detailed information about the scientific literature. The current draft of the ISA to support this
8 review of the NO₂ primary NAAQS is presented in the Integrated Science Assessment for
9 Oxides of Nitrogen - Health Criteria (Second External Review Draft), henceforth referred to as
10 the draft ISA (EPA, 2008a).

11 The third component of the review process is a risk and exposure assessment, the first
12 draft of which is described in this document. The purpose of this draft document is to
13 communicate EPA's assessment of exposures and risks associated with ambient NO₂. It is
14 supported by a more detailed technical support document, henceforth referred to as the draft
15 TSD. This first draft of the risk and exposure assessment develops estimates of human
16 exposures and risks associated with current ambient levels of NO₂ and with levels that just meet
17 the current standard. The second draft of this document will also consider levels of NO₂ that just
18 meet any potential alternative standards that are identified for consideration. The results of the
19 risk and exposure assessment will be considered alongside the health evidence, as evaluated in
20 the final ISA, to inform the policy assessment and rulemaking process (see below). The draft
21 plan for conducting the risk and exposure assessment to support the NO₂ primary NAAQS is
22 presented in the Nitrogen Dioxide Health Assessment Plan: Scope and Methods for Exposure
23 and Risk Assessment, henceforth referred to as the Health Assessment Plan (EPA, 2007b).

24 The fourth component of the process is the policy assessment and rulemaking. The
25 Agency's views on policy options will be published in the Federal Register as an advance notice
26 of proposed rulemaking (ANPR). This policy assessment will address the adequacy of the
27 current standard and of any potential alternative standards, which will be defined in terms of
28 indicator, averaging time, form,¹ and level. To accomplish this, the policy assessment will
29 consider the results of the final risk and exposure assessment as well as the scientific evidence

¹ 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.

1 (including evidence from the epidemiological, controlled human exposure, and animal
2 toxicological literatures) evaluated in the final ISA. Taking into consideration CASAC advice
3 and recommendations as well as public comment on the ANPR, the Agency will publish a
4 proposed rule, to be followed by a public comment period. Taking into account comments
5 received on the proposed rule, the Agency will issue a final rule to complete the rulemaking
6 process.

7 **1.2 HISTORY**

8 **1.2.1 History of the NO₂ NAAQS**

9 On April 30, 1971, EPA promulgated identical primary and secondary NAAQS for NO₂
10 under section 109 of the Act. The standards were set at 0.053 parts per million (ppm), annual
11 average (36 FR 8186). In 1982, EPA published *Air Quality Criteria for Oxides of Nitrogen*
12 (EPA, 1982), which updated the scientific criteria upon which the initial NO₂ standards were
13 based. On February 23, 1984, EPA proposed to retain these standards (49 FR 6866). After
14 taking into account public comments, EPA published the final decision to retain these standards
15 on June 19, 1985 (50 FR 25532).

16 On July 22, 1987, EPA announced that it was undertaking plans to revise the 1982 air
17 quality criteria (52 FR 27580). In November 1991, EPA released an updated draft air quality
18 criteria document for CASAC and public review and comment (56 FR 59285). The draft
19 document provided a comprehensive assessment of the available scientific and technical
20 information on health and welfare effects associated with NO₂ and other oxides of nitrogen. The
21 CASAC reviewed the draft document at a meeting held on July 1, 1993 and concluded in a
22 closure letter to the Administrator that the document “provides a scientifically balanced and
23 defensible summary of current knowledge of the effects of this pollutant and provides an
24 adequate basis for EPA to make a decision as to the appropriate NAAQS for NO₂” (Wolff,
25 1993). The Air Quality Criteria Document for the Oxides of Nitrogen was then finalized (EPA,
26 1993).

27 The EPA also prepared a Staff Paper that summarized an air quality assessment for NO₂
28 conducted by the Agency (McCurdy, 1994), summarized and integrated the key studies and
29 scientific evidence contained in the revised air quality criteria document, and identified the
30 critical elements to be considered in the review of the NO₂ NAAQS. The CASAC reviewed two

1 drafts of the Staff Paper and concluded in a closure letter to the Administrator (Wolff, 1995) that
2 the document provided a “scientifically adequate basis for regulatory decisions on nitrogen
3 dioxide.” In September of 1995, EPA finalized the Staff Paper entitled, “Review of the National
4 Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical
5 Information” (EPA, 1995).

6 In October 1995, the Administrator announced her proposed decision not to revise either
7 the primary or secondary NAAQS for NO₂ (60 FR 52874; October 11, 1995). A year later, the
8 Administrator made a final determination not to revise the NAAQS for NO₂ after careful
9 evaluation of the comments received on the proposal (61 FR 52852, October 8, 1996). The level
10 for both the existing primary and secondary NAAQS for NO₂ is 0.053 parts per million (ppm)
11 (100 micrograms per cubic meter of air [$\mu\text{g}/\text{m}^3$]), annual arithmetic average, calculated as the
12 arithmetic mean of the 1-hour NO₂ concentrations.

13 **1.2.2 Health Evidence from Previous Review**

14 The prior Air Quality Criteria Document (AQCD) for Oxides of Nitrogen (EPA, 1993)
15 concluded that there were two key health effects of greatest concern at ambient or near-ambient
16 levels of NO₂, increased airways responsiveness in asthmatic individuals after short-term
17 exposures and increased occurrence of respiratory illness in children with longer-term exposures.
18 Evidence also was found for increased risk of emphysema, but this was of major concern only
19 with exposures to levels of NO₂ much higher than then-current ambient levels. The evidence
20 regarding airways responsiveness was drawn largely from controlled human exposure studies.
21 The evidence for respiratory illness was drawn from epidemiological studies that reported
22 associations between respiratory symptoms and indoor exposures to NO₂ in people living in
23 homes with gas stoves. The biological plausibility of the epidemiological results was supported
24 by toxicological studies that detected changes in lung host defenses following NO₂ exposure.
25 Subpopulations considered potentially more susceptible to the effects of NO₂ included
26 individuals with preexisting respiratory disease, children, and the elderly.

27 **1.2.3 Assessment from Previous Review**

28 In the previous review of the NO₂ NAAQS, risks were assessed by comparing ambient
29 monitoring data, which was used as a surrogate for exposure, with health benchmark levels
30 identified from controlled human exposure studies. At the time of the review, a few studies

1 indicated the possibility for adverse health effects due to short-term (e.g., 1-hour) exposures
2 between 0.20 ppm and 0.30 ppm NO₂. Therefore, the focus of the assessment was on the
3 potential for short-term (i.e., 1-hour) exposures to NO₂ levels above potential health benchmarks
4 in this range. The assessment used monitoring data from the years 1988-1992 and screened for
5 sites with one or more hourly exceedances of potential short-term health effect benchmarks.
6 Predictive models were then constructed to relate the frequency of hourly concentrations above
7 short-term health effect benchmarks to a range of annual average concentrations, including the
8 current standard. Based on the results of this analysis, both CASAC (Wolff, 1995) and the
9 Administrator (60 FR 52874) concluded that the minimal occurrence of short-term peak
10 concentrations at or above a potential health effect benchmark of 0.20 ppm (1-hr average)
11 indicated that the existing annual standard would provide adequate health protection against
12 short-term exposures. This conclusion was instrumental in providing the rationale for the
13 decision in the last review to retain the existing annual standard.

14 **1.3 SCOPE OF THE RISK AND EXPOSURE ASSESSMENT FOR THE** 15 **CURRENT REVIEW**

16 **1.3.1 Species of Nitrogen Oxides Included in Analyses**

17 The nitrogen oxides (NO_x) include multiple gaseous (e.g., NO₂, NO, HONO) and
18 particulate (e.g., nitrate) species. As discussed in the integrated review plan (2007a), the current
19 review of the NO₂ NAAQS will focus on the gaseous species of NO_x and will not consider health
20 effects directly associated with particulate species of NO_x. Of the gaseous species, EPA has
21 historically determined it appropriate to specify the indicator of the standard in terms of NO₂
22 because the majority of the information regarding health effects and exposures is for NO₂. The
23 current draft ISA has found this to be the case and, therefore, NO₂ will be used as the indicator
24 for the gaseous NO_x in the risk and exposure assessment described in this document.

25 **1.3.2 Scenarios Addressed in First Draft Assessment**

26 The first draft of the risk and exposure assessment, described in this document, details the
27 assessment of risks and exposures associated with recent ambient levels of NO₂ and with levels
28 associated with just meeting the current standard. The second draft of this document will also
29 describe the assessment of risks and exposures associated with just meeting potential alternative

1 standards. Completion of the second draft of the risk and exposure assessment will follow the
2 completion of the final ISA, thereby allowing the choice of potential alternative standards to be
3 informed by the information in the final ISA.

2. SOURCES, AMBIENT LEVELS, AND EXPOSURES

2.1 SOURCES OF NO₂

Ambient levels of NO₂ are the product of both direct NO₂ emissions and emissions of other NO_x (e.g., NO) which can then be converted to NO₂ (for a more detailed discussion see the draft ISA, section 2.2). Nationally, anthropogenic sources account for approximately 87% of total NO_x emissions. Mobile sources (both on-road and off-road) account for about 60% of total anthropogenic emissions of NO_x, while stationary sources (e.g., electrical utilities and industry) account for the remainder (annex Table 2.6-1). Highway vehicles represent the major mobile source component. In the United States, approximately half the mobile source emissions are contributed by diesel engines and half are emitted by gasoline-fueled vehicles and other sources (annex section 2.6.2 and Table 2.6-1). Apart from these anthropogenic sources, there are also natural sources of NO_x including microbial activity in soils, lightning, and wildfires (draft ISA, section 2.2.1 and annex section 2.6.2).

2.2 AMBIENT LEVELS OF NO₂

According to monitoring data, nationwide levels of ambient NO₂ (annual average) decreased 41% between 1980 and 2006 (draft ISA, Figure 2.4-4). Between 2003 and 2005, national mean concentrations of NO₂ were about 15 ppb for averaging periods ranging from a day to a year. The average daily maximum hourly NO₂ concentrations were approximately 30 ppb. These values are about twice as high as the 24-h averages. The highest maximum hourly concentrations (~200 ppb) between 2003 and 2005 are more than a factor of ten higher than the mean hourly or 24-h concentrations (draft ISA, Figure 2.4-2). The highest levels of NO₂ in the United States can be found in and around Los Angeles, in the Midwest, and in the Northeast.

Nitrogen dioxide is monitored mainly in large urban areas and, therefore, data from the NO₂ monitoring network is generally more representative of urban areas than rural areas. Levels in non-urban areas can be estimated with modeling. Model-based estimates indicate that NO₂ levels in many non-urban areas of the United States are less than 1 ppb. Levels in these areas can approach policy-relevant background concentrations, which are those concentrations that would occur in the United States in the absence of anthropogenic emissions in continental North America (defined here as the United States, Canada, and Mexico). For NO₂, policy-relevant

1 background concentrations are estimated to range from 0.1 ppb to 0.3 ppb (draft ISA, section
2 2.4.6.1).

3 Ambient levels of NO₂ exhibit both seasonal and diurnal variation. In southern cities,
4 such as Atlanta, higher concentrations are found during winter, consistent with the lowest mixing
5 layer heights being found during that time of the year. Lower concentrations are found during
6 summer, consistent with higher mixing layer heights and increased rates of photochemical
7 oxidation of NO₂. For cities in the Midwest and Northeast, such as Chicago and New York City,
8 higher levels tend to be found from late winter to early spring with lower levels occurring from
9 summer through the fall. In Los Angeles the highest levels tend to occur from autumn through
10 early winter and the lowest levels from spring through early summer. Mean and peak
11 concentrations in winter can be up to a factor of two larger than in the summer at sites in Los
12 Angeles. In terms of daily variability, NO₂ levels typically peak during the morning rush hours.
13 Monitor siting plays a key role in evaluating diurnal variability as monitors located further away
14 from traffic will show cycles that are less pronounced over the course of a day than monitors
15 located closer to traffic.

16 **2.3 EXPOSURE TO NO₂**

17 Human exposure to an airborne pollutant is defined as contact between a person and the
18 pollutant at a specific concentration for a specified period of time (draft ISA, section 2.5.1). The
19 integrated exposure of a person to a given pollutant is the sum of the exposures over all time
20 intervals for all microenvironments in which the individual spends time. Microenvironments in
21 which people are exposed to air pollutants such as NO₂ typically include residential indoor
22 environments and other indoor locations, near-traffic outdoor environments and other outdoor
23 locations, and in vehicles (draft ISA, Figure 2.5-1).

24 There is a large amount of variability in the time that individuals spend in different
25 microenvironments, but on average people spend the majority of their time (about 87%) indoors.
26 Most of this time is spent at home with less time spent in an office/workplace or other indoor
27 locations (draft ISA, Figure 2.5-1). On average, people spend about 8% of their time outdoors
28 and 6% of their time in vehicles. Significant variability surrounds each of these broad estimates,
29 particularly when considering influential personal attributes such as age or gender; when
30 accounting for daily, weekly, or seasonal factors influencing personal behavior; or even when

1 characterizing individual variability in time spent in various locations (McCurdy and Graham,
2 2003; Graham and McCurdy, 2004). Typically, the time spent outdoors or in vehicles could vary
3 by 100% or more depending on which of these influential factors are considered. One potential
4 consequence of this is that exposure misclassification can result when total human exposure is
5 not disaggregated between relevant microenvironments and the variability in time spent in these
6 locations is not taken into account.

7 Such misclassification, which can occur in epidemiological studies that rely on ambient
8 pollutant levels as a surrogate for exposure, may obscure the true relationship between ambient
9 air pollutant exposures and health outcomes. Thus, use of ambient pollutant levels as a surrogate
10 for exposures can introduce uncertainty that should be considered when interpreting the
11 epidemiological literature. This uncertainty in exposure estimates can result from differences
12 between ambient levels and actual exposures as well as from the NO₂ monitoring approach itself.

13 Results have been mixed regarding the ability of ambient levels of NO₂ to act as a
14 surrogate for personal exposures to NO₂. Studies examining the association between ambient
15 NO₂ and personal exposure to NO₂ have generated mixed results due to 1) the prevalence of
16 indoor sources of NO₂; 2) the spatial heterogeneity of NO₂ in study areas; 3) the seasonal and
17 geographic variability in the infiltration of ambient NO₂; 4) differences in the time spent in
18 different microenvironments; and 5) differences in study design (draft ISA, section 2.5.6.2). As
19 a result, some researchers have concluded that ambient NO₂ may be a reasonable proxy for
20 personal exposure, while others have noted that caution must be exercised. Overall, the body of
21 evidence examined in the draft ISA demonstrates that ambient NO₂ concentrations are associated
22 with personal exposures; however, the strength of that association varies considerably.

23 The current approach to monitoring ambient NO₂ can also introduce uncertainty into
24 exposure estimates. For example, the method for estimating NO₂ levels (i.e., subtraction of NO
25 from a measure of total NO_x) is subject to interference by NO_x oxidation products. Limited
26 evidence suggests that these compounds result in an overestimate of NO₂ levels by roughly 20 to
27 25% at typical ambient levels. Smaller relative errors are estimated to occur in measurements
28 taken near strong NO_x sources since most of the mass emitted as NO or NO₂ would not yet have
29 been further oxidized. Relatively larger errors appear in locations more distant from strong local
30 NO_x sources. Additionally, many NO₂ monitors are elevated above ground level in the cores of
31 large cities. Because most sources of NO₂ are near ground level, this produces a gradient of NO₂

1 with higher levels near ground level and lower levels being detected at the elevated monitor.
2 One comparison has found an average of a 2.5-fold increase in NO₂ concentration measured at 4
3 meters above the ground compared to 15 meters above the ground. Levels are likely even higher
4 at elevations below 4 meters (draft ISA, section 2.5.3.3). Another source of uncertainty in
5 exposure estimates can result from monitor location. NO₂ monitors are sited for compliance
6 with air quality standards rather than for capturing small-scale variability in NO₂ concentrations
7 near sources such as roadway traffic. Significant gradients in NO₂ concentrations near roadways
8 have been observed in several studies, and NO₂ concentrations have been found to be correlated
9 with distance from roadway and traffic volume (draft ISA, section 2.5.3.2).
10

3. AT RISK POPULATIONS

3.1 OVERVIEW

Specific subpopulations are at increased risk for suffering NO₂-related health effects. This could occur because they are affected by lower levels of NO₂ than the general population (susceptibility), because they experience a larger health impact than the general population to a given level of exposure (susceptibility), and/or because they are exposed to higher levels of NO₂ than the general population (vulnerability). In discussions of susceptibility, the draft ISA focuses on disease-mediated (e.g., asthma, cardiovascular disease) and age-mediated susceptibility (i.e., children and elderly) (draft ISA, sections 4.3.1 and 4.3.2). In discussions of vulnerability, the draft ISA focuses on age-mediated vulnerability (i.e., children and elderly) and vulnerability in individuals who spend a large amount of time on or near roadways due to the location of their residence, their occupation, or the fact that they spend time commuting in traffic (draft ISA, section 4.3.5). These groups are discussed in more detail below.

3.2 DISEASE AND ILLNESS

Recent evidence strengthens the conclusion, drawn in the 1993 Criteria Document, that asthmatics are likely more susceptible than the general population to the effects of NO₂ exposure. In addition, recent evidence broadens this likely susceptible population to include those with other pulmonary conditions and individuals with upper respiratory viral infections (draft ISA, section 4.3.1). These conclusions are based on an array of both short- and long-term studies reporting associations between NO₂ and respiratory and cardiac health effects. The most extensive supporting evidence is available for asthmatics. In addition to the large number of epidemiological studies that have reported associations between NO₂ exposure and health effects in asthmatics, human clinical studies demonstrate that airways hyperresponsiveness in asthmatics is the most sensitive clinical indicator of response to NO₂ (draft ISA, section 4.3.1).

3.3 AGE

The draft ISA identifies both children (i.e., <18 years of age) and older adults (i.e., >65 years of age) as groups that are potentially more susceptible than the general population to the health effects associated with NO₂ exposure (draft ISA, section 4.3.2). In children, the

1 developing lung is highly susceptible to damage from exposure to environmental toxicants
2 (Dietert et al., 2000) likely because eighty percent of alveoli are formed postnatally and changes
3 in the lung continue through adolescence (draft ISA, section 4.3.2). The basis for the increased
4 susceptibility in the elderly is not known, but one hypothesis is that it may be related to changes
5 in antioxidant defenses in the fluid lining the respiratory tract (draft ISA, section 4.3.2). In
6 addition, the generally declining health status of many elderly individuals may increase their
7 risks for pollution-mediated effects (draft ISA, section 4.3.2).

8 **3.4 PROXIMITY TO ROADWAYS**

9 The draft ISA also includes discussion of vulnerable populations that experience
10 increased NO₂ exposures on or near roadways (draft ISA, section 4.3.5). Many studies find that
11 indoor, personal, and outdoor NO₂ levels are strongly associated with proximity to traffic or to
12 traffic density (draft ISA, section 2.5.4). Due to high air exchange rates, NO₂ levels inside a
13 vehicle could rapidly approach levels outside the vehicle during commuting (draft ISA, section
14 4.3.5). Mean in-vehicle NO₂ levels are between 2 and 3 times ambient levels measured at fixed
15 sites nearby (draft ISA, sections 2.5.4 and 4.3.5). Therefore, individuals with occupations that
16 require them to be in traffic or close to traffic (e.g., bus and taxi drivers, highway patrol officers,
17 toll collectors) and those who spend time commuting in traffic could be exposed to relatively
18 high levels of NO₂ compared to ambient levels. Due to the high peak exposures while driving,
19 total personal exposure could be underestimated if exposures while commuting are not
20 considered. In some cases, exposure in traffic can dominate personal exposure to NO₂ (Lee et
21 al., 2000; Son et al., 2004) (draft ISA, section 2.5.4).

22

4. HEALTH EFFECTS

4.1 INTRODUCTION

The draft ISA, along with its associated annexes, provides a comprehensive review and assessment of the scientific evidence related to the health effects associated with NO₂ exposures. For these health effects, the draft ISA characterizes judgments about causality with a hierarchy (for discussion see draft ISA, section 1.6) that contains the following five levels.

- Sufficient to infer a causal relationship
- Sufficient to infer a likely causal relationship (i.e., more likely than not)
- Suggestive but not sufficient to infer a causal relationship
- Inadequate to infer the presence or absence of a causal relationship
- Suggestive of no causal relationship

Judgments about causality are informed by a series of decisive factors that are based on those set forth by Sir Austin Bradford Hill in 1965 (draft ISA, Table 1.6-1). These decisive factors include strength of the observed association, availability of experimental evidence, consistency of the observed association, biological plausibility, coherence of the evidence, temporal relationship of the observed association, and the presence of an exposure-response relationship. For purposes of the characterization of NO₂ health risks, staff have judged it appropriate to focus on endpoints for which the draft ISA concludes that the available evidence is sufficient to infer either a causal or a likely causal relationship.

4.2 ADVERSE RESPIRATORY EFFECTS FOLLOWING SHORT-TERM EXPOSURES

4.2.1 Overview

The draft ISA concludes that, when taken together, recent studies provide scientific evidence that NO₂ is associated with a range of respiratory effects and are *sufficient to infer a likely causal relationship* between short-term NO₂ exposure and adverse effects on the respiratory system (draft ISA, section 5.3.2.1). This finding is supported by a large body of epidemiologic evidence, in combination with findings from human and animal experimental studies. The epidemiologic evidence for respiratory effects can be characterized as consistent, in

1 that associations are reported in studies conducted in numerous locations with a variety of
2 methodological approaches. Considering this large body of epidemiologic studies alone, the
3 findings are coherent in the sense that the studies report associations with respiratory health
4 outcomes that are logically linked together. A number of these epidemiologic studies have been
5 conducted in locations where the ambient NO₂ levels are well below the level of the current
6 NAAQS. Health effects associations have been observed in epidemiologic studies reporting
7 maximum ambient concentrations as high as 100 to 300 ppb, concentrations within the range of
8 the controlled animal and human exposures used in current toxicological and clinical studies
9 reporting respiratory effects (see draft ISA, Tables 5.3-2 and 5.3-3). This evidence is discussed
10 in more detail below.

11 **4.2.2 Effects Based on Controlled Human Exposure Studies**

12 **4.2.2.1 Overview**

13 Controlled human exposure studies have addressed the consequences of short-term (e.g.,
14 15-minutes to several hours) NO₂ exposures for a number of health endpoints including airways
15 responsiveness, host defense and immunity, inflammation, and lung function (draft ISA, section
16 3.1). The draft ISA concludes that in asthmatics, NO₂ may increase the allergen-induced airways
17 inflammatory response at exposures as low as 0.26-ppm for 30 min (draft ISA, Figure 3.1-2) and
18 NO₂ exposures between 0.2 and 0.3 ppm for 30 minutes can result in small but significant
19 increases in non-specific airways responsiveness (draft ISA, section 5.3.2.1). In contrast, the
20 draft ISA concludes that 1) limited evidence indicates that NO₂ may increase susceptibility to
21 injury by subsequent viral challenge at exposures as low as 0.6 ppm for 3 hours; 2) evidence
22 exists for increased airways inflammation at NO₂ concentrations less than 2.0 ppm; and 3) the
23 direct effects of NO₂ on lung function in asthmatics have been inconsistent at exposure
24 concentrations below 1 ppm (draft ISA, section 5.3.2.1). As a result, although studies on all of
25 these endpoints provide qualitative support for the ability of NO₂ to cause adverse effects on
26 respiratory health, the focus for purposes of characterizing risks associated with ambient NO₂ is
27 airways responsiveness (see below).

1 4.2.2.2 Airways Responsiveness

2 Inhaled pollutants such as NO₂ may have direct effects on lung function, or they may
3 enhance the inherent responsiveness of the airways to a challenge with a bronchoconstricting
4 agent (draft ISA, section 3.1.3). Asthmatics are generally much more sensitive to nonspecific
5 bronchoconstricting agents (e.g., cholinergic drugs, cold air, histamine, etc.) than non-asthmatics,
6 and airways challenge testing is used as a diagnostic test in asthma. An increase in airways
7 responsiveness in asthmatics is one indicator of increased severity of disease and worsened
8 asthma control while effective treatment often reduces airways responsiveness. Aerosolized
9 allergens can also be used in controlled airways challenge testing in the laboratory. The degree
10 of responsiveness to allergens is a function of the concentration of inhaled allergen, the degree of
11 sensitization to the allergen, and the degree of nonspecific airways responsiveness. Following
12 inhalation of a non-specific bronchoconstricting agent or an allergen, asthmatics may experience
13 both an “early” response, with a decline in lung function within minutes after the challenge, and
14 a “late” response, with a decline in lung function hours after the exposure. The early response
15 primarily reflects release of histamine and other inflammatory mediators by airways mast cells
16 while the late response reflects enhanced airways inflammation and mucous production.
17 Airways responsiveness can be measured by assessing changes in pulmonary function (e.g.,
18 decline in FEV₁) or changes in the inflammatory response (e.g., using markers in
19 bronchoalveolar lavage (BAL) fluid or induced sputum) (draft ISA, section 3.1.3.1).

20 Folinsbee (1992) conducted a meta-analysis using individual level data from 19 clinical
21 NO₂ exposure studies measuring airways responsiveness in asthmatics (draft ISA, section
22 3.1.3.2). These studies included NO₂ exposure levels between 0.1 ppm and 1.0 ppm and most of
23 them used non-specific bronchoconstricting agents such as methacholine, carbachol, histamine,
24 or cold air. The largest effects were observed for subjects at rest. Among subjects exposed at
25 rest, 76% experienced increased airways responsiveness following exposure to NO₂ levels
26 between 0.2 and 0.3 ppm. Because this meta-analysis evaluated only the direction of the change
27 in airways responsiveness, it is not possible to discern the magnitude of the change from these
28 data. However, the results do suggest that short-term exposures to NO₂ at near-ambient levels
29 (<0.3 ppm) can alter airways responsiveness in people with mild asthma (draft ISA, section
30 3.1.3.2).

1 Several studies published since the last review address the question of whether low-level
2 exposures to NO₂ enhance the response to specific allergen challenge in mild asthmatics (draft
3 ISA, section 3.1.3.1). These recent studies suggest that NO₂ may enhance the sensitivity to
4 allergen-induced decrements in lung function, and increase the allergen-induced airways
5 inflammatory response. Strand et al. (1997) demonstrated that single 30-minute exposures to
6 0.26-ppm NO₂ increased the late phase response to allergen challenge 4 hours after exposure, as
7 measured by changes in lung function. In a separate study (Strand et al., 1998), 4 daily repeated
8 exposures to 0.26-ppm NO₂ for 30 minutes increased both the early and late-phase responses to
9 allergen, as measured by changes in lung function. Barck et al. (2002) used the same exposure
10 and challenge protocol in the earlier Strand study (0.26 ppm for 30 min, with allergen challenge
11 4-h after exposure), and performed BAL 19 hours after the allergen challenge to determine NO₂
12 effects on the allergen-induced inflammatory response. Compared with air followed by allergen,
13 NO₂ followed by allergen caused an increase in the BAL recovery of polymorphonuclear (PMN)
14 cells and eosinophil cationic protein (ECP) as well as a reduction in total BAL fluid volume and
15 cell viability. ECP is released by degranulating eosinophils, is toxic to respiratory epithelial
16 cells, and is thought to play a role in the pathogenesis of airways injury in asthma. Subsequently,
17 Barck et al. (2005) exposed 18 mild asthmatics to air or 0.26 ppm NO₂ for 15 minutes on day 1,
18 followed by two 15 minute exposures separated by 1 hour on day 2, with allergen challenge after
19 exposures on both days 1 and 2. Sputum was induced before exposure on day 1 and after
20 exposures (morning of day 3). Compared to air plus allergen, NO₂ plus allergen resulted in
21 increased levels of ECP in both sputum and blood and increased myeloperoxidase levels in
22 blood. All exposures in these studies (Barck et al., 2002, 2005; Strand et al., 1997, 1998) used
23 subjects at rest. They used an adequate number of subjects, included air control exposures,
24 randomized exposure order, and separated exposures by at least 2 weeks. Together, they indicate
25 the possibility for effects on allergen responsiveness in some asthmatics following brief
26 exposures to 0.26 ppm NO₂. However, other recent studies have failed to find effects using
27 similar, but not identical, approaches (draft ISA, section 3.1.3.1). The differing findings may
28 relate in part to differences in timing of the allergen challenge, the use of multiple versus single-
29 dose allergen challenge, the use of BAL versus sputum induction, exercise versus rest during
30 exposure, and differences in subject susceptibility (draft ISA, section 3.1.3.1). Table 1 (below)

1 provides summary information on the key controlled human exposure studies identified in the
2 draft ISA that evaluated airways responsiveness.

3 **4.2.2.3 Conclusions**

4 Based on the draft ISA's evaluation of controlled human exposure studies, staff have
5 judged that the strongest basis for the characterization of NO₂ risks is airway responsiveness in
6 asthmatics. Asthmatic volunteers have been exposed to NO₂ in the absence of other pollutants
7 that often confound associations in the epidemiology literature. Therefore, these studies provide
8 evidence for a direct relationship between exposure to NO₂ and this respiratory health effect.
9 However, because many of the studies of airways responsiveness evaluate only a single level of
10 NO₂ and because of methodological differences between the studies, staff have judged that the
11 data are not sufficient to derive an exposure-response relationship in the range of interest.
12 Therefore, the most appropriate approach to characterizing risks based on the controlled human
13 exposure studies evidence for airways responsiveness is to compare estimated NO₂ air quality
14 and exposure levels with potential health effect benchmark levels. Estimates of hourly peak air
15 quality concentrations and personal exposures to ambient NO₂ concentrations at and above
16 specified potential health effect benchmark levels provides some perspective on the public health
17 impacts of health effects that we cannot currently evaluate in quantitative risk assessments. Staff
18 recognizes that there is high inter-individual variability in responsiveness such that only a subset
19 of asthmatic individuals exposed at and above a given benchmark level would actually be
20 expected to experience any such potential adverse health effects.

21 To identify these potential health effect benchmarks, staff have relied on the draft ISA's
22 evaluation of the NO₂ human exposures studies. Controlled human exposure studies involving
23 allergen challenge in asthmatics suggest that NO₂ exposure may enhance the sensitivity to
24 allergen-induced decrements in lung function and increase the allergen-induced airways
25 inflammatory response at exposures as low as 0.26-ppm NO₂ for 30 min (draft ISA, Figure 3.1-2
26 and section 5.3.2.1). Exposure to NO₂ also has been found to enhance the inherent
27 responsiveness of the airways to subsequent non-specific challenges (draft ISA, sections 3.1.4.2
28 and 5.3.2.1). In general, small but significant increases in non-specific airways responsiveness

Table 1. Summary of Key Controlled Human Exposure Studies of Airways Responsiveness

1

Study	NO₂ Exposure Level (ppm)	Exposure Duration	Study Population	Allergen versus non-specific	Metric Used	Number of Subjects	Exercise	Statistically Significant	Statistically Non Significant
Tunncliffe, 1994	0.4	1-hour	Mild asthmatics	Allergen	Lung function	8	No	X	
Devalia, 1994	0.4	6-hours	Mild asthmatics	Allergen	Lung function	8	No		X
Strand, 1997	0.26	30-minutes	Mild asthmatics	Allergen	Lung function	18	No	X	
Strand, 1998	0.26	30-minutes (4x per day)	Mild to Moderate asthmatics	Allergen	Lung function	16	No	X	
Barck, 2005	0.26	15-minutes (3x over 2 days)	Mild asthmatics	Allergen	Lung function	18	No		X
Barck, 2005	0.26	15-minutes (3x over 2 days)	Mild asthmatics	Allergen	Inflammatory Markers (sputum, blood)	18	No	X	
Barck, 2002	0.26	30-minutes	Mild asthmatics	Allergen	Inflammatory Markers (BAL)	13	No	X	
Bylin, 1985	0.3	20-minutes	Mild asthmatics	Non-specific	Lung function	8	No	X	
Mohsenin, 1987	0.5	1-hour	Asthmatics	Non-specific	Lung function	8	No	X	
Strand, 1996	0.26	30-minutes	Mild asthmatics	Non-specific	Lung function	19	No	X	
Jörres, 1990	0.25	30-minutes	Mild asthmatics	Non-specific	Lung function	14	No	X	
Rubenstein, 1990	0.3	30-minutes	Asthmatics	Non-specific	Lung function	9	Yes		X
Jörres, 1991	0.25	30-minutes	Mild asthmatics	Non-specific	Lung function	11	Yes		X
Witten, 2005	0.4	3-hours	Mild asthmatics	Allergen	Inflammatory Markers (sputum)	15	Yes		X
Jörres, 1991	0.25	30-minutes	Mild asthmatics	Non-specific	Lung Function	11	Yes		X
Jenkins, 1999	0.4	3-hours	Mild asthmatics	Allergen	Lung function	11	Yes	X	
Jenkins, 1999	0.2	6-hours	Mild asthmatics	Allergen	Lung function	11	Yes		X
Witten, 2005	0.4	3-hours	Mild asthmatics	Allergen	Lung function	15	Yes		X
Roger, 1990	0.15-0.6	75-minutes	Mild asthmatics	Non-specific	Lung function	21	Yes		X

1 have been observed in the range of 0.2 to 0.3 ppm NO₂ for 30 minute exposures in asthmatics.
2 Therefore, in the risk characterization described in Chapters 5-7 of this document, staff judge
3 that 1-hour NO₂ levels in this range are appropriate to consider as potential health benchmarks
4 for comparison to air quality levels and exposure estimates. To characterize health risks with
5 respect to this range, potential health effect benchmark values of 0.20 ppm (200 ppb), 0.25 ppm
6 (250 ppb), and 0.30 ppm (300 ppb) have been employed to reflect the lower- middle- and upper-
7 end of the range identified in the draft ISA as the lowest levels at which controlled human
8 exposure studies have provided sufficient evidence for the occurrence of NO₂-related airway
9 responsiveness.

10 **4.2.3 Epidemiology Literature**

11 ***4.2.3.1 Hospital Admissions and Emergency Department Visits***

12 Epidemiologic evidence exists for positive associations between short-term ambient NO₂
13 concentrations below the current NAAQS and increased numbers of emergency department
14 visits and hospital admissions for respiratory causes, especially asthma (draft ISA, section
15 5.3.2.1). Total respiratory causes for emergency department visits and hospitalizations typically
16 include asthma, bronchitis and emphysema (collectively referred to as COPD), pneumonia, upper
17 and lower respiratory infections, and other minor categories. Temporal associations between
18 emergency department visits or hospital admissions for respiratory diseases and ambient levels
19 of NO₂ have been the subject of over 50 peer-reviewed research publications since the last
20 review. These studies have examined morbidity in different age groups and have often utilized
21 multi-pollutant models to evaluate potential confounding effects of co-pollutants.

22 Of the emergency department visit and hospital admission studies reviewed in the NO_x
23 draft ISA, 6 were conducted in the United States (draft ISA, Table 5.3-4). Of these 6 studies,
24 only 3 evaluated associations with NO₂ using multi-pollutant models (Peel et al., 2005 and
25 Tolbert et al., 2007 in Atlanta; Ito et al., 2007 in New York City). In the study by Peel and
26 colleagues, investigators evaluated emergency department visits among all ages in Atlanta, GA
27 during the period of 1993 to 2000. Using single pollutant models, the authors reported a 2.4%
28 (95% CI: 0.9, 4.1) increase in respiratory emergency department visits associated with a 30-ppb
29 increase in 1-h max NO₂ levels. For asthma visits, a 4.1% (95% CI: 0.8%, 7.6%) increase was
30 detected only in individuals 2 to 18 years of age. Tolbert and colleagues reanalyzed these data

1 with 4 additional years of information and found essentially similar results in single pollutant
2 models (2.0% increase, 95% CI: 0.5, 3.3). This same study found that the associations were
3 positive, but not statistically-significant, in multi-pollutant models that included PM₁₀ or ozone
4 (O₃). In the study by Ito and colleagues, investigators evaluated emergency department visits for
5 asthma in New York City during the years 1999 to 2002. The authors found a 12 % (95% CI:
6 7%, 15%) increase in risk per 20 ppb increase in 24-hour ambient NO₂. Risk estimates were
7 robust and remained statistically significant in multi-pollutant models that included PM_{2.5}, O₃,
8 CO, and SO₂.

9 *4.2.3.2 Respiratory Illness and Symptoms*

10 *Studies of Ambient NO₂*

11 Epidemiologic studies using community ambient monitors have found associations
12 between ambient NO₂ concentrations and respiratory symptoms (draft ISA, sections 3.1.4.2 and
13 5.3.2.1, Figure 3.1-6) in cities where NO₂ concentrations were within the range of 24-hour
14 average concentrations observed in recent years. Several studies have been published since the
15 last review of the NO₂ NAAQS including 3 multi-city studies in urban areas covering the
16 continental United States and southern Ontario. These are the Harvard Six Cities Study (Six
17 Cities; Schwartz et al., 1994), the National Cooperative Inner-City Asthma Study (NCICAS;
18 Mortimer et al., 2002), and the Childhood Asthma Management Program (CAMP; Schilderout et
19 al., 2006).

20 Schwartz et al (1994) studied 1,844 schoolchildren, followed for 1 year, as part of the Six
21 Cities Study that included the cities of Watertown, MA, Baltimore, MD, Kingston-Harriman,
22 TN, Steubenville, OH, Topeka, KS, and Portage, WI. Respiratory symptoms were recorded
23 daily. The authors reported a significant association between 4-day mean NO₂ levels and
24 incidence of cough among all children in single-pollutant models, with an odds ratio (OR) of
25 1.61 (95% CI: 1.08, 2.43) standardized to a 20-ppb increase in NO₂. The incidence of cough
26 increased up to approximately mean NO₂ levels (~13 ppb) (p = 0.01), after which no further
27 increase was observed. The significant association between cough and 4-day mean NO₂ level
28 remained unchanged in models that included O₃, but was attenuated and lost statistical
29 significance in two-pollutant models that included PM₁₀ (OR = 1.37 [95% CI: 0.88, 2.13]) or
30 SO₂ (OR = 1.42 [95% CI: 0.90, 2.28]).

1 Mortimer et al. (2002) studied the risk of asthma symptoms among 864 asthmatic
2 children in the eight cities that were part of the NCICAS. The eight study locations included
3 New York City, NY, Baltimore, MD, Washington, DC, Cleveland, OH, Detroit, MI, St Louis,
4 MO, and Chicago, IL. Subjects were followed daily for four 2-week periods over the course of
5 nine months with morning and evening asthma symptoms and peak flow recorded. The greatest
6 effect was observed for morning symptoms using a 6-day moving average, with a reported OR of
7 1.48 (95% CI: 1.02, 2.16). Although effects were generally robust in multi-pollutant models that
8 included O₃ (OR for 20-ppb increase in NO₂ = 1.40 [95% CI: 0.93, 2.09]), O₃ and SO₂ (OR for
9 NO₂ = 1.31 [95% CI: 0.87, 2.09]), or O₃, SO₂, and PM₁₀ (OR for NO₂ = 1.45 [95% CI: 0.63,
10 3.34]), they were not statistically-significant.

11 Schildcrout et al. (2006) investigated the association between ambient NO₂ and
12 respiratory symptoms and rescue inhaler use as part of the CAMP study. The study reported on
13 990 asthmatic children living within 50 miles of an NO₂ monitor in Boston, MA, Baltimore, MD,
14 Toronto, ON, St. Louis, MO, Denver, CO, Albuquerque, NM, or San Diego, CA. Symptoms and
15 use of rescue medication were recorded daily, resulting in each subject having an approximate
16 average of two months of data. The authors reported the strongest association between NO₂ and
17 increased risk of cough for a 2-day lag, with an OR of 1.09 (95% CI: 1.03, 1.15) for each 20-ppb
18 increase in NO₂ occurring 2 days before measurement. Multi-pollutant models that included CO,
19 PM₁₀, or SO₂ produced similar results (see Figure 3.1-5, panel A of the draft ISA). Additionally,
20 increased NO₂ exposure was associated with increased use of rescue medication, with the
21 strongest association for a 2-day lag, both for single- and multi-pollutant models (e.g., for an
22 increase of 20-ppb NO₂ in the single-pollutant model, the RR for increased inhaler usage was
23 1.05 (95% CI: 1.01, 1.09).

24 ***Studies of Indoor NO₂***

25 Evidence supporting increased respiratory morbidity following NO₂ exposures is also
26 found in studies of indoor NO₂ (draft ISA, section 3.1.4.1). For example, in a randomized
27 intervention study in Australia (Pilotto et al., 2004), students attending schools that switched out
28 unvented gas heaters, a major source of indoor NO₂, experienced a decrease in both levels of
29 NO₂ and in respiratory symptoms (e.g., difficulty breathing, chest tightness, and asthma attacks)
30 compared to students in schools that did not switch out unvented gas heaters (levels were 47.0

1 ppb in control schools and 15.5 ppb in intervention schools) (draft ISA, section 2.7). An earlier
2 indoor study by Pilotto and colleagues (1997) found that students in classrooms with higher
3 levels of NO₂ also had higher rates of respiratory symptoms (e.g., sore throat, cold) and
4 absenteeism than students in classrooms with lower levels of NO₂. This study detected a
5 significant concentration-response relationship, strengthening the argument that NO₂ is causally
6 related to respiratory morbidity. A number of other indoor studies conducted in homes have also
7 detected significant associations between indoor NO₂ and respiratory symptoms (draft ISA,
8 section 3.1.4.1).

9 ***4.2.3.3 Conclusions Regarding the Epidemiology Literature***

10 As mentioned above (see section 1.1), the NO₂ epidemiological literature will be
11 considered during the policy assessment and rulemaking stage of the NAAQS review process as
12 part of an evidence-based approach to assessing the adequacy of potential alternative standards.
13 This use of the epidemiological literature will be reflected in Agency rulemaking documents
14 (i.e., ANPR, proposed rulemaking, and final rulemaking). However, the appropriateness of the
15 epidemiological literature for use as the basis of a quantitative risk assessment is a separate issue
16 and is discussed below.

17 The preferred approach for conducting a risk assessment based on concentration-response
18 relationships from the epidemiological literature would be to rely on studies of ambient NO₂
19 conducted in multiple locations throughout the United States that employ both single-pollutant
20 and multi-pollutant models. This approach would provide a range of concentration-response
21 functions that are relevant to specific cities in the United States. However, the relatively small
22 number of NO₂ epidemiological studies conducted in the United States and the difficulty in
23 separating direct effects of NO₂ from those associated with a traffic-related pollutant mixture that
24 includes NO₂ (draft ISA, section 5.4) would increase the quantitative uncertainty associated with
25 a risk assessment based on the epidemiological literature. These factors make it particularly
26 difficult to quantify with confidence the unique contribution of NO₂ to respiratory health effects.
27 Therefore, staff judge it unlikely that a quantitative risk assessment based on the available NO₂
28 epidemiological literature would meaningfully inform a decision to retain or revise the standard.
29 This judgment, along with consideration of the resource requirements associated with conducting
30 such an assessment, have led staff to conclude that it is not appropriate to conduct a quantitative

1 assessment of NO₂ risks based on the epidemiological literature to support this review of the
2 NO₂ NAAQS.

3 **4.2.4 Toxicology Literature**

4 Although the animal toxicology literature is not used as a quantitative basis for evaluating
5 NO₂ risks in this assessment, toxicology studies are important for their ability to provide
6 mechanistic insights into health effects that have been observed in humans and because they can
7 support the plausibility of associations observed in the epidemiological literature. For example,
8 animal studies provide evidence that NO₂ can impair the respiratory host defense system
9 sufficiently to render animals more susceptible to respiratory infections. Mortality rates
10 following infection with a respiratory virus have been evaluated in the presence and absence of
11 NO₂. Susceptibility to bacterial and viral pulmonary infections, as measured by this approach,
12 increases with NO₂ exposures as low as 0.5 ppm (draft ISA, section 3.1.1 and 5.3.2.1). In
13 addition, increased airways responsiveness has been detected in animals exposed to NO₂ levels
14 between 1 and 4 ppm (draft ISA, section 5.3.2.1 and Table 5.3-3). Six-week exposures to 4.0
15 ppm NO₂ or longer exposures (e.g., 12 week) to lower levels (e.g., 1 ppm) of NO₂ have caused
16 airways hyperresponsiveness to histamine in guinea pigs (draft ISA, section 3.1.3.2).
17 Toxicologic studies have also detected indications of increased inflammation following NO₂
18 exposures < 1.0 ppm in vitamin C-deficient guinea pigs (draft ISA, section 3.1.2). Thus, the
19 toxicology literature provides qualitative support for the NO₂ findings reported in humans.

20 **4.3 OTHER ADVERSE EFFECTS FOLLOWING SHORT-TERM** 21 **EXPOSURES**

22 The epidemiologic evidence is *suggestive but not sufficient to infer a casual relationship*
23 between short-term exposure to NO₂ and nonaccidental and cardiopulmonary-related mortality.
24 Results from several large U.S. and European multi-city studies and a meta-analysis study
25 indicated positive associations between ambient NO₂ concentrations and the risk of all-cause
26 (nonaccidental) mortality, with effect estimates ranging from 0.5 to 3.6% excess risk in mortality
27 per standardized increment (draft ISA, section 3.3.1, Figure 3.3-2, section 5.3.2.3). In general,
28 the NO₂ effect estimates were robust to adjustment for co-pollutants. Both cardiovascular and
29 respiratory mortality have been associated with increased NO₂ concentrations in epidemiologic

1 studies (draft ISA, Figure 3.3-3); however, similar associations were observed for other
2 pollutants, including PM and SO₂. The range of risk estimates for mortality excess was
3 generally smaller than that for other pollutants such as PM. In addition, while NO₂ exposure,
4 alone or in conjunction with other pollutants, may contribute to increased mortality, evaluation
5 of the specificity of this effect is difficult. Clinical studies showing hematologic effects and
6 animal toxicological studies showing biochemical, lung host defense, permeability, and
7 inflammation changes with short-term exposures to NO₂ provide limited evidence of plausible
8 pathways by which risks of morbidity and, potentially, mortality may be increased, but no
9 coherent picture is evident at this time (draft ISA, section 5.3.2.3).

10 The available evidence on the effects of short-term exposure to NO₂ on cardiovascular
11 health effects is *inadequate to infer the presence or absence of a causal relationship* at this time.
12 Evidence from epidemiologic studies of heart rate variability, repolarization changes, and cardiac
13 rhythm disorders among heart patients with ischemic cardiac disease are inconsistent (draft ISA,
14 sections 3.2.1 and 5.3.2.2). In most studies, associations with PM were found to be similar or
15 stronger than associations with NO₂. Generally positive associations between ambient NO₂
16 concentrations and hospital admissions or emergency department visits for cardiovascular
17 disease have been reported in single-pollutant models (draft ISA, section 3.2.2); however, most
18 of these effect estimate values were diminished in multi-pollutant models that also contained CO
19 and PM indices (draft ISA, section 5.3.2.2). Mechanistic evidence of a role for NO₂ in the
20 development of cardiovascular diseases from studies of biomarkers of inflammation, cell
21 adhesion, coagulation, and thrombosis is lacking (draft ISA, sections 3.2.1.4 and 5.3.2.2).
22 Furthermore, the effects of NO₂ on various hematological parameters in animals are inconsistent
23 and, thus, provide little biological plausibility for effects of NO₂ on the cardiovascular system.

24 **4.4 ADVERSE EFFECTS FOLLOWING LONG-TERM EXPOSURES**

25 The epidemiologic and toxicological evidence examining the effect of long-term
26 exposure to NO₂ on respiratory morbidity is *suggestive but not sufficient to infer a casual*
27 *relationship* at this time. A number of epidemiologic studies examined the effects of long-term
28 exposure to NO₂ and reported positive associations with decrements in lung function and
29 partially irreversible decrements in lung function growth (draft ISA, section 3.4.1, Figures 3.4-1
30 and 3.4-2, section 5.3.2.4). However, similar associations have also been found for PM, O₃, and

1 proximity to traffic (<500 m) and the high correlation among traffic-related pollutants made it
2 difficult to accurately estimate the independent effects in these long-term exposure studies.
3 Results from the available epidemiologic evidence investigating the association between long-
4 term exposure to NO₂ and increases in asthma prevalence and incidence are suggestive but not
5 always consistent (draft ISA, sections 3.4.2 and 5.3.2.4). Epidemiologic studies conducted in
6 both the United States and Europe also have produced inconsistent results regarding an
7 association between long-term exposure to NO₂ and respiratory symptoms (draft ISA, sections
8 3.4.3 and 5.3.2.4). While some positive associations were noted, a large number of symptom
9 outcomes were examined and the results across specific outcomes were inconsistent. Animal
10 toxicological studies demonstrated that NO₂ exposure resulted in morphological changes in the
11 centriacinar region of the lung and in bronchiolar epithelial proliferation (draft ISA, section
12 3.4.4), which may provide some biological plausibility for the observed epidemiologic
13 associations between long-term exposure to NO₂ and respiratory morbidity. Susceptibility to
14 these morphological effects was found to be influenced by many factors, such as age,
15 compromised lung function, and acute infections.

16 The available epidemiologic and toxicological evidence is *inadequate to infer the*
17 *presence or absence of a causal relationship* for carcinogenic, cardiovascular, and reproductive
18 and developmental effects related to long-term NO₂ exposure. Epidemiologic studies conducted
19 in Europe have shown an association between long-term NO₂ exposure and increased incidence
20 of cancer (draft ISA, section 5.3.2.5). However, the animal toxicological studies have provided
21 no clear evidence that NO₂ acts as a carcinogen (draft ISA, sections 3.5.1 and 5.3.2.5). The very
22 limited epidemiologic and toxicological evidence does not suggest that long-term exposure to
23 NO₂ has cardiovascular effects (draft ISA, sections 3.5.2 and 5.3.2.5). The epidemiologic
24 evidence is not consistent for associations between NO₂ exposure and growth retardation;
25 however, some evidence is accumulating for effects on preterm delivery (draft ISA, sections
26 3.5.3 and 5.3.2.5). Scant animal evidence supports a weak association between NO₂ exposure
27 and adverse birth outcomes and provides little mechanistic information or biological plausibility
28 for the epidemiologic findings.

29 The epidemiologic evidence is *inadequate to infer the presence or absence of a causal*
30 *relationship* between long-term exposure to NO₂ and mortality (draft ISA, section 5.3.2.6). In the
31 United States and European cohort studies examining the relationship between long-term

1 exposure to NO₂ and mortality, results were generally inconsistent (draft ISA, section 3.6, Figure
2 3.6-2, and section 5.3.2.6). Further, when associations were suggested, they were not specific to
3 NO₂, but also implicated PM and other traffic indicators. The relatively high correlations
4 reported between NO₂ and PM indices make it difficult to interpret these observed associations at
5 this time (draft ISA, section 5.3.2.6).

6

5. OVERVIEW OF RISK AND EXPOSURE ASSESSMENT

5.1 INTRODUCTION

Human exposure, regardless of the pollutant, depends on where an individual is located and what they are doing at a given moment of time. The magnitude of the exposure can depend on a variety of factors, such as personal attributes (e.g., age or gender), emission sources (e.g., automobile exhaust, indoor gas stoves), and physical-chemical properties of the pollutant (e.g., atmospheric chemistry). The risk of an adverse health effect following exposure to a pollutant is also dependent on a number of factors, such as the individual's personal attributes (age, gender, preexisting health conditions) and the toxic properties of the pollutant (e.g., as indicated by dose- or concentration-response relationships). An important feature of a combined exposure assessment and health risk characterization is to maintain their expected degree of correlation, considering common influential factors and the variability that occurs in personal behavior and exposure concentrations across time and space.

One method to assess exposure to air pollutants is through analysis of air quality concentrations. Ambient monitoring can serve as an indicator of potential exposures that a population residing in an area might have. Depending on the spatial density of the monitoring network and the frequency of sample collection, the measured concentrations can provide a useful record of ambient concentrations that vary over time and across a geographic area. Ambient NO₂ concentrations have been linked with adverse health responses and thus are considered a reasonable surrogate for exposure. However, the actual exposures that individuals experience might be influenced by other sources not measured by the ambient monitor (e.g., indoor sources). In addition, while temporal variability can be well represented with continuous ambient monitoring (e.g., hourly measures throughout the year), the spatial and temporal variability in human activities is not considered, further adding to exposure error.

Another method for determining people's exposure to a substance is through personal measurements of the pollutant(s). Personal exposures can provide a reasonable estimate of an individual's total exposure since it accounts for different concentrations an individual encounters over time, including high concentrations that may result from outdoor and indoor source emissions. As described in section 2.5 of the NO_x ISA however, the availability of personal

1 exposure measurements for NO₂ is limited to only a few studies performed in U.S., each
2 containing a limited number of study subjects. The measurement of personal NO₂ exposure is
3 further restricted by the sampling device detection capabilities, resulting in measurement periods
4 of days to weeks when measured. This time-averaging of personal exposure concentrations may
5 provide some information most relevant to health effects associated with long-term exposures to
6 NO₂, but is less informative for evaluating health effects that result from hourly or daily (or even
7 multiple peak) exposures.

8 Inhalation exposure models are useful in realistically estimating personal exposures,
9 particularly those exposure models that can simulate human activity patterns over variable
10 periods of time. The value of these advanced models is further supported by recognizing that
11 exposure measurements cannot be performed for a large population and/or cannot be used to
12 evaluate alternative exposure scenarios such as simulating just meeting the current or alternative
13 standards. Exposure models are capable of performing any number of simulations (e.g., an entire
14 population, selected individuals) and in any location (e.g., urban area, CMSA, census block), the
15 scope of which depends on the availability of relevant input data. Inhalation exposure models
16 are typically driven by estimates of ambient outdoor concentrations of the pollutants, since the
17 contribution of ambient conditions to total exposure is of primary interest. These outdoor
18 concentrations, which can vary by time of day as well as by location, may be provided by
19 measurements, by air quality models, or by a combination of these. In addition, exposure models
20 can estimate concentrations associated with indoor source emissions to provide perspective on
21 the relative contribution such sources have on total exposure. Thus, the complexity of modeling
22 exposure and the usefulness of the results generated is driven by the temporal and spatial
23 variability in ambient and other concentrations persons may be exposed to, the ability to capture
24 variability (both inter- and intra-personal) in human activities, and whether the most important
25 sources contributing to total exposure are represented.

26 Each of these elements of exposure and risk have been considered in the development of
27 the approach for conducting these assessments in the draft document entitled *Nitrogen Dioxide*
28 *Health Assessment Plan: Scope and Methods for Exposure and Risk Assessment* (EPA, 2007b).
29 That draft document was reviewed by CASAC and the public at a public meeting on October 24-
30 25, 2007. Comments received at that meeting informed the approach adopted by staff for
31 conducting the risk and exposure assessment presented herein.

1 This draft assessment summarizes the results of a risk characterization and exposure
2 assessment associated with recent ambient levels of NO₂ and with ambient levels of NO₂
3 simulated to just meeting the current NO₂ standard of 0.053 ppm annual average. The second
4 draft assessment and the final assessment also will evaluate exposures and health risks associated
5 with any potential alternative standards that are identified for consideration (also see section
6 1.3.2 of this document). Additional details are available in the Exposure and Risk technical
7 support document (draft TSD) (EPA, 2008b) that supports this assessment.

8 **5.2 GOALS**

9 The goals of this draft NO₂ risk and exposure assessment, for both recent ambient air
10 quality conditions and for where ambient concentrations just meet the current standard, are to 1)
11 estimate short-term exposures and potential human health risks associated with ambient NO₂; 2)
12 evaluate the quantitative relationship between long-term average NO₂ air quality and short-term
13 levels of NO₂ that exceed health effect benchmark levels; 3) determine factors contributing to
14 persons estimated to be most frequently exposed to concentration at or above selected 1-hour
15 concentrations; and 4) identify important assumptions and uncertainties associated with the
16 estimates of exposure and the risk characterization.

17 **5.3 GENERAL APPROACH**

18 Exposures were assessed in a two-step process. In the first step, scenario-driven air
19 quality analyses were performed using ambient NO₂ concentrations for years 1995 through 2006.
20 This air quality data, as well as other NO₂ concentrations derived from ambient levels, were used
21 as a surrogate to estimate potential human exposure. All U.S. monitoring sites where NO₂ data
22 have been collected are represented by this analysis and, as such, the results generated are
23 considered a broad characterization of national air quality and human exposures that might be
24 associated with these concentrations.

25 In the second step, detailed modeling of population exposures was conducted. For this
26 exposure analysis, a probabilistic approach was used to model individual exposures considering
27 the time people spend in different microenvironments and variable NO₂ concentrations that occur
28 within these microenvironments across time, space, and microenvironment type. This approach
29 to assessing exposures was more resource intensive than using ambient levels as a surrogate for

1 exposure, therefore staff included only a few specific locations in the U.S. for potential inclusion
2 in this part of the assessment. Although the geographic scope of this analysis was restricted, the
3 approach used provides realistic estimates of NO₂ exposures, particularly those exposures
4 associated with important emission sources of NO_x and NO₂, and serves to complement to the
5 broad air quality characterization.

6 For the characterization of risks in both the air quality analysis and the exposure
7 modeling, staff used the range of health short-term potential health effect benchmark values
8 based on the draft ISA (i.e., 1-hr NO₂ levels ranging from 200 to 300 ppb). To assess potential
9 health risks, benchmark values of 200, 250, and 300 ppb were selected and compared to both
10 NO₂ air quality levels and estimates of NO₂ exposure. When NO₂ air quality was used as a
11 surrogate for exposure, the output of the analysis were estimates of the number of times per year
12 specific locations experience 1-hr levels of NO₂ that have been shown to potentially cause
13 adverse health effects in susceptible individuals. When personal exposures were simulated, the
14 output of the analysis were estimates of the number of individuals at risk for experiencing daily
15 maximum 1-hr levels of exposure to NO₂ of ambient origin that have been shown to potentially
16 cause adverse health effects in susceptible individuals. The rationale and details for each of the
17 approaches used and the range of potential health effect benchmarks identified is described
18 below in Chapter 6 (Air Quality Characterization and Associated Health Risk) and Chapter 7
19 (Exposure Assessment and Associated Health Risk).

20 **5.4 ADDITIONAL CONSIDERATIONS**

21 A primary goal of this draft of the risk and exposure assessments is to evaluate the ability
22 of the current NO₂ standard of 0.053 ppm annual average to protect public health. All areas of
23 the United States have annual average levels below the current standard. Therefore, in order to
24 evaluate the ability of the current standard to protect public health, NO₂ concentrations need to
25 be adjusted such that they simulate levels of NO₂ that just meet the current annual standard.

26 Two different adjustment procedures, although mathematically equivalent, were used for
27 the two different approaches to estimate NO₂ exposures. For the air quality characterization, a
28 proportional roll-up of air quality concentrations was performed. The exposure modeling used a
29 proportional roll-down of the potential health effect benchmark levels. Each of these is briefly
30 described below.

1 These procedures were necessary to provide insights into the degree of exposure and risk
2 which would be associated with an increase in ambient NO₂ levels such that the levels were just
3 at or near the current standard in the urban areas analyzed. Staff recognizes that it is extremely
4 unlikely that NO₂ concentrations in these urban areas would rise to meet the current NAAQS and
5 that there is considerable uncertainty with the simulation of conditions that just meet the current
6 annual standard.

7 **5.4.1 Adjustment of Ambient Air Quality**

8 Based on the form of the standard and observed trends in ambient monitoring, ambient
9 NO₂ concentrations were proportionally rolled-up at each location using the maximum annual
10 average concentration that occurred in each year. While annual average concentrations have
11 declined significantly over the time period of analysis, the variability in the concentrations, both
12 the annual average and 1-hour concentrations, have remained relatively constant (see section 2.5
13 in the draft TSD). Therefore, proportional adjustment factors F for each location (i) and year (j)
14 were derived by the following:

$$15 F_{ij} = 53 / C_{\max,ij} \quad \text{eq (1)}$$

16
17 where,

18 F_{ij} = Adjustment factor (unitless)
19
20 $C_{\max,ij}$ = Maximum annual average NO₂ concentration at a monitor in a location i and
21 year j (ppb)
22
23

24 In these cases where staff simulated a proportional roll-up in ambient NO₂ concentrations
25 using eq (1), it is assumed that the current temporal and spatial distribution of air concentrations
26 (as characterized by the current air quality data) is maintained and increased NO_x emissions
27 contribute to increased NO₂ concentrations, with the highest monitor (in terms of annual
28 averages) being adjusted so that it just meets the current 0.053 ppm annual average standard.
29 Values for each air quality adjustment factor used for each location evaluated in the air quality
30 and risk characterization are given in the draft TSD (section 2.5). For each location and calendar
31 year, all the hourly concentrations in a location were multiplied by the same constant value F to
32 make the highest annual mean equal to 53 ppb for that location and year. For example, of
33 several monitors measuring NO₂ in Boston for year 1995, the maximum annual mean

1 concentration was 30.5 ppb, giving an adjustment factor of $F = 53/30.5 = 1.74$ for that year. All
2 hourly concentrations measured at all monitoring sites in that location would then be multiplied
3 by 1.74, resulting in an upward scaling of hourly NO₂ concentrations for that year. Therefore,
4 one monitoring site in Boston for year 1995 would have an annual average concentration of
5 0.053 ppm, while all other monitoring sites would have an annual average concentration below
6 that value, although still proportionally scaled up by 1.74. Then, using the adjusted hourly
7 concentrations to simulate just meeting the current standard, the metrics of interest (e.g., annual
8 mean NO₂ concentration, the number of potential health effect benchmark exceedances) were
9 estimated for each site-year.

10 **5.4.2 Adjustment of Potential Health Effect Benchmark Levels**

11 Rather than proportionally modify the air quality concentrations used for input to the
12 exposure model, a proportional roll-down of the potential health effect benchmark level was
13 performed. This was done to reduce the processing time associated with the exposure modeling
14 simulations since there were tens of thousands of receptors modeled in each location. In
15 addition, because the adjustment is proportional, the application of a roll-down of the selected
16 benchmark level is mathematically equivalent to a proportional roll-up of the air quality
17 concentrations. The same approach used in the air quality adjustment described above was used
18 in the exposure modeling to scale the benchmark levels downward to simulate just meeting the
19 current standard. For example, an adjustment factor of 1.59 was determined for Philadelphia for
20 year 2001, based on a maximum predicted annual average NO₂ concentration of 33 ppb for a
21 modeled receptor placed at an ambient monitoring location. Therefore, the 1-hour potential
22 health effect benchmark levels of 200, 250, and 300 ppb were proportionally rolled-down to 126,
23 157, and 189 ppb, respectively for year 2001. This procedure was applied for each year within
24 each location where an exposure modeling was performed to simulate just meeting the current
25 standard.

6. AMBIENT AIR QUALITY AND HEALTH RISK

CHARACTERIZATION

6.1 OVERVIEW

Ambient monitoring data for each of the years 1995 through 2006 were used in this analysis to characterize NO₂ air quality across the U.S. This air quality data, as well as other NO₂ concentrations derived from ambient levels, were used as a surrogate to estimate potential human exposure. Because the current standard is based on annual average levels of NO₂ while the most definitive health effects evidence is associated with short-term (i.e., 30-minute to 1-hour, or one to several day) exposures, the air quality analysis required the development of a model that relates annual average and short-term levels of NO₂. To characterize this relationship and to estimate the number of exceedances of the potential health effect benchmarks in specific locations, several possible models were explored (i.e., exponential regression, logistic regression, a regression assuming a Poisson distribution, and an empirical model). An empirical model, employing the annual average and hourly concentrations, was chosen to avoid some of the difficulties in extrapolating outside the range of the data. A detailed discussion justifying the selection of this approach is provided in Appendix D of the draft TSD.

A total of four air quality scenarios were evaluated using the empirical model for each of two distinct ambient monitoring periods, resulting in a total of eight separate analyses. The available NO₂ air quality were divided into two groups; one contained data from years 1995-2000, representing an *historical* data set; the other contained the monitoring years 2001-2006, representing *recent* ambient monitoring. Each of these monitoring year-groups were evaluated considering the NO₂ concentrations as they were reported and representing the conditions at that time (termed in this assessment “*as is*”). This served as the first air quality scenario. The second scenario considered the ambient NO₂ concentrations simulated to just meeting the current standard of 0.053 ppm annual average. The 3rd and 4th scenarios followed in similar fashion, however these scenarios used the ambient monitoring data to estimate NO₂ concentrations that might occur on roadways to generate on-road concentrations for *as is* air quality and for ambient concentrations just meeting the current standard. Again, each of these four scenarios was evaluated using both the historical and recent data air quality data sets.

1 Since all of the NO₂ ambient monitoring sites are represented by this analysis, the
2 generated results are considered a broad characterization of national air quality and human
3 exposures that might be associated with these concentrations. The output of this air quality
4 characterization was used to estimate the number of times per year specific locations experience
5 levels of NO₂ that could cause adverse health effects in susceptible individuals. Each location
6 that was evaluated contained one to several monitors operating for a few to several years,
7 generating a number of site-years of data. The number of site-years in a location were used to
8 generate a distribution of two exposure and risk characterization metrics; the annual average
9 concentrations and the numbers of exceedances that did (observed data) or could occur
10 (simulated data) in a year for that location. The mean and median values were reported to
11 represent the central tendency of each metric for the four scenarios in each air quality year-
12 group, while the minimum value served to represent the lower bound. Since there were either
13 multiple site-years or numerous simulations performed at each location using all available site-
14 years of data, results for the upper percentiles included the 95th, 98th and 99th percentiles of the
15 distribution.

16 **6.2 APPROACH**

17 There were three broad steps to allow for the characterization of the air quality. The first
18 step involved collecting, compiling, and screening the ambient air quality data collected since the
19 prior review in 1995. A screening of the data followed to ensure consistency with the NO₂
20 NAAQS requirements. Then, criteria based on the current standard and the potential health
21 effect benchmark levels were used to identify specific locations for analysis using descriptive
22 statistical analysis of the screened data set. All other monitoring data not identified by the
23 selected criteria were grouped into one of two non-specific categories. These locations (both the
24 specific and non-specific) served as the geographic centers of the analysis, where application of
25 the empirical model was done to estimate concentrations and exceedances of potential health
26 effect benchmark levels. In addition to use of the ambient concentrations (*as is*), and ambient
27 concentrations just meeting the current standard, on-road concentrations were estimated in this
28 air quality characterization to approximate the potential exposure and risk metrics associated
29 with these concentrations.

6.2.1 Air Quality Data Screen

NO₂ air quality data and associated documentation from the years 1995 through 2006 were downloaded from EPA's Air Quality System (AQS) for this purpose (EPA, 2007c, d). A *site* was defined by the state, county, site code, and parameter occurrence code (POC), which gives a 10-digit monitor ID code. As required by the NO₂ NAAQS, a valid year of monitoring data is needed to calculate the annual average concentration. A valid year at a monitoring site was comprised of 75% of valid days in a year, with at least 18 hourly measurements for a valid day (thus at least 274 or 275 valid days depending on presence of a leap year and a minimum of 4,932 or 4,950 hours). This served as the screening criterion for data used in the analysis.

Site-years of data are the total numbers of years the collective monitors in a location were in operation. Of a total of 5,243 site-years of data in the entire NO₂ 1-hour concentration database, 1,039 site-years did not meet the above criterion and were excluded from any further analyses. In addition, since shorter term average concentrations are of interest, the remaining site-years of data were further screened for 75% completeness on hourly measures in a year (i.e., containing a minimum of 6,570 or 6,588, depending on presence of a leap year). Twenty-seven additional site-years were excluded, resulting in 4,177 complete site-years in the analytical database. Table 2 provides a summary of the site-years included in the analysis, relative to those excluded, by location and by two site-year groups.² The air quality data from AQS were separated into these two groups, one representing historic data (1995-2000) and the other representing more recent data (2001-2006) to represent temporal variability in NO₂ concentrations within each location. The selection of locations was a companion analysis to the screening, however, it is discussed in a separate section.

² 14 of 18 named locations and the 2 grouped locations contained enough data to be considered valid for year 2006.

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Table 2. Counts of complete site-years of NO₂ monitoring data.

Location	Number of Site-Years				Site-Years % Complete	
	Complete		Incomplete		1995-2000	2001-2006
	1995-2000	2001-2006	1995-2000	2001-2006		
Boston	58	47	16	34	78%	58%
Chicago	47	36	20	22	70%	62%
Cleveland	11	11	2	2	85%	85%
Denver	26	10	10	4	72%	71%
Detroit	12	12	4	1	75%	92%
Los Angeles	193	177	16	19	92%	90%
Miami	24	20	1	4	96%	83%
New York	93	81	12	24	89%	77%
Philadelphia	46	39	6	8	88%	83%
Washington	69	66	21	18	77%	79%
Atlanta	24	29	5	1	83%	97%
Colorado Springs	26	0	4	4	87%	0%
El Paso	14	30	11	0	56%	100%
Jacksonville	6	4	0	2	100%	67%
Las Vegas	16	35	4	9	80%	80%
Phoenix	22	27	8	25	73%	52%
Provo	6	6	0	0	100%	100%
St. Louis	56	43	3	9	95%	83%
Other CMSA	1135	1177	249	235	82%	83%
Not MSA	200	243	112	141	64%	63%
Total	4177		1066		80%	

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6.2.2 Selection of Locations for Air Quality Analysis

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Criteria were established for selecting sites with high annual means and/or frequent exceedances of potential health effect benchmarks. Selected locations were those that had a maximum annual mean NO₂ level at a particular monitor greater than or equal to 25.7 ppb, which represents the 90th percentile across all locations and site-years, and/or had at least one reported 1-hour NO₂ level greater than or equal to 200 ppb, the lowest level of the potential health effect benchmarks. A *location* in this context would include a geographic area that encompasses more than a single air quality monitor (e.g., particular city, metropolitan statistical area (MSA), or consolidated metropolitan statistical area or CMSA). First, all monitors were identified as either belonging to a CMSA, a MSA, or neither. Then, locations of interest were identified through statistical analysis of the ambient NO₂ air quality data for each site within a location.

1 Fourteen locations met both selection criteria and an additional four met at least one of
2 the criteria (see Table 3).³ In addition to these 18 specific locations, the remaining sites were
3 grouped into two broad location groupings. The *Other CMSA* location contains all the other sites
4 that are in MSAs or CMSAs but are not in any of the 18 specified locations. The *Not MSA*
5 location contains all the sites that are not in an MSA or CMSA. The final database for analysis
6 included air quality data from a total of 205 monitors within the named locations, 331 monitors
7 in the Other CMSA group, and 92 monitors in the Not MSA group.

8 **6.2.3 Estimation of On-Road Concentrations using Ambient Concentrations**

9 Since mobile sources can account for a large part of personal exposures to ambient NO₂
10 in some individuals, the potential impact of roadway levels of NO₂ was evaluated. A strong
11 relationship has been reported between NO₂ levels measured on roadways and NO₂ measured at
12 increasing distance from the road. This relationship has been described previously (e.g., Cape et
13 al., 2004) using an exponential decay equation of the form:

14

$$15 \quad C_x = C_b + C_v e^{-kx} \quad \text{eq (2)}$$

16 where,

17

18 C_x = NO₂ concentration at a given distance (x) from a roadway (ppb)

19 C_b = NO₂ concentration (ppb) at a distance from a roadway, not directly influenced
20 by road or non-road source emissions.

21 C_v = NO₂ concentration contribution from vehicles on a roadway (ppb)

22 k = Rate constant describing NO₂ combined formation/decay with perpendicular
23 distance from roadway (meters⁻¹)

24 x = Distance from roadway (meters)

³ New Haven, CT, while meeting both criteria, did not have any recent exceedances of 200 ppb and contained one of the lowest maximum concentration-to-mean ratios, therefore was not separated out as a specific location for analysis.

1 **Table 3. Locations selected for Tier I NO₂ Air Quality Characterization, associated**
 2 **abbreviations, and values of selection criteria.**
 3

Location				Maximum # of Exceedances of 200 ppb	Maximum Annual Mean (ppb)
Type ¹	Code	Description	Abbreviation		
CMSA*	1122	Boston-Worcester-Lawrence, MA-NH-ME-CT	Boston	1	31.1
CMSA	1602	Chicago-Gary-Kenosha, IL-IN-WI	Chicago	0	33.6
CMSA*	1692	Cleveland-Akron, OH	Cleveland	1	28.1
CMSA*	2082	Denver-Boulder-Greeley, CO	Denver	2	36.8
CMSA*	2162	Detroit-Ann Arbor-Flint, MI	Detroit	12	25.9
CMSA*	4472	Los Angeles-Riverside-Orange County, CA	Los Angeles	5	50.6
CMSA	4992	Miami-Fort Lauderdale, FL	Miami	3	16.8
CMSA*	5602	New York-Northern New Jersey-Long Island, NY-NJ-CT-PA	New York	3	42.2
CMSA*	6162	Philadelphia-Wilmington-Atlantic City, PA-NJ-DE-MD	Philadelphia	3	34.0
CMSA*	8872	Washington-Baltimore, DC-MD-VA-WV	Washington DC	2	27.2
MSA*	0520	Atlanta,GA	Atlanta	1	26.6
MSA*	1720	Colorado Springs,CO	Colorado Springs	69	34.8
MSA*	2320	El Paso,TX	El Paso	2	35.1
MSA	3600	Jacksonville,FL	Jacksonville	2	15.9
MSA*	4120	Las Vegas,NV-AZ	Las Vegas	11	27.1
MSA*	6200	Phoenix-Mesa,AZ	Phoenix	37	40.5
MSA	6520	Provo-Orem,UT	Provo	0	28.9
MSA*	7040	St. Louis,MO-IL	St. Louis	8	27.2
MSA/CMSA	-	Other MSA/CMSA	Other CMSA	10	31.9
-	-	Other Not MSA	Not MSA	2	19.7

¹ CMSA is consolidated metropolitan statistical area; MSA is metropolitan statistical area according to the 1999 Office of Management and Budget definitions (January 28, 2002 revision).

* Indicates locations that satisfied both the annual average and exceedance criteria.

4
 5
 6 Much of the decline in NO₂ concentrations with distance from the road has been shown
 7 to occur within the first few meters (approximately 90% within 10 meter distance), returning to
 8 near ambient levels between 200 to 500 meters (Rodes and Holland, 1981; Bell and Ashenden,
 9 1997; Gilbert et al., 2003; Pleijel et al., 2004). Theoretically, NO₂ concentrations can increase at
 10 a distance from the road due to chemical interaction of NO_x with O₃, the magnitude of which can
 11 be driven by certain meteorological conditions (e.g., wind direction). However, this relationship

1 developed from NO₂ measurement studies was used to estimate NO₂ concentrations that occur
2 on the roadway and not used to estimate NO₂ concentrations at a distance from the road. At a
3 distance of 0 meters, referred to here as *on-road*, the equation reduces to the sum of the non-
4 source influenced NO₂ concentration and the concentration contribution expected from vehicle
5 emissions on the roadway using

$$C_r = C_a (1 + m) \quad \text{eq (2)}$$

8 where,

10 C_r = 1-hour on-road NO₂ concentration (ppb)

11 C_a = 1-hour ambient monitoring NO₂ concentration (ppb) either *as is* or modified
12 to just meet the current standard

13 m = Modification factor derived from estimates of C_v/C_b (from eq (1))

15 and assuming that $C_a = C_b$.⁴

17 To estimate on-roadway NO₂ levels as a function of the level recorded at ambient
18 monitors and the distance of those monitors from a roadway, empirical data from the scientific
19 literature have been used. A literature review was conducted to identify published studies
20 containing NO₂ concentrations on roadways and at varying distances from roadways (see section
21 2.6.1 of the draft TSD for more detail). Ratios identified from this literature review were used to
22 estimate m empirically (draft TSD, section 2.6.2). To estimate NO₂ levels on roadways, each
23 monitor site was randomly assigned one on-road factor (m) for summer months and one for non-
24 summer months from the derived empirical distribution. On-road factors were assigned
25 randomly because we expect the empirical relationship between C_v and C_b to vary from place to
26 place and we do not have sufficient information to match specific ratios with specific locations.
27 Hourly NO₂ levels were estimated for each site-year of data in a location using eq (2) and the
28 randomly assigned on-road factors. The process was simulated 100 times for each site-year of
29 hourly data. For example, the Boston CMSA location had 210 random selections from the on-
30 road distributions applied independently to the total site-years of data (105). Following 100

⁴ Note that C_a differs from C_b since C_a may include the influence of on-road as well as non-road sources. However, it is expected that for most monitors the influence of on-road emissions is minimal so that $C_a \cong C_b$.

1 simulations, a total of 10,500 site-years of data were generated using this procedure (along with
2 21,000 randomly assigned on-road values selected from the appropriate empirical distribution).

3 Simulated on-road NO₂ concentrations were then used to generate concentration
4 distributions for the annual average concentrations and distributions for the number of
5 exceedances of short-term health potential health effect benchmark levels. Mean and median
6 values are reported to represent the central tendency of each parameter estimate. Since there
7 were multiple site-years and numerous simulations performed at each location using all valid
8 site-years of data, results for the upper percentiles were expanded to the 95th, 98th and 99th
9 percentiles of the distribution. In using the Boston CMSA data as an example, 4700 site years of
10 on-road concentration hourly data were simulated, and both the annual average concentration
11 and numbers of exceedances of potential health effect benchmark levels were calculated. The
12 95th, 98th and 99th percentiles were the 4465th, the 4606th, and the 4653th highest values,
13 respectively, of the 4700 calculated and ranked values. Roadways with high vehicle densities are
14 likely better represented by on-road concentration estimates at the upper tails of the distribution.

15 **6.3 AIR QUALITY AND HEALTH RISK CHARACTERIZATION**
16 **RESULTS**

17 **6.3.1 Ambient Air Quality (As Is)**

18 As described earlier, the air quality data obtained from AQS were separated into two
19 groups, one representing historic data (1995-2000) and the other representing more recent data
20 (2001-2006). A summary of the descriptive statistics for ambient NO₂ concentrations at each
21 selected location is provided in Table 4. Detailed descriptive statistics regarding concentration
22 distributions for particular locations and specific monitoring years are provided in the draft TSD
23 (section 2.4 and Appendices B and C). None of the locations contained an exceedance of the
24 current standard of 0.053 ppm. The highest observed annual average concentrations were
25 measured in Los Angeles, New York, and Phoenix during the historic monitoring period,
26 however as with most of the locations, recent concentrations are lower across all percentiles of
27 the distribution.

28 The estimated number of exceedances of the three potential health effect benchmark
29 levels (200, 250, and 300 ppb NO₂ for 1-hr) is shown in Tables 5 and 6. The exceedances of
30 each benchmark were totaled for the year at each monitor; a monitor value of 10 could represent

1 ten 1-hr exceedances that occurred in one day, 10 exceedances in 10 days, or some combination
2 of multiple hours or days that totaled 10 exceedances for the year. In general, the number of
3 benchmark exceedances was low across all locations. The average number of exceedances of the
4 lowest potential health effect benchmark level across each location was typically none or one.
5 Considering that there are 8760 hours in a year, this amounts to small fraction of the year
6 (0.01%) containing an exceedance. For locations predicted to have a larger number of yearly
7 average exceedances, estimates were primarily driven by a single site-year of data. For example,
8 the Colorado Springs mean estimate is 3 exceedances per year for the years 1995-2000; however,
9 this mean was driven by a single site-year that contained 69 exceedances of 200 ppb. That
10 particular monitor (ID 0804160181) does not appear to have any unusual attributes (e.g., the
11 closest major road is beyond a distance of 160 meters and the closest stationary source emitting >
12 5 tons per year (tpy) is over 4 km away) except that a power generating utility (NAICS code
13 221112) located 7.2 km from the monitor has estimated emissions of 4205 tpy. It is not known
14 at this time whether this particular facility is influencing the observed concentration exceedances
15 at this specific monitoring site. Similarly, in Phoenix a single year from one monitor (ID
16 0401330031) was responsible for all observed exceedances of 200 ppb. This monitor is located
17 78 m from the roadway and 9 of 10 stationary sources located within 10 km of this monitor
18 emitted less than 60 tpy (one emitted 272 tpy). It is not known if observed exceedances of 200
19 ppb at this monitor are a result of proximity of major roads or stationary sources. Detroit
20 contained the largest number of exceedances of 200 ppb (a maximum of 12) for air quality data
21 from years 2001-2006 (Table 6). Again, all of those exceedances occurred at one monitor (ID
22 2616300192) during one year (2002). The number of exceedances of higher potential
23 benchmark concentration levels was less than for 200 ppb. Most locations had no exceedances
24 of 250 or 300 ppb, with higher numbers confined to the same aforementioned cities where
25 exceedances of 200 ppb was observed.

1 **Table 4. Monitoring site-years and annual average NO₂ concentrations for two monitoring periods, historic and recent air**
 2 **quality data (as is).**
 3

Location	1995-2000							2001-2006						
	Site-Years	Annual Mean (ppb) ¹						Site-Years	Annual Mean (ppb) ¹					
	mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99	
Boston	58	18	5	19	31	31	31	47	15	5	13	25	30	30
Chicago	47	24	9	24	32	34	34	36	24	16	23	32	32	32
Cleveland	11	23	17	23	28	28	28	11	19	14	19	24	24	24
Denver	26	16	6	9	35	35	35	10	26	18	27	37	37	37
Detroit	12	19	12	19	26	26	26	12	19	14	19	23	23	23
Los Angeles	193	26	4	26	45	46	46	177	22	4	22	36	37	40
Miami	24	10	6	9	17	17	17	20	9	6	8	15	16	16
New York	93	27	11	27	41	42	42	81	23	10	24	36	40	40
Philadelphia	46	23	15	21	33	34	34	39	20	14	19	29	30	30
Washington	69	20	9	22	26	27	27	66	18	7	19	25	26	26
Atlanta	24	14	5	15	25	27	27	29	12	3	14	19	23	23
Colorado Springs ²	26	16	7	17	24	35	35	0	-	-	-	-	-	-
El Paso	14	23	14	23	35	35	35	30	15	8	16	21	22	22
Jacksonville	6	15	14	15	16	16	16	4	14	13	14	15	15	15
Las Vegas	16	14	3	8	27	27	27	35	11	1	9	22	23	23
Phoenix	22	30	24	30	36	40	40	27	25	11	24	35	37	37
Provo	6	24	23	24	24	24	24	6	24	21	23	29	29	29
St. Louis	56	18	5	19	26	26	27	43	15	8	15	22	25	25
Other CMSA	1135	14	1	14	24	26	28	1177	12	1	12	20	22	24
Not MSA	200	8	0	7	16	19	19	243	7	1	6	14	16	16

¹ The mean is the sum of the annual means for each monitor in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the annual mean.

² Colorado Springs monitoring data were collected as part of short-term study completed in September 2001, therefore there are no 2001-2006 data.

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1 **Table 5. Number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year, 1995-2000 historic**
 2 **NO₂ air quality (as is).**
 3

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	0	0	0	1	1	1	0	0	0	1	1	1	0	0	0	0	0	0
Denver	0	0	0	1	2	2	0	0	0	0	1	1	0	0	0	0	0	0
Detroit	0	0	0	3	3	3	0	0	0	1	1	1	0	0	0	1	1	1
Los Angeles	0	0	0	1	2	4	0	0	0	0	0	2	0	0	0	0	0	0
Miami	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
New York	0	0	0	0	1	3	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	0	0	0	0	3	3	0	0	0	0	0	0	0	0	0	0	0	0
Washington	0	0	0	0	1	2	0	0	0	0	1	1	0	0	0	0	0	0
Atlanta	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
Colorado Springs	3	0	0	3	69	69	1	0	0	0	23	23	0	0	0	0	4	4
El Paso	0	0	0	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Las Vegas	1	0	0	11	11	11	0	0	0	3	3	3	0	0	0	3	3	3
Phoenix	2	0	0	0	37	37	0	0	0	0	3	3	0	0	0	0	0	0
Provo	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
St. Louis	0	0	0	0	0	8	0	0	0	0	0	4	0	0	0	0	0	0
Other CMSA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Not MSA	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

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1 **Table 6. Number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year, 2001-2006 recent**
 2 **NO₂ air quality (as is).**
 3

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Denver	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Detroit	1	0	0	12	12	12	1	0	0	8	8	8	0	0	0	5	5	5
Los Angeles	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0
Miami	0	0	0	2	3	3	0	0	0	2	3	3	0	0	0	2	3	3
New York	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	0	0	0	0	1	1	0	0	0	0	1	1	0	0	0	0	0	0
Washington	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Atlanta	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
El Paso	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	1	0	1	2	2	2	0	0	0	1	1	1	0	0	0	0	0	0
Las Vegas	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Phoenix	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Provo	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
St. Louis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other CMSA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Not MSA	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	0

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

4
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6.3.2 Ambient Air Quality Adjusted to Just Meet the Current Standard

Table 7 presents descriptive statistics for the ambient NO₂ levels in each location after applying an air quality adjustment procedure that rolls-up NO₂ concentrations to simulate just meeting the current annual standard. Note that the 99th percentile annual average level for all locations is 53 ppb for these simulations, except for the other CMSA location. This is a direct consequence of the air quality adjustment procedure that sets the highest monitor in a location to the current standard with the other monitors adjusted proportionally, and the number of site-years available for each location. Mean and median values are similar when comparing the historic annual average concentrations with the more recent estimates following the air quality adjustment procedure with one exception, Denver. This is probably because the mean of Denver's annual average ambient concentrations (as is) was also higher for the more recent air quality analysis period (26 ppb) versus the historic data set (16 ppb, see Table 4). This suggests that the air quality adjustment procedure affected the two sets of data comparably.

As expected, the number of estimated potential benchmark exceedances is greater when air quality is modified to just meet the current standard than air quality levels *as is* (compare Tables 8 and 9 to Tables 5 and 6). The cities with the largest estimated number of potential benchmark exceedances are the same as those predicted to have largest number of exceedances in the "*as-is*" scenario (i.e., Colorado Springs, Detroit, Phoenix). The rationale explaining these results is also the same. That is, the results are due to the influence of a single monitor within their respective monitoring network. Miami and Jacksonville are also predicted to have a relatively large number of exceedances. This result is most likely due to the small network size in these locations (n=1 for Jacksonville, n=5 for Miami). Having few ambient monitors in a given location could bias the mean estimate in either direction, but most likely biases estimates high in these locations because of the unusually large number of peak concentrations in one or more years (see draft TSD section 2.4 and Appendices B and C). In addition, Miami contained some of the lowest annual average concentrations which results in high air quality simulation factors across all years of data. That factor, coupled with a high coefficient of variance (COV) (~130%) for hourly concentrations at two of the monitors in Miami (IDs 1201180021, 1208600271) clearly played a significant role in the higher estimated number of exceedances (see draft TSD section 2.4 and Appendices B and C). Denver also contained a high COV

1 (~110%) for hourly concentrations using the earlier air quality period (1995-2000), likely
2 associated with the higher estimate of exceedances at this location (99th percentile of 141)
3 following the air quality adjustment procedure compared with only 2 observed exceedances
4 when considering the “*as-is*” air quality. Both the mean and maximum estimate of exceedances
5 for Provo (ID 4904900021) during 2001-2006 were also likely influenced by the small network
6 size (n=1) in this location and one particular year (2006) that contained numerous concentrations
7 above 150 ppb prior to the concentration roll-up.

8 **6.3.3 On-Road Concentrations Derived From Ambient Air Quality (As Is)**

9 Descriptive statistics for estimated on-road NO₂ levels are presented in Table 10. These
10 estimated on-road levels were generated using the simulation procedure described above (section
11 5.2.3). The simulated on-road annual average concentrations are, on average, a factor of 1.8
12 higher than their respective ambient levels. This falls within the range of ratios reported in the
13 draft ISA (about 2-fold higher concentrations on roads) (draft ISA, section 2.5.4). Los Angeles,
14 New York, Phoenix, and Denver (recent data only for this location) are predicted to have the
15 highest on-road NO₂ levels. This is a direct result of these locations already containing the
16 highest “*as-is*” levels prior to the on-road simulation.

17 The median of the simulated concentration estimates for Los Angeles were compared
18 with NO₂ measurements provided by Westerdahl et al. (2005) for arterial roads and freeways in
19 the same general location during spring 2003. Although the averaging time is not the same,
20 comparison of the medians is judged to be appropriate.⁵ The estimated median on-road level for
21 2001-2006 is 41 ppb which falls within the range of 31 ppb to 55 ppb identified by Westerdahl et
22 al. (2005).

23 On average, most locations are predicted to have fewer than 10 exceedances per year for
24 the 200 ppb potential health effect benchmark while the median frequency of exceedances in
25 most locations is estimated to be 1 or less per year (Tables 11 and 12). There are generally fewer

⁵Table 10 considers annual average of hourly measurements while Westerdahl et al. (2005) reported between 2 to 4 hour average concentrations. Over time, the mean of 2-4 hour averages will be similar to the mean of hourly concentrations, with the main difference being in the variability (and hence the various percentiles of the distribution outside the central tendency).

1 **Table 7. Estimated annual average NO₂ concentrations for two monitoring periods, historic and recent air quality data**
 2 **adjusted to just meet the current standard (0.053 ppm annual average).**
 3

Location	1995-2000							2001-2006						
	Site-Years	Annual Mean (ppb) ¹						Site-Years	Annual Mean (ppb) ¹					
	mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99	
Boston	58	32	10	33	53	53	53	47	32	11	28	53	53	53
Chicago	47	39	15	40	53	53	53	36	41	27	39	53	53	53
Cleveland	11	47	37	53	53	53	53	11	48	41	53	53	53	53
Denver	26	29	10	29	53	53	53	10	47	33	53	53	53	53
Detroit	12	45	26	51	53	53	53	12	49	42	50	53	53	53
Los Angeles	193	31	4	32	52	53	53	177	33	5	33	53	53	53
Miami	24	34	19	31	53	53	53	20	35	19	32	53	53	53
New York	93	35	14	35	53	53	53	81	35	15	35	53	53	53
Philadelphia	46	39	25	35	53	53	53	39	41	26	40	53	53	53
Washington	69	42	20	45	53	53	53	66	40	19	44	53	53	53
Atlanta	24	32	11	31	53	53	53	29	34	9	40	53	53	53
Colorado Springs ²	26	38	14	45	53	53	53	0	-	-	-	-	-	-
El Paso	14	43	30	40	53	53	53	30	42	24	43	53	53	53
Jacksonville	6	53	53	53	53	53	53	4	53	53	53	53	53	53
Las Vegas	16	29	7	17	53	53	53	35	28	4	21	53	53	53
Phoenix	22	45	36	44	53	53	53	27	40	19	40	53	53	53
Provo	6	53	53	53	53	53	53	6	53	53	53	53	53	53
St. Louis	56	37	11	39	53	53	53	43	38	19	38	53	53	53
Other CMSA	1135	26	1	26	43	48	50	1177	25	1	26	43	48	51
Not MSA	200	22	1	20	51	53	53	243	22	3	20	46	53	53

¹ The mean is the sum of the annual means for each monitor in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the annual mean.

² Colorado Springs monitoring data were collected as part of short-term study completed in September 2001, therefore there are no 2001-2006 data.

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Table 8. Estimated number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year, 1995-2000 NO₂ air quality adjusted to just meet the current standard (0.053 ppm annual average).

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	0	0	0	1	1	2	0	0	0	0	1	1	0	0	0	0	0	1
Chicago	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	3	0	0	24	24	24	1	0	0	10	10	10	0	0	0	3	3	3
Denver	8	0	0	19	141	141	2	0	0	5	28	28	1	0	0	4	9	9
Detroit	13	0	13	25	25	25	4	0	2	15	15	15	2	0	1	10	10	10
Los Angeles	1	0	0	5	8	9	0	0	0	0	2	2	0	0	0	0	0	2
Miami	10	0	8	27	34	34	2	0	0	6	15	15	1	0	0	2	8	8
New York	0	0	0	1	2	3	0	0	0	0	1	3	0	0	0	0	0	1
Philadelphia	0	0	0	1	12	12	0	0	0	0	9	9	0	0	0	0	5	5
Washington	1	0	0	4	9	17	0	0	0	1	3	3	0	0	0	1	2	2
Atlanta	4	0	0	19	21	21	0	0	0	2	3	3	0	0	0	1	1	1
Colorado Springs	30	0	0	180	241	241	15	0	0	123	135	135	8	0	0	72	83	83
El Paso	4	0	1	14	14	14	1	0	0	6	6	6	0	0	0	2	2	2
Jacksonville	12	2	15	20	20	20	2	0	1	7	7	7	0	0	0	1	1	1
Las Vegas	3	0	0	28	28	28	1	0	0	13	13	13	1	0	0	11	11	11
Phoenix	12	0	0	57	198	198	4	0	0	4	92	92	1	0	0	0	31	31
Provo	1	0	0	5	5	5	0	0	0	0	0	0	0	0	0	0	0	0
St. Louis	0	0	0	1	1	15	0	0	0	0	0	14	0	0	0	0	0	13
Other CMSA	0	0	0	1	3	6	0	0	0	0	1	1	0	0	0	0	0	1
Not MSA	4	0	0	18	53	87	1	0	0	4	15	42	1	0	0	1	8	21

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

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Table 9. Estimated number of exceedances of short-term (1-hour) health effect benchmark levels in a year, 2001-2006 NO₂ air quality adjusted to just meet the current standard (0.053 ppm annual average).

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	0	0	0	1	5	5	0	0	0	0	1	1	0	0	0	0	0	0
Chicago	1	0	0	2	15	15	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	1	0	1	4	4	4	0	0	0	1	1	1	0	0	0	1	1	1
Denver	2	0	1	7	7	7	0	0	0	2	2	2	0	0	0	1	1	1
Detroit	8	0	1	45	45	45	4	0	0	34	34	34	3	0	0	28	28	28
Los Angeles	0	0	0	1	5	6	0	0	0	0	0	1	0	0	0	0	0	1
Miami	17	0	11	66	69	69	3	0	0	18	23	23	1	0	0	11	19	19
New York	0	0	0	1	2	5	0	0	0	0	1	1	0	0	0	0	0	0
Philadelphia	1	0	0	2	25	25	0	0	0	1	7	7	0	0	0	0	1	1
Washington	0	0	0	2	5	6	0	0	0	1	1	2	0	0	0	0	1	1
Atlanta	8	0	0	48	56	56	1	0	0	9	10	10	0	0	0	2	5	5
El Paso	7	0	6	24	27	27	1	0	0	3	6	6	0	0	0	0	1	1
Jacksonville	31	7	22	72	72	72	15	1	7	46	46	46	7	0	1	25	25	25
Las Vegas	1	0	0	3	12	12	0	0	0	0	2	2	0	0	0	0	0	0
Phoenix	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
Provo	88	0	0	526	526	526	34	0	0	205	205	205	0	0	0	1	1	1
St. Louis	0	0	0	2	5	5	0	0	0	1	1	1	0	0	0	0	1	1
Other CMSA	0	0	0	1	3	5	0	0	0	0	1	2	0	0	0	0	0	1
Not MSA	3	0	0	17	44	57	1	0	0	4	14	20	1	0	0	2	8	9

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

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1 **Table 10. Estimated annual average on-road concentrations for two monitoring periods, historic and recent ambient air**
 2 **quality (as is).**
 3

Location	1995-2000							2001-2006						
	Site-Years	Annual Mean NO ₂ (ppb) ¹						Site-Years	Annual Mean NO ₂ (ppb) ¹					
	mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99	
Boston	5800	33	7	33	59	67	71	4700	27	7	25	51	57	60
Chicago	4700	44	11	44	68	75	79	3600	43	20	42	66	72	76
Cleveland	1100	42	22	41	61	65	67	1100	36	18	35	51	54	58
Denver	2600	29	8	19	67	78	81	1000	48	23	46	74	83	87
Detroit	1200	35	15	34	52	57	59	1200	34	18	34	47	52	54
Los Angeles	19300	48	5	47	87	97	104	17700	41	5	40	71	80	85
Miami	2400	19	7	17	33	38	39	2000	17	7	15	30	33	36
New York	9300	50	14	49	81	91	96	8100	43	12	41	70	79	85
Philadelphia	4600	43	19	40	68	76	80	3900	37	18	34	57	63	68
Washington	6900	37	12	38	56	61	64	6600	33	9	33	52	57	61
Atlanta	2400	26	6	25	49	57	60	2900	21	4	23	40	43	47
Colorado Springs	2600	30	9	30	51	64	73	-	-	-	-	-	-	-
El Paso	1400	42	17	40	67	75	82	3000	27	10	27	42	45	48
Jacksonville	600	28	18	27	37	39	41	400	25	17	25	34	36	37
Las Vegas	1600	26	4	16	56	62	63	3500	20	2	15	45	50	53
Phoenix	2200	54	30	52	76	83	88	2700	45	14	43	70	79	84
Provo	600	43	29	42	58	62	64	600	43	26	41	61	69	70
St. Louis	5600	33	7	33	51	58	61	4300	27	10	27	44	49	52
Other CMSA	113500	26	1	25	47	53	57	117700	21	1	21	39	45	48
Not MSA	20000	14	0	12	31	35	39	24300	12	1	11	27	31	33

¹ The mean is the sum of the annual means for each monitor in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the annual mean.

1 **Table 11. Estimated number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year on-**
 2 **roads, 1995-2000 historic NO₂ air quality (as is).**
 3

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	3	0	0	14	37	54	1	0	0	2	10	15	0	0	0	0	1	3
Chicago	12	0	0	79	142	183	2	0	0	15	31	53	0	0	0	2	6	10
Cleveland	10	0	0	74	108	129	2	0	0	12	30	49	1	0	0	1	10	17
Denver	7	0	0	41	94	102	2	0	0	9	17	33	1	0	0	4	6	7
Detroit	10	0	2	48	72	86	4	0	1	21	34	35	2	0	0	14	21	26
Los Angeles	45	0	4	236	417	550	13	0	0	71	146	211	4	0	0	21	48	78
Miami	0	0	0	4	6	8	0	0	0	1	4	6	0	0	0	0	3	4
New York	20	0	1	109	230	384	5	0	0	28	65	129	1	0	0	5	14	31
Philadelphia	5	0	0	31	60	84	1	0	0	4	11	15	0	0	0	1	4	7
Washington	4	0	0	23	43	58	0	0	0	3	7	11	0	0	0	1	2	2
Atlanta	4	0	0	31	57	87	1	0	0	3	11	21	0	0	0	1	1	2
Colorado Springs	20	0	0	170	264	320	11	0	0	106	181	216	6	0	0	47	119	159
El Paso	7	0	2	33	58	76	2	0	0	9	19	30	1	0	0	5	7	11
Jacksonville	0	0	0	1	2	4	0	0	0	0	1	1	0	0	0	0	0	0
Las Vegas	6	0	0	37	66	97	1	0	0	11	15	19	1	0	0	6	11	11
Phoenix	36	0	3	256	319	390	14	0	0	107	200	280	7	0	0	26	103	181
Provo	2	0	0	9	33	34	0	0	0	0	1	4	0	0	0	0	0	0
St. Louis	2	0	0	14	25	35	0	0	0	1	8	12	0	0	0	0	4	10
Other CMSA	1	0	0	6	18	32	0	0	0	1	3	6	0	0	0	0	1	2
Not MSA	1	0	0	2	7	14	0	0	0	1	2	4	0	0	0	0	1	2

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

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Table 12. Estimated number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year on-roads, 2001-2006 historic NO₂ air quality (as is).

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	1	0	0	2	8	17	0	0	0	0	1	4	0	0	0	0	0	0
Chicago	10	0	0	50	142	188	2	0	0	11	29	44	0	0	0	1	6	8
Cleveland	3	0	0	21	36	42	1	0	0	4	7	9	0	0	0	1	3	3
Denver	8	0	1	39	69	82	2	0	0	8	15	20	0	0	0	1	7	7
Detroit	5	0	0	29	44	45	2	0	0	16	22	28	1	0	0	13	14	21
Los Angeles	11	0	0	70	131	183	2	0	0	13	29	48	0	0	0	2	7	13
Miami	0	0	0	3	7	13	0	0	0	2	5	5	0	0	0	2	4	5
New York	9	0	0	48	90	143	2	0	0	8	19	25	0	0	0	1	3	6
Philadelphia	1	0	0	6	14	29	0	0	0	1	1	2	0	0	0	0	1	1
Washington	1	0	0	6	14	21	0	0	0	0	1	2	0	0	0	0	0	0
Atlanta	1	0	0	8	16	25	0	0	0	1	3	6	0	0	0	0	1	2
El Paso	1	0	0	6	9	15	0	0	0	1	1	2	0	0	0	0	0	0
Jacksonville	3	0	1	15	23	24	2	0	0	8	15	15	1	0	0	5	8	8
Las Vegas	1	0	0	6	15	23	0	0	0	0	1	3	0	0	0	0	0	0
Phoenix	3	0	0	21	44	61	0	0	0	2	5	7	0	0	0	0	0	0
Provo	70	0	0	547	662	662	33	0	0	234	606	612	13	0	0	3	423	435
St. Louis	1	0	0	2	7	14	0	0	0	0	1	2	0	0	0	0	0	1
Other CMSA	0	0	0	1	5	10	0	0	0	0	1	1	0	0	0	0	0	0
Not MSA	0	0	0	1	4	8	0	0	0	0	2	3	0	0	0	0	1	2

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

1 predicted exceedances of the potential health effect benchmark levels when considering recent
2 air quality compared with the historic air quality. Areas with a relatively high number of
3 exceedances (e.g., Provo) are likely influenced by the presence of a small number of monitors
4 and one or a few exceptional site-years where levels reached the upper percentiles.

5 The number of predicted benchmark exceedances across large urban areas may be used to
6 broadly represent particular locations within those types of areas. For example, Chicago, New
7 York, and Los Angeles are large CMSAs, have several monitoring sites, and have a large number
8 of roadways. Each of these locations was estimated to have, on average, about 10 exceedances
9 of 200 ppb per year on-roads. Assuming that the on-road exceedances distribution is
10 proportionally representing the distribution of roadways within each location, about one-half of
11 the roads in these areas would not have any on-road concentrations in excess of 200 ppb. This is
12 because the median value for exceedances of 200 ppb in most locations was estimated as zero.
13 However, Tables 11 and 12 indicate that there is also a possibility of tens to just over a hundred
14 exceedances in a year as an upper bound estimate on certain roads/sites.

15 **6.3.4 On-Road Concentrations Derived From Ambient Air Quality Adjusted to Just** 16 **Meet the Current Standard**

17 Table 13 presents descriptive statistics for estimated on-road NO₂ concentrations
18 assuming each location just meets the current 0.053 ppm annual standard. These on-road
19 concentrations were generated using the simulation procedure described above (see section
20 5.2.1.3) applied to air quality data that has been modified to simulate each location just meeting
21 the annual standard. On average, these simulated on-road annual average concentrations are
22 about 1.8 times higher than the accompanying ambient concentrations (Table 7). Tables 14 and
23 15 present estimates for the number of exceedances of the three selected potential health effect
24 benchmark levels (i.e., 200, 250, and 300 ppb NO₂ 1-hr).

25 The mean number of estimated exceedances of 200 ppb ranges from tens to several
26 hundreds (Tables 14 and 15), sharply increased from the previous on-road estimates using the air
27 quality (*as is*). Some of the highest exceedance estimates occurred in the locations described
28 previously as being influenced by a few concentrations at the upper percentiles of their
29 distributions in a small number of years and/or monitoring sites (e.g., Miami, Colorado Springs,
30 Provo). Compared to the means, median estimated exceedances of 200 ppb are lower, on

1 **Table 13. Estimated annual average on-road concentrations for two monitoring periods, air quality data adjusted to just meet**
 2 **the current standard (0.053 ppm annual average).**
 3

Location	1995-2000							2001-2006						
	Site-Years	Annual Mean (ppb) ¹						Site-Years	Annual Mean (ppb) ¹					
		mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99
Boston	5800	58	13	57	103	117	125	4700	58	14	53	105	120	126
Chicago	4700	72	18	72	112	123	130	3600	74	35	72	113	124	130
Cleveland	1100	86	47	84	123	128	136	1100	88	53	86	123	130	146
Denver	2600	53	12	49	112	124	129	1000	85	42	85	124	130	141
Detroit	1200	81	33	83	124	129	133	1200	90	54	87	123	129	134
Los Angeles	19300	56	6	55	102	114	122	17700	61	7	60	105	116	123
Miami	2400	62	24	56	111	124	128	2000	63	25	57	112	126	129
New York	9300	64	18	62	104	117	123	8100	63	18	61	103	119	125
Philadelphia	4600	71	31	67	111	123	128	3900	74	33	71	111	125	128
Washington	6900	77	26	77	116	124	130	6600	73	23	74	114	124	128
Atlanta	2400	57	14	55	111	126	129	2900	61	12	66	111	126	129
Colorado Springs	2600	69	18	73	118	127	131	-	-	-	-	-	-	-
El Paso	1400	77	38	74	122	129	138	3000	75	30	74	112	124	128
Jacksonville	600	96	67	95	128	131	144	400	96	67	94	129	139	145
Las Vegas	1600	53	8	34	113	125	130	3500	50	5	36	112	124	129
Phoenix	2200	82	46	78	115	127	129	2700	72	24	71	110	125	127
Provo	600	96	67	95	129	139	144	600	95	67	93	128	131	138
St. Louis	5600	68	14	68	106	118	124	4300	69	25	67	106	118	126
Other CMSA	113500	46	1	46	84	95	103	117700	46	1	45	84	95	102
Not MSA	20000	39	1	35	90	104	115	24300	39	3	35	89	101	109

¹ The mean is the sum of the annual means for each monitor in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the annual mean.

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1 **Table 14. Estimated number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year on-**
 2 **roads, 1995-2000 historic NO₂ air quality adjusted to just meet the current standard (0.053 ppm annual average).**
 3

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	78	0	13	411	677	790	23	0	1	131	257	334	8	0	0	43	106	131
Chicago	172	0	61	727	1001	1170	59	0	7	303	512	643	22	0	0	137	230	322
Cleveland	321	1	195	1045	1221	1439	124	0	38	566	663	761	51	0	5	304	380	392
Denver	214	0	23	1261	1921	2215	97	0	5	511	1142	1574	45	0	1	228	582	908
Detroit	405	2	284	1227	1439	1589	175	2	97	576	776	872	80	0	40	317	424	482
Los Angeles	100	0	18	489	791	927	33	0	2	173	318	432	12	0	0	62	127	184
Miami	363	1	260	1045	1334	1427	162	0	93	579	737	791	72	0	32	316	396	430
New York	77	0	11	412	693	930	23	0	1	127	258	420	8	0	0	40	91	171
Philadelphia	114	0	27	570	797	942	32	0	4	181	308	364	9	0	0	52	104	138
Washington	219	0	101	852	1070	1185	73	0	18	351	457	525	27	0	2	158	220	270
Atlanta	251	0	42	1094	1472	1640	106	0	7	535	843	947	45	0	1	277	435	514
Colorado Springs	304	0	77	1320	1756	1879	120	0	11	565	769	930	60	0	1	294	371	416
El Paso	178	0	82	692	951	1105	57	0	24	215	347	447	21	0	8	78	162	200
Jacksonville	610	40	549	1426	1515	1801	263	2	195	773	839	1002	114	0	66	407	443	470
Las Vegas	238	0	26	1107	1674	1882	89	0	5	574	688	860	36	0	1	280	369	422
Phoenix	250	0	105	953	1326	1435	83	0	17	379	466	563	33	0	3	181	296	364
Provo	443	1	230	1643	1871	2058	135	0	32	543	697	817	43	0	2	208	303	339
St. Louis	148	0	48	620	871	966	46	0	6	259	356	432	16	0	0	99	163	200
Other CMSA	52	0	6	268	444	592	15	0	0	84	156	231	5	0	0	25	57	90
Not MSA	95	0	7	549	928	1203	39	0	1	221	438	635	17	0	0	91	198	318

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

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Table 15. Estimated number of exceedances of short-term (1-hour) potential health effect benchmark levels in a year on-roads, 2001-2006 recent NO₂ air quality adjusted to just meet the current standard (0.053 ppm annual average).

Location	Exceedances of 200 ppb 1-hour ¹						Exceedances of 250 ppb 1-hour ¹						Exceedances of 300 ppb 1-hour ¹					
	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99	mean	min	med	p95	p98	p99
Boston	87	0	12	458	753	990	23	0	1	137	263	330	7	0	0	38	93	132
Chicago	176	0	61	805	1022	1139	59	0	7	335	560	620	23	0	0	128	295	354
Cleveland	387	14	268	1117	1322	1735	149	0	65	573	676	846	62	0	15	326	407	428
Denver	277	0	113	964	1233	1560	87	0	22	337	430	557	28	0	5	125	203	283
Detroit	440	17	309	1214	1444	1628	166	0	90	513	689	744	67	0	25	265	322	385
Los Angeles	106	0	23	533	788	893	31	0	2	186	290	363	10	0	0	59	115	150
Miami	406	3	306	1173	1345	1416	193	0	113	669	855	923	88	0	35	367	542	588
New York	84	0	14	458	709	872	25	0	1	149	295	413	8	0	0	49	110	177
Philadelphia	174	0	60	726	973	1184	51	0	7	239	383	521	16	0	1	77	153	227
Washington	208	0	83	874	1171	1310	63	0	10	327	426	558	21	0	1	127	181	224
Atlanta	335	0	135	1293	1647	1755	143	0	21	687	973	1093	61	0	4	339	510	656
El Paso	389	4	257	1251	1604	1737	144	0	66	530	858	971	54	0	20	221	350	441
Jacksonville	607	56	542	1385	1642	1743	273	5	202	789	924	1027	125	1	74	436	490	557
Las Vegas	278	0	43	1319	1929	2196	101	0	6	680	828	1045	42	0	0	354	502	565
Phoenix	149	0	19	758	1172	1352	33	0	1	203	303	370	7	0	0	48	70	95
Provo	516	1	345	1664	1966	2115	228	0	72	729	818	847	134	0	5	643	693	694
St. Louis	182	0	69	762	1100	1216	59	0	8	302	468	576	20	0	1	127	211	260
Other CMSA	64	0	6	333	569	740	19	0	0	105	207	300	6	0	0	31	72	120
Not MSA	101	0	7	569	874	1095	39	0	1	232	419	569	16	0	0	95	184	264

¹ The mean number of exceedances represents the number of exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p95, p98, and p99 represent the minimum, median, 95th, 98th, and 99th percentiles of the distribution for the number of exceedances in any one year within the monitoring period.

1 average by about 60%, indicating the presence of highly influential data at the upper percentiles
2 of the distribution at each location. This is evident when considering the 95th – 99th percentiles,
3 where several hundred to around two thousand exceedances of 200 ppb were estimated.
4 However, the estimated number of exceedances is lower for locations containing more site-years
5 of data than for the locations with the fewest site-years. This trend is consistent with those
6 described earlier, whereas estimates of exceedances in the simulated data for the large urban
7 areas are stabilized by greater sample size (both the number of monitors and 1-hour values). The
8 median number of exceedances of 200 ppb at the locations containing a larger monitoring
9 network (i.e. at least 40 site-years per year-group) was estimated to be between 10 and 100 per
10 year. Upper bounds for the locations with the greatest number of monitoring sites approach
11 around 1,000 estimated on-road exceedances per year upon just meeting the current standard.

12 It should be noted that the estimated on-road concentrations and number exceedances for
13 many of the locations were higher for the 2001-2006 rolled-up data when compared with the
14 1995-2000 rolled-up data. To obtain generally comparable results across the two time periods,
15 the assumption for the concentration roll-up was that a similar level of variability be maintained
16 from year-to-year (or year-group to year-group). As described in section 2.5 of the draft TSD, a
17 slight increase in hourly COV occurred from 1995-2006 (~10% for all locations). The effect
18 may have finally emerged in this combined simulation by generating a greater number of
19 concentrations above the potential health effect benchmarks that may have previously been just
20 below the threshold in the earlier on-road simulations considering the *as is* ambient
21 concentrations.

22 **6.4 UNCERTAINTY AND VARIABILITY**

23 This uncertainty analysis first identifies the sources of the assessment that do or do not
24 contribute to uncertainty, and provide a rationale for why this is the case. A qualitative
25 evaluation follows for the types and components of uncertainty, resulting in a matrix describing,
26 for each source of uncertainty, both the direction and magnitude of influence has on exposure
27 estimates. The bias direction indicates how the source of uncertainty is judged to influence
28 estimated concentrations, either the concentrations are likely “over-“ or “under-estimated”. In
29 the instance where two types or components of uncertainty result in offsetting direction of

1 influence, the uncertainty was judged as “both”. The magnitude indicates an estimated size of
2 influence the uncertainty has on estimated concentrations. “Minimal” uncertainty was noted
3
4 where quantitative evidence indicates the influence is either conditional and/or limited to few
5 components in type. A characterization of “moderate” was assigned where multiple components
6 of uncertainty existed within a given type and act in similar direction, however the presence of
7 all at once may be dependent on certain conditions. “Major” uncertainty was used where
8 multiple components of uncertainty exist within a given type, the components have few limiting
9 conditions, and the components consistently act in similar bias direction. “Unknown” was
10 assigned where there was no evidence reviewed to judge the uncertainty associated with the
11 source. Table 16 provides a summary of the sources of uncertainty identified in the air quality
12 characterization and the judged bias and magnitude of each.

13 **6.4.1 Air Quality Data**

14 One basic assumption is that the AQS NO₂ air quality data used are quality assured
15 already. Reported concentrations contain only valid measures, since values with quality
16 limitations are either removed or flagged. There is likely no selective bias in retention of data
17 that is not of reasonable quality, it is assumed that selection of high concentration poor quality
18 data would be just as likely as low concentration data of poor quality. Given the numbers of
19 measurements used for this analysis, it is likely that even if a few low quality data are present in
20 the data set, they would not have any significant effect on the results presented here. Therefore,
21 the air quality data and database used likely contributes minimally to uncertainty. Temporally,
22 the data are hourly measurements and appropriately account for variability in concentrations that
23 are commonly observed for NO₂ and by definition are representative of an entire year. In
24 addition, having more than one monitor does account for some of the spatial variability in a
25 particular location. However, the degree of representativeness of the monitoring data used in this
26 analysis can be evaluated from several perspectives, one of which is how well the temporal and
27 spatial variability are represented. In particular, missing hourly measurements at a monitor may
28 introduce bias (if different periods within a year or different years have different numbers of
29 measured values) and increase the uncertainty. Furthermore, the spatial representativeness will
30 be poor if the monitoring network is not dense enough to resolve the spatial variability (causing

1 increased uncertainty) or if the monitors are not evenly distributed (causing a bias). Additional
2 uncertainty regarding temporal and spatial representation by the monitors is expanded below.

3 **6.4.2 Measurement Technique for Ambient NO₂**

4 One source of uncertainty for NO₂ air quality data is due to interference with other
5 oxidized nitrogen compounds. The ISA points out positive interference, commonly from HNO₃,
6 of up to 50%, particularly during the afternoon hours, resulting in overestimation of
7 concentrations. Also, negative vertical gradients exist for monitors (2.5 times higher at 4 meter
8 vs. 15 meter vertical siting (draft ISA, section 2.5.3.3), thus monitors positioned on rooftops may
9 underestimate exposures. Only 7 of the 177⁶ monitors in the named locations contained
10 monitoring heights of 15 meters or greater, with nearly 60% at 4 meters or less height, and 80%
11 at 5 meters or less in height. Not accounting for this potential vertical gradient in NO₂
12 concentrations may generate underestimates of exceedances for some site-years, however the
13 overall impact of inferences made for the locations included in this assessment is likely minimal
14 since most monitors sited at less than 4-5 meters in vertical height.

15 **6.4.3 Temporal Representation**

16 Data are valid hourly measures and are of similar temporal scale as identified health
17 effect benchmark concentrations. There are frequent missing values within a given valid year
18 which contribute to the uncertainty as well as introducing a possible bias if some seasons, day
19 types (e.g., weekday/weekend), or time of the day (e.g., night or day) are not equally represented.
20 Since a 75 percent daily and hourly completeness rule was applied, some of these uncertainties
21 and biases were reduced in these analyses. Data were not interpolated in the analysis. Similarly,
22 there may be bias and uncertainty if the years monitored vary significantly between locations.
23 Although monitoring locations within a region do change over time, the NO₂ network has been
24 reasonably stable over the 1995-2006 period, particularly at locations with larger monitoring
25 networks, so the impact to uncertainty is expected to be minimal regarding both bias direction
26 and magnitude. It should also be noted that use of the older data in some of the analyses here
27 carries the assumption that the sources present at that time are the same as current sources,
28 adding uncertainty to results if this is not the case. Separating the data into two 5 year groups

⁶ 28 monitors did not have height reported (therefore, 177 + 28 = 205 total number of monitors in named locations)

1 (historic and recent) before analysis reduces the potential impact from changes in national- or
2 location-specific source influences and is judged to have a minimal magnitude.

3 **6.4.4 Spatial Representation**

4 Relative to the physical area, there are only a small number of monitors in each location.
5 Since most locations have sparse siting, the monitoring data are assumed to be spatially
6 representative of the locations analyzed here. This includes areas between the ambient monitors
7 that may or may not be influenced by similar local sources of NO₂. For these reasons the
8 uncertainty and bias due to the spatial network may be moderate, although the monitoring
9 network design should have addressed these issues within the available resources and other
10 monitoring constraints. This air quality characterization used all monitors meeting the 75
11 percent completeness criteria, without taking into account the monitoring objectives or land use
12 for the monitors. Thus, there will be some lack of spatial representation and likely moderate
13 uncertainty due to the inclusion/exclusion of some monitors that are very near local sources
14 (including mobile sources).

15 **6.4.5 Air Quality Adjustment Procedure**

16 The primary uncertainty of the empirical method used to estimate exceedances under the
17 current-standard scenario is due to the uncertainty of the true relationship between the annual
18 mean concentrations and the number of exceedances. The empirical method assumes that if the
19 annual means change then all the hourly concentrations will change proportionately. However,
20 different sources have different temporal emission profiles, so that applied changes to the annual
21 mean concentrations at monitors may not correspond well to all parts of the concentration
22 distribution equally. Similarly, emissions changes that affect the concentrations at the site with
23 the highest annual mean concentration will not necessarily impact lower concentration sites
24 proportionately. This could result in overestimations in the number of exceedances at lower
25 concentration sites within a location, however it is likely to be minimal given that the highest
26 concentrations typically were measured at the monitoring sites with the highest annual average
27 concentrations within the location (draft TSD, Appendix C). This minimal bias would apply to
28 areas that contain several monitors, such as Boston, New York, or Los Angeles. Universal
29 application of the proportional simulation approach at each of the locations was done for
30 consistency and was designed to preserve the inherent variability in the concentration profile. A

1 few locations were noted that may have an exceptional number of exceedances as a result of the
2 air quality adjustment approach, particularly those locations with few monitoring sites that
3 contained very low annual average concentrations and/or atypical variability in hourly
4 concentrations. These locations (e.g., Miami, Jacksonville, Provo) could contain moderate
5 overestimations at the upper tails of the concentration distribution, leading to bias in number of
6 estimated exceedances at both the upper percentiles and the mean for the scenarios using the air
7 quality simulated to just meet the current standard.

8 **6.4.6 On-Road Concentration Simulation**

9 On-road and ambient monitoring NO₂ concentrations have been shown to be correlated
10 significantly on a temporal basis (e.g., Cape et al., 2004) and motor vehicles are a significant
11 emission source of NO_x, providing support for estimating on-road concentrations using ambient
12 monitoring data. The relationship used in this analysis to estimate on-road NO₂ concentrations
13 was derived from data collected in measurement studies containing mostly long-term averaging
14 times, typically 14-days or greater in duration (e.g., Roorda-Knape, 1998; Pleijel et al., 2004;
15 Cape et al, 2004), although one study was conducted over a one-hour time averaging period
16 (Rodes and Holland, 1981). This is considered appropriate in this analysis to estimate on-road
17 hourly concentrations from hourly ambient measures, assuming a direct relationship exists
18 between the short-term peaks to time-averaged concentrations (e.g., hourly on-road NO₂
19 concentrations are correlated with 24-hour averages). While this should not impact the overall
20 contribution relationship between vehicles and ambient concentrations on roads, the decay
21 constant *k* will differ for shorter averaging times. The on-road concentration estimation also
22 assumes that concentration changes that occur on-road and at the monitor are simultaneous (i.e.,
23 within the hour time period of estimation). Since time-activity patterns of individuals are not
24 considered in this analysis, there is no bias in the number of estimated exceedances. The long-
25 term data used to develop the model were likely collected over variable meteorological
26 conditions (e.g., shifting wind direction) and other influential attributes (e.g., rate of
27 transformation of NO to NO₂ during the daytime versus nighttime hours) than would be observed
28 across shorter time periods. This could result in either over- or under-estimations of
29 concentrations, depending on the time of day. The variability in NO₂ concentration within an
30 hour is also not considered in this analysis, that is, the on-road concentration at a given site will

1 likely vary during the 1-hour time period. If considering personal exposures to individuals
2 within vehicles that are traveling on a road, it is likely that their exposure concentrations would
3 also vary due to differing roadway concentrations. This could also result in either over- or
4 under-estimations of concentrations, depending on the duration of travel and type of road
5 traveled on.

6 On-road concentrations were not modified in this analysis to account for in-vehicle
7 penetration and decay. This indicates that in-vehicle concentrations would be overestimated if
8 using the on-road concentrations as a surrogate, given that reactive pollutants (e.g., PM_{2.5}) tend
9 to have a lower indoor/outdoor (I/O) concentration ratio (Rodes et al., 1998). Chan and Chung
10 (2003) report mean (I/O) ratios of NO₂ for a few roadways and driving conditions in Hong Kong.
11 On highways and urban streets, the value is centered about 0.6 to 1.0, indicating decay of NO₂ as
12 it enters the vehicle.

13 At locations where traffic counts are very low (e.g., on the order of hundreds/day) the on-
14 road contribution has been shown to be negligible (Bell and Ashenden, 1997; Cape et al., 2004),
15 therefore any rural areas just meeting the standard with minimal traffic volumes would likely
16 have resulted in small overestimations of NO₂ concentrations using eq (2). For any monitor that
17 is sited in close proximity of the roadway (14 monitors were sited at <10 m from a major road),
18 on-road concentrations may have been overestimated using eq (2), since the assumption is that
19 the ambient concentration is equivalent to the non-source impacted concentration. In some
20 locations (i.e., Boston, Chicago, Denver, Los Angeles, Miami, St. Louis, and Washington DC),
21 at least half of the monitors used in this analysis are sited < 100 m from a major road (see draft
22 TSD, Table 5, section 2.3.3), a distance noted by some researchers as possibly receiving notable
23 impact from vehicle emissions (e.g., Beckerman et al., 2008). In addition, NO_x is primarily
24 emitted as NO (e.g., Heeb et al., 2008; Shorter et al., 2005), with substantial secondary formation
25 due predominantly to $\text{NO} + \text{O}_3 \rightarrow \text{NO}_2 + \text{O}_2$. Numerous studies have demonstrated the O₃
26 reduction that occurs near major roads, reflecting the transfer of odd oxygen to NO to form NO₂,
27 a process that can impact NO₂ concentrations both on- and downwind of the road. Some studies
28 report NO₂ concentrations increasing just downwind of roadways and that are inversely
29 correlated with O₃ (e.g., Beckerman et al., 2008), suggesting that peak concentration of NO₂ may
30 not always occur on the road, but at a distance downwind. Uncertainty regarding where the peak
31 concentration occurs (on-road or at a distance from the road) in combination with the form of the

1 exponential model used to estimate the on-road concentrations (the highest concentration occurs
2 at zero distance from road) could also lead to overestimation. However, the interpretation of the
3 estimate is what may be most uncertain, that is whether the exceedances are occurring on the
4 road or nearby.

5 Another source of uncertainty is the extent to which the near-road study locations
6 represent the locations studied in these analyses. The on-road and near-road data were collected
7 in a few locations, most of them outside of the United States. The source mixes (i.e., the vehicle
8 fleet) in study locations may not be representative of the U.S. fleet. Without detailed information
9 characterizing the emissions patterns for the on-road study areas, there was no attempt to match
10 the air quality characterization locations to specific on-road study areas, which might have
11 improved the precision of the estimates. However, since concentration ratios were selected
12 randomly from all the near-road studies and applied to each monitor individually, and since we
13 estimated overall minimum and upper bounds using multiple simulations, the analysis provides a
14 reasonable lower and upper bound estimate of the uncertainty.

15 **6.4.7 Health Benchmark**

16 The choice of potential health effect benchmarks, and the use of those benchmarks to
17 assess risks, can introduce uncertainty into the risk assessment. For example, the potential health
18 effect benchmarks used were based on studies where volunteers were exposed to NO₂ for
19 varying lengths of time. Typically, the NO₂ exposure durations were between 30 minutes and 2
20 hours. This introduces some uncertainty into the characterization of risk, which compared the
21 potential health effect benchmarks to estimates of exposure over a 1-hour time period. Use of a
22 1-hour averaging time could over- or under-estimate risks. In addition, the human exposure
23 studies evaluated airways responsiveness in mild asthmatics. For ethical reasons, more severely
24 affected asthmatics and asthmatic children were not included in these studies. Severe asthmatics
25 and/or asthmatic children may be more susceptible than mildly asthmatic adults to the effects of
26 NO₂ exposure. Therefore, the potential health effect benchmarks based on these studies could
27 underestimate risks in populations with greater susceptibility.

1 **Table 16. Summary of qualitative uncertainty analysis for the air quality and health risk**
 2 **characterization.**
 3

Source	Type	Bias Direction	Magnitude
Air Quality Data	Database quality	both	minimal
Ambient Measurement	Interference	over	moderate
	Vertical siting	under	minimal
Temporal Representation	Scale	none	none
	Missing data	both	minimal
	Years monitored	both	minimal
	Source changes	over	minimal
Spatial Representation	Scale	both	moderate
	Monitor objectives	both	moderate
Air Quality Adjustment	Temporal scale	over	moderate
	Spatial scale	over	moderate
On-Road Simulation	Temporal scale	both	minimal
	Decay	over	minimal
	Spatial scale	over	moderate
	Model used	over	minimal
	Non US studies used	unknown	unknown
Health Benchmarks	Averaging time	unknown	minimal
	Susceptibility	under	moderate
Notes: Bias Direction: indicates the direction the source of uncertainty is judged to influence either the concentration or risk estimates. Magnitude: indicates the estimated size of influence. minimal – influence is either conditional and/or limited to few components in type moderate – multiple components of uncertainty existed within a given type and act in similar direction, however the presence of all at once may be dependent on certain conditions. major – multiple components of uncertainty exist within a given type, the components have few limiting conditions, and the components consistently act in similar bias direction.			

4

7. EXPOSURE ASSESSMENT AND HEALTH RISK

CHARACTERIZATION

7.1 OVERVIEW

This section documents the methodology and data used in the inhalation exposure assessment and associated health risk characterization for NO₂ conducted in support of the current review of the NO₂ primary NAAQS. Two important components of the analysis include the approach for estimating temporally and spatially variable NO₂ concentrations and simulating human contact with these pollutant concentrations. Both air quality and exposure modeling approaches have been used to generate estimates of 1-hour NO₂ exposures within selected urban areas of the U.S. across a 3-year period (2001-2003). Exposures and risk were characterized considering recent air quality conditions (*as is*) and for air quality adjusted to just meet the current NO₂ standard (0.053 ppm annual average). Details on the approaches used are provided below and in Chapter 3 in the draft TSD. Briefly, the discussion includes the following:

- Description of the inhalation exposure model and associated input data
- Evaluation of estimated NO₂ exposures
- Assessment of the quality and limitations of the input data for supporting the goals of the NO₂ NAAQS exposure and risk characterization.

The combined dispersion and exposure modeling approach was both time and labor intensive. To date, only the exposure and risk results for the Philadelphia case-study are complete and are presented in this draft document. Location-specific input data for Philadelphia and the other selected case-study areas are presented where collected (mainly meteorological data) to provide information on the relative variability of the input data to be used.

7.2 OVERVIEW OF HUMAN EXPOSURE MODELING USING APEX

The purpose of this exposure analysis is to allow comparisons of population exposures to ambient NO₂ among and within selected locations, and to characterize risks associated with current air quality levels and with just meeting the current 0.053 ppm annual average standard. This section provides a brief overview of the model used by EPA to estimate NO₂ population

1 exposure. Details about the application of the model to estimate NO₂ population exposure are
2 provided in the following sections and in Chapter 3 of the draft TSD.

3 The EPA has developed the Air Pollutants Exposure Model (APEX) model for estimating
4 human population exposure to criteria and air toxic pollutants. APEX serves as the human
5 inhalation exposure model within the Total Risk Integrated Methodology (TRIM) framework
6 (EPA 2006a; 2006b) and was recently used to estimate population exposures in 12 urban areas
7 for the O₃ NAAQS review (EPA, 2007g; 2007h).

8 APEX is a probabilistic model designed to account for sources of variability that affect
9 people's exposures. APEX simulates the movement of individuals through time and space and
10 estimates their exposure to a given pollutant in indoor, outdoor, and in-vehicle
11 microenvironments. The model stochastically generates a sample of simulated individuals using
12 census-derived probability distributions for demographic characteristics. The population
13 demographics are drawn from the year 2000 Census at the tract, block-group, or block level, and
14 a national commuting database based on 2000 census data provides home-to-work commuting
15 flows. Any number of simulated individuals can be modeled, and collectively they approximate
16 a random sampling of people residing in a particular study area.

17 Daily activity patterns for individuals in a study area, an input to APEX, are obtained
18 from detailed diaries that are compiled in the Consolidated Human Activity Database (CHAD)
19 (McCurdy et al., 2000; EPA, 2002). The diaries are used to construct a sequence of activity
20 events for simulated individuals consistent with their demographic characteristics, day type, and
21 season of the year, as defined by ambient temperature regimes (Graham and McCurdy, 2004).
22 The time-location-activity diaries input to APEX contain information regarding an individuals'
23 age, gender, race, employment status, occupation, day-of-week, daily maximum hourly average
24 temperature, the location, start time, duration, and type of each activity performed. Much of this
25 information is used to best match the activity diary with the generated personal profile, using
26 age, gender, employment status, day of week, and temperature as first-order characteristics. The
27 approach is designed to capture the important attributes contributing to an individuals' behavior,
28 and of likely importance in this assessment (i.e., time spent outdoors) (Graham and McCurdy,
29 2004). Furthermore, these diary selection criteria give credence to the use of the variable data
30 that comprise CHAD (e.g., data collected were from different seasons, different states of origin,
31 etc.).

1 APEX has a flexible approach for modeling microenvironmental concentrations, where
2 the user can define the microenvironments to be modeled and their characteristics. Typical
3 indoor microenvironments include residences, schools, and offices. Outdoor microenvironments
4 include for example near roadways, at bus stops, and playgrounds. Inside cars, trucks, and mass
5 transit vehicles are microenvironments which are classified separately from indoors and
6 outdoors. APEX probabilistically calculates the concentration in the microenvironment
7 associated with each event in an individual's activity pattern and sums the event-specific
8 exposures within each hour to obtain a continuous series of hourly exposures spanning the time
9 period of interest. The estimated pollutant concentrations account for the effects of ambient
10 (outdoor) pollutant concentration, penetration factors, air exchange rates, decay/deposition rates,
11 proximity to important outdoor sources, and indoor source emissions, each depending on the
12 microenvironment, available data, and estimation method selected by the user. And, since the
13 modeled individuals represent a random sample of the population of interest, the distribution of
14 modeled individual exposures can be extrapolated to the larger population.

15 The model simulation can be summarized in the following five steps:

- 16 1. **Characterize the study area.** APEX selects census blocks within a study area – and
17 thus identifies the potentially exposed population – based on user-defined criteria and
18 availability of air quality and meteorological data for the area.
- 19 2. **Generate simulated individuals.** APEX stochastically generates a sample of
20 hypothetical individuals based on the census data for the study area and human
21 profile distribution data
- 22 3. **Construct a sequence of activity events.** APEX constructs an exposure event
23 sequence spanning the period of the simulation for each of the simulated individuals
24 and based on the activity pattern data.
- 25 4. **Calculate hourly concentrations in microenvironments.** APEX users define
26 microenvironments that people in the study area would visit by assigning location
27 codes in the activity pattern to the user-specified microenvironments. The model then
28 calculates hourly concentrations of a pollutant in each of these microenvironments for
29 the period of simulation, based on the user-provided microenvironment descriptions,
30 the hourly air quality data, and for some of the indoor microenvironments, indoor

1 sources of NO₂. Microenvironmental concentrations are calculated for each of the
2 simulated individuals.

- 3 5. **Estimate exposures.** APEX estimates a concentration for each exposure event based
4 on the microenvironment occupied during the event. These values can be averaged
5 by clock hour to produce a sequence of hourly average exposures spanning the
6 specified exposure period. These hourly values may be further aggregated to produce
7 daily, monthly, and annual average exposure values.

8 **7.3 CHARACTERIZATION OF STUDY AREAS**

9 **7.3.1 Study Area Selection**

10 The selection of areas to include in the exposure analysis takes into consideration the
11 location of field and epidemiological studies, the availability of ambient monitoring and other
12 input data, the desire to represent a range of geographic areas, population demographics, general
13 climatology, and results of the ambient air quality characterization.

14 Locations of interest were initially identified through a similar statistical analysis of the
15 ambient NO₂ air quality data described above for each site within a location. Criteria were
16 established for selecting sites with high annual means and/or high numbers of exceedances of
17 health effect benchmark concentrations. The analysis considered all ambient monitoring data
18 combined (1995-2006), as well as the more recent air quality data (2001-2006) separately.

19 The 90th percentile served as the point of reference for the annual means, and across all
20 complete site-years for recent ambient monitoring (2001 through 2006), this value was 23.52
21 ppb. Seventeen locations contained one or more site-years with an annual average concentration
22 at or above the 90th percentile. When combined with the number of 1-hour NO₂ concentrations
23 at or above 200 ppb, only two locations fit these criteria, Philadelphia and Los Angeles.
24 Considering the short-term criterion alone, Detroit contained the greatest number of exceedances
25 of 200 ppb (numbering 12 for years 2001-2006). Two additional locations were selected by
26 considering geographic/climatologic representation and also their historic ambient
27 concentrations. Atlanta (1 exceedance of 200 ppb and a maximum annual average concentration
28 of 26.63 ppb for years 1995-2006) and Phoenix (maximum annual mean concentration of 37.09
29 ppb for 2001-2006 and 37 exceedances of 200 ppb for years 1995-2006) were selected to

1 represent the southern and western region of the US from the pool of remaining locations with
2 either exceedances of the 90th percentile annual mean concentration or 200 ppb 1-hour.

3
4 To summarize, the following 5 urban areas were selected for a detailed exposure analysis:

- 5 • Philadelphia, PA
- 6 • Atlanta, GA
- 7 • Detroit, MI
- 8 • Los Angeles, CA
- 9 • Phoenix, AZ

10 The exposure periods modeled were 2001 through 2003 to envelop the most recent year
11 of travel demand modeling (TDM) data available for the respective study locations (i.e., 2002)
12 and to include a 3 years of meteorological data to achieve stability in the dispersion/exposure
13 model estimates.

14 **7.3.2 Study Area Descriptions**

15 The APEX study area has traditionally been on the scale of a city or slightly larger
16 metropolitan area, although it is now possible to model larger areas such as combined statistical
17 areas (CSAs). In this analysis the study area is defined by a single or few counties. The
18 demographic data used by the model to create personal profiles is provided at the census block
19 level. For each block the model requires demographic information representing the distribution
20 of age, gender, race, and work status within the study population. Each block has a location
21 specified by latitude and longitude for some representative point (e.g., geographic center). The
22 current release of APEX includes input files that already contain this demographic and location
23 data for all census tracts, block groups, and blocks in the 50 United States, based on the 2000
24 Census.

25 The first area study area selected for a detailed exposure analysis was Philadelphia
26 County since the TDM data were readily available and was one of two locations where both
27 selection criteria were met using recent air quality (the other being Los Angeles). Philadelphia
28 County is a large part of the Philadelphia-Wilmington-Atlantic City CMSA, comprised of 16,857
29 blocks and containing a total population of 1,475,651 persons (representing approximately 97%
30 of the county population).

7.4 CHARACTERIZATION OF AMBIENT HOURLY AIR QUALITY DATA USING AERMOD

7.4.1 Overview

Air quality data used for input to APEX were generated using AERMOD, a steady-state, Gaussian plume model (EPA, 2004). For each identified case-study location, the following steps were performed

1. **Collect and analyze general input parameters.** Meteorological data, processing methodologies used to derive input meteorological fields (e.g., temperature, wind speed, precipitation), and information on surface characteristics and land use are needed to help determine pollutant dispersion characteristics, atmospheric stability and mixing heights.
2. **Estimate emissions.** The emission sources modeled included, major stationary emission sources, on-road emissions that occur on major roadways, and fugitive emissions.
3. **Define receptor locations.** Three sets of receptors were identified for the dispersion modeling, including ambient monitoring locations, census block centroids, and links along major roadways.
4. **Estimate concentrations at receptors.** Hourly concentrations were estimated for each year of the simulation (years 2001 through 2003) by combining concentration contributions from each of the emission sources and accounting for sources not modeled.

The AERMOD hourly concentrations were then used as input to the APEX model to estimate population exposure concentrations. Details regarding both modeling approaches and input data used are provided below and in Chapter 3 of the draft TSD. Hourly NO₂ concentrations were estimated for each of 3 years (2001-2003) at each of the defined receptor locations (census blocks and roadway links) using hourly NO_x emission estimates and dispersion modeling. Relevant input data collected for Philadelphia as well some of the data collected as part of the other selected case-study locations to be evaluated in the second draft risk and exposure assessment are presented below.

7.4.2 General Model Inputs

7.4.2.1 Meteorological Data

All meteorological data used for the AERMOD dispersion model simulations were processed with the AERMET meteorological preprocessor, version 06341. Raw surface meteorological data for the 2001 to 2003 period were obtained from the Integrated Surface Hourly (ISH) Database,⁷ maintained by the National Climatic Data Center (NCDC). The ISH data used for this study consists of typical hourly surface parameters (including air and dew point temperature, atmospheric pressure, wind speed and direction, precipitation amount, and cloud cover) from hourly Automated Surface Observing System (ASOS) stations. No on-site observations were used.

Surface meteorological stations for this analysis were those at the major airports of each of the five cities in the study:

- Philadelphia: Philadelphia International (KPHL)
- Atlanta: Atlanta Hartsfield International (KATL)
- Detroit: Detroit Metropolitan (KDTW)
- Los Angeles: Los Angeles International (KLAX)
- Phoenix: Phoenix Sky Harbor International (KPHX).

The selection of surface meteorological stations for each city minimized the distance from the station to city center, minimized missing data, and maximized land-use representativeness of the station site compared to the city center. The total number of surface observations per station were compiled and the percentage of those observations accepted by AERMET (i.e., those observations that were both not missing and within the expected ranges of values) were typically $\geq 99\%$.

Mandatory and significant levels of upper-air data were obtained from the NOAA Radiosonde Database.⁸ Upper air observations show less spatial variation than do surface observations; thus they are both representative of larger areas and measured with less spatial frequency than are surface observations. The selection of upper-air station locations for each

⁷ <http://www1.ncdc.noaa.gov/pub/data/techrpts/tr200101/tr2001-01.pdf>

⁸ <http://raob.fsl.noaa.gov/>

1 city minimized both the proximity of the station to city center and the amount of missing data in
2 the records. The selected stations were:

- 3 • Philadelphia: Washington Dulles Airport (KIAD)
- 4 • Atlanta: Peachtree City (KFFC)
- 5 • Detroit: Detroit/Pontiac (KDTX)
- 6 • Los Angeles: Miramar Naval Air Station near San Diego (KNKX)
- 7 • Phoenix: Tucson (KTWC).

8 The total number of upper-air observations per station per height interval, and the
9 percentage of those observations accepted by AERMET were typically $\geq 99\%$ for the pressure,
10 height, and temperature parameters however, dewpoint temperature, wind direction, and wind
11 speed parameters had lower acceptance rates (sometimes $\leq 75\%$), particularly when considering
12 greater atmospheric heights.

13 *7.4.2.2 Surface Characteristics and Land Use Analysis*

14 In addition to the standard meteorological observations of wind, temperature, and cloud
15 cover, AERMET analyzes three principal variables to help determine atmospheric stability and
16 mixing heights: the Bowen ratio⁹, surface albedo¹⁰ as a function of the solar angle, and surface
17 roughness¹¹.

18 The January 2008 version of AERSURFACE was used to estimate land-use patterns and
19 calculate the Bowen ratio, surface albedo, and surface roughness as part of the AERMET
20 processing. AERSURFACE uses the US Geological Survey (USGS) National Land Cover Data
21 1992 archives (NLCD92)¹². Three to four land-use sectors were manually identified around the
22 surface meteorological stations using this land-use data. These land-use sectors are used to
23 identify the Bowen ratio and surface albedo, which are assumed to represent an area around the
24 station of radius 10 km, and to calculate surface roughness by wind direction.

⁹ For any moist surface, the Bowen Ratio is the ratio of heat energy used for sensible heating (conduction and convection) to the heat energy used for latent heating (evaporation of water or sublimation of snow). The Bowen ratio ranges from about 0.1 for the ocean surface to more than 2.0 for deserts. Bowen ratio values tend to decrease with increasing surface moisture for most land-use types.

¹⁰ The ratio of the amount of electromagnetic radiation reflected by the earth's surface to the amount incident upon it. Value varies with surface composition. For example, snow and ice vary from 80% to 85% and bare ground from 10% to 20%.

¹¹ The presence of buildings, trees, and other irregular land topography that is associated with its efficiency as a momentum sink for turbulent air flow, due to the generation of drag forces and increased vertical wind shear.

¹² <http://seamless.usgs.gov/>

1 A monthly temporal resolution was used for the Bowen ratio, albedo, and surface
 2 roughness for all five meteorological sites. Because the five sites were located at airports, a
 3 lower surface roughness was calculated for the ‘Commercial/Industrial/Transportation’ land-use
 4 type to reflect the dominance of transportation land cover rather than commercial buildings. Los
 5 Angeles and Phoenix are arid regions, which increases the calculated albedo and Bowen ratio
 6 values and decreases the surface roughness values assigned to the ‘Shrubland’ and ‘Bare
 7 Rock/Sand/Clay’ land-use types to reflect a more desert-like area. Philadelphia and Detroit each
 8 have at least one winter month of continuous snow cover, which tends to increase albedo,
 9 decrease Bowen ratio, and decrease surface roughness for most land-use types during the winter
 10 months compared to snow-free areas.

11 Seasons were assigned for each site based on 1971-2000 NCDC 30-year climatic normals
 12 and on input from the respective state climatologists. Table 17 provides the seasonal definitions
 13 for each city. Further discussion of the land use and surface analysis, as well as a discussion of
 14 the difference in results from employing the new AERSURFACE tool is given in Appendix E of
 15 the draft TSD.

16

17 **Table 17. Seasonal specifications by selected case-study locations.**

18

Location	Winter (continuous snow)	Winter (no snow)	Spring	Summer	Fall
Philadelphia	Dec, Jan, Feb		Mar, Apr, May	Jun, Jul, Aug	Sep, Oct, Nov
Atlanta		Dec, Jan, Feb	Mar, Apr, May	Jun, Jul, Aug	Sep, Oct, Nov
Detroit	Dec, Jan, Feb, Mar		Apr, May	Jun, Jul, Aug	Sep, Oct, Nov
Los Angeles			Apr, May, Jun	Jul, Aug, Sep	Oct, Nov, Dec, Jan, Feb, Mar
Phoenix			Apr, May, Jun	Jul, Aug, Sep	Oct, Nov, Dec, Jan, Feb, Mar
Season definitions provided by the AERSURFACE manual:					
Winter (continuous snow):		Winter with continuous snow on ground			
Winter (no snow):		Late autumn after frost and harvest, or winter with no snow			
Spring:		Transitional spring with partial green coverage or short annuals			
Summer:		Midsummer with lush vegetation			
Fall:		Autumn with unharvested cropland			

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Meteorological Data Analysis

The AERMET application location and elevation were taken as the center of each modeled city, estimated using Google Earth version 4.2.0198.2451 (beta). They are as follows:

- Philadelphia: 39.952 °N, 75.164 °W, 12 m
- Atlanta: 33.755 °N, 84.391 °W, 306 m
- Detroit: 42.332 °N, 83.048 °W, 181 m
- Los Angeles: 34.053 °N, 118.245 °W, 91 m
- Phoenix: 33.448 °N, 112.076 °W, 330 m

For each site in this study, the 2001-2003 AERSURFACE processing was run three times – once assuming the entire period was drier than normal, once assuming the entire period was wetter than normal, and once assuming the entire period was of average precipitation accumulation. These precipitation assumptions influence the Bowen ratio, as discussed above.

To create meteorological input records that best represent the given city for each of the three years, the resulting surface output files for each site were then pieced together on a month-by-month basis, with selection based on the relative amount of precipitation in each month. Any month where the actual precipitation amount received was at least twice the 1971-2000 NCDC 30-year climatic normal monthly precipitation amount was considered wetter than normal, while any month that received less than half the normal amount of precipitation amount was considered drier than normal; all other months were considered to have average surface moisture conditions.

Surface moisture conditions were variable when considering the month-location combinations to 30-year climatic normals, with much of the precipitation across the 3-year period reflective of typical to dry conditions (Table 18).

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Table 18. Characterization of monthly precipitation levels in selected case-study locations compared to NCDC 30-year climatic normals, 2001-2003.

Location	Number of Months with Precipitation Level ¹		
	Avg	Dry	Wet
Philadelphia	27	7	2
Atlanta	28	7	1
Detroit	26	9	1
Los Angeles	12	15	9
Phoenix	11	22	3

¹**Precipitation Level Definitions**
Avg: <2 times the normal precipitation level and >1/2 the normal amount.
Dry: ≤1/2 the normal monthly precipitation amount.
Wet: ≥2 times the normal precipitation level.

5 **7.4.2.3 Additional AERMOD Input Specifications**

6 Since each of the case-study locations were MSA/CMSAs, all emission sources were
7 characterized as urban. The AERMOD *toxics* enhancements were also employed to speed
8 calculations from area sources. NO_x chemistry was applied to all sources to determine NO₂
9 concentrations. For the each of the roadway, fugitive, and airport emission sources, the ozone
10 limiting method (OLM) was used, with plumes considered ungrouped. Because an initial NO₂
11 fraction of NO_x is anticipated to be about 10% or less (Finlayson-Pitts and Pitts, 2000; Yao et al.,
12 2005), a conservative value of 10% for all sources was selected. For all point source simulations
13 the Plume Volume Molar Ratio Method (PVMRM) was used to estimate the conversion of NO_x
14 to NO₂, with the following settings:

- 15 1. Hourly series of O₃ concentrations were taken from EPA’s AQS database¹³. The
16 complete national hourly record of monitored O₃ concentrations were filtered for the
17 four monitors within Philadelphia County (stations 421010004, 421010014,
18 421010024, and 421010136). The hourly records of these stations were then
19 averaged together to provide an average Philadelphia County concentrations of O₃ for
20 each hour of 2001-2003.

¹³ <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqdata.htm>

- 1 2. The equilibrium value for the NO₂:NO_x ratio was taken as 75%, the national average
2 ambient ratio.¹⁴
- 3 3. The initial NO₂ fraction of NO_x is anticipated to be about 10% or less. A default
4 value of 10% was used for all stacks (Finlayson-Pitts and Pitts, 2000).

5 **7.4.3 Emissions Estimates**

6 ***7.4.3.1 On-Road Emissions Preparation***

7 Information on traffic data in the Philadelphia area was obtained from the Delaware
8 Valley Regional Planning Council (DVRPC¹⁵) via their most recent, baseline travel demand
9 modeling (TDM) simulation – that is, the most recent simulation calibrated to match observed
10 traffic data.

11 ***Emission Sources and Locations***

12 The TDM simulation's shapefile outputs include annual average daily traffic (AADT)
13 volumes and a description of the loaded highway network. The description of the network
14 consists of a series of nodes joining individual model links (i.e., roadway segments) to which the
15 traffic volumes are assigned, and the characteristics of those links, such as endpoint location,
16 number of lanes, link distance, and TDM-defined link daily capacity.¹⁶

17 The full set of links in the DVRPC network was filtered to include only those roadway
18 types considered *major* (i.e., freeway, parkway, major arterial, ramp), and that had AADT values
19 greater than 15,000 vehicles per day (one direction). Then, link locations from the TDM were
20 modified to represent the best known locations of the actual roadways, since there was not
21 always a direct correlation between the two. The correction of link locations was done based on
22 the locations of the nodes that define the end points of links with a GIS analysis, as follows.

23 A procedure was developed to relocate TDM nodes to more realistic locations. The
24 nodes in the TDM represent the endpoints of links in the transportation planning network and are
25 specified in model coordinates. The model coordinate system is a Transverse Mercator

¹⁴ Appendix W to CFR 51, page 466. http://www.epa.gov/scram001/guidance/guide/appw_03.pdf.

¹⁵ <http://www.dvrpc.org/>

¹⁶ The TDM capacity specifications are not the same as those defined by the Highway Capacity Manual (HCM). Following consultation with DVRPC, the HCM definition of capacity was used in later calculations discussed below.

1 projection of the TranPlan Coordinate System with a false easting of 31068.5, false northing of -
 2 200000.0, central meridian: -75.00000000, origin latitude of 0.0, scale factor of 99.96, and in
 3 units of miles. The procedure moved the node locations to the true road locations and translated
 4 to dispersion model coordinates. The Pennsylvania Department of Transportation (PA DOT)
 5 road network database¹⁷ was used as the specification of the true road locations. The nodes were
 6 moved to coincide with the nearest major road of the corresponding roadway type using a built-
 7 in function of ArcGIS. Once the nodes had been placed in the corrected locations, a line was
 8 drawn connecting each node pair to represent a link of the adjusted planning network.

9 To determine hourly traffic on each link, the AADT volumes were converted to hourly
 10 values by applying DVRPC’s seasonal and hourly scaling factors. The heavy-duty vehicle
 11 fraction – which is assumed by DVRPC to be about 6% in all locations and times – was also
 12 applied¹⁸. Another important variable, the number of traffic signals occurring on a given link,
 13 was obtained from the TDM link-description information. Table 19 summarizes the AADT
 14 volumes used in the simulations for each road type.

15
 16 **Table 19. Statistical summary of AADT volumes (one direction) for Philadelphia County**
 17 **AERMOD simulations.**
 18

Statistic	Road Type	CBD	Fringe	Suburban	Urban
Count	Arterial	186	58	210	580
	Freeway	11	10	107	98
	Ramp	0	4	3	1
Minimum AADT	Arterial	15088	15282	15010	15003
	Freeway	15100	18259	15102	15100
	Ramp		16796	15679	16337
Maximum AADT	Arterial	44986	44020	48401	44749
	Freeway	39025	56013	68661	68661
	Ramp		40538	24743	16337
Average AADT	Arterial	21063	21196	20736	22368
	Freeway	25897	40168	33979	31294
	Ramp		24468	18814	16337

19
¹⁷ <http://www.pasda.psu.edu/>

¹⁸ As shown by Figure 1, NO_x emissions from HDVs tend to be higher than their LDV counterparts by about a factor of 10. However, the HDV fraction is less than 10% of the total VMT in most circumstances, mitigating their influence on composite emission factors, although this mitigating effect is less pronounced at some times than others. For example, nighttime on freeways tend to show a smaller reduction in HDV volume than in total volume, and thus an increased HDV fraction. This effect is not captured in most TDMs or emission postprocessors and – both to maintain consistency with the local MPO’s vehicle characterizations and emissions modeling and due to lack of other relevant data – was also not included here. The net result of this is likely to be slightly underestimated emissions from major freeways during late-night times.

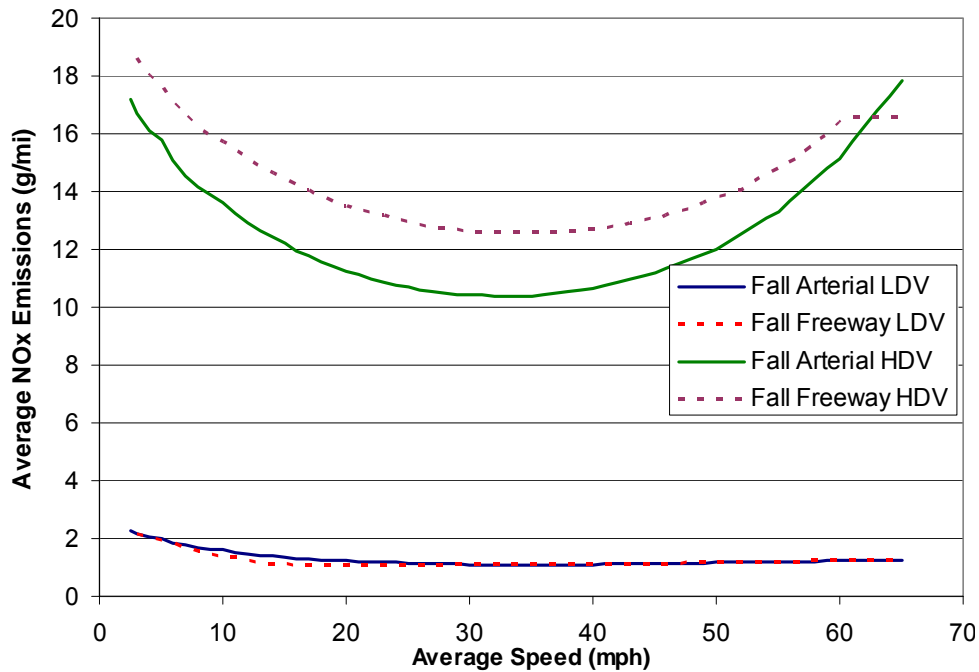
1 ***Emission Source Strength***

2 On-road mobile emission factors were derived from the MOBILE6.2 emissions model as
3 follows. The DVRPC-provided external data files describing the VMT distribution by speed,
4 functional class, and hour, as well as the registration distribution and *Post-1994 Light Duty*
5 *Gasoline Implementation* for Philadelphia County were all used in the model runs without
6 modification. To further maintain consistency with the recent DVRPC inventory simulations
7 and maximize temporal resolution, the DVRPC's seasonal particulate matter (PM) MOBILE6
8 input control files were also used.¹⁹ These files include county-specific data describing the
9 vehicle emissions inspection and maintenance (I/M) Programs, on-board diagnostics (OBD) start
10 dates, vehicle miles traveled (VMT) mix, vehicle age distributions, default diesel fractions, and
11 representative minimum and maximum temperatures, humidity, and fuel parameters. The
12 simulations are designed to calculate average running NO_x emission factors.

13 These input files were modified for the current project to produce running NO_x emissions
14 in grams per mile for a specific functional class (Freeway, Arterial, or Ramp) and speed.
15 Iterative MOBILE6.2 simulations were conducted to create tables of average Philadelphia
16 County emission factors resolved by speed (2.5 to 65 mph), functional class, season, and year
17 (2001, 2002, or 2003) for each of eight combined MOBILE vehicle classes.²⁰ The resulting
18 tables were then consolidated into speed, functional class, and seasonal values for combined
19 light- and heavy-duty vehicles. Figure 1 shows an example of the calculated emission factors for
20 Autumn, 2001.

¹⁹ The present emissions model input files were based on MPO-provided PM, rather than NO_x input files for a few reasons. First, MPO-provided PM files were used because they contain quarterly rather than annual or biannual information. In all cases the output species were modified to produce gaseous emissions. Further, many of the specified input parameters do not affect PM emissions, but were included by the local MPO to best represent local conditions, which were preserved in the present calculations of NO_x emissions. This usage is consistent with the overall approach of preserving local information wherever possible.

²⁰ HDDV - Heavy-Duty Diesel Vehicle, HDGV - Heavy-Duty Gasoline Vehicle, LDDT - Light-Duty Diesel Truck, LDDV - Light-Duty Diesel Vehicle, LDGT12 - Light-Duty Gasoline Truck with gross vehicle weight rating ≤ 6,000 lbs and a loaded vehicle weight of ≤ 5,750 lbs, LDGT 34 - Light-Duty Gasoline Truck with gross vehicle weight rating between 6,001 - 8,500 and a loaded vehicle weight of ≤ 5,750 lbs, LDGV - Light-Duty Gasoline Vehicle, MC - Motorcycles.



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3 **Figure 1. Example of Light- and heavy-duty vehicle NO_x emissions grams/mile (g/mi) for**
4 **arterial and freeway functional classes, Philadelphia 2001.**
5

6 To determine the emission strengths for each link for each hour of the year, the
7 Philadelphia County average MOBILE6.2 speed-resolved emissions factor tables were merged
8 with the TDM link data, which had been processed to determine time-resolved speeds. The
9 spatial-mean speed of each link at each time was calculated following the methodology of the
10 Highway Capacity Manual.²¹ Table 20 shows the resulting average speed for each functional
11 class within each TDM region.

12
13 **Table 20. Average calculated speed by link type for Philadelphia County.**
14

	Average Speed (mph)			
	CBD	Fringe	Suburban	Urban
Ramp	N/A	35	35	35
Arterial	34	31	44	32
Freeway	51	62	66	62
Notes: N/A not available				

15
16 The resulting emission factors were then coupled with the TDM-based activity estimates
17 to calculate emissions from each of the 1,268 major roadway links. However, many of the links

²¹ As defined in Chapter 9 of Recommended Procedure for Long-Range Transportation Planning and Sketch Planning, NCHRP Report 387, National Academy Press, 1997. 151 pp., ISBN No: 0-309-060-58-3.

1 were two sides of the same roadway segment. To speed model execution time, those links that
 2 could be combined into a single emission source were merged together. This was done for the
 3 628 links (314 pairs) where opposing links were spatially paired and exhibited similar activity
 4 levels within 20% of each other.

5 ***Other Emission Parameters***

6 Each roadway link is characterized as a rectangular area source with the width given by
 7 the number of lanes and an assumed universal lane width of 12 ft (3.66 m). The length and
 8 orientation of each link is determined as the distance and angle between end nodes from the
 9 adjusted TDM locations. In cases where the distance is such that the aspect ratio is greater than
 10 100:1, the links were disaggregated into sequential links, each with a ratio less than that
 11 threshold. There were 27 links that exceeded this ratio and were converted to 55 segmented
 12 sources. Thus, the total number of area sources included in the dispersion simulations is 982.
 13 Table 21 shows the distribution of on-road area source sizes. Note that there are some road
 14 segments whose length was zero after GIS adjustment of node location. This is assumed to be
 15 compensated by adjacent links whose length will have been expanded by a corresponding
 16 amount.

17
 18 **Table 21. On-road area source sizes for Philadelphia County.**
 19

	Segment Width (m)	Lanes	Segment Length (m)
Minimum	3.7	1.0	0.0
Median	11.0	3.0	220.6
Average	13.7	3.8	300.2
1- σ Deviation	7.7	2.1	259.5
Maximum	43.9	12.0	1340.2

20
 21 Resulting daily emission estimates were temporally allocated to hour of the day and
 22 season using MOBILE6.2 emission factors, coupled with calculated hourly speeds from the
 23 postprocessed TDM and allocated into SEASHR emission profiles for the AERMOD dispersion
 24 model. That is, 96 emissions factors are attributed to each roadway link to describe the emission
 25 strengths for 24 hours of each day of each of four seasons and written to the AERMOD input
 26 control file.

1 The release height of each source was determined as the average of the light- and heavy-
2 duty vehicle fractions, with an assumed light- and heavy-duty emission release heights of 1.0 ft
3 (0.3048 m) and 13.1 ft (4.0 m), respectively.²² Because AERMOD only accepts a single release
4 height for each source, the 24-hour average of the composite release heights is used in the
5 modeling.

6 Since surface-based mobile emissions are anticipated to be terrain following, no elevated
7 or complex terrain was included in the modeling. That is, all sources are assumed to lie in a flat
8 plane.

9 ***7.4.3.2 Stationary Sources Emissions Preparation***

10 Data for the parameterization of major point sources in Philadelphia comes primarily from
11 two sources: the 2002 National Emissions Inventory (NEI; EPA, 2007e) and Clean Air Markets
12 Division (CAMD) Unit Level Emissions Database (EPA, 2007f). The NEI database contains
13 stack locations, emissions release parameters (i.e., height, diameter, exit temperature, exit
14 velocity), and annual NO_x emissions. The CAMD database has information on hourly NO_x
15 emission rates for all the units in the US, where the units are the boilers or equivalent, each of
16 which can have multiple stacks. These two databases generally contain complimentary
17 information, and were first evaluated for matching facility data. Then, annual emissions data
18 from the NEI were used to scale the hourly CAMD data where discrepancies existed between
19 estimated annual emissions.

20 ***Data Source Alignment and Scaling***

21 To align the information between the two emission databases and extract the most useful
22 portion of each for dispersion modeling, the following methodology was used.

- 23 1. Collect data for all stacks within Philadelphia County.
- 24 2. Combine individual stacks that have identical stack physical parameters and were co-
25 located within about 10 m, to be simulated as a single stack with their emissions
26 summed.
- 27 3. Retain facilities with total emissions from all stacks exceeding 100 tpy NO_x.
- 28 4. Remove fugitive releases, to be analyzed as a separate source group.

²² 4.0 m includes plume rise from truck exhaust stacks. See [Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach](#), State of California Air Resources Board, Final Report, April 2006.

1 This resulted in 19 distinct, combined stacks from the NEI (Table 22). Then, the CAMD
2 database was queried for facilities that matched the facilities identified from the NEI database.
3 Facility matching was done on the facility name, Office of Regulatory Information Systems
4 (ORIS) identification code (when provided) and facility total emissions to ensure a best match
5 between the facilities. Once facilities were paired, individual units and stacks in the data bases
6 were paired based on annual emission totals. Most facilities contained similar total annual NO_x
7 emissions when comparing the two databases, although at one facility, nearly half of the NEI
8 emissions (without fugitives) do not appear in the CAMD database, while another identified in
9 the NEI was not included at all in the CAMD. The reason for this is unknown and no
10 information was readily available on the relative accuracy of the two databases.

11 Hourly emissions in the CAMD database were scaled using a factor to match the NEI
12 annual total emissions based on each of the matched stacks/units. This includes accounting for
13 where emissions were reduced or absent from the CAMD database. Then for consistency, the
14 2001 and 2003 hourly emission profiles were also determined using the same scaling factors, but
15 applied to the respective CAMD emission profile. Details for source parameters and scaling
16 factors are provided in Chapter 3 of the draft TSD.

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Table 22. Stationary NOx emission sources modeled in Philadelphia County.

Stack No.	NEI Site ID	Facility Name	SIC Code	NAICS Code	ORIS Code	Stack Emiss. (tpy)	Stack X (deg)	Stack Y (deg)
817	NEIPA2218	EXELON GENERATION CO - DELAWARE STATION	4911	221112	3160	4.82	-75.1358	39.96769
818	NEIPA2218	EXELON GENERATION CO - DELAWARE STATION	4911	221112	3160	287.8	-75.1358	39.96769
819	NEI40720	JEFFERSON SMURFIT CORPORATION (U S)	2631	32213		0.148	-75.2391	40.03329
820	NEI40720	JEFFERSON SMURFIT CORPORATION (U S)	2631	32213		113.8	-75.2391	40.03329
821	NEI40720	JEFFERSON SMURFIT CORPORATION (U S)	2631	32213		114.46	-75.2391	40.03329
855	NEI40723	Sunoco Inc. - Philadelphia	2911	32411		26.2	-75.2027	39.92535
856	NEI40723	Sunoco Inc. - Philadelphia	2911	32411		1.3	-75.2003	39.91379
857	NEI40723	Sunoco Inc. - Philadelphia	2911	32411		1.4	-75.203	39.92539
858	NEI40723	Sunoco Inc. - Philadelphia	2911	32411		19.3	-75.2027	39.92535
859	NEI40723	Sunoco Inc. - Philadelphia	2911	32411		1032.8	-75.2124	39.90239
860	NEI7330	SUNOCO CHEMICALS (FORMER ALLIED SIGNAL)	2869	325998		0.033	-75.0715	40.00649
861	NEI7330	SUNOCO CHEMICALS (FORMER ALLIED SIGNAL)	2869	325998		49.1	-75.0715	40.00649
862	NEI7330	SUNOCO CHEMICALS (FORMER ALLIED SIGNAL)	2869	325998		34.6	-75.0715	40.00649
863	NEI7330	SUNOCO CHEMICALS (FORMER ALLIED SIGNAL)	2869	325998		77.2	-75.0715	40.00649
864	NEIPA101353	TRIGEN - SCHUYLKILL	4961	22		128.6	-75.1873	39.94239
865	NEIPA101353	TRIGEN - SCHUYLKILL	4961	22		61.5	-75.1873	39.94239
866	NEIPA101356	GRAYS FERRY COGENERATION PARTNERS	4911	22	54785	143.2	-75.1873	39.94239
867	NEIPA101356	GRAYS FERRY COGENERATION PARTNERS	4911	22	54785	90.3	-75.1873	39.94239
868	NEIPA2222	TRIGEN - EDISON	4961	62		130.5	-75.1569	39.94604

3

4 ***7.4.3.3 Fugitive and Airport Emissions Preparation***

5 Fugitive emission releases, as totaled in the NEI database, were modeled as area sources
6 with the profile of these releases determined by the overall facility profile of emissions. In
7 addition, emissions associated with the Philadelphia International Airport were estimated.

8 ***Fugitive Releases***

9 Thirty five *combined stacks* were identified during the point source analysis (see previous
10 section) that were associated with facilities considered major emitters, but where the emissions
11 from the stacks are labeled *Fugitive* in the NEI. These stacks have zero stack diameter, zero

1 emission velocity, and exit temperature equal to average ambient conditions (295 K). Thus, we
 2 determined it was not appropriate to include these in the point source group simulation.

3 These 35 stacks occur at only two facilities in the County: Exelon Generation Co –
 4 Delaware Station (NEI Site ID: NEIPA2218) and Sunoco Inc. – Philadelphia (NEI Site ID:
 5 NEI40723). Consequently, they were grouped by facility. The Sunoco emissions were grouped
 6 into two distinct categories based on release heights. Thus, to accommodate all of these sources
 7 most efficiently, a total of three area source groups were created: one for Sunoco emissions at 3.0
 8 m, one for Sunoco emissions greater than 23.0 m, and one for Exelon. Their combined area
 9 source parameters are given in Table 23.

10

11 **Table 23. Emission parameters for the three Philadelphia County fugitive NOx area**
 12 **emission sources.**

13

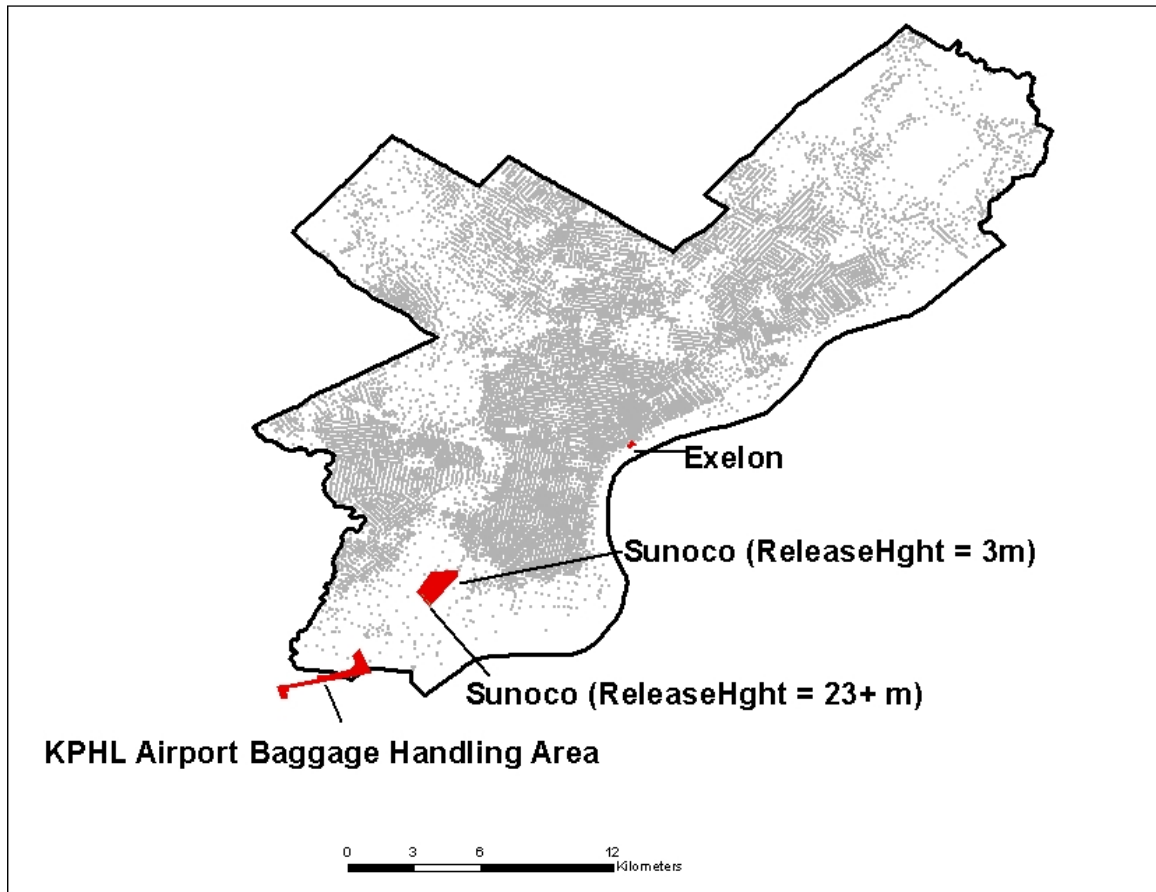
No.	NEI Site ID	Facility Name	Combined		Average Stack Height (m)	Stacks Used for Profile **	Scaled Emissions (tpy) *		
			# of Stacks	NEI 2002 Emissions (tpy)			2001	2002	2003
1	NEI PA2218	Exelon Generation Co - Delaware Station	2	5.2	6.5	817+818	4.8	5.2	6.4
2	NEI 40723	Sunoco Inc. - Philadelphia	26	1,680.4	3	855+856 + 857+ 858+ 859	1,873.8	1,681.4	2,202.4
3	NEI 40723	Sunoco Inc. - Philadelphia	7	350.8	26.7	855+856 +857+ 858+ 859	391.2	351.0	459.8

14

15 In the case of the Sunoco emissions, the vertices of the area sources were determined by a
 16 convex hull encapsulating all the points. In the case of Exelon, only two points are provided,
 17 which is insufficient information to form a closed polygon. Instead, the boundary of the facility
 18 was digitized into a 20-sided polygon. Figure 2 shows the locations of these polygons.

19 Emission profiles for the fugitive releases were determined from the CAMD hourly
 20 emission database in a method similar to that for the point sources. We determined scaling
 21 factors based on the ratio of the 2002 fugitive releases described by the NEI to the total, non-
 22 fugitive point source releases from the same facility. All stacks within that facility were

1 combined on an hourly basis for each year and the fugitive to non-fugitive scaling factor applied,
2 ensuring that the same temporal emission profile was used for fugitives as for other releases from
3 the facility, since the origins of the emissions should be parallel. We created external hourly
4 emissions files for each of the three fugitive area sources with appropriate units (grams per
5 second per square meter).



6
7
8 **Figure 2. Locations of the four ancillary area sources. Also shown are centroid receptor**
9 **locations.**

10
11 ***Philadelphia International Airport***

12 Another significant source of NO_x emissions in Philadelphia County not captured in the
13 earlier simulations is from operation of the Philadelphia International Airport (PHL). PHL is the
14 only major commercial airport in the County and is the largest airport in the Delaware Valley.

1 The majority of NO_x emissions in the NEI²³ database attributable to airports in
 2 Philadelphia County are from non-road mobile sources, specifically ground support equipment.
 3 There is another airport in the County: Northeast Philadelphia Airport. However, because it
 4 serves general aviation, is generally much smaller in operations than PHL, and has little ground
 5 support equipment activity – which is associated primarily with commercial aviation – all airport
 6 emissions in the County were attributed to PHL. The PHL emissions were taken from the non-
 7 road section of the 2002 NEI, and are shown by Table 24.

8
 9 **Table 24. Philadelphia International airport (PHL) NO_x emissions.**

10

State and County	SCC	NO _x (tpy)	SCC Level 1 Description	SCC Level 3 Description	SCC Level 6 Description	SCC Level 8 Description
Philadelphia, PA	2265008005	4.6	Mobile Sources	Off-highway Vehicle Gasoline, 4-Stroke	Airport Ground Support Equipment	Airport Ground Support Equipment
	2267008005	5.1	Mobile Sources	LPG	Airport Ground Support Equipment	Airport Ground Support Equipment
	2270008005	196.2	Mobile Sources	Off-highway Vehicle Diesel	Airport Ground Support Equipment	Airport Ground Support Equipment
	2275020000	0.01	Mobile Sources	Aircraft	Commercial Aircraft	Total: All Types
	2275050000	2.5	Mobile Sources	Aircraft	General Aviation	Total
PHL Total		208.4				

11
 12 As with the fugitive sources discussed above, the airport emissions are best
 13 parameterized as area sources. The boundary of the area source was taken as the region of
 14 operation of baggage handling equipment, including the terminal building and the region
 15 surrounding the gates. This region was digitized into an 18-sided polygon of size 1,326,000 m²,
 16 and included in the AERMOD input control file.

17 The activity profile for PHL was taken to have seasonal and hourly variation (SEASHR),
 18 based on values from the EMS-HAP model.²⁴ These factors are disaggregated in the EMS-HAP
 19 model database based on source classification codes (SCCs), which were linked to those from

²³ <http://www.epa.gov/ttn/chief/net/2002inventory.html>

²⁴ EPA 2004, User's Guide for the Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP) Version 3.0, EPA-454/B-03-006.

1 the NEI database. The EMS-HAP values provide hourly activity factors by season, day type, and
 2 hour; to compress to simple SEASHR modeling, the hourly values from the three individual day
 3 types were averaged together. The total emissions for each SCC were then disaggregated into
 4 seasonal and hourly components and the resulting components summed to create total PHL
 5 emissions for each hour of the four annual seasons. These parameterized emissions were then
 6 normalized to the total cargo handling operational area, to produce emission factors in units of
 7 grams per second per square meter and included in the AERMOD input file. Figure 2 also
 8 shows the location of the PHL area source.

9 **7.4.4 Receptor Locations**

10 Three sets of receptors were chosen to represent the locations of interest. First, all NO_x
 11 monitor locations, shown by Table 25, within the Philadelphia CMSA were included as receptor
 12 locations. Although all receptors are assumed to be on a flat plane, they are placed at the
 13 standard breathing height of 5.9 ft (1.8 m).

14
 15 **Table 25. Philadelphia CMSA NO_x monitors.**
 16

CMSA	Site ID	Latitude	Longitude
Philadelphia- Wilmington- Atlantic City, PA-NJ-DE-MD	100031003	39.7611	-75.4919
	100031007	39.5511	-75.7308
	100032004	39.7394	-75.5581
	340070003	39.923	-75.0976
	420170012	40.1072	-74.8822
	420450002	39.8356	-75.3725
	420910013	40.1122	-75.3092
	421010004	40.0089	-75.0978
	421010029	39.9572	-75.1731
	421010047	39.9447	-75.1661

17
 18 The second receptor locations were selected to represent the locations of census block
 19 centroids near major NO_x sources. GIS analysis was used to determine all block centroids in
 20 Philadelphia County that lie within a 0.25 mile (400 m) of the roadway segments and also all
 21 block centroids that lie within 6.2 miles (10 km) of any major point source. 12,982 block
 22 centroids were selected due to their proximity to major roadways; 16,298 centroids were selected
 23 due to their proximity to major sources. The union of these sets produced 16,857 unique block

1 centroid receptor locations, each of which was assigned a height of 5.9 ft (1.8 m). The location
2 of centroids that met either distance criteria and included in the modeling is shown by Figure 3.

3

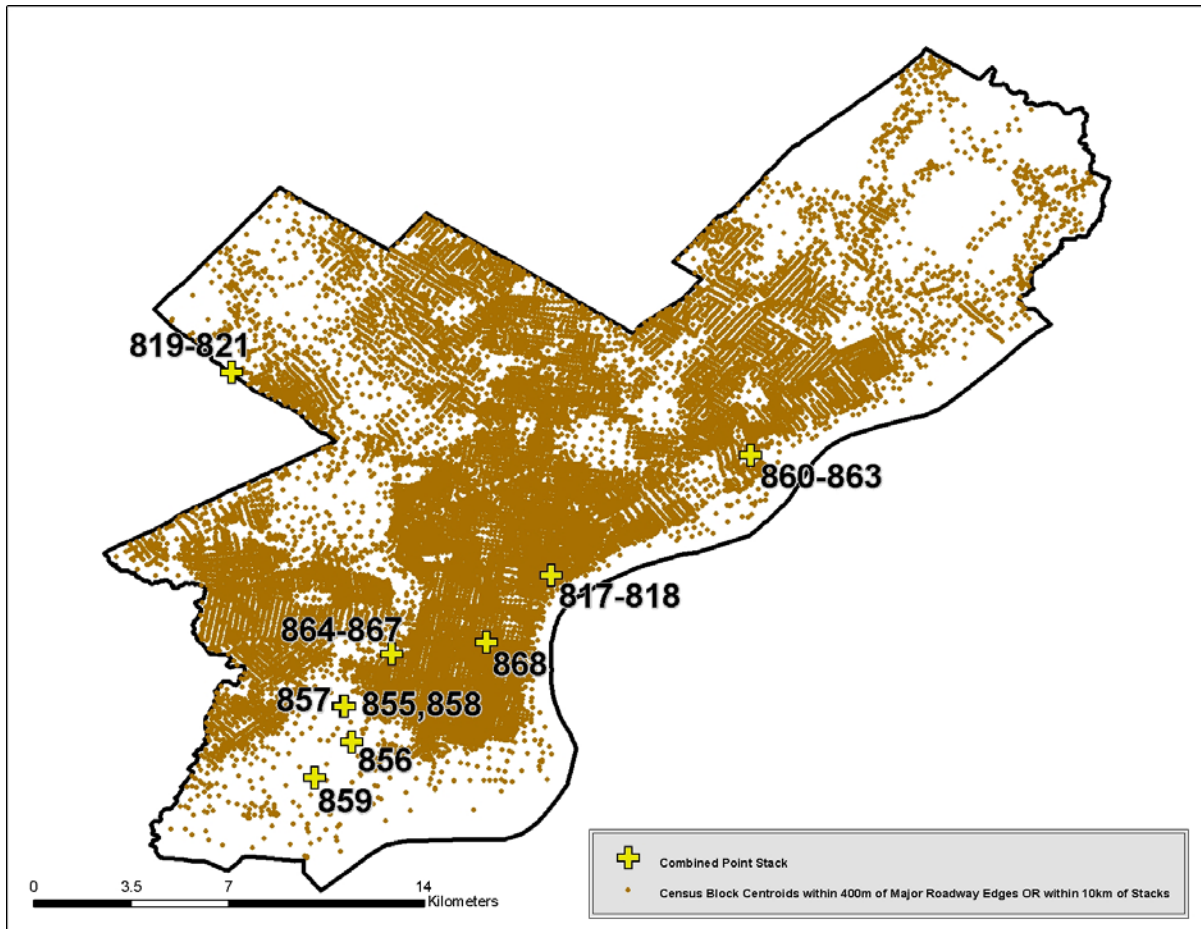
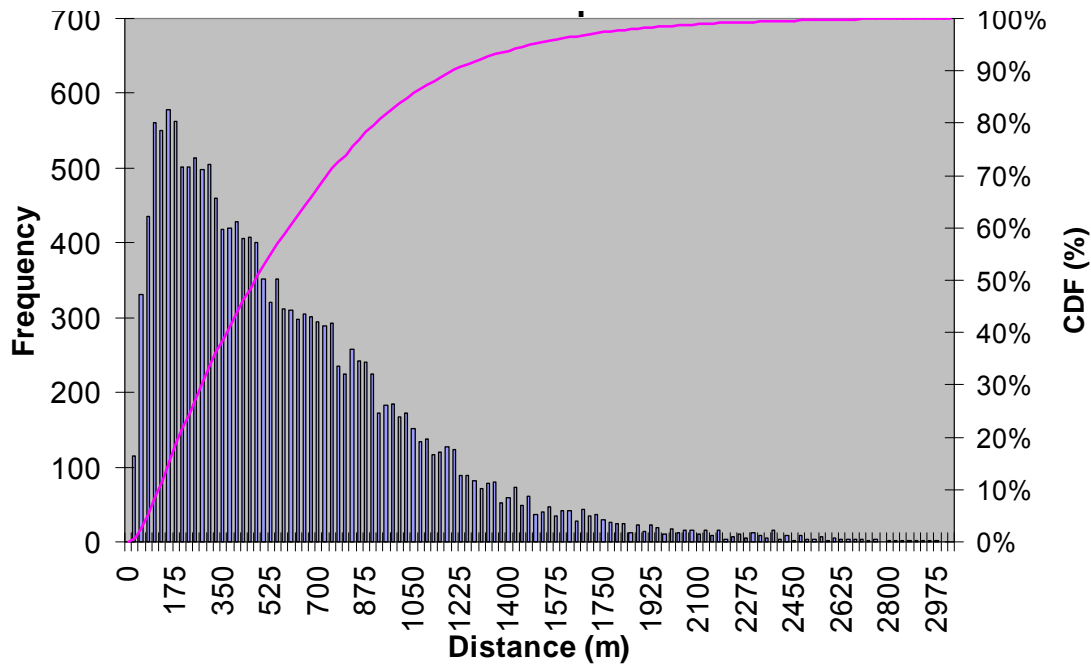


Figure 3. Centroid locations within fixed distances to major point and mobile sources.

The third set of receptors was chosen to represent the on-road microenvironment. For this set, one receptor was placed at the center of each of the 982 sources.

The distance relationship between the road segments and block centroids can be estimated by looking at the distance between the road-centered and the block centroid receptors. Figure 4 shows the histogram of the shortest distance between each centroid receptor and its nearest roadway-centered receptor.



1
2 **Figure 4. Frequency distribution of distance between each Census receptor and its nearest**
3 **road-centered receptor.**
4

5 The centroids selected were those within 10 km of any major point source or 400 m from
6 any receptor edge, so the distances to the nearest major road segment can be significantly greater
7 than 400 m. The mode of the distribution is about 150 m and the median distance to the closest
8 roadway segment center is about 450 m. However, these values represent the distances of the
9 block centroids to road centers instead of road edges, so that they overestimate the actual
10 distances to the zone most influenced by roadway by an average of 14 m and a range of 4 m to
11 44 m (see Table 21 above).

12 **7.4.5 Estimate Air Quality Concentrations**

13 The hourly concentrations estimated from each of the three source categories were
14 combined at each receptor. Then a local concentration, reflecting the concentration contribution
15 from emission sources not included in the simulation, was added to the sum of the concentration
16 contributions from each of these sources at each receptor. The local concentration was estimated
17 from the difference between the model predictions at the local NO₂ monitors and the observed
18 values. It should be noted that this local concentration may also include any model error present
19 in estimating concentration at the local monitoring sites. Table 26 presents a summary of the
20 estimated local concentration added to the AERMOD hourly concentration data.
21

1 **Table 26. Comparison of ambient monitoring and AERMOD predicted NO₂**
 2 **concentrations.**
 3

Year and Monitor ID	Annual Average NO ₂ concentration (ppb)			
	Monitor	AERMOD Initial	Difference ¹	AERMOD Final ²
2001				
4210100043	26	7	18	19
4210100292	28	22	6	33
4210100471	30	20	10	32
mean			11	
2002				
4210100043	24	7	17	18
4210100292	28	21	7	32
4210100471	29	19	10	31
mean			11	
2003				
4210100043	24	7	17	13
4210100292	25	22	3	28
4210100471*	25	26	-1	32
mean			6	
¹ the difference represents concentrations attributed to sources not modeled by AERMOD and model error. ² the mean difference between measured and modeled was added uniformly at each receptor hourly concentration to generate the AERMOD final concentrations. * monitor did not meet completeness criteria used in the air quality characterization.				

4

5 **7.5 POPULATION MODELED**

6 A detailed consideration of the population residing in each modeled area was included
 7 where the exposure modeling was performed. The assessment included the general population
 8 residing in each modeled area and susceptible subpopulations identified in the ISA. These
 9 include population subgroups defined from either an exposure or health perspective. The
 10 population subgroups identified by the ISA and that were modeled in the exposure assessment
 11 include asthmatics of all ages and asthmatic children (ages 5-18). In addition to these population
 12 subgroups, activities for those susceptible to potentially greater exposure to NO₂ were
 13 considered, including those commuting on roadways and persons residing near major roadways.
 14 While the total population exposure was estimated, the focus of the analysis was on the
 15 susceptible individuals.

1 **7.5.1 Simulated Individuals**

2 APEX takes population characteristics into account to develop accurate representations of
3 study area demographics. Population counts and employment probabilities by age and gender
4 are used to develop representative profiles of hypothetical individuals for the simulation. Block-
5 level population counts by age in one-year increments, from birth to 99 years, come from the
6 2000 Census of Population and Housing Summary File 1 (SF-1). This file contains the 100-
7 percent data, which is the information compiled from the questions asked of all people and about
8 every housing unit. The total population considered in this analysis was approximately 1.48
9 million persons, of which there a total simulated population of 163,000 asthmatics. The model
10 simulated approximately 281,000 children, of which there were about 48,000 asthmatics. Due to
11 random sampling, the actual number of specific subpopulations modeled will vary slightly by
12 year.

13 **7.5.2 Employment Probabilities**

14 Employment data from the 2000 Census provide employment probabilities for each
15 gender and specific age groups for every Census tract. The employment age groupings were: 16-
16 19, 20-21, 22-24, 25-29, 30-34, 35-44, 45-54, 55-59, 60-61, 62-64, 65-69, 70-74, and >75 years
17 of age. Children under the age of 16 are assigned employment probabilities of zero.

18 **7.5.3 Commuting Patterns**

19 To ensure that individuals' daily activities are accurately represented within APEX, it is
20 important to integrate working patterns into the assessment. Commuting data were originally
21 derived from the 2000 Census and were collected as part of the Census Transportation Planning
22 Package (CTPP) (US DOT, 2007). CTPP contains tabulations by place of residence, place of
23 work, and the flows between the residence and work.

24 It is assumed that all persons with home-to-work distances up to 120 km are daily
25 commuters, and that persons who travel further than 120 km do not commute daily. Therefore
26 the list of commuting destinations for each home tract is restricted to only those work tracts that
27 are within 120 km of the home tract.

28 APEX allows the user to specify how to handle individuals who commute to destinations
29 outside the study area. One option is to drop them from the simulation. If they are included, the
30 user specifies values for two additional parameters, called L_M and L_A (Multiplicative and

1 Additive factors for commuters who Leave the area). While a commuter is at work, if the
2 workplace is outside the study area, then the ambient concentration cannot be determined from
3 any air district (since districts are inside the study area). Instead, it is assumed to be related to
4 the average concentration $C_{AVE(t)}$ over all air districts at the time in question. The ambient
5 concentration outside the study area at time t , $C_{OUT(t)}$, is estimated as:

$$C_{OUT(t)} = L_M * C_{AVE(t)} + L_A$$

6
7
8
9 The microenvironmental concentration (for example, in an office outside the study area)
10 is determined from this ambient concentration by the same model (mass balance or factor) as
11 applied inside the study area. The parameters L_M and L_A were both set to zero for this modeling
12 analysis; thus, exposures to individuals are set to zero when they are outside of the study area.
13 Although this tends to underestimate exposures, it is a small effect and this was done since we
14 have not estimated ambient concentrations of NO_2 in counties outside of the modeled areas.

15 School age children did not have commuting to and from school. This results in the
16 implicit assumption that children attend a school with ambient NO_2 concentrations similar to
17 concentrations near their residence.

18 **7.6 CONSTRUCTION OF LONGITUDINAL ACTIVITY SEQUENCES**

19 Exposure models use human activity pattern data to predict and estimate exposure to
20 pollutants. Different human activities, such as spending time outdoors, indoors, or driving, will
21 result in varying pollutant exposure concentrations. To accurately model individuals and their
22 exposure to pollutants, it is critical to understand their daily activities.

23 The Consolidated Human Activity Database (CHAD) provides data for where people
24 spend time and the activities performed. CHAD was designed to provide a basis for conducting
25 multi-route, multi-media exposure assessments (McCurdy et al., 2000; EPA, 2002). Table 27
26 summarizes the studies in CHAD used in this modeling analysis, providing nearly 16,000 diary-
27 days of activity data (3,075 diary-days for ages 5-18) collected between 1982 and 1998.

1 **Table 27. Studies in CHAD used for the exposure analysis.**

2

Study name	Geographic coverage	Study time period	Subject ages	Diary-days	Diary-days (ages 5-18)	Diary type and study design	Reference
Baltimore	One building in Baltimore	01/1997-02/1997, 07/1998-08/1998	72 - 93	292	0	Diary	Williams et al. (2000)
California Adolescents (CARB)	California	10/1987-09/1988	12 - 17	181	181	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Adults (CARB)	California	10/1987-09/1988	18 - 94	1,552	36	Recall; Random	Robinson et al. (1989), Wiley et al. (1991a)
California Children (CARB)	California	04/1989- 02/1990	<1 - 11	1,200	683	Recall; Random	Wiley et al. (1991b)
Cincinnati (EPRI)	Cincinnati metro. area	03/1985-04/1985, 08/1985	<1 - 86	2,587	740	Diary; Random	Johnson (1989)
Denver (EPA)	Denver metro. area	11/1982- 02/1983	18 - 70	791	7	Diary; Random	Johnson (1984) Akland et al. (1985)
Los Angeles: Elementary School	Los Angeles	10/1989	10 - 12	51	51	Diary	Spier et al. (1992)
Los Angeles: High School	Los Angeles	09/1990-10/1990	13 - 17	42	42	Diary	Spier et al. (1992)
National: NHAPS-Air	National	09/1992-10/1994	<1 - 93	4,326	634	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
National: NHAPS-Water	National	09/1992-10/1994	<1 - 93	4,332	691	Recall; Random	Klepeis et al. (1996), Tsang and Klepeis (1996)
Washington, D.C. (EPA)	Wash., D.C. metro. area	11/1982-02/1983	18 - 98	639	10	Diary; Random	Hartwell et al. (1984), Akland et al. (1985)
Total diary days				15,993	3,075		

1 Typical time-activity pattern data available for inhalation exposure modeling consist of a
2 sequence of location/activity combinations spanning 24-hours, with 1 to 3 diary-days for any
3 single individual. Exposure modeling typically requires information on activity patterns over
4 longer periods of time, e.g., a full year. For example, even for pollutant health effects with short
5 averaging times (e.g., NO₂ 1-hour average concentration) it may be desirable to know the
6 frequency of exceedances of a concentration over a long period of time (e.g., the annual number
7 of exceedances of a 1-hour average NO₂ concentration of 200 ppb for each simulated individual).

8 Long-term multi-day activity patterns can be estimated from single days by combining
9 the daily records in various ways, and the method used for combining them will influence the
10 variability of the long-term activity patterns across the simulated population. This in turn will
11 influence the ability of the model to accurately represent either long-term average high-end
12 exposures, or the number of individuals exposed multiple times to short-term high-end
13 concentrations.

14 A new algorithm has been developed and incorporated into APEX to represent the day-
15 to-day correlation of activities for individuals. The algorithms first use cluster analysis to divide
16 the daily activity pattern records into groups that are similar, and then select a single daily record
17 from each group. This limited number of daily patterns is then used to construct a long-term
18 sequence for a simulated individual, based on empirically-derived transition probabilities. This
19 approach is intermediate between the assumption of no day-to-day correlation (i.e., re-selection
20 of diaries for each time period) and perfect correlation (i.e., selection of a single daily record to
21 represent all days).

22 The steps in the algorithm are as follows:

- 23 1. For each demographic group (age, gender, employment status), temperature range,
24 and day-of-week combination, the associated time-activity records are partitioned into
25 3 groups using cluster analysis. The clustering criterion is a vector of 5 values: the
26 time spent in each of 5 microenvironment categories (indoors – residence; indoors –
27 other building; outdoors – near road; outdoors – away from road; in vehicle).
- 28 2. For each simulated individual, a single time-activity record is randomly selected from
29 each cluster.
- 30 3. A Markov process determines the probability of a given time-activity pattern
31 occurring on a given day based on the time-activity pattern of the previous day and

1 cluster-to-cluster transition probabilities. The cluster-to-cluster transition
2 probabilities are estimated from the available multi-day time-activity records. If
3 insufficient multi-day time-activity records are available for a demographic group,
4 season, day-of-week combination, then the cluster-to-cluster transition probabilities
5 are estimated from the frequency of time-activity records in each cluster in the CHAD
6 database.

7 Further details regarding the Cluster-Markov algorithm and supporting evaluations are
8 provided in Appendix F of the draft TSD.

9 **7.7 CALCULATING MICROENVIRONMENTAL CONCENTRATIONS**

10 Probabilistic algorithms are used to estimate the pollutant concentration associated with
11 each exposure event. The estimated pollutant concentrations account for temporal and spatial
12 variability in ambient (outdoor) pollutant concentration and factors affecting indoor
13 microenvironment, such as a penetration, air exchange rate, and pollutant decay or deposition
14 rate. APEX calculates air concentrations in the various microenvironments visited by the
15 simulated person by using the ambient air data estimated for the relevant blocks/receptors, the
16 user-specified algorithm, and input parameters specific to each microenvironment. The method
17 used by APEX to estimate the microenvironment depends on the microenvironment, the data
18 available for input to the algorithm, and the estimation method selected by the user. At this time,
19 APEX calculates hourly concentrations in all the microenvironments at each hour of the
20 simulation for each of the simulated individuals using one of two methods: by mass balance or a
21 transfer factors method.

22 The mass balance method simulates an enclosed microenvironment as a well-mixed
23 volume in which the air concentration is spatially uniform at any specific time. The
24 concentration of an air pollutant in such a microenvironment is estimated using the following
25 processes:

- 26 • Inflow of air into the microenvironment
- 27 • Outflow of air from the microenvironment
- 28 • Removal of a pollutant from the microenvironment due to deposition, filtration, and
29 chemical degradation
- 30 • Emissions from sources of a pollutant inside the microenvironment.

1 A transfer factors approach is simpler than the mass balance model, however, most
2 parameters are derived from distributions rather than single values to account for observed
3 variability. It does not calculate concentration in a microenvironment from the concentration in
4 the previous hour as is done by the mass balance method, and it has only two parameters. A
5 proximity factor is used to account for proximity of the microenvironment to sources or sinks of
6 pollution, or other systematic differences between concentrations just outside the
7 microenvironment and the ambient concentrations (at the measurements site or modeled
8 receptor). The second, a penetration factor, quantifies the amount of outdoor pollutant penetrates
9 into the microenvironment.

10 **7.7.1 Microenvironments Modeled**

11 In APEX, microenvironments represent the exposure locations for simulated individuals.
12 For exposures to be estimated accurately, it is important to have realistic microenvironments that
13 match closely to the locations where actual people spend time on a daily basis. As discussed
14 above, the two methods available in APEX for calculating pollutant levels within
15 microenvironments are: 1) factors and 2) mass balance. A list of microenvironments used in this
16 study, the calculation method used, and the type of parameters used to calculate the
17 microenvironment concentrations can be found in Table 26.

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Table 28. List of microenvironments modeled and calculation methods used.

Microenvironment	Calculation Method	Parameter Types used¹
Indoors – Residence	Mass balance	AER and DE
Indoors – Bars and restaurants	Mass balance	AER and DE
Indoors – Schools	Mass balance	AER and DE
Indoors – Day-care centers	Mass balance	AER and DE
Indoors – Office	Mass balance	AER and DE
Indoors – Shopping	Mass balance	AER and DE
Indoors – Other	Mass balance	AER and DE
Outdoors – Near road	Factors	PR
Outdoors – Public garage - parking lot	Factors	PR
Outdoors – Other	Factors	None
In-vehicle – Cars and Trucks	Factors	PE and PR
In-vehicle - Mass Transit (bus, subway, train)	Factors	PE and PR
¹ AER=air exchange rate, DE=decay-deposition rate, PR=proximity factor, PE=penetration factor		

4

7.7.2 Microenvironment Descriptions

5

7.7.2.1 Microenvironment 1: Indoor-Residence

6

The Indoor-Residence microenvironment uses several variables that affect NO₂ exposure: whether or not air conditioning is present, the average outdoor temperature, the NO₂ removal rate, and an indoor concentration source.

8
9

Air conditioning prevalence rates

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Since the selection of an air exchange rate distribution is conditioned on the presence or absence of an air-conditioner, for each modeled area the air conditioning status of the residential microenvironments is simulated randomly using the probability that a residence has an air conditioner. For this study, location-specific air conditioning prevalence was calculated using the data and survey weights from the American Housing Survey of 2003 (AHS, 2003a; 2003b). Table 29 contains the values for air conditioning prevalence used for each modeled location.

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Table 29. Air conditioning (A/C) prevalence estimates with 95% confidence intervals.

AHS Survey	Housing Units	A/C Prevalence (%)	se	L95	U95
Philadelphia	1,943,492	90.6	1.3	88.1	93.2
Atlanta	797,687	97.0	1.2	94.7	99.3
Detroit	1,877,178	81.4	1.8	78.0	84.9
Los Angeles	3,296,819	55.1	1.7	51.7	58.4
Phoenix	-	-	-	-	-

Notes:
se – Standard error
L95 – Lower limit on 95th confidence interval
U95 – Upper limit on 95th confidence interval

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Air exchange rates

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Air exchange rate data for the indoor residential microenvironment were obtained from EPA (2007g). Briefly, data were reviewed, compiled and evaluated from the extant literature to generate location-specific AER distributions categorized by influential factors, namely temperature and presence of air conditioning. In general, lognormal distributions provided the best fit, and are defined by a geometric mean (GM) and standard deviation (GSD). To avoid unusually extreme simulated AER values, bounds of 0.1 and 10 were selected for minimum and maximum AER, respectively.

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Fitted distributions were available for one of the cities modeled in this assessment, Los Angeles. For the other four of the locations to be modeled, a distribution was selected from one of the other locations thought to have similar characteristics to the city to be modeled, qualitatively considering factors that might influence AERs. These factors include the age composition of housing stock, construction methods, and other meteorological variables not explicitly treated in the analysis, such as humidity and wind speed patterns. The distributions used for each of the modeled locations are provided in Table 30.

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Table 30. Geometric means (GM) and standard deviations (GSD) for air exchange rates by city, A/C type, and temperature range.

Area Modeled	Study City	A/C Type	Temp (°C)	N	GM	GSD
	Houston	Central or Room A/C	<=20	15	0.4075	2.1135
			20-25	20	0.4675	1.9381
			25-30	65	0.4221	2.2579
			>30	14	0.4989	1.7174
		No A/C	<=10	13	0.6557	1.6794
			10-20	28	0.6254	2.9162
	Inland California	Central or Room A/C	<=25	226	0.5033	1.9210
			>25	83	0.8299	2.3534
		No A/C	<=10	17	0.5256	3.1920
			10-20	52	0.6649	2.1743
			20-25	13	1.0536	1.7110
			>25	14	0.8271	2.2646
Los Angeles	Los Angeles	Central or Room A/C	<=20	721	0.5894	1.8948
			20-25	273	1.1003	2.3648
			25-30	102	0.8128	2.4151
			>30	12	0.2664	2.7899
		No A/C	<=10	18	0.5427	3.0872
			10-20	390	0.7470	2.0852
			20-25	148	1.3718	2.2828
			>25	25	0.9884	1.9666
Philadelphia and Detroit	New York City	Central or Room A/C	<=10	20	0.7108	2.0184
			10-25	42	1.1392	2.6773
			>25	19	1.2435	2.1768
		No A/C	<=10	48	1.0165	2.1382
			10-20	59	0.7909	2.0417
			>20	32	1.6062	2.1189
Atlanta (No A/C)	Outside California	Central or Room A/C	<=10	179	0.9185	1.8589
			10-20	338	0.5636	1.9396
			20-25	253	0.4676	2.2011
			25-30	219	0.4235	2.0373
			>30	24	0.5667	1.9447
		No A/C	<=10	61	0.9258	2.0836
			10-20	87	0.7333	2.3299
			>20	44	1.3782	2.2757
Atlanta (A/C)	Research Triangle Park, NC	Central or Room A/C	<=10	157	0.9617	1.8094
			10-20	320	0.5624	1.9058
			20-25	196	0.3970	1.8887
			>25	145	0.3803	1.7092

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NO₂ removal rate

For this analysis, the same NO₂ removal rate distribution was used for all microenvironments that use the mass balance method. This removal rate is based on data

1 provided by Spicer et al. (1993). A total of 6 experiments, under variable source emission
2 characteristics including operation of gas stove, were conducted in an unoccupied test house. A
3 statistical distribution could not be described with the limited data, therefore a uniform
4 distribution was approximated by the bounds of the 6 values, a minimum of 1.02 and a maximum
5 of 1.45 h⁻¹.

6 ***Indoor source contributions***

7 A number of studies, as described in section 2.5.5 of the NO_x ISA, have noted the
8 importance of gas cooking appliances as sources of NO₂ emissions. An indoor emission source
9 term was included in the APEX simulations to estimate NO₂ exposure to gas cooking (hereafter
10 referred to as “indoor sources”). Three types of data were used to implement this factor:

- 11 • The fraction of households in the Philadelphia MSA that use gas for cooking fuel
- 12 • The range of contributions to indoor NO₂ concentrations that occur from cooking
13 with gas
- 14 • The diurnal pattern of cooking in households.

15 ***Households using gas for cooking fuel.*** The fraction of households in Philadelphia
16 County that use gas cooking fuel (i.e., 55%) was taken from the US Census Bureau’s American
17 Housing Survey for the Philadelphia Metropolitan Area: 2003.

18 ***Concentration Contributions.*** Data used for estimating the contribution to indoor NO₂
19 concentrations that occur during cooking with gas fuel were derived from a study sponsored by
20 the California Air Resources Board (CARB, 2001). For this study a test house was set up for
21 continuous measurements of NO₂ indoors and outdoors, among several other parameters, and
22 conducted under several different cooking procedures and stove operating conditions.

23 A uniform distribution of concentration contributions for input to APEX was estimated as
24 follows.

- 25 • The concurrent outdoor NO₂ concentration measurement was subtracted from each
26 indoor concentration measurement, to yield net indoor concentrations
- 27 • Net indoor concentrations for duplicate cooking tests (same food cooked the same
28 way) were averaged for each indoor room, to yield average net indoor concentrations
- 29 • The minimum and maximum average net indoor concentrations for any test in any
30 room were used as the lower and upper bounds of a uniform distribution

1 This resulted in a minimum average net indoor concentration of 4 ppb and a maximum
2 net average indoor concentration of 188 ppb.

3 ***Diurnal Pattern of Cooking Events.*** An analysis by Johnson et al (1999) of survey data
4 on gas stove usage collected by Koontz et al (1992) showed an average number of meals
5 prepared each day with a gas stove of 1.4. The diurnal allocation of these cooking events was
6 estimated as follows.

- 7 • Food preparation time obtained from CHAD diaries was stratified by hour of the day,
8 and summed for each hour, and summed for total preparation time.
- 9 • The fraction of food preparation occurring in each hour of the day was calculated as
10 the total number of minutes for that hour divided by the overall total preparation time.
11 The result was a measure of the probability of food preparation taking place during
12 any hour, given one food preparation event per day.
- 13 • Each hourly fraction was multiplied by 1.4, to normalize the expected value of daily
14 food preparation events to 1.4.

15 The estimated probabilities of cooking by hour of the day are presented in Table 31.
16 For this analysis it was assumed that the probability that food preparation would include stove
17 usage was the same for each hour of the day, so that the diurnal allocation of food preparation
18 events would be the same as the diurnal allocation of gas stove usage. It was also assumed that
19 each cooking event lasts for exactly 1 hour, implying that the average total daily gas stove usage
20 is 1.4 hours.

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Table 31. Probability of gas stove cooking by hour of the day.

Hour of Day	Probability of Cooking (%) ¹
0	0
1	0
2	0
3	0
4	0
5	5
6	10
7	10
8	10
9	5
10	5
11	5
12	10
13	5
14	5
15	5
16	15
17	20
18	15
19	10
20	5
21	5
22	0
23	0

¹Values rounded to the nearest 5%. Data sum to 145% due to rounding and scaling to 1.4 cooking events/day.

4

7.7.2.2 Microenvironments 2-7: All Other Indoor Microenvironments

6 The remaining five indoor microenvironments, which represent Bars and Restaurants,
7 Schools, Day Care Centers, Office, Shopping, and Other environments, were all modeled using
8 the same data and functions. As with the Indoor-Residence microenvironment, these
9 microenvironments use both AER and removal rates to calculate exposures within the
10 microenvironment. The air exchange rate distribution (GM = 1.109, GSD = 3.015, Min = 0.07,
11 Max = 13.8) was developed based on an indoor air quality study (Persily et al, 2005; see EPA,
12 2007g for details in derivation). The decay rate is the same as used in the Indoor-Residence
13 microenvironment discussed previously. The Bars and Restaurants microenvironment included
14 an estimated contribution from indoor sources as was described for the Indoor-Residence, only

1 there was an assumed 100% prevalence rate and the cooking with the gas appliance occurred at
2 any hour of the day.

3 **7.7.2.3 Microenvironments 8 and 9: Outdoor Microenvironments**

4 Two outdoor microenvironments, the Near Road and Public Garage/Parking Lot, used the
5 transfer factors method to calculate pollutant exposure. Penetration factors are not applicable to
6 outdoor environments (effectively, PEN=1). The distribution for proximity factors were
7 developed from the dispersion model estimated concentrations, using the relationship between
8 the on-road to receptor estimated concentrations.

9 **7.7.2.4 Microenvironment 10: Outdoors-General**

10 The general outdoor environment concentrations are well represented by the modeled
11 concentrations. Therefore, both the penetration factor and proximity factor for this
12 microenvironment were set to 1.

13 **7.7.2.5 Microenvironments 11 and 12: In Vehicle- Cars and Trucks, and Mass Transit**

14 Penetration factors were developed from data provided in Chan and Chung (2003).
15 Inside-vehicle and outdoor NO₂ concentrations were measured with for three ventilation
16 conditions, air-recirculation, fresh air intake, and with windows opened. Since major roads were
17 the focus of this assessment, reported indoor/outdoor ratios for highway and urban streets were
18 used here. Mean values range from about 0.6 to just over 1.0, with higher values associated with
19 increased ventilation (i.e., window open). A uniform distribution was selected for the
20 penetration factor for Inside-Cars/Trucks (ranging from 0.6 to 1.0) due to the limited data
21 available to describe a more formal distribution and the lack of data available to reasonably
22 assign potentially influential characteristics such as use of vehicle ventilation systems for each
23 location. Mass transit systems, due to the frequent opening and closing of doors, was assigned a
24 point estimate of 1.0 based on the reported mean values for open windows ranging from 0.96 and
25 1.0. Proximity factors were developed from the dispersion model estimated concentrations,
26 using the relationship between the on-road to receptor estimated concentrations.

1 **7.8 EXPOSURE AND HEALTH RISK CALCULATIONS**

2 APEX calculates the time series of exposure concentrations that a simulated individual
3 experiences during the simulation period. APEX determines the exposure using hourly ambient
4 air concentrations, calculated concentrations in each microenvironment based on these ambient
5 air concentrations (and indoor sources if present), and the minutes spent in a sequence of
6 microenvironments visited according to the composite diary. The hourly exposure concentration
7 at any clock hour during the simulation period is determined using the following equation:

8
$$C_i = \frac{\sum_{j=1}^N C_{ME(j)}^{hourlymean} t_{(j)}}{T}$$

9 where,

10 C_i = Hourly exposure concentration at clock hour i of the simulation period
11 (ppb)

12 N = Number of events (i.e., microenvironments visited) in clock hour i of
13 the simulation period.

14 $C_{ME(j)}^{hourlymean}$ = Hourly mean concentration in microenvironment j (ppm)

15 $t_{(j)}$ = Time spent in microenvironment j (minutes)

16 T = 60 minutes

17
18 From the hourly exposures, APEX calculates time series of 1-hour average exposure
19 concentrations that a simulated individual would experience during the simulation period.
20 APEX then statistically summarizes and tabulates the hourly (or daily, annual average)
21 exposures. In this analysis, the exposure indicator is 1-hr exposures above selected health effect
22 benchmark levels. From this, APEX can calculate two general types of exposure estimates:
23 counts of the estimated number of people exposed to a specified NO₂ concentration level and the
24 number of times per year that they are so exposed; the latter metric is in terms of person-
25 occurrences or person-days. The former highlights the number of individuals exposed at least
26 *one or more* times per modeling period to the health effect benchmark level of interest. APEX
27 can also report counts of individuals with multiple exposures. This person-occurrences measure

1 estimates the number of times per season that individuals are exposed to the exposure indicator
2 of interest and then accumulates these estimates for the entire population residing in an area.

3 APEX tabulates and displays the two measures for exposures above levels ranging from
4 200 to 300 ppb by 50 ppb increments for 1-hour average exposures. These results are tabulated
5 for the population and subpopulations of interest.

6 To simulate just meeting the current standard, dispersion modeled concentration were not
7 rolled-up as done in the air quality characterization. A proportional approach was used as done
8 in the Air Quality Characterization, but to reduce processing time, the potential health effect
9 benchmark levels were proportionally reduced by the similar factors described for each specific
10 location and simulated year. Since it is a proportional adjustment, the end effect of adjusting
11 concentrations upwards versus adjusting benchmark levels downward within the model is the
12 same. The difference in the exposure and risk modeling was that the modeled air quality
13 concentrations were used to generate the adjustment factors. Table 32 provides the adjustment
14 factors used and the adjusted potential health effect benchmark concentrations to simulate just
15 meeting the current standard. When modeling indoor sources, the indoor concentration
16 contributions needed to be scaled downward by the same proportions.

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Table 32. Adjustment factors and potential health effect benchmark levels used by APEX to simulate just meeting the current standard.

Simulated Year (factor)	Potential Health Effect Benchmark Level (ppb)	
	Actual	Adjusted
2001 (1.59)	150	94
	200	126
	250	157
	300	189
2002 (1.63)	150	92
	200	122
	250	153
	300	184
2003 (1.64)	150	91
	200	122
	250	152
	300	183

5

6 **7.9 EXPOSURE MODELING AND HEALTH RISK**
7 **CHARACTERIZATION RESULTS**

8 **7.9.1 Overview**

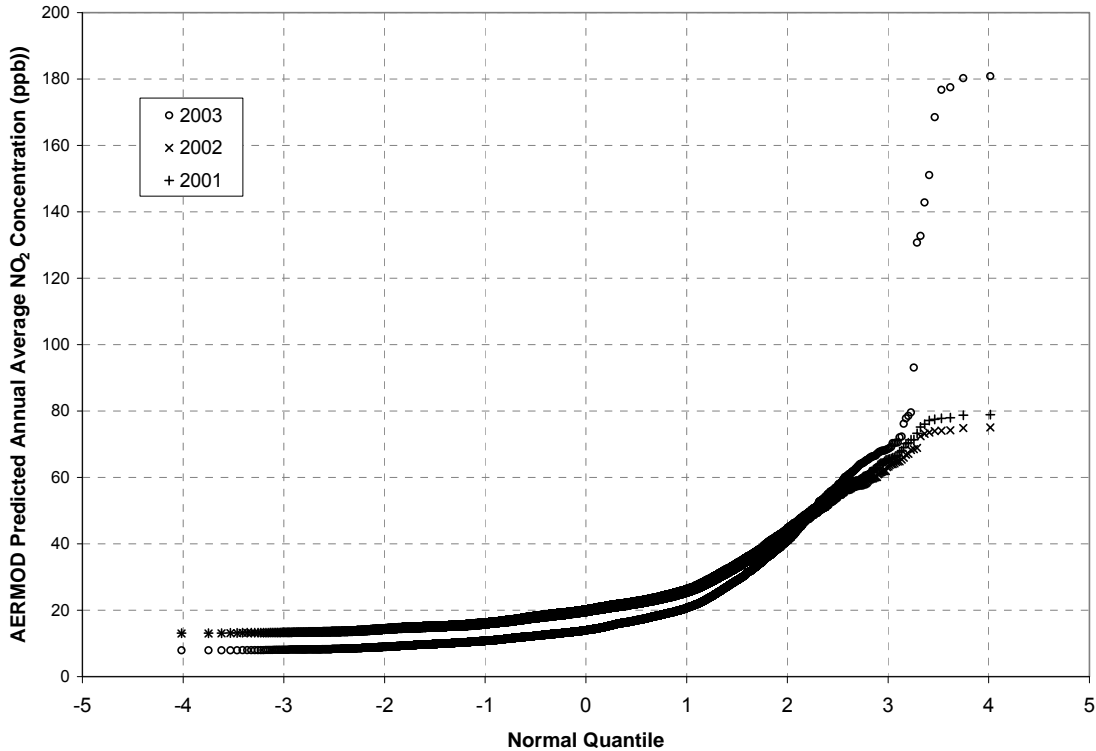
9 The results of the exposure and risk characterization are presented here for Philadelphia
10 County. Several scenarios were considered for the exposure assessment, including two
11 averaging time for NO₂ concentrations (annual and 1-hour), inclusion of indoor sources, and for
12 evaluating just meeting the current standard. To date, year 2002 served as the base year for all
13 scenarios, years 2001 and 2003 were only evaluated for a limited number of scenarios.
14 Exposures were simulated for four groups; children and all persons, and the asthmatic population
15 within each of these.

16 The exposure results summarized below focus on the population group where exposure
17 estimations are of greatest interest, namely asthmatic individuals. However, due to certain
18 limitations in the data summaries output from APEX, some exposure data could only be output
19 for the entire population modeled (i.e., all persons - includes asthmatics and healthy persons of

1 all ages). The summary data for the entire population (e.g., annual average exposure
2 concentrations, time spent in microenvironments at or above a potential health effect benchmark
3 level) can be representative of the asthmatic population since the asthmatic population does not
4 have its microenvironmental concentrations and activities estimated any differently from those of
5 the total population.

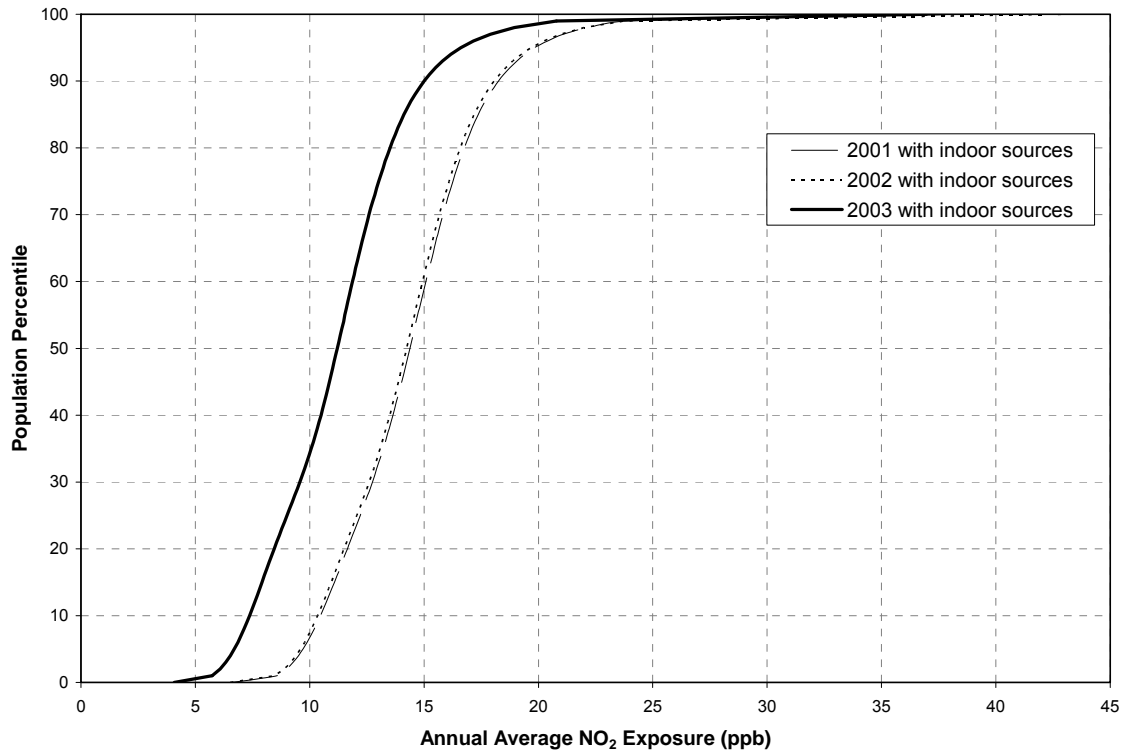
6 **7.9.2 Annual Average Exposure Concentrations (as is)**

7 Since the current NO₂ standard is 0.053 ppm annual average, the predicted air quality
8 concentrations, the measured ambient monitoring concentrations, and the estimated exposures
9 were summarized by annual average concentration. The distribution for the AERMOD predicted
10 NO₂ concentrations at each of the 16,857 receptors for years 2001 through 2003 are illustrated in
11 Figure 5. Variable concentrations were estimated by the dispersion model over the three year
12 period (2001-2003). The NO₂ concentration distribution was similar for years 2001 and 2002,
13 with mean annual average concentrations of about 21 ppb and a COV of just over 30%. On
14 average, NO₂ annual average concentrations were lowest during simulated year 2003 (mean
15 annual average concentration was about 16 ppb), largely a result of the comparably lower local
16 concentration added (Table 26). While the mean annual average concentrations were lower than
17 those estimated for 2001 and 2002, a greater number of annual average concentrations were
18 estimated above 53 ppb for year 2003. In addition, year 2003 also contained greater variability
19 in annual average concentrations as indicated by a COV of 53%.



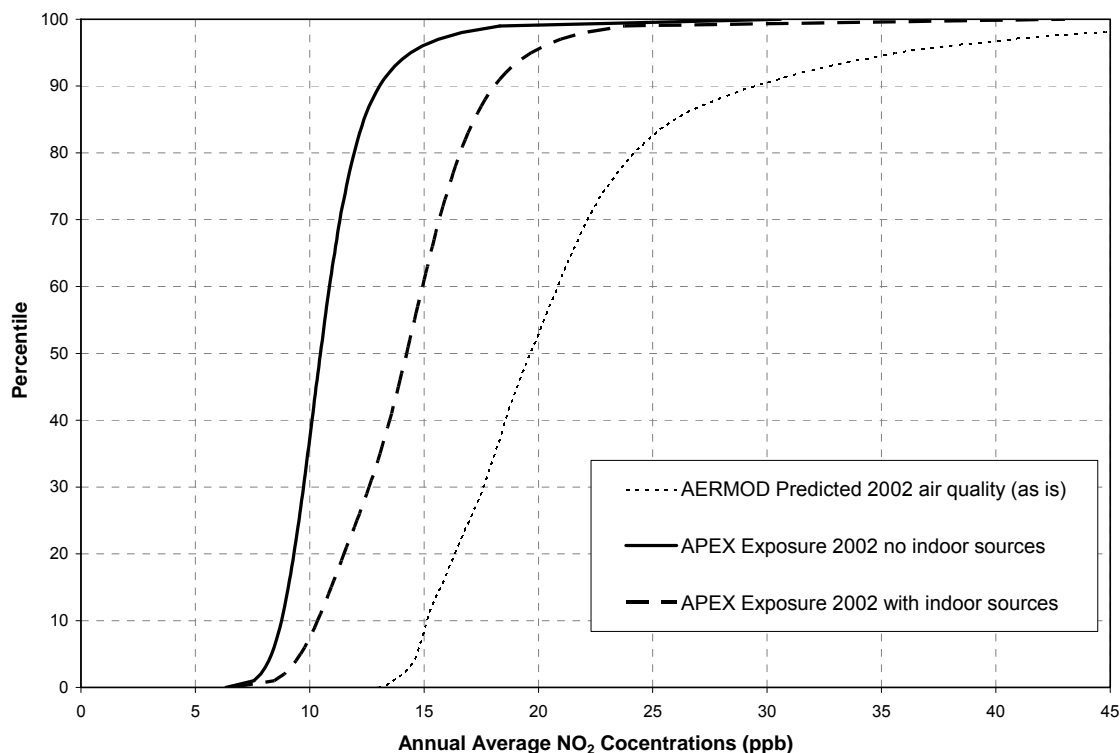
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 2 **Figure 5. Distribution of AERMOD predicted annual average NO₂ concentrations at each**
 3 **of the 16,857 receptors in Philadelphia County for years 2001-2003.**
 4

5 The hourly concentrations output from AERMOD were input into the exposure model,
 6 providing a range of estimated exposures output by APEX. Figure 6 illustrates the annual
 7 average exposure concentrations for the entire simulated population (both asthmatics and healthy
 8 individual of all ages), for each of the years analyzed and where indoor sources were modeled.
 9 While years 2001 and 2002 contained very similar population exposure concentration
 10 distributions, the modeled year 2003 contained about 20% lower annual average concentrations.
 11 The lower exposure concentrations for year 2003 are similar to what was observed for the
 12 predicted air quality (Figure 5), however, all persons were estimated to contain exposures below
 13 an annual average concentration of 53 ppb, even considering indoor source concentration
 14 contributions. Again, while the figure summarizes the entire population, the data are
 15 representative of what would be observed for the population of asthmatics or asthmatic children.
 16



1
 2 **Figure 6. Estimated annual average total NO₂ exposure concentrations for all simulated**
 3 **persons in Philadelphia County, using modeled 2001-2003 air quality (as is), with**
 4 **modeled indoor sources.**

5
 6 The AERMOD predicted air quality and the estimated exposures for year 2002 were
 7 compared using their respective annual average NO₂ concentrations (Figure 7). As a point of
 8 reference, the annual average concentration for 2002 ambient monitors ranged from 24 ppb to 29
 9 ppb. Many of the AERMOD predicted annual average concentrations were below that of the
 10 lowest ambient monitoring concentration of 24 ppb, although a few of the receptors contained
 11 concentrations above the highest measured annual average concentration. Estimated exposure
 12 concentrations were below that of both the modeled and measured air quality. For example,
 13 exposure concentrations were about 5 ppb less than the modeled air quality when the exposure
 14 estimation included indoor sources, and about 10 ppb less for when exposures were estimated
 15 without indoor sources. In comparing the estimated exposures with and without indoor sources,
 16 indoor sources were estimated to contribute between 1 and 5 ppb to the total annual average
 17 exposures.



1
 2 **Figure 7. Comparison of AERMOD predicted and ambient monitoring annual average**
 3 **NO₂ concentrations (as is) and APEX exposure concentrations (with and without**
 4 **modeled indoor sources) in Philadelphia County for year 2002.**

5 **7.9.3 One-Hour Exposures (as is)**

6 Since there is interest in short-term exposures, a few analyses were performed using the
 7 APEX estimated exposure concentrations. As part of the standard analysis, APEX reports the
 8 maximum exposure concentration for each simulated individual in the simulated population.
 9 This can provide insight into the proportion of the population experiencing any NO₂ exposure
 10 concentration level of interest. In addition, exposures are estimated for each of the selected
 11 potential health effect benchmark levels (200, 250, and 300 ppb, 1-hour average). An
 12 exceedance was recorded when the maximum exposure concentration observed for the individual
 13 was above the selected level in a day (therefore, the maximum number of exceedances is 365 for
 14 a single person). Estimates of repeated exposures are also recorded, that is where 1-hour
 15 exposure concentrations were above a selected level in a day added together across multiple days
 16 (therefore, the maximum number of multiple exceedances is also 365). Persons of interest in this
 17 exposure analysis are those with particular susceptibility to NO₂ exposure, namely individuals
 18 with asthma. The health effect benchmark levels are appropriate for estimating the potential risk

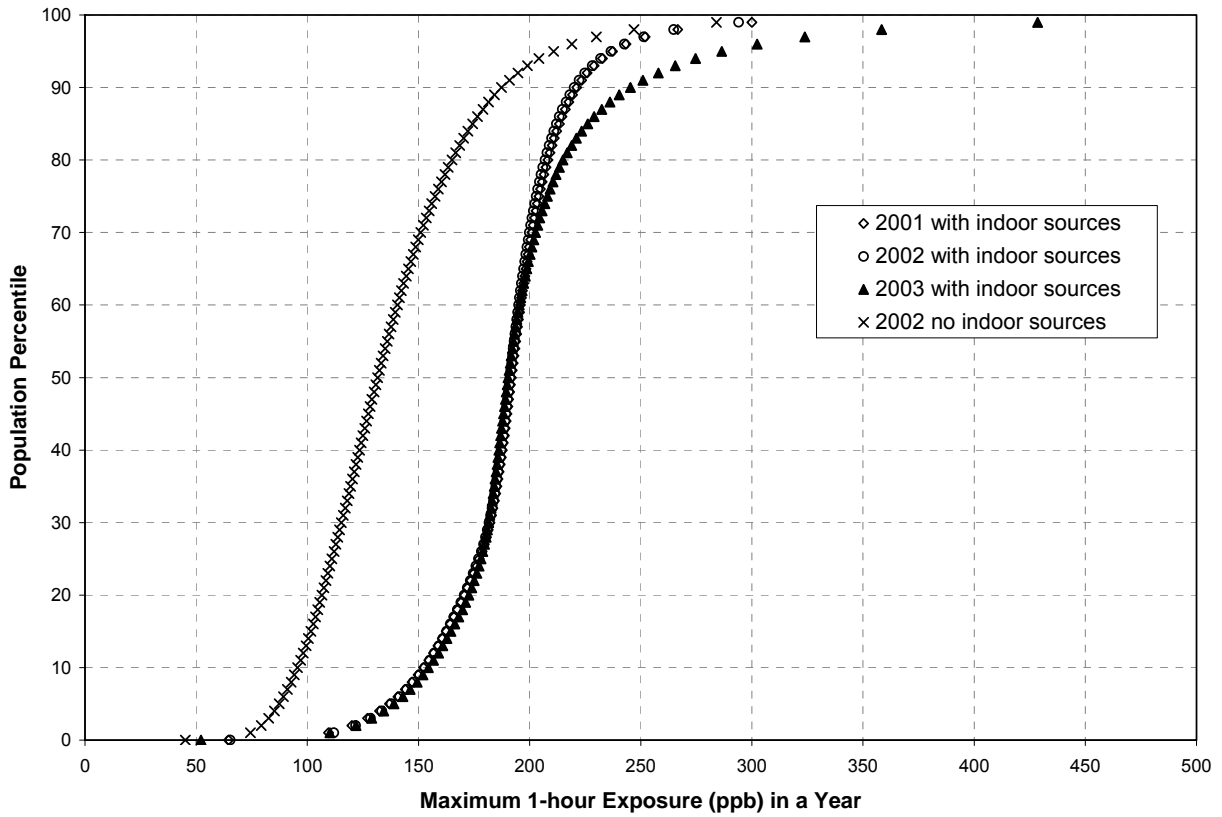
1 of adverse health effects for asthmatics. The majority of the results presented in this section are
2 for the simulated asthmatic population. However, the exposure analysis was performed for the
3 total population to assess numbers of persons exposed to these levels and to provide additional
4 information relevant to the asthmatic population (such as time spent in particular
5 microenvironments).

6 ***7.9.3.1 Maximum Estimated Exposure Concentrations***

7 A greater variability was observed in maximum exposure concentrations for the 2003
8 year simulation compared with years 2001 and 2002 (Figure 8). While annual average exposure
9 concentrations for the total population were the lowest of the 3-year simulation, year 2003
10 contained a greater number of individual maximum exposures at and above the lowest potential
11 health effect benchmark level. When indoor sources are not modeled however, over 90% of the
12 simulated persons do not have an occurrence of a 1-hour exposure above 200 ppb in a year.

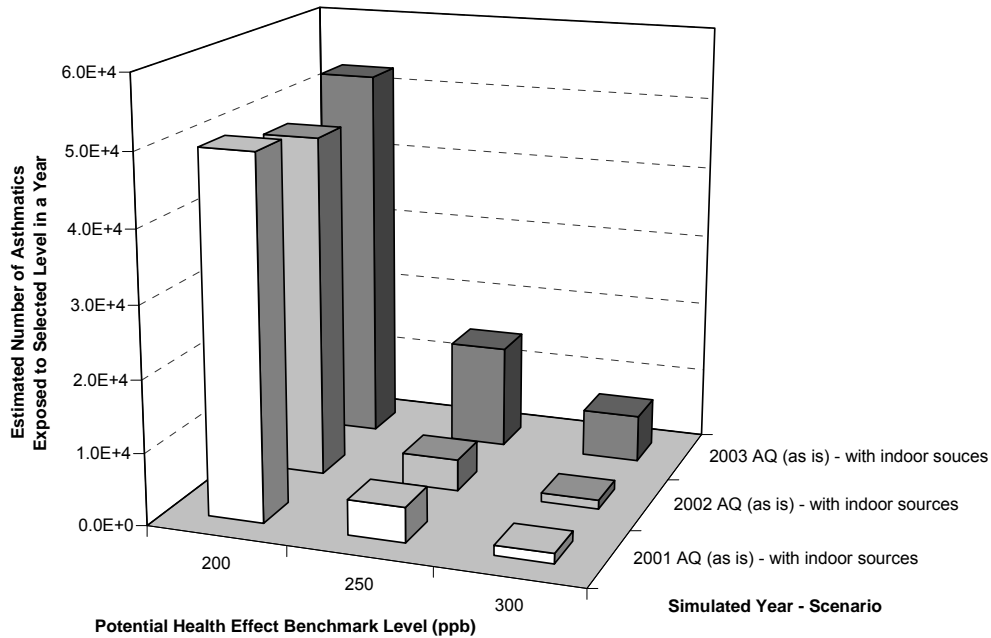
13 ***7.9.3.2 Number of Estimated Exposures above Selected Levels***

14 When considering the total asthmatic population simulated in Philadelphia County and using
15 current air quality of 2001-2003, nearly 50,000 persons were estimated to be exposed at least one
16 time to a one-hour concentration of 200 ppb in a year (Figure 9). These exposures include both
17 the NO₂ of ambient origin and that contributed by indoor sources. The number of asthmatics
18 exposed to greater concentrations (e.g., 250 or 300 ppb) drops dramatically and is estimated to be
19 somewhere between 1,000 – 15,000 depending on the 1-hour concentration level and the year of
20 air quality data used. Exposures simulated for year 2003 contained the greatest number of
21 asthmatics exposed in a year consistently for all potential health effect benchmark levels, while
22 year 2002 contained the lowest number of asthmatics. Similar trends across the benchmark
23 levels and the simulation years were observed for asthmatic children, albeit with lower numbers
24 of asthmatic children with exposures at or above the potential health effect benchmark levels.
25 For example, nearly 12,000 were estimated to be exposed to at least a one-hour NO₂
26 concentration of 200 ppb in a year (Figure 10). Additional exposure estimates were

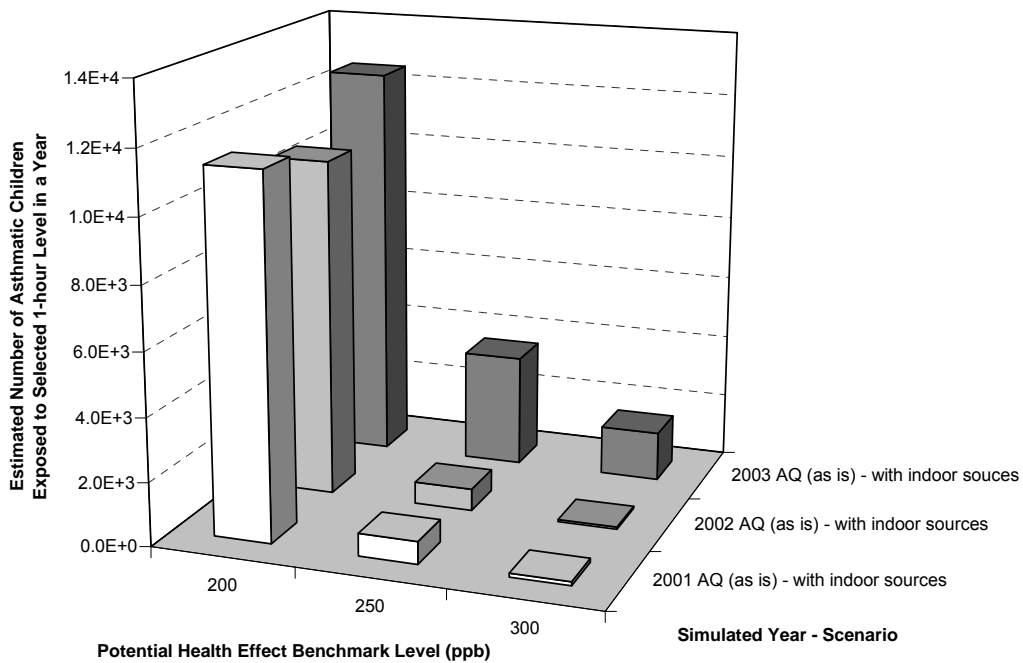


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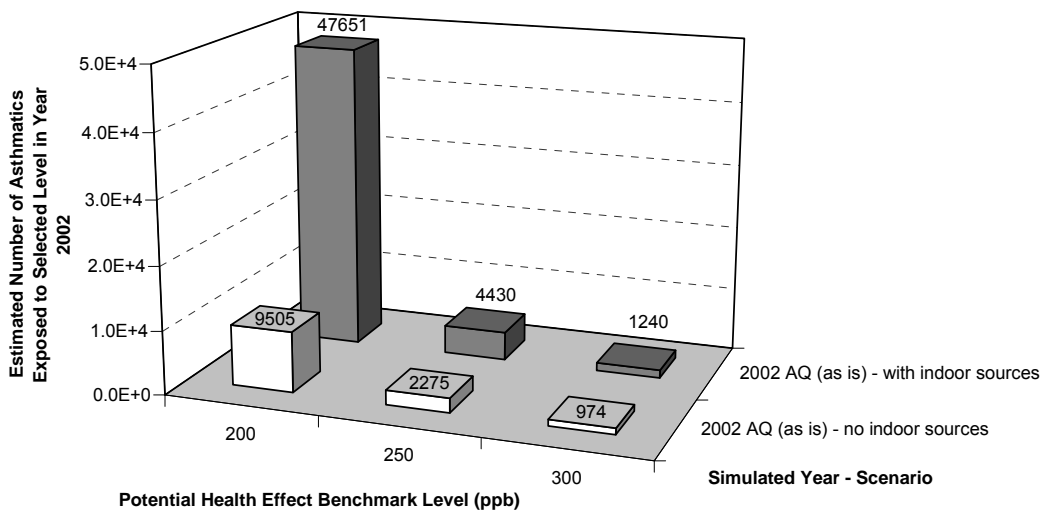
Figure 8. Estimated maximum NO₂ exposure concentration for all simulated persons in Philadelphia County, using modeled 2001-2003 air quality (as is), with and without modeled indoor sources. Values above the 99th percentile are not shown.



1
 2 **Figure 9. Estimated number of all simulated asthmatics in Philadelphia County with at**
 3 **least one NO₂ exposure at or above the potential health effect benchmark levels,**
 4 **using modeled 2001-2003 air quality (as is), with modeled indoor sources.**
 5



6
 7 **Figure 10. Estimated number of simulated asthmatic children in Philadelphia County with**
 8 **at least one NO₂ exposure at or above the potential health effect benchmark**
 9 **levels, using modeled 2001-2003 air quality (as is), with modeled indoor sources.**



1
 2 **Figure 11. Comparison of the estimated number of all simulated asthmatics in**
 3 **Philadelphia County with at least one NO₂ exposure at or above potential health effect**
 4 **benchmark levels, using modeled 2002 air quality (as is) , with and without modeled indoor**
 5 **sources.**
 6

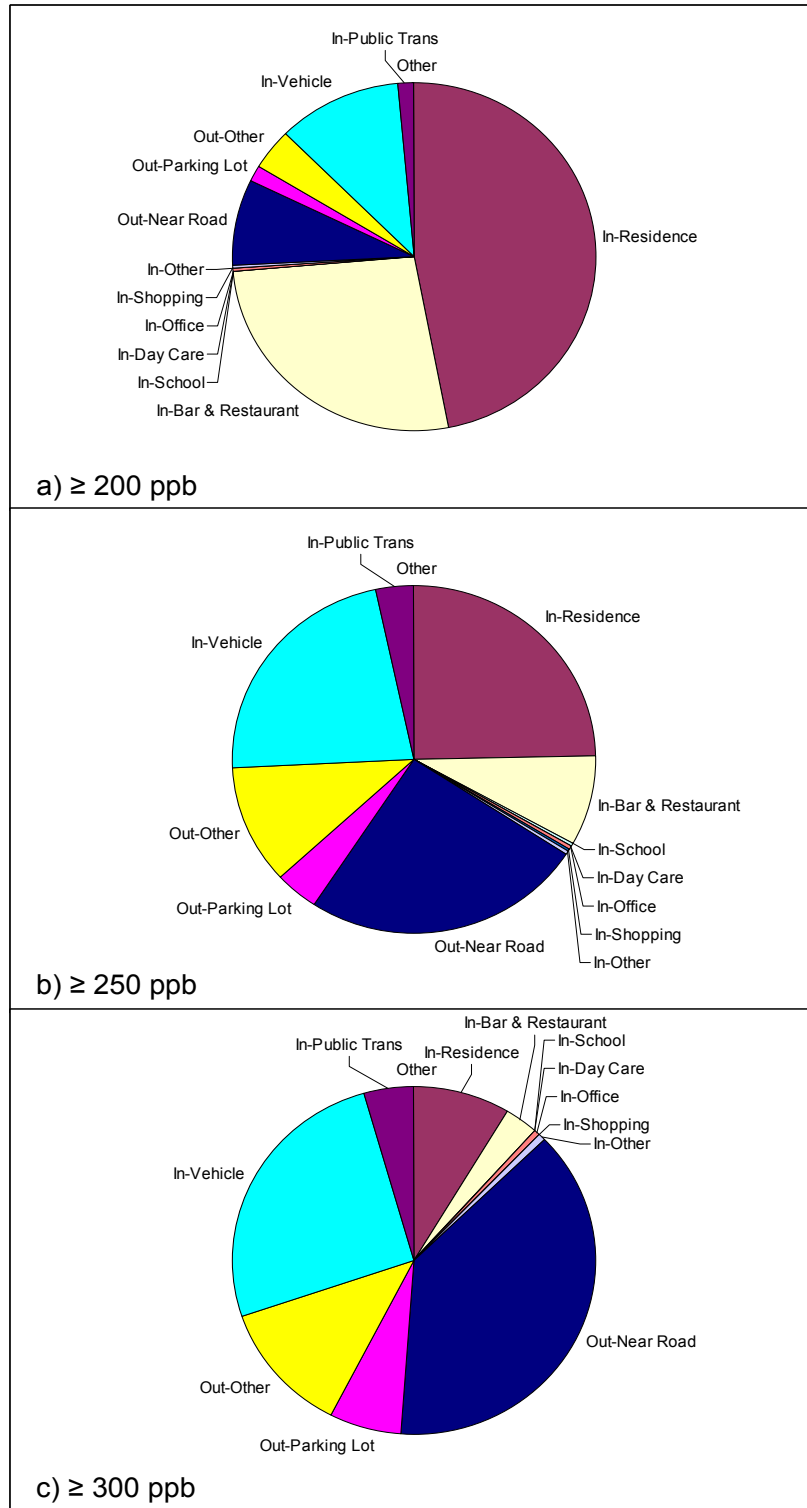
7 generated using the modeled 2002 air quality (as is) and where the contribution from indoor
 8 sources was not included in the exposure concentrations. APEX allows for the same persons to
 9 be simulated, i.e., demographics of the population were conserved, as well as using the same
 10 individual time-location-activity profiles generated for each person. Figure 11 compares the
 11 estimated number of asthmatics experiencing exposures above the potential health effect
 12 benchmarks, both with indoor sources and without indoor sources included in the model runs.
 13 The number of asthmatics at or above the selected concentrations is reduced by between 50-80%,
 14 depending on benchmark level, when not including indoor source (i.e., gas cooking)
 15 concentration contributions.

16 An evaluation of the time spent in the 12 microenvironments was performed to estimate
 17 where simulated individuals are exposed to concentrations above the potential health effect
 18 benchmark levels. Currently, the output generated by APEX is limited to compiling the
 19 microenvironmental time for the total population (includes both asthmatic individuals and
 20 healthy persons) and is summarized to the total time spent above the selected potential health

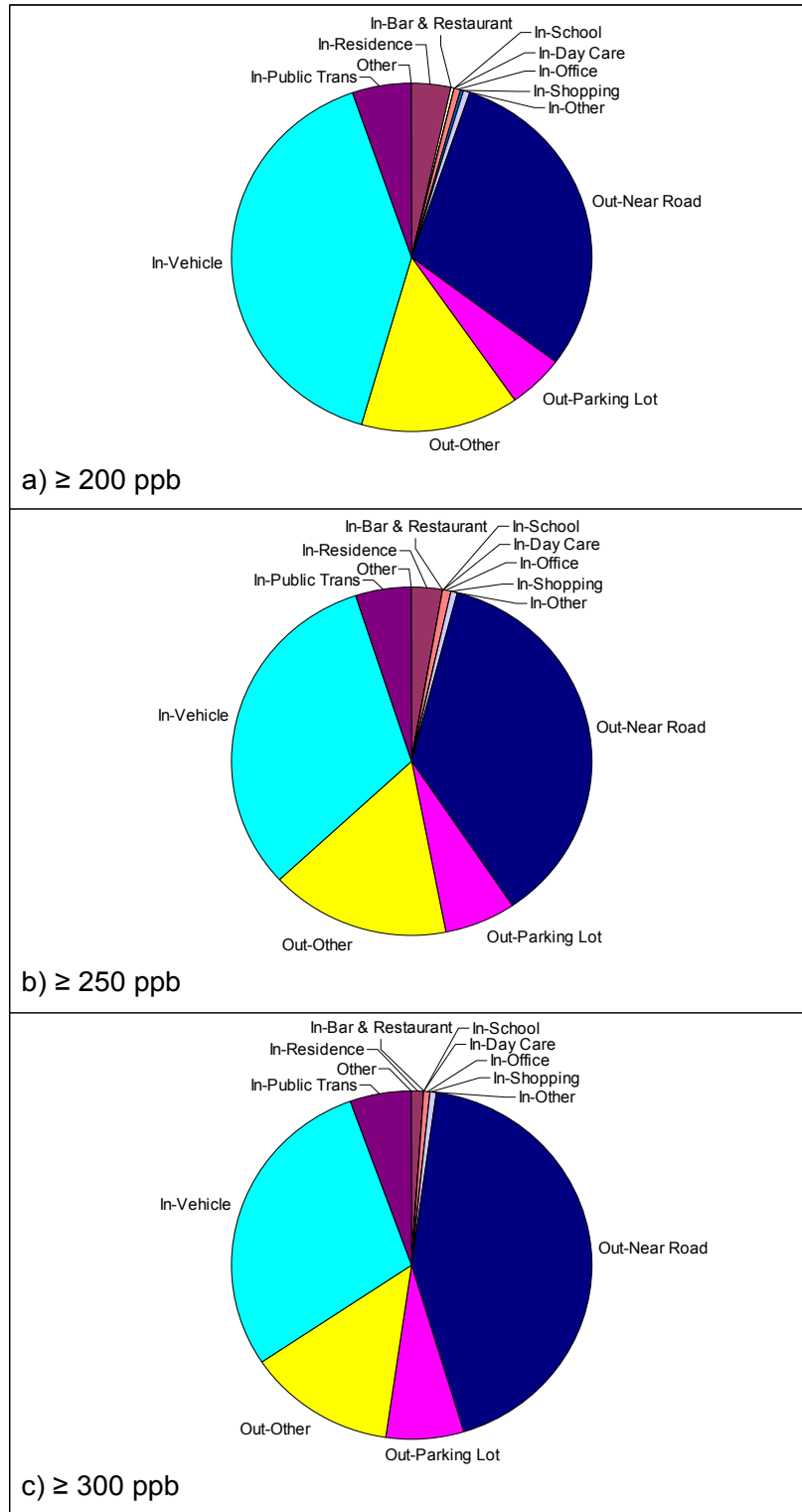
1 effect benchmark levels. As mentioned above, the data still provide a reasonable approximation
2 for each of the population subgroups (e.g., asthmatics or asthmatic children) since their
3 microenvironmental concentrations and activities are not estimated any differently from those of
4 the total population by APEX.

5 As an example, Figure 12 (a, b, c) summarizes the percent of total time spent in each
6 microenvironment for simulation year 2002 that was associated with estimated exposure
7 concentrations at or above 200, 250, and 300 ppb (results for years 2001 and 2003 were similar).
8 Estimated exposures included the contribution from one major category of indoor sources (i.e.,
9 gas cooking). The time spent in the indoor residence and bars/restaurants were the most
10 important for concentrations ≥ 200 ppb, contributing to approximately 75% of the time persons
11 were exposed (Figure 12a). This is likely a result of the indoor source concentration contribution
12 to each individual's exposure concentrations. The importance of the particular
13 microenvironment however changes with differing potential health effect benchmark levels.
14 This is evident when considering the in-vehicle and outdoor near-road microenvironments,
15 progressing from about 19% of the time exposures were at the lowest potential health effect
16 benchmark level (200 ppb) to a high of 64% of the time exposures were at the highest
17 benchmark level (300 ppb, Figure 12c).

18 The microenvironments where higher exposure concentrations occur were also evaluated
19 for the exposure estimates generated without indoor source contributions. Figure 13 illustrates
20 that the time spent in the indoor microenvironments contributes little to the estimated exposures
21 above the selected benchmark levels. The contribution of these microenvironments varied only
22 slightly with increasing benchmark concentration, ranging from about 2-5%. Most of the time
23 associated with high exposures was associated with the transportation microenvironments (In-
24 Vehicle or In-Public Transport) or outdoors (Out-Near Road, Out-Parking Lot, Out-Other). The
25



1
 2 **Figure 12. Fraction of time all simulated persons in Philadelphia County spend in the**
 3 **twelve microenvironments associated with the potential NO₂ health effect**
 4 **benchmark levels, a) ≥ 200 ppb, b) ≥ 250 ppb, and c) ≥ 300 ppb, year 2002**
 5 **simulation with indoor sources.**



1
 2 **Figure 13. Fraction of time all simulated persons in Philadelphia County spend in the**
 3 **twelve microenvironments associated with the potential NO₂ health effect**
 4 **benchmark levels, a) ≥ 200 ppb, b) ≥ 250 ppb, and c) ≥ 300 ppb, year 2002**
 5 **simulation without indoor sources.**

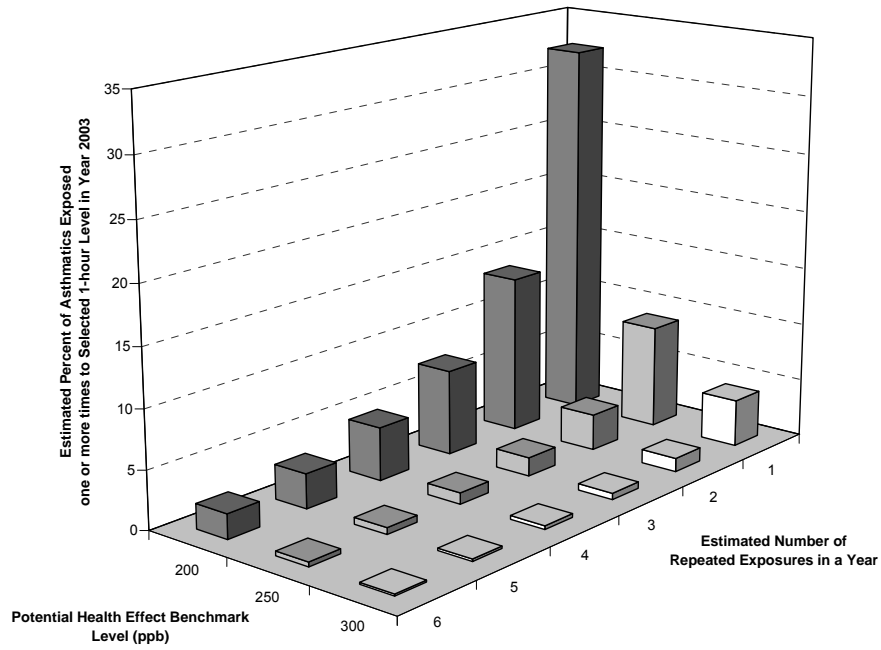
1 importance of time spent outdoors near roadways exhibited the greatest change in contribution
2 with increased health benchmark level, increasing from around 30 to 44% of time associated
3 with concentrations of 200 and 300 ppb, respectively.

4 ***7.9.3.3 Number of Repeated Exposures Above Selected Levels***

5 In the analysis of persons exposed, the results show the number or percent of those with
6 at least one exposure at or above the selected potential health effect benchmark level. Given that
7 the benchmark is for a small averaging time (i.e., one-hour) it may be possible that individuals
8 are exposed to concentrations at or above the potential health effect benchmark levels more than
9 once in a given year. Since APEX simulates the longitudinal diary profile for each individual,
10 the number of times above a selected level is retained for each person. Figure 14 presents such
11 an analysis for the year 2003, the year containing the greatest number of exposure concentrations
12 at or above the selected benchmarks. Estimated exposures include both those resulting from
13 exposures to NO₂ of ambient origin and those resulting from indoor source NO₂ contributions.
14 While a large fraction of individuals experience at least one exposure to 200 ppb or greater over
15 a 1-hour time period in a year (about 32 percent), only around 14 percent were estimated to
16 contain at least 2 exposures. Multiple exposures at or above the selected benchmarks greater
17 than or equal to 3 or more times per year are even less frequent, with around 5 percent or less of
18 asthmatics exposed to 1-hour concentrations greater than or equal to 200 ppb 3 or more times in
19 a year.

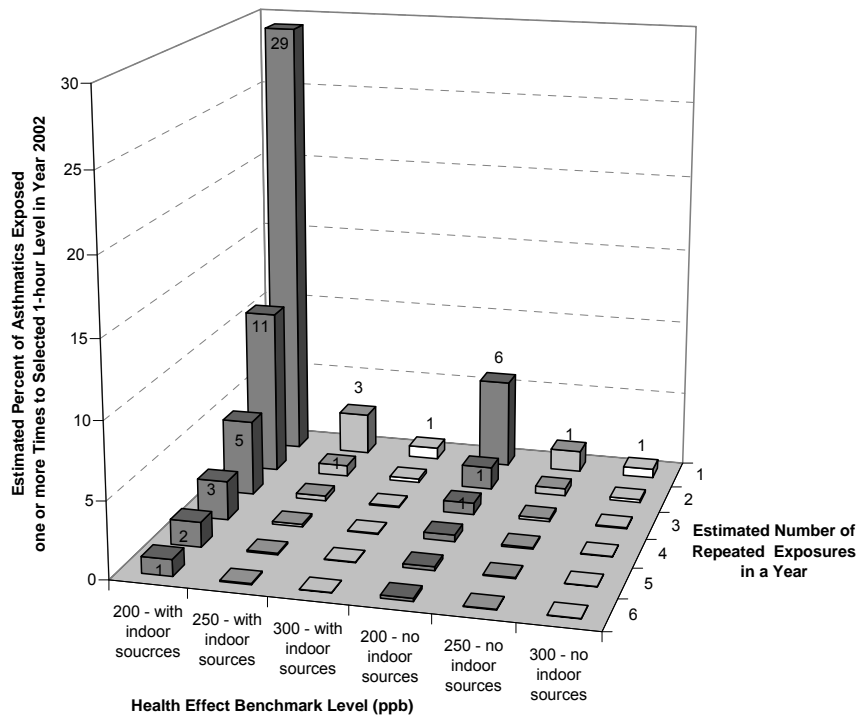
20 Exposure estimates for year 2002 are presented to provide an additional perspective,
21 including a lower bound of repeated exposures for this population subgroup and for exposure
22 estimates generated with and without modeled indoor sources (Figure 15). Most asthmatics
23 exposed to a 200 ppb concentration are exposed once per year and only around 11 percent would
24 experience 2 or more exposures at or above 200 ppb when including indoor source contributions.
25 The percent of asthmatics experiencing multiple exposures at and above 250 and 300 ppb is
26 much lower, typically less than 1 percent of all asthmatics are exposed at the higher potential
27 benchmark levels. Also provided in Figure 14 are the percent of asthmatics exposed to selected
28 levels in the absence of indoor sources. Again, without the indoor source contribution, there are
29 reduced occurrences of multiple exposures at all of the potential health effect benchmark levels
30 compared with when indoor sources were modeled.

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Figure 14. Estimated percent of all asthmatics in Philadelphia County with repeated NO₂ exposures above potential health effect benchmark levels, using 2003 modeled air quality (as is), with modeled indoor sources.



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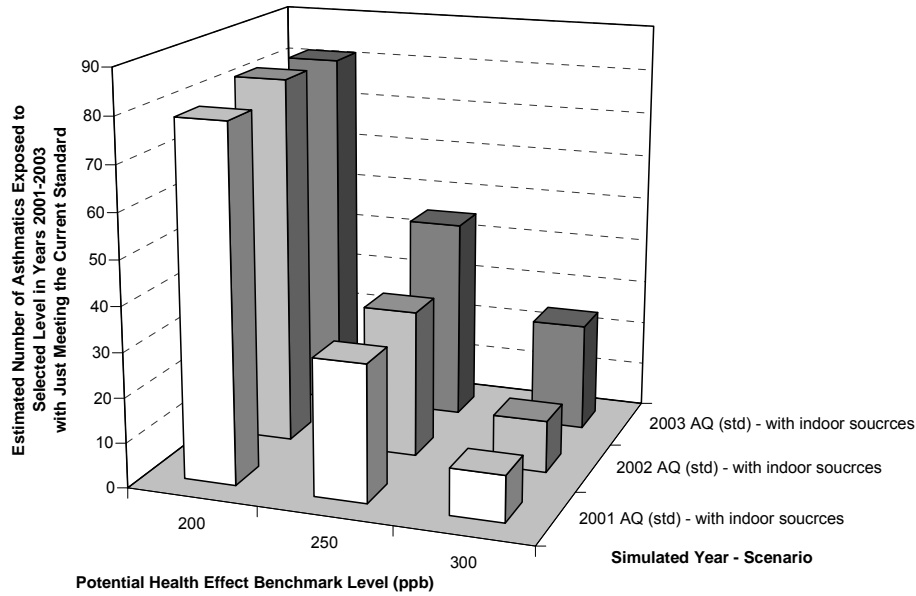
1 **Figure 15. Estimated percent of all asthmatics in Philadelphia County with repeated NO₂**
2 **exposures above potential health effect benchmark levels, using modeled 2002 air**
3 **quality (as is), with and without indoor sources.**

4 **7.9.4 One-Hour Exposures Associated with Just Meeting the Current Standard**

5 To simulate just meeting the current NO₂ standard, the potential health effect
6 benchmark level was adjusted in the exposure model, rather than adjusting all of the hourly
7 concentrations for each receptor and year simulated (see section 5.4.2 and section 7.8 above).
8 Similar estimates of short-term exposures (i.e., 1-hour) were generated for the total population
9 and population subgroups of interest (i.e., asthmatics and asthmatic children).

10 *7.9.4.1 Number of Estimated Exposures above Selected Levels*

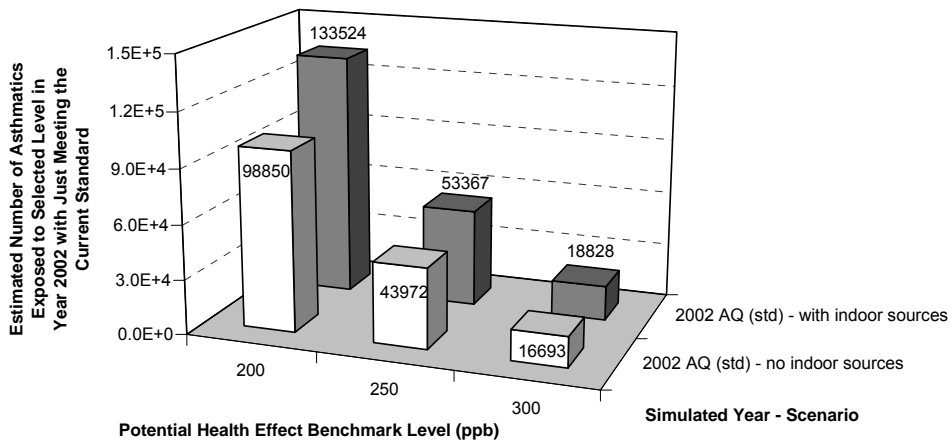
11 In considering exposures estimated to occur associated with air quality simulated to just
12 meet the current annual average NO₂ standard, the number of persons experiencing
13 concentrations at or above the potential health effect benchmarks increased. To allow for
14 reasonable comparison, the number of persons affected considering each scenario is expressed as
15 the percent of the subpopulation of interest. Figure 16 illustrates the percent of asthmatics
16 estimated to experience at least one exposure at or above the selected potential health effect
17 benchmark concentrations, with just meeting the current standard and including indoor source
18 contributions. While it was estimated that about 30% percent of asthmatics would be exposed to
19 200 ppb (1-hour average) at least once in a year for as is air quality, it was estimated that around
20 80 percent of asthmatics would experience at least one concentration above the lowest potential
21 health effect benchmark level in a year representing just meeting the current standard. Again,
22 estimates for asthmatic children exhibited a similar trend, with between 75 to 80 percent exposed
23 to a concentration at or above the lowest potential health effect benchmark level at least once per
24 year for a year just meeting the current standard (data not shown). The percent of all asthmatics
25 experiencing the higher benchmark levels is reduced to between 31 and 45 percent for the 250
26 ppb, 1-hour benchmark, and between 10 and 24 percent for the 300 ppb, 1-hour benchmark level
27 associated with air quality representing just meeting the current annual average standard.
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Figure 16. Estimated percent of all asthmatics in Philadelphia with at least one exposure at or above the potential health effect benchmark level, using modeled 2001-2003 air quality just meeting the current standard, with modeled indoor sources.

In evaluating the influence of indoor source contribution for the scenario just meeting the current standard, the numbers of individuals exposed at selected levels are reduced without indoor sources, ranging from about 26 percent lower for the 200 ppb level to around 11 percent for the 300 ppb level when compared with exposure estimates that accounted for indoor sources (Figure 17).



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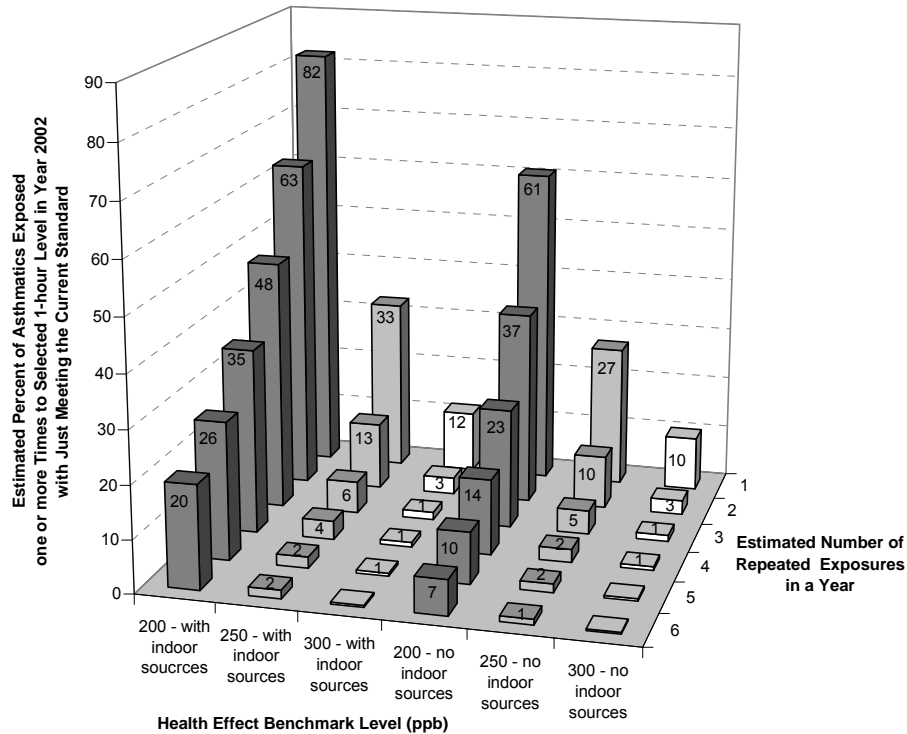
Figure 17. Estimated number of all asthmatics in Philadelphia with at least one exposure at or above the potential health effect benchmark level, using modeled 2002 air quality just meeting the current standard, with and without modeled indoor sources.

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7.9.4.2 Number of Repeated Exposures Above Selected Levels

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For air quality simulated to just meet the current standard, repeated exposures at the selected potential health effect benchmarks are more frequent than that estimated for the modeled as is air quality. Figure 19 illustrates this using the simulated asthmatic population for year 2002 data as an example. Many asthmatics that are exposed at or above the selected levels are exposed more than one time. Repeated exposures above the potential health effect benchmark levels are reduced however, when not including the contribution from indoor sources. The percent of asthmatics exposed drops with increasing benchmark level, with progressively fewer persons experiencing multiple exposures for each benchmark level.



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4 **Figure 18. Estimated percent of asthmatics in Philadelphia County with repeated**
5 **exposures above health effect benchmark levels, using modeled 2002 air quality**
6 **just meeting the current standard, with and without modeled indoor sources.**
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9 **7.10 VARIABILITY AND UNCERTAINTY**

10 **7.10.1 Introduction**

11 The methods and the model used in this assessment conform to the most contemporary
12 modeling methodologies available. APEX is a powerful and flexible model that allows for the
13 realistic estimation of air pollutant exposure to individuals. Since it is based on human activity
14 diaries and accounts for the most important variables known to affect exposure, it has the ability
15 to effectively approximate actual conditions. In addition, the input data selected were the best
16 available data to generate the exposure results. However, there are constraints and uncertainties
17 with the modeling approach and the input data that limit the realism and accuracy of the model
18 results.

1 All models have limitations that require the use of assumptions. Limitations of APEX lie
2 primarily in the uncertainties associated with data distributions input to the model. Broad
3 uncertainties and assumptions associated with these model inputs, utilization, and application
4 include the following, with more detailed analysis summarized below and presented previously
5 (see EPA, 2007g; Langstaff, 2007). In addition, while at this time only the analyses for
6 Philadelphia County were complete, uncertainties are discussed regarding some of the location-
7 specific input gathered to date. Identified uncertainties include:

- 8
9 • The CHAD activity data used in APEX are compiled from a number of studies in
10 different areas, and for different seasons and years. Therefore, the combined data set
11 may not constitute a representative sample for a particular study scenario.
- 12 • Commuting pattern data were derived from the 2000 U.S. Census. The commuting
13 data address only home-to-work travel. The population not employed outside the
14 home is assumed to always remain in the residential census tract. Furthermore,
15 although several of the APEX microenvironments account for time spent in travel, the
16 travel is assumed to always occur in basically a composite of the home and work
17 block. No other provision is made for the possibility of passing through other blocks
18 during travel.
- 19 • APEX creates seasonal or annual sequences of daily activities for a simulated
20 individual by sampling human activity data from more than one subject. Each
21 simulated person essentially becomes a composite of several actual people in the
22 underlying activity data.
- 23 • The APEX model currently does not capture certain correlations among human
24 activities that can impact microenvironmental concentrations (for example, cigarette
25 smoking leading to an individual opening a window, which in turn affects the amount
26 of outdoor air penetrating the microenvironment).
- 27 • Certain aspects of the personal profiles are held constant, though in reality they
28 change as individuals age. This is only important for simulations with long
29 timeframes, particularly when simulating young children (e.g., over a year or more).

1 **7.10.2 Input Data Evaluation**

2 Modeling results are heavily dependent on the quality of the data that are input to the
3 system. As described above, several studies were reviewed, and data from these studies were
4 used to develop the parameters and factors that were used to build the microenvironments in this
5 assessment. A constraint on this effort is that there are a limited number of NO₂ exposure studies
6 to use for evaluation.

7 The input data used in this assessment were selected to best simulate actual conditions
8 that affect human exposure. Using well characterized data as inputs to the model lessens the
9 degree of uncertainty in exposure estimates. Still, the limitations and uncertainties of each of the
10 data streams affect the overall quality of the model output. These issues and how they
11 specifically affect each data stream are discussed in this section.

12 **7.10.3 Meteorological Data**

13 Meteorological data are taken directly from monitoring stations in the assessment areas.
14 One strength of these data is that it is relatively easy to see significant errors if they appear in the
15 data. Because general climactic conditions are known for each area simulation, it would have
16 been apparent upon review if there were outliers in the dataset. However, there are limitations in
17 the use of these data. Because APEX only uses one temperature value per day, the model does
18 not represent hour-to-hour variations in meteorological conditions throughout the day that may
19 affect both NO₂ formation and exposure estimates within microenvironments.

20 **7.10.4 Air Quality Data**

21 Air quality data used in the exposure modeling was determined through use of EPA's
22 recommended regulatory air dispersion model, AERMOD (version 07026 (EPA, 2004)), with
23 meteorological data discussed above and emissions data based on the EPA's National Emissions
24 Inventory for 2002 (EPA, 2007b) and the CAMD Emissions Database (EPA, 2007c) for
25 stationary sources and mobile sources determined from local travel demand modeling and EPA's
26 MOBILE6.2 emission factor model. All of these are high quality data sources. Parameterization
27 of meteorology and emissions in the model were made in as accurate a manner as possible to
28 ensure best representation of air quality for exposure modeling. Further, minor sources not
29 included in the dispersion modeling were captured and any remaining long-term errors in the
30 results corrected through use of local concentrations derived from monitor observations. Thus,

1 the resulting air quality values are free of systematic errors to the best approximation available
2 through application of modeled data.

3 **7.10.5 Population and Commuting Data**

4 The population and commuting data are drawn from U.S. Census data from the year
5 2000. This is a high quality data source for nationwide population data in the U.S. However, the
6 data do have limitations. The Census used random sampling techniques instead of attempting to
7 reach all households in the U.S., as it has in the past. While the sampling techniques are well
8 established and trusted, they introduce some uncertainty to the system. The Census has a quality
9 section (<http://www.census.gov/quality/>) that discusses these and other issues with Census data.

10 In addition to these data quality issues, certain simplifying assumptions were made in
11 order to better match reality or to make the data match APEX input specifications. For example,
12 the APEX dataset does not differentiate people that work at home from those that commute
13 within their home tract, and individuals that commute over 120 km a day were assumed to not
14 commute daily. In addition to emphasizing some of the limitations of the input data, these
15 assumptions introduce uncertainty to the results.

16 Furthermore, the estimation of block-to-block commuter flows relied on the assumption
17 that the frequency of commuting to a workplace block within a tract is proportional to the
18 amount of commercial and industrial land in the block. This assumption introduces additional
19 uncertainty.

20 **7.10.6 Activity Pattern Data**

21 It is probable that the CHAD data used in the system is the most subject to limitations
22 and uncertainty of all the data used in the system. Much of the data used to generate the daily
23 diaries are over 20 years old. Table 44 indicates the ages of the CHAD diaries used in this
24 modeling analysis. While the specifics of people's daily activities may not have changed much
25 over the years, it is certainly possible that some differences do exist. In addition, the CHAD data
26 are taken from numerous surveys that were performed for different purposes. Some of these
27 surveys collected only a single diary-day while others went on for several days. Some of the
28 studies were designed to not be representative of the U.S. population, although a large portion of
29 the data are from National surveys. Furthermore, study collection periods occur at different
30 times of the year, possibly resulting in seasonal differences. A few of these limitations are

1 corrected by the approaches used in the exposure modeling (e.g., weighting by US population
2 demographics for a particular location, adjusting for effects of temperature on human activities).

3 A sensitivity analysis was performed to evaluate the impact of the activity pattern
4 database on APEX model results for O₃ (see Langstaff (2007) and EPA (2007g)). Briefly,
5 exposure results were generated using APEX with all of the CHAD diaries and compared with
6 results generated from running APEX using only the CHAD diaries from the National Human
7 Activity Pattern Study (NHAPS), a nationally representative study in CHAD. There was very
8 good agreement between the APEX results for the 12 cities evaluated, whether all of CHAD or
9 only the NHAPS component of CHAD is used. The absolute difference in percent of persons
10 above a particular concentration level ranged from -1% to about 4%, indicating that the exposure
11 model results are not being overly influenced by any single study in CHAD. It is likely that
12 similar results would be obtained here for NO₂ exposures, although remains uncertain due to
13 different averaging times (1-hour vs. 8-hour average).

14 **7.10.7 Air Exchange Rates**

15 There are several components of uncertainty in the residential air exchange rate
16 distributions used for this analysis. EPA (2007d) details an analysis of uncertainty due to
17 extrapolation of air exchange rate distributions between-CMSAs and within-CMSA uncertainty
18 due to sampling variation. In addition, the uncertainty associated with estimating daily air
19 exchange rate distributions from air exchange rate measurements with varying averaging times is
20 discussed. The results of those investigations are briefly summarized here.

21 ***7.10.7.1 Extrapolation among cities***

22 Location-specific distributions were assigned in the APEX model, as detailed in the
23 indoors-residential microenvironment. Since specific data for all of the locations targeted in this
24 analysis were not available, data from another location were used based on similar influential
25 characteristics. Such factors include age composition of housing stock, construction methods,
26 and other meteorological variables not explicitly treated in the analysis, such as humidity and
27 wind speed patterns. In order to assess the uncertainty associated with this extrapolation,
28 between-CSA uncertainty was evaluated by examining the variation of the geometric means and
29 standard deviations across cities and studies.

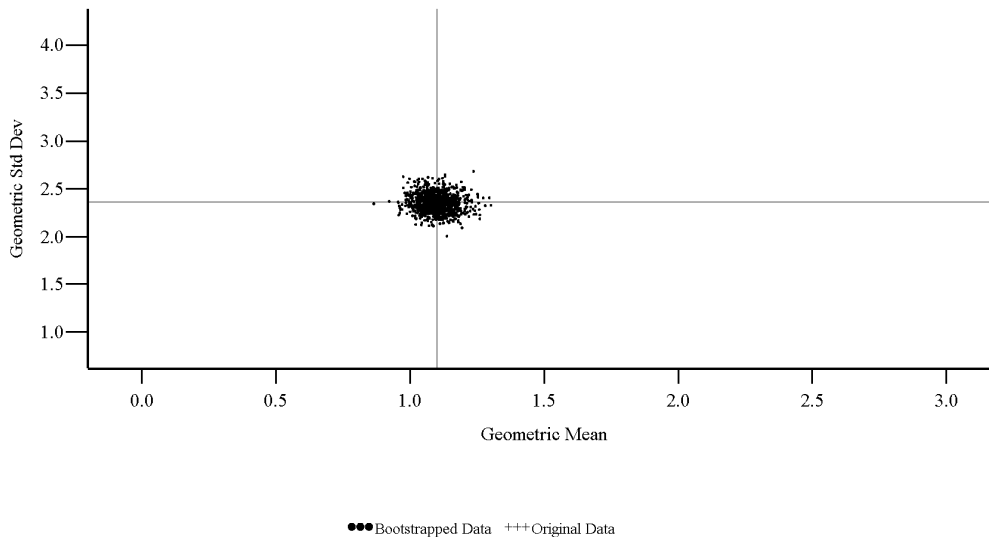
1 The analysis showed a relatively wide variation across different cities in the air exchange
2 rate geometric mean and standard deviation, stratified by air-conditioning status and temperature
3 range. This implies that the air exchange rate modeling results would be very different if the
4 matching of modeled locations to study locations was changed. For example, the NO₂ exposure
5 estimates may be sensitive to the assumption that the Philadelphia air exchange rate distributions
6 can be represented by the New York City air exchange rate data.

7 **7.10.7.2 Within CSA uncertainty**

8 There is also variation within studies for the same location (e.g., Los Angeles), but this is
9 much smaller than the variation across CMSAs. This finding tends to support the approach of
10 combining different studies for a CMSA. In addition, within-city uncertainty was assessed by
11 using a bootstrap distribution to estimate the effects of sampling variation on the fitted geometric
12 means and standard deviations for each CMSA. The bootstrap distributions assess the
13 uncertainty due to random sampling variation but do not address uncertainties due to the lack of
14 representativeness of the available study data or the variation in the lengths of the AER
15 monitoring periods.

16 1,000 bootstrap samples were randomly generated for each AER subset (of size N),
17 producing a set of 1,000 geometric mean and geometric standard deviation pairs. The analysis
18 indicated that the geometric standard deviation uncertainty for a given CSA/air-conditioning-
19 status/temperature-range combination tended to have a range of at most from *fitted GSD-1.0 hr⁻¹*
20 to *fitted GSD+1.0 hr⁻¹*, but the intervals based on larger AER sample sizes were frequently much
21 narrower. The ranges for the geometric means tended to be approximately from *fitted GM-0.5*
22 *hr⁻¹* to *fitted GM+0.5 hr⁻¹*, but in some cases were much smaller. Figure 12 illustrates such
23 results for Los Angeles as an example.

24



1
 2 **Figure 19. Geometric mean and standard deviation of air exchange rate bootstrapped for**
 3 **Los Angeles residences with A/C, temperature range from 20-25 degrees**
 4 **centigrade (from EPA, 2007g).**
 5

6 ***7.10.7.3 Variation in measurement averaging times***

7 Although the averaging periods for the air exchange rates in the study data varied from
 8 one day to seven days, the analyses did not take the measurement duration into account and
 9 treated the data as if they were a set of statistically independent daily averages. To investigate
 10 the uncertainty of this assumption, correlations between consecutive 24-hour air exchange rates
 11 measured at the same house were investigated using data from the Research Triangle Park Panel
 12 Study (EPA, 2007g). The results showed extremely strong correlations, providing support for
 13 the simplified approach of treating multi-day averaging periods as if they were 24-hour averages.

14 **7.10.8 Air Conditioning Prevalence**

15 Because the selection of an air exchange rate distribution is conditioned on the presence
 16 or absence of an air-conditioner, for each modeled area, the air conditioning status of the
 17 residential microenvironments was simulated randomly using the probability that a residence has
 18 an air conditioner, i.e., the residential air conditioner prevalence rate. For this study we used
 19 location-specific data from the American Housing Survey of 2003. EPA (2007g) details the
 20 specification of uncertainty estimates in the form of confidence intervals for the air conditioner
 21 prevalence rate, and compares these with prevalence rates and confidence intervals developed
 22 from the Energy Information Administration’s Residential Energy Consumption Survey (RECS)

1 of 2001 for more aggregate geographic subdivision (e.g., states, multi-state Census divisions and
2 regions).

3 Air conditioning prevalence rates for the 5 locations from the American Housing Survey
4 (Table 48) ranged from 55% for Los Angeles to 97% for Atlanta. Reported standard errors were
5 relatively small, ranging from less than 1.2% for Atlanta to 1.8% for Detroit. The corresponding
6 95% confidence intervals are also small and range from approximately 4.6% to 6.9%. The
7 RECS prevalence estimates and confidence intervals compared with the similar locations using
8 AHS data were mixed. Good agreements between the AHS and RECS confidence intervals was
9 found for Atlanta and Detroit. Poor agreement with the AHS for either the Census Region or
10 Census Division estimates was shown for Los Angeles and Philadelphia, with estimates of those
11 owning A/C lower when considering the RECS data. However, since the AHS survey results are
12 city-specific and were based on a more recent survey, the AHS prevalence estimates were used
13 for the APEX modeling.

14 Furthermore, some residences use evaporative coolers, also known as “swamp coolers,”
15 for cooling. The estimation of air exchange rate distributions from measurement data used here
16 did not take into account the presence or absence of an evaporative cooler. Based on statistical
17 comparison tests (i.e., F-test, Kruskal-Wallis, Mood) for where information was available to
18 generate AER distributions with and without swamp cooler ownership, it was determined that
19 presence or absence of such data did not alter the statistical air exchange model (EPA, 2007d).

20 **7.10.9 Indoor Source Estimation**

21 Other indoor NO₂ emission sources, such as gas pilot lights, gas heating, or gas clothes
22 drying were not included in this analysis, due to lack of data for characterization.

23 The data used to estimate the average number of daily food preparation events is
24 somewhat dated (1992) and may therefore be unrepresentative of current conditions, and may
25 lead to under- or over-estimates of exposure to exceedances of threshold concentrations of
26 concern. For example, if the population of Philadelphia County in 2003 prepares food at home
27 less frequently than the 1992 survey population, then the number of such exposures may be over-
28 estimated.

29 As noted above, it was assumed that the probability that a food preparation event
30 included stove use was the same no matter what hour of the day the food preparation event

1 occurred. If such probabilities differ, then the diurnal allocation of cooking events may differ
2 from the actual pattern. To the extent that the gas stove usage patterns may correlate with
3 ambient concentration patterns, the number of exposures to exceedances of threshold
4 concentrations of concern may be under- or over-estimated. For example, if gas stove usage and
5 ambient concentrations are positively correlated (e.g., if cooking tends to occur during evening
6 rush hour) and the diurnal allocation assumed here results in a lower correlation (e.g., if the
7 diurnal allocation understates the probability of gas stove usage at times of high ambient
8 concentrations) then the number of such exposures may be under-estimated. Or, for another
9 example, if the diurnal pattern allocation assumed here understates the probability of gas stove
10 usage at times when simulated subjects are assumed to be at home, then the number of such
11 exposures may be under-estimated.

12 The durations of the CARB cooking tests ranged from 21 minutes to 3 hours with an
13 average of about 70 minutes. But for implementation in APEX it was assumed that each cooking
14 event lasts exactly an hour. That is, the randomly selected net concentration contribution was
15 added to hourly average indoor concentration for the hour it was selected to occur. Because the
16 mass balance algorithm leads to carryover from one hour to the next, some of the indoor cooking
17 impact will influence subsequent hours. However, the impact of the cooking event may be
18 overstated or understated for cooking events longer or shorter than 1 hour.

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