



# **Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard**

EPA document # EPA-452/R-08-008a  
November 2008

# **Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard**

U.S. Environmental Protection Agency  
Office of Air Quality Planning and Standards  
Research Triangle Park, North Carolina

## **Disclaimer**

This 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. For questions concerning this document, please contact Dr. Stephen Graham (919-541-4344; [graham.stephen@epa.gov](mailto:graham.stephen@epa.gov)), Mr. Harvey Richmond (919-541-5271; [richmond.harvey@epa.gov](mailto:richmond.harvey@epa.gov)), or Dr. Scott Jenkins (919-541-1167; [jenkins.scott@epa.gov](mailto:jenkins.scott@epa.gov)).

# Table of Contents

List of Tables .....	v
List of Figures .....	ix
List of Acronyms/Abbreviation.....	xiii
<b>1. INTRODUCTION .....</b>	<b>1</b>
1.1 OVERVIEW .....	1
1.2 HISTORY .....	5
1.2.1 History of the NO <sub>2</sub> NAAQS.....	5
1.2.2 Health Evidence from Previous Review .....	6
1.2.3 Assessment from Previous Review .....	7
1.3 SCOPE OF THE RISK AND EXPOSURE ASSESSMENT FOR THE CURRENT REVIEW .....	7
<b>2. SOURCES, AMBIENT LEVELS, AND EXPOSURES .....</b>	<b>9</b>
2.1 SOURCES OF NO <sub>2</sub> .....	9
2.2 AMBIENT LEVELS OF NO <sub>2</sub> .....	9
2.2.1 Background on NO <sub>2</sub> monitoring network.....	9
2.2.2 Trends in ambient concentrations of NO <sub>2</sub> .....	13
2.2.3 Uncertainty Associated with the Ambient NO <sub>2</sub> Monitoring Method .....	14
2.3 EXPOSURE TO NO <sub>2</sub> .....	15
2.3.1 Overview .....	15
2.3.2 Uncertainty Associated with Ambient Levels as a Surrogate for Exposure .....	16
<b>3. AT RISK POPULATIONS .....</b>	<b>17</b>
3.1 OVERVIEW .....	17
3.2 SUSCEPTIBILITY: PRE-EXISTING DISEASE .....	18
3.3 SUSCEPTIBILITY: AGE .....	19
3.4 SUSCEPTIBILITY: GENETICS.....	19
3.5 SUSCEPTIBILITY: GENDER .....	20
3.6 VULNERABILITY: PROXIMITY (ON OR NEAR) TO ROADWAYS.....	20
3.7 VULNERABILITY: SOCIOECONOMIC STATUS .....	21
3.8 CONCLUSIONS.....	21
<b>4. HEALTH EFFECTS .....</b>	<b>23</b>
4.1 INTRODUCTION .....	23
4.2 ADVERSE RESPIRATORY EFFECTS FOLLOWING SHORT-TERM EXPOSURES.....	26
4.2.1 Overview .....	26
4.2.2 Respiratory Emergency Department Visits and Hospitalizations .....	27
4.2.3 Respiratory Symptoms.....	28
4.2.4 Lung Host Defenses and Immunity.....	31
4.2.5 Airway Response .....	32
4.2.6 Airway Inflammation.....	35
4.2.7 Lung Function.....	36
4.2.8 Conclusions and Coherence of Evidence for Short-Term Respiratory Effects.....	36
4.3 OTHER ADVERSE EFFECTS FOLLOWING SHORT-TERM EXPOSURES .....	37
4.4 ADVERSE EFFECTS FOLLOWING LONG-TERM EXPOSURES .....	38

4.4.1 Respiratory Morbidity.....	38
4.4.2 Mortality .....	40
4.4.3 Other Long-Term Effects.....	40
4.5 RELEVANCE OF SPECIFIC HEALTH EFFECTS TO THE NO <sub>2</sub> RISK CHARACTERIZATION .....	41
4.5.1 Overview .....	41
4.5.2 Epidemiology .....	42
4.5.3 Controlled Human Exposure Studies .....	44
<b>5. IDENTIFICATION OF POTENTIAL ALTERNATIVE STANDARDS FOR ANALYSIS.....</b>	<b>46</b>
5.1 INTRODUCTION .....	46
5.2 INDICATOR.....	46
5.3 AVERAGING TIME .....	46
5.4 FORM .....	48
5.5 LEVEL .....	49
<b>6. OVERVIEW OF APPROACHES TO ASSESSING EXPOSURES AND RISKS .....</b>	<b>55</b>
6.1 INTRODUCTION .....	55
6.2 POTENTIAL HEALTH BENCHMARK LEVELS .....	57
6.3 SIMULATING THE CURRENT AND ALTERNATIVE STANDARDS.....	59
6.3.1 Adjustment of Ambient Air Quality .....	59
6.3.2 Adjustment of Potential Health Effect Benchmark Levels.....	64
<b>7. AMBIENT AIR QUALITY ASSESSMENT AND HEALTH RISK CHARACTERIZATION .....</b>	<b>67</b>
7.1 OVERVIEW .....	67
7.2 APPROACH .....	69
7.2.1 Air Quality Data Screen.....	70
7.2.2 Selection of Locations for Air Quality Analysis .....	71
7.2.3 Site Characteristics of Ambient NO <sub>2</sub> Monitors .....	73
7.2.4 Estimation of On-Road Concentrations using Ambient Concentrations.....	80
7.2.5 Air Quality Concentration Metrics.....	83
7.3 AIR QUALITY AND HEALTH RISK CHARACTERIZATION RESULTS.....	85
7.3.1 Ambient Air Quality (As Is).....	85
7.3.2 On-Road Concentrations Derived From Ambient Air Quality (As Is) .....	97
7.3.3 Ambient Air Quality Adjusted to Just Meet the Current and Alternative Standards .....	102
7.3.4 On-Road Concentrations Derived From Ambient Air Quality Adjusted to Just Meet the Current and Alternative Standards.....	114
7.4 UNCERTAINTY ANALYSIS .....	122
7.4.1 Air Quality Database .....	124
7.4.2 Measurement Technique for Ambient NO <sub>2</sub> .....	125
7.4.3 Temporal Representation .....	125
7.4.4 Spatial Representation .....	127
7.4.5 Air Quality Adjustment Procedure.....	130
7.4.6 On-Road Concentration Simulation.....	133
7.4.7 Health Benchmark.....	143
7.5 KEY OBSERVATIONS .....	144
<b>8. EXPOSURE ASSESSMENT AND HEALTH RISK CHARACTERIZATION .....</b>	<b>146</b>
8.1 OVERVIEW .....	146
8.2 OVERVIEW OF HUMAN EXPOSURE MODELING USING APEX.....	149
8.3 CHARACTERIZATION OF STUDY AREA .....	151
8.3.1 Study Area Selection .....	151
8.3.2 Study Area Description .....	152
8.3.3 Time Period of Analysis .....	152
8.3.4 Populations Analyzed.....	152
8.4 CHARACTERIZATION OF AMBIENT AIR QUALITY USING AERMOD.....	154
8.4.1 Overview .....	154

8.4.2	<i>General Model Inputs</i>	154
8.4.2.1	Meteorological Inputs	154
8.4.2.2	Surface Characteristics and Land Use Analysis	155
8.4.2.4	Other AERMOD Input Specifications	155
8.4.3	<i>Major Link On-Road Emission Estimates</i>	156
8.4.3.1	Emission Sources and Locations	156
8.4.3.2	Emission Source Strength	158
8.4.3.3	Other Emission Parameters	160
8.4.4	<i>Minor Link On-road Emission Estimates</i>	161
8.4.5	<i>Adjustment of On-road Mobile Source Strengths to 2002 NEI Vehicle Emissions</i>	163
8.4.5	<i>Stationary Sources Emissions Preparation</i>	164
8.4.6	<i>Airport Emissions Preparation</i>	165
8.4.7	<i>Receptor Locations</i>	167
8.4.8	<i>Modeled Air Quality Evaluation</i>	168
8.4.8.1	Comparison of Hourly Cumulative Density Functions	168
8.4.8.2	Comparison of annual average diurnal concentration profiles	170
8.4.8.3	Comparison of estimated on-road NO <sub>2</sub> concentrations	173
8.4.8	Using unadjusted AERMOD predicted NO <sub>2</sub> concentrations	177
8.5	SIMULATED POPULATION	177
8.6	CONSTRUCTION OF LONGITUDINAL ACTIVITY SEQUENCES	179
8.7	CALCULATING MICROENVIRONMENTAL CONCENTRATIONS	179
8.7.1	<i>Microenvironments Modeled</i>	180
8.7.2	<i>Microenvironment Descriptions</i>	181
8.7.2.1	Microenvironment 1: Indoor-Residence	181
8.7.2.2	Microenvironments 2-7: All other indoor microenvironments	184
8.7.2.3	Microenvironments 8 and 9: Outdoor Microenvironments	184
8.7.2.4	Microenvironment 10: Outdoors-General	185
8.7.2.5	Microenvironments 11 and 12: In Vehicle- Cars and Trucks, and Mass Transit	185
8.8	EXPOSURE MEASURES AND HEALTH RISK CHARACTERIZATION	185
8.8.1	<i>Adjustment for Just Meeting the Current and Alternative Standards</i>	187
8.9	EXPOSURE MODELING AND HEALTH RISK CHARACTERIZATION RESULTS	189
8.9.1	<i>Overview</i>	189
8.9.2	<i>Annual Average Exposure Concentrations (as is)</i>	190
8.9.3	<i>Daily Average Exposures (as is)</i>	192
8.9.4	<i>One-Hour Exposures</i>	197
8.9.4.1	Overview	197
8.9.4.2	Estimated Number of 1-hour Exposures Above Selected Levels (as is)	197
8.9.4.3	Estimated Number of 1-hour Exposures Above Selected Levels (current standard)	207
8.9.4.4	Estimated Number of 1-hour Exposures Above Selected Levels (alternative standards)	209
8.10	KEY OBSERVATIONS	213
8.11	REPRESENTATIVENESS OF EXPOSURE RESULTS	214
8.11.1	<i>Introduction</i>	214
8.11.2	<i>Description of Data Compiled and Summarized</i>	214
8.11.2.1	HAPEM6 Near-Road Population Data Base	215
8.11.2.2	American Housing Survey (AHS) Data	218
8.11.2.3	Federal Highway Administration (FHWA) Data	220
8.11.4	<i>Discussion</i>	221
8.12	UNCERTAINTY ANALYSIS	223
8.12.1	<i>Dispersion Modeling Uncertainties</i>	224
8.12.1.1	AERMOD Algorithms	224
8.12.1.2	Meteorological Inputs	226
8.12.1.3	Mobile Source Characterization	228
8.12.1.4	On-Road Emissions Estimates	230
8.12.1.5	O <sub>3</sub> Monitoring Data for OLM and PVMRM Options	234
8.12.1.6	Use of Unadjusted AERMOD NO <sub>2</sub> Concentrations	235
8.12.2	<i>Exposure Modeling Uncertainties</i>	237
8.12.2.1	Population Data Base	237
8.12.2.2	Commuting Data Base	238
8.12.2.2	Human Time-Location-Activity Pattern Data	239

8.12.2.3 Longitudinal Profile .....	240
8.12.2.4 Meteorological Data .....	242
8.12.2.5 Air Exchange Rates (AER) .....	243
8.12.2.6 Air Conditioning Prevalence .....	246
8.12.2.7 Indoor Source Estimation .....	248
<b>9. CHARACTERIZATION OF HEALTH RISKS USING DATA FROM EPIDEMIOLOGICAL STUDIES .....</b>	<b>252</b>
9.1 INTRODUCTION .....	252
9.2 GENERAL APPROACH .....	253
9.3 AIR QUALITY INFORMATION .....	258
9.4 CONCENTRATION-RESPONSE FUNCTIONS .....	259
9.5 BASELINE HEALTH EFFECTS INCIDENCE DATA .....	261
9.6 ADDRESSING UNCERTAINTY AND VARIABILITY .....	262
9.7 RISK ESTIMATES FOR EMERGENCY DEPARTMENT VISITS .....	269
<b>10. EVIDENCE- AND EXPOSURE/RISK-BASED CONSIDERATIONS RELATED TO THE PRIMARY NO<sub>2</sub> NAAQS .....</b>	<b>275</b>
10.1 INTRODUCTION .....	275
10.2 GENERAL APPROACH .....	276
10.3 ADEQUACY OF THE CURRENT ANNUAL STANDARD .....	278
10.3.1 Evidence-based considerations .....	279
10.3.2 Exposure- and risk-based considerations .....	282
10.3.2.1 Key uncertainties .....	284
10.3.2.2 Assessment results .....	287
10.3.3 Conclusions regarding the adequacy of the current standard .....	290
10.4 POTENTIAL ALTERNATIVE STANDARDS .....	291
10.4.1 Indicator .....	291
10.4.2 Averaging Time .....	291
10.4.3 Form .....	296
10.4.4 Level .....	299
10.4.4.1 Evidence-based considerations .....	299
10.4.4.2 Exposure- and risk-based considerations .....	304
10.4.4.3 Conclusions regarding level .....	309
<b>11. REFERENCES .....</b>	<b>310</b>
<b>APPENDICES</b>	
<b>APPENDIX A - Supplement to the NO<sub>2</sub> Air Quality Characterization</b>	
<b>APPENDIX B - Supplement to the NO<sub>2</sub> Exposure Assessment</b>	
<b>APPENDIX C - Nitrogen Dioxide Health Risk Assessment for Atlanta, GA</b>	

## List of Tables

<u>Number</u>	<u>Page</u>
Table 2-1. NO <sub>x</sub> Network Distribution of Monitor Objectives .....	12
Table 2-2. NO <sub>x</sub> Network Distribution across Measurement Scales. ....	13
Table 3-1. Overview of Susceptibility and Vulnerability Factors .....	18
Table 4-1. Weight of Evidence for Causal Determination .....	24
Table 4-2. Causality judgments made in the ISA for endpoints associated with short-term NO <sub>2</sub> exposures.....	25
Table 4-3. Causality judgments made in the ISA for endpoints associated with long-term NO <sub>2</sub> exposures.....	26
Table 4-4. Proposed Mechanisms Whereby NO <sub>2</sub> and Respiratory Virus Infections May Exacerbate Upper and Lower Airway Symptoms.....	32
Table 4-5. Fraction of nitrogen dioxide-exposed asthmatics with increased nonspecific airway hyperresponsiveness.....	34
Table 7-1. Counts of complete and incomplete site-years of NO <sub>2</sub> ambient monitoring data. ....	71
Table 7-2. Locations selected for NO <sub>2</sub> Air Quality Characterization, associated abbreviations, and values of selection criteria. ....	73
Table 7-3. Percent of ambient NO <sub>2</sub> monitors with selected monitoring objectives, using all valid site-years of historical air quality (1995-2000).....	75
Table 7-4. Percent of ambient NO <sub>2</sub> monitors with selected monitoring objectives, using all valid site-years of recent air quality (2001-2006). ....	75
Table 7-5. Percent of ambient NO <sub>2</sub> monitors with selected measurement scales, using all valid site-years of historical air quality (1995-2000).....	76
Table 7-6. Percent of ambient NO <sub>2</sub> monitors with selected measurement scales, using all valid site-years of recent air quality (2001-2006). ....	77
Table 7-7. Percent of ambient NO <sub>2</sub> monitors with selected land use, using all valid site-years of historical air quality (1995-2000).....	77
Table 7-8. Percent of ambient NO <sub>2</sub> monitors with selected land use, using all valid site-years of recent air quality (2001-2006).....	78
Table 7-9. Distance of ambient monitors to the nearest major sources in selected locations.....	79
Table 7-10. Derived $C_v/C_b$ ratios ( $m$ ) for two season groups used for adjusting ambient NO <sub>2</sub> concentrations to simulate on-road NO <sub>2</sub> concentrations.....	82
Table 7-11. Monitoring site-years and annual average NO <sub>2</sub> concentrations, using recent air quality data ( <i>as is</i> ) and monitors sited $\geq 100$ m of a major road.....	86
Table 7-12. Monitoring site-years and annual average NO <sub>2</sub> concentrations, using recent air quality data ( <i>as is</i> ) and monitors sited $>20$ m and $<100$ m of a major road. ....	87
Table 7-13. Monitoring site-years and annual average NO <sub>2</sub> concentrations, using recent air quality data ( <i>as is</i> ) and monitors sited $\leq 20$ m of a major road.....	88
Table 7-14. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited $\geq 100$ m of a major road. ....	91
Table 7-15. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited $\geq 100$ m of a major road. ....	92

Table 7-16. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited >20 m and <100 m of a major road.....	93
Table 7-17. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited >20 m and <100 m of a major road.....	94
Table 7-18. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited ≤20 m from a major road.....	95
Table 7-19. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and monitors sited ≤20 m from a major road.....	96
Table 7-20. Estimated annual average NO <sub>2</sub> concentrations on-roads, using recent air quality data ( <i>as is</i> ) and an on-road adjustment factor.....	99
Table 7-21. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using 2001-2003 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and an on-road adjustment factor.....	100
Table 7-22. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using 2004-2006 recent NO <sub>2</sub> air quality ( <i>as is</i> ) and an on-road adjustment factor.....	101
Table 7-23. Estimated annual mean NO <sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98 <sup>th</sup> percentile alternative standard and monitors sited ≥ 100 m of a major road.....	109
Table 7-24. Estimated annual mean NO <sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98 <sup>th</sup> percentile alternative standard and monitors sited >20 m and <100 m from a major road. ....	110
Table 7-25. Estimated annual mean NO <sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98 <sup>th</sup> percentile alternative standard and monitors sited ≤20 m from a major road. ....	111
Table 7-26. Estimated mean number of daily maximum exceedances of 100 ppb 1-hour NO <sub>2</sub> concentrations in a year, using 2001-2003 air quality <i>as is</i> and that adjusted to just meeting the current and alternative standards (98 <sup>th</sup> percentile) for monitors sited ≥100 m, >20 m and <100 m, and ≤20 m of a major road. ....	112
Table 7-27. Estimated mean number of daily maximum exceedances of 150 ppb 1-hour NO <sub>2</sub> concentrations in a year, using 2001-2003 air quality <i>as is</i> and air quality adjusted to just meeting the current and alternative standards (98 <sup>th</sup> percentile) for monitors sited ≥100 m, >20 m and <100 m, and ≤20 m of a major road. ....	113
Table 7-28. Estimated annual mean NO <sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using recent air quality (2001-2003) adjusted to just meeting a 1-hour 100 ppb 98 <sup>th</sup> percentile alternative standard and an on-road adjustment factor.....	119
Table 7-29. Estimated mean number of daily maximum exceedances of 100 ppb 1-hour NO <sub>2</sub> concentrations on-roads in a year, using air quality <i>as is</i> and air quality adjusted to just	

meeting the current and alternative standards (98 <sup>th</sup> percentile) and an on-road adjustment factor.....	120
Table 7-30. Estimated mean number of daily maximum exceedances of 150 ppb 1-hour NO <sub>2</sub> concentrations on-roads in a year, using air quality <i>as is</i> and air quality adjusted to just meeting the current and alternative standards (98 <sup>th</sup> percentile) and an on-road adjustment factor.....	121
Table 7-31. Summary of qualitative uncertainty analysis for the air quality and health risk characterization.....	123
Table 7-32. Percent difference in 1-hour NO <sub>2</sub> concentrations for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality.....	129
Table 7-33. Number of 1-hour NO <sub>2</sub> concentrations above 100 ppb for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality.....	130
Table 7-34. Comparison of empirical distribution of on-road adjustment factors used in on-road concentration estimation with a fitted lognormal distribution.....	140
Table 7-35. Absolute difference in the estimated number of exceedances of potential health effect benchmarks on-roads using either a fitted lognormal distribution or empirical distribution of the on-road adjustment factors and 2004-2006 air quality <i>as is</i> and air quality adjusted to just meet the current annual standard.....	142
Table 8-1. Statistical summary of average annual daily traffic (AADT) volumes (one direction) for Atlanta AERMOD simulations.....	157
Table 8-2. Average heavy duty vehicle (HDV) fraction for Atlanta AERMOD simulations. ..	158
Table 8-3. Average calculated speed by link type in Atlanta modeling domain.....	160
Table 8-4. On-road area source sizes.....	160
Table 8-5. On-road emissions from major and minor links in Atlanta, 2002.....	162
Table 8-6. On-road vehicle emission strengths by county for Atlanta modeling domain: modeled vs NEI 2002.....	163
Table 8-7. Summary statistics of estimated on-road hourly NO <sub>2</sub> concentrations (ppb) and the numbers of hourly concentrations above 100, 150, and 200 ppb in a year using both the AERMOD and the on-road ambient monitor simulation approaches in Atlanta.	176
Table 8-8. Asthma prevalence rates by age and gender used for Atlanta.....	178
Table 8-9. List of microenvironments modeled and calculation methods used.....	181
Table 8-10. Geometric means (GM) and standard deviations (GSD) for air exchange rates by A/C type and temperature range used for Atlanta exposure assessment.....	182
Table 8-11. Data used to estimate removal rate constant for indoor microenvironments.....	183
Table 8-12. Probability of gas stove cooking by hour of the day.....	183
Table 8-13. Adjusted potential health effect benchmark levels used by APEX to simulate just meeting the current standard and various alternative standards considered.....	187
Table 8-14. Percent of population within selected distances of a major road in several locations.....	217
Table 8-15. Residential A/C prevalence and roadway distance statistics for housing units in several locations (AHS, 2008).....	219
Table 8-16. Population and roadway statistics for several locations (FHWA, 2002).....	220
Table 8-17. Summary of qualitative uncertainty analysis for the exposure assessment.....	223
Table 8-18. National vehicle miles traveled by roadway category and vehicle type.....	232
Table 8-19. Observed peak hour truck percentages on Interstate 75 (I-75) using 2002 traffic count data.....	232

Table 8-20. Comparison of exposure results using a 0.55 versus 0.97 A/C prevalence for 2002 air quality without indoor sources.....	248
Table 8-21. Comparison of exposure results using a uniform versus lognormal NO <sub>2</sub> indoor decay distribution for 2002 air quality without indoor sources. ....	249
Table 9-1. Characterization of Key Uncertainties in the Emergency Department Visits Health Risk Assessment for the Atlanta Region.....	264
Table 9-2. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2005 NO <sub>2</sub> Concentrations.* .....	270
Table 9-3. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2006 NO <sub>2</sub> Concentrations.* .....	271
Table 9-4. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2007 NO <sub>2</sub> Concentrations.* .....	272
Table 10-1. Ratios of short-term to annual average NO <sub>2</sub> concentrations .....	293
Table 10-2. Ratios of 1-h daily maximum NO <sub>2</sub> concentrations to 24-h average concentrations (ppm).....	295
Table 10-3. Mean annual NO <sub>2</sub> concentrations for 2004-2006 given just meeting alternative 1-h standards (98 <sup>th</sup> percentile) .....	296
Table 10-4. NO <sub>2</sub> concentrations (ppm) corresponding to 2 <sup>nd</sup> -9 <sup>th</sup> daily maximum and 98 <sup>th</sup> /99 <sup>th</sup> percentile forms (2004-2006).....	298
Table 10-5. Mean number of days per year (averaged over the 2004-2006 time period) estimated to have ambient (central site monitor) 1-h daily maximum NO <sub>2</sub> concentrations $\geq$ 0.10 ppm assuming 98 <sup>th</sup> and 99 <sup>th</sup> percentile forms of a 0.20 ppm standard.....	299
Table 10-6. Percent of counties that may be above the level of the standard, given different levels (based on years 2004-2006) .....	305

## List of Figures

<u>Number</u>	<u>Page</u>
Figure 1-1. Overview of the analyses described in this document and their interconnections.....	4
Figure 5-1. NO <sub>2</sub> effect estimates (95% CI) for ED visits/HA and associated 1-h daily maximum NO <sub>2</sub> levels (98 <sup>th</sup> and 99 <sup>th</sup> percentile values in boxes) .....	52
Figure 5-2. NO <sub>2</sub> effect estimates for respiratory symptoms and associated 1-h daily maximum NO <sub>2</sub> levels (98 <sup>th</sup> and 99 <sup>th</sup> percentile values in boxes) .....	53
Figure 6-1. Comparison of measured daily maximum NO <sub>2</sub> concentration percentiles in Atlanta for three high concentration years (1985, 1986, 1988) versus three low concentration years (2005-2007) at one ambient monitor. ....	61
Figure 6-2. Distributions of hourly NO <sub>2</sub> concentrations at twelve ambient monitors in the Boston CMSA, as is (top) and air quality adjusted to just meet the current standard (bottom), Year 1995. ....	62
Figure 6-3. Comparison of adjusted ambient monitoring concentrations (CS) or adjusted benchmark level (dashed line) to simulate just meeting the current annual average standard in Atlanta for year 2001. ....	65
Figure 6-4. Comparison of the upper percentiles for where ambient monitoring NO <sub>2</sub> concentrations (CS) and the benchmark level (dashed line) were adjusted to simulate just meeting the current annual standard in Atlanta for year 2001. The hourly NO <sub>2</sub> concentration distributions are provided in Figure 6-3. ....	66
Figure 7-1. Illustration of three roadway distance categories used to characterize ambient monitors in the Air Quality Characterization. ....	80
Figure 7-2. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using recent NO <sub>2</sub> air quality (2001-2003) adjusted to just meeting the current annual standard (0.053 ppm). Left graph: monitors ≥100m from a major road; Middle graph: monitors >20 m and <100 m from a major road; Right graph: monitors ≤20 m from a major road. ....	105
Figure 7-3. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using recent NO <sub>2</sub> air quality (2004-2006) adjusted to just meeting the current annual standard (0.053 ppm). Left graph: monitors ≥100m from a major road; Middle graph: monitors >20 m and <100 m from a major road; Right graph: monitors ≤20 m from a major road). ....	106
Figure 7-4. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks (100 ppb, top; 200 ppb, bottom) in Chicago in a year, using recent NO <sub>2</sub> air quality data (2001-2006) adjusted to just meeting alternative 1-hour standard levels (98 <sup>th</sup> percentile, left; and 99 <sup>th</sup> percentile, right) and monitors sited ≥100 m, > 20 m and < 100 m, ≤ 20 m of major roads. ....	107
Figure 7-5. Estimated mean number of daily maximum exceedances of 200 ppb in four locations (Phoenix, Los Angeles, Washington DC, and St. Louis) in a year, using recent NO <sub>2</sub> air quality data (2001-2006) adjusted to just meeting alternative 1-hour 98 <sup>th</sup> percentile standard levels and monitors sited ≥100 m, > 20 m and < 100 m, ≤ 20 m of major roads. ....	108
Figure 7-6. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using recent NO <sub>2</sub> air quality	

adjusted to just meeting the current annual standard (0.053 ppm) and an on-road adjustment factor. Left graph: 2001-2003 air quality; Right graph: 2004-2006 air quality.....	116
Figure 7-7. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks (100 ppb, top; 200 ppb, bottom) on-roads in Chicago in a year, using recent NO <sub>2</sub> air quality (2001-2006) adjusted to just meeting alternative 1-hour standard levels (98 <sup>th</sup> percentile, left; and 99 <sup>th</sup> percentile) and an on-road adjustment factor.....	117
Figure 7-8. Estimated mean number of daily maximum exceedances of 200 ppb on-roads in four locations (Phoenix, Los Angeles, Washington DC, and St. Louis) in a year, using recent NO <sub>2</sub> air quality (2001-2006) adjusted to just meeting alternative 1-hour 98 <sup>th</sup> percentile standard levels and an on-road adjustment factor. ....	118
Figure 7-9. Distribution of 1-hour NO <sub>2</sub> concentrations for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality. ....	129
Figure 7-10. Comparison of measured daily maximum NO <sub>2</sub> concentration percentiles in Philadelphia for one high concentration years (1984) versus a low concentration years (2007) at four ambient monitors. ....	133
Figure 7-11. Comparison of the distribution of estimated C <sub>v</sub> /C <sub>b</sub> ratios or <i>m</i> for the <i>not summer</i> category with fitted distributions. ....	141
Figure 7-12. Comparison of the distribution of estimated C <sub>v</sub> /C <sub>b</sub> ratios or <i>m</i> for the <i>summer</i> category with fitted distributions. ....	141
Figure 8-1. General flow used for NO <sub>2</sub> exposure assessment. ....	148
Figure 8-2. Four county modeling domain used for Atlanta exposure assessment. ....	153
Figure 8-3. The 478 U.S. Census tracts representing area sources for on-road mobile emissions that do not occur on major roadway links.....	162
Figure 8-4. Location of major roadway links and major stationary emission sources in Atlanta modeling domain.....	166
Figure 8-5. Location of modeled receptors in Atlanta modeling domain.....	168
Figure 8-6. Comparison of measured ambient monitor NO <sub>2</sub> concentration distribution with the modeled monitor receptor and receptors within 4 km of the monitors at three locations in Atlanta for Year 2002. ....	171
Figure 8-7. Comparison of measured ambient monitor NO <sub>2</sub> concentration diurnal profile with the modeled monitor receptor and receptors within 4 km of the monitors at three locations in Atlanta for Year 2002. ....	172
Figure 8-8. Comparison of on-road/non-road ratios developed from AERMOD concentration estimates for year 2002 and those derived from data reported in published NO <sub>2</sub> measurement studies. ....	175
Figure 8-9. Comparison of annual average AERMOD predicted NO <sub>2</sub> concentrations (on-road and non-road receptors) and APEX modeled NO <sub>2</sub> exposures (with and without modeled indoor sources) in Atlanta modeling domain for year 2002.....	191
Figure 8-10. Comparison of estimated annual average NO <sub>2</sub> exposures for Years 2001-2003 in Atlanta modeling domain without modeled indoor sources. ....	192
Figure 8-11. Distribution of measured daily average personal NO <sub>2</sub> exposures for individuals in Atlanta, stratified by two seasons (fall or spring) and cooking fuel (gas or electric). Minimum (min), median (p50), and maximum (max) were obtained from each	

individual's multi-day exposure measurements. The figure generated here was based on personal exposure measurements obtained from Suh (2008). ..... 195

Figure 8-12. Distribution of estimated daily average NO<sub>2</sub> exposures for individuals in Atlanta, stratified by two seasons (fall or spring) and with and without indoor sources, for Year 2002 APEX simulation. Lower bound (2.5th percentile, p2.5), median (p50), and upper bound (97.5th percentile, p97.5) were calculated from each simulated persons 365 days of exposure. A random sample of 5% of persons (about 2,500 individuals) is presented in each figure to limit the density of the graphs. .... 196

Figure 8-13. Estimated number of all simulated asthmatics in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark levels, using modeled 2001-2003 air quality (as is), without indoor sources. .... 199

Figure 8-14. Estimated number of simulated asthmatic children in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark levels, using modeled 2001-2003 air quality (as is), without modeled indoor sources. .... 199

Figure 8-15. Estimated number of all simulated asthmatics in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above potential health effect benchmark levels, using modeled 2002 air quality (as is), both with and without modeled indoor sources..... 201

Figure 8-16. Estimated number asthmatic person-days in the Atlanta model domain with an NO<sub>2</sub> exposure at or above potential health effect benchmark levels, using modeled 2002 air quality (as is), both with and without modeled indoor sources..... 201

Figure 8-17. Fraction of time all simulated persons in the Atlanta model domain spend in the twelve microenvironments that corresponds with exceedances of the potential NO<sub>2</sub> health effect benchmark levels, a)  $\geq 100$  ppb, b)  $\geq 200$  ppb, and c)  $\geq 300$  ppb, year 2002 air quality (as is) without indoor sources. .... 204

Figure 8-18. Fraction of time all simulated persons in the Atlanta model domain spend in the twelve microenvironments that corresponds with exceedances of the potential NO<sub>2</sub> health effect benchmark levels, a)  $\geq 100$  ppb, b)  $\geq 200$  ppb, and c)  $\geq 300$  ppb, year 2002 air quality (as is) with indoor sources. .... 205

Figure 8-19. Estimated percent of all asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above potential health effect benchmark levels, using modeled 2002 air quality (as is), without indoor sources. .... 206

Figure 8-20. Estimated percent of all asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above potential health effect benchmark levels, using modeled 2002 air quality (as is), with indoor sources. .... 207

Figure 8-21. Estimated number of all asthmatics in the Atlanta modeling domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark level, using modeled 2002 air quality just meeting the current standard (cur std), with and without modeled indoor sources..... 208

Figure 8-22. Estimated percent of asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above health effect benchmark levels, using modeled 2002 air quality just meeting the current standard, without modeled indoor sources..... 209

Figure 8-23. Estimated percent of asthmatics in the Atlanta modeling domain with NO<sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting potential alternative standards, without indoor sources..... 211

Figure 8-24. Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO <sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 50 ppb level 99 <sup>th</sup> percentile form alternative standard, without indoor sources. ....	211
Figure 8-25. Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO <sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 50 ppb level 99 <sup>th</sup> percentile form alternative standard, with indoor sources.....	212
Figure 8-26. Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO <sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 100 ppb level 99 <sup>th</sup> percentile form alternative standard, without indoor sources. ....	212
Figure 8-27. Comparison of estimated population and total roadway miles in 18 locations (data from FHWA (2002) provided in Table 8-16). ....	221
Figure 8-28. Comparison of high ranked AERMOD 1-hour NO <sub>2</sub> concentrations (µg/m <sup>3</sup> ) from mobile sources across four NO <sub>2</sub> monitoring locations based on JST vs. ATL meteorological inputs for 2002. ....	228
Figure 8-29. Comparison of the average ratios of predicted/observed concentrations of NO <sub>2</sub> across four ambient monitors based on weekday vs. weekend only.....	234
Figure 8-30. Example comparison of estimated geometric mean and geometric standard deviations of AER (h-1) for homes with air conditioning in several cities. ....	244
Figure 8-31. Example of boot strap simulation results used in evaluating random sampling variation of AER (h-1) distributions (RTP data).....	245
Figure 8-32. Example of boot strap simulation results used in evaluating random sampling variation of AER (h-1) distributions (outside CA). ....	246
Figure 9-1. Major components of nitrogen dioxide health risk assessment for emergency department visits. ....	256

## List of Acronyms/Abbreviations

AADT	Annual average daily traffic
A/C	Air conditioning
AER	Air exchange rate
AERMOD	American Meteorological Society (AMS)/EPA Regulatory Model
AHS	American Housing Survey
APEX	EPA's Air Pollutants Exposure model, version 4
ANOVA	One-way analysis of variance
AQS	EPA's Air Quality System
AS	Asthma symptoms
BAL	bronchoalveolar lavage
BRFSS	Behavioral Risk Factor Surveillance System
C	Cough
CAA	Clean Air Act
CAMD	EPA's Clean Air Markets Division
CAMP	Childhood Asthma Management Program
CASAC	Clean Air Scientific Advisory Committee
CDC	Centers for Disease Control
CHAD	EPA's Consolidated Human Activity Database
CHF	Congestive Heart Failure
Clev/Cinn	Cleveland and Cincinnati, Ohio
CMSA	Consolidated metropolitan statistical area
CO	Carbon monoxide
COPD	Chronic Obstructive Pulmonary Disease
COV	Coefficient of Variation
C-R	Concentration-Response
CTPP	Census Transportation Planning Package
DVRPC	Delaware Valley Regional Planning Council
ECP	Eosinophil cationic protein
EDR	Emergency department visits for respiratory disease
EDA	Emergency department visits for asthma
EDAC	Emergency department visits for asthma – children
FHWA	Federal Highway Administration
HAAC	Hospital admissions for asthma - children
ER	Emergency room
EPA	United States Environmental Protection Agency
EOC	Exposure of Concern
GM	Geometric mean
GSD	Geometric standard deviation
GST	Glutathione S-transferase (e.g., GSTM1, GSTP1, GSTT1)
h	Hour
HNO <sub>3</sub>	Nitric acid
HONO	Nitrous acid
ID	Identification
ISA	Integrated Science Assessment

ISH	Integrated Surface Hourly Database
km	Kilometer
L95	Lower limit of the 95 <sup>th</sup> confidence interval
LA	Los Angeles, California
m	Meter
max	Maximum
ME	Microenvironment
med	Median
MI	Myocardial Infarction
min	Minimum
MS	Morning symptoms
MSA	Metropolitan statistical area
NAAQS	National Ambient Air Quality Standards
NAICS	North American Industrial Classification System
NCEA	National Center for Environmental Assessment
NEI	National Emissions Inventory
NEM	NAAQS Exposure Model
NCDC	National Climatic Data Center
NHAPS	National Human Activity Pattern Study
NHIS	National Health Interview Survey
NO <sub>2</sub>	Nitrogen dioxide
NO <sub>x</sub>	Oxides of nitrogen
NO <sub>3</sub> <sup>-</sup>	Nitrate ion
NWS	National Weather Service
NYC	New York City
NYDOH	New York Department of Health
O <sub>3</sub>	Ozone
OAQPS	Office of Air Quality Planning and Standards
OR	Odds ratio
ORD	Office of Research and Development
ORIS	Office of Regulatory Information Systems identification code
POC	Parameter occurrence code
ppb	Parts per billion
PEN	Penetration factor
PM	Particulate matter
PMN	Polymorphonuclear
ppm	Parts per million
PRB	Policy-Relevant Background
PROX	Proximity factor
PVMRM	Plume Volume Molar Ratio Method
RECS	Residential Energy Consumption Survey
RIU	Rescue inhaler use
RR	Relative risk
SAS	Statistical Analysis Software
SB	Shortness of breath
SEP	Social-economic position

SIC	Standard Industrial Code
SD	Standard deviation
se	Standard error
TDM	Travel Demand Modeling
tpy	Tons per year
TRIM	EPA's Total Risk Integrated Methodology
U95	Upper limit of the 95 <sup>th</sup> confidence interval
US DOT	United States Department of Transportation
US EPA	United States Environmental Protection Agency
USGS	United States Geological Survey
VMT	Vehicle miles traveled
W	Wheeze

# 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 (NO<sub>2</sub>). 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 or 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 NO<sub>2</sub> NAAQS, contains four major components. Each of these components, as they relate to the review of the NO<sub>2</sub> 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 will guide the review. The integrated review plan for this review of the NO<sub>2</sub> primary NAAQS is presented in the *Integrated Review Plan for the Primary National Ambient Air Quality Standard for Nitrogen Dioxide* (EPA, 2007a). The policy-relevant questions identified in this document to guide the review are:

- Has new information altered the scientific support for the occurrence of health effects following short- and/or long-term exposure to levels of nitrogen oxides (NO<sub>x</sub>) found in the ambient air?
- What do recent studies focused on the near-roadway environment tell us about health effects of NO<sub>x</sub>?
- At what levels of NO<sub>x</sub> exposure do health effects of concern occur?
- Has new information altered conclusions from previous reviews regarding the plausibility of adverse health effects caused by NO<sub>x</sub> exposure?
- To what extent have important uncertainties identified in the 1996 review been reduced and/or have new uncertainties emerged?
- What are the air quality relationships between short-term and long-term exposures to NO<sub>x</sub>?

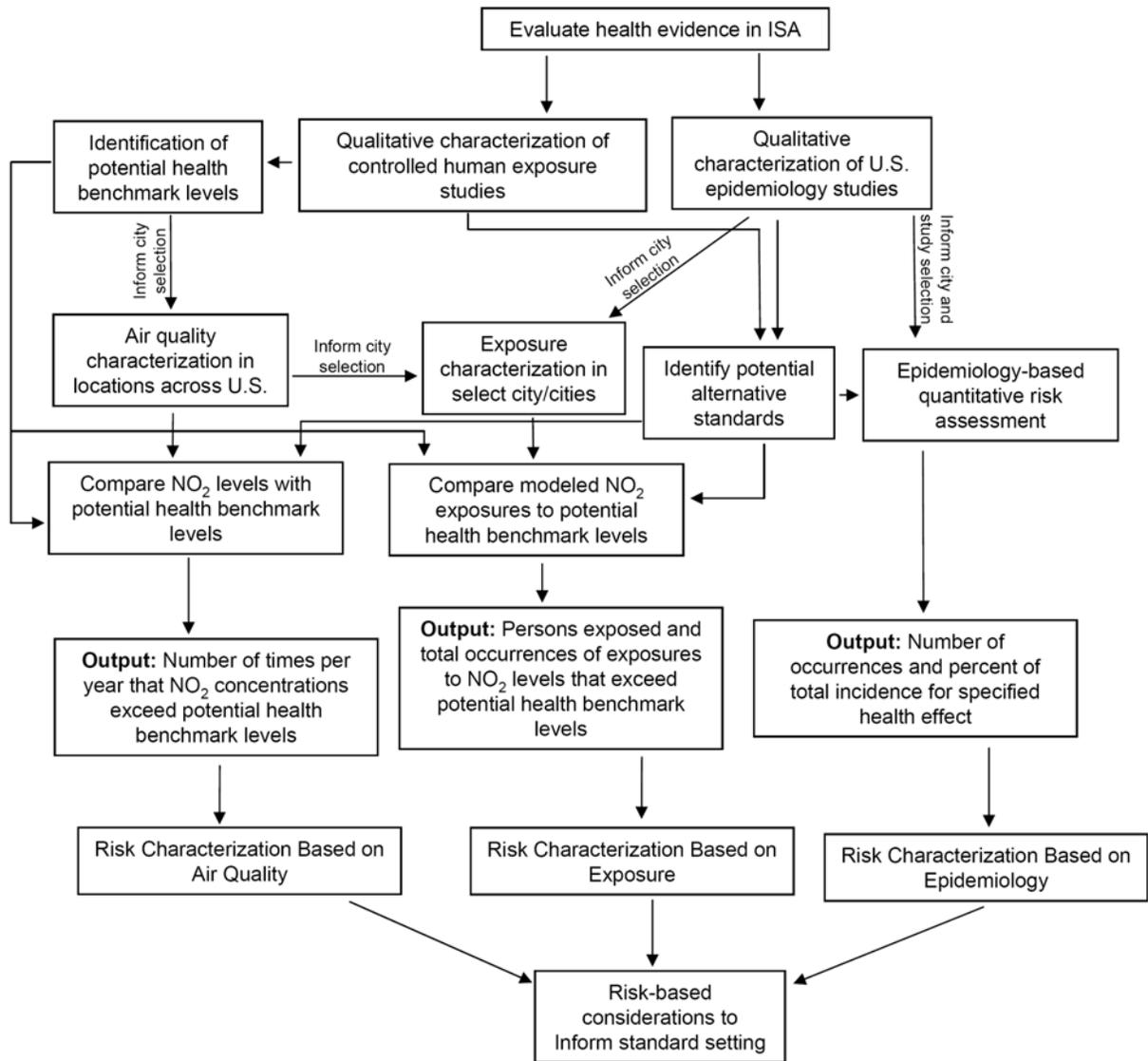
Additional questions will become relevant if the evidence suggests that revision of the current standard might be appropriate. These questions are:

- Is there evidence for the occurrence of adverse health effects at levels of NO<sub>x</sub> lower than those observed previously? If so, at what levels and what are the important uncertainties associated with that evidence?
- Do exposure estimates suggest that exposures of concern for NO<sub>x</sub>-induced health effects will occur with current ambient levels of NO<sub>2</sub> or with levels that just meet current, or potential alternative, standards? If so, are these exposures of sufficient magnitude such that the health effects might reasonably be judged to be important from a public health perspective? What are the important uncertainties associated with these exposure estimates?
- Do the evidence, the air quality assessment, and the risk/exposure assessment provide support for considering different standard indicators or averaging times?
- What range of levels is supported by the evidence, the air quality assessment, and the risk/exposure assessments? What are the uncertainties and limitations in the evidence and the assessments?

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

The second component of the review process is a science assessment. A concise synthesis of the most policy-relevant science has been compiled into the Integrated Science Assessment (ISA). The ISA is supported by a series of annexes that contain more detailed information about the scientific literature. The ISA to support this review of the NO<sub>2</sub> primary NAAQS is presented in the *Integrated Science Assessment for Oxides of Nitrogen - Health Criteria*, henceforth referred to as the ISA (EPA, 2008a).

The third component of the review process is a risk and exposure assessment, which is described in this document. The purpose of this document is to communicate EPA's assessment of exposures and risks associated with ambient NO<sub>2</sub>. In this assessment, we have developed estimates of human exposures and risks associated with current ambient levels of NO<sub>2</sub>, with levels that just meet the current standard, and with levels that just meet potential alternative standards. Figure 1-1 (below) presents a schematic overview of the analyses described in this document and how those analyses fit together. Each of the steps highlighted in Figure 1-1 is described in more detail in subsequent sections.



**Figure 1-1. Overview of the analyses described in this document and their interconnections**

The results of the risk and exposure assessment is considered alongside the health evidence, as evaluated in the final ISA, to inform the policy assessment and rulemaking process, as discussed below in chapter 10. The draft plan for conducting the risk and exposure assessment to support the NO<sub>2</sub> primary NAAQS is presented in the *Nitrogen Dioxide Health Assessment Plan: Scope and Methods for Exposure and Risk Assessment*, henceforth referred to as the Health Assessment Plan (EPA, 2007b). The first draft of the risk and exposure assessment is presented in *Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard: First Draft* (EPA, 2008b). The second draft is presented in *Risk and Exposure*

*Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard: Second Draft* (EPA, 2008c).

The fourth component of the process is the policy assessment and rulemaking. The Agency's views on policy options will be published in the Federal Register as an advance notice of proposed rulemaking (ANPR). This policy assessment will address the adequacy of the current standard and of potential alternative standards, which will be defined in terms of indicator, averaging time, form,<sup>1</sup> and level. To accomplish this, the policy assessment will consider the results of the final risk and exposure assessment as well as the scientific evidence (including evidence from the epidemiologic, controlled human exposure, and animal toxicological literatures) evaluated in the ISA, drawing from the discussion in chapter 10. Taking into consideration CASAC advice and recommendations, as well as public comment on the ANPR, the Agency will publish a proposed rule, to be followed by a public comment period. Taking into account comments received on the proposed rule, the Agency will issue a final rule to complete the rulemaking process.

## **1.2 HISTORY**

### **1.2.1 History of the NO<sub>2</sub> NAAQS**

On April 30, 1971, EPA promulgated identical primary and secondary NAAQS for NO<sub>2</sub> under section 109 of the Act. The standards were set at 0.053 parts per million (ppm), annual average (36 FR 8186). In 1982, EPA published *Air Quality Criteria for Oxides of Nitrogen* (EPA, 1982), which updated the scientific criteria upon which the initial NO<sub>2</sub> standards were based. On February 23, 1984, EPA proposed to retain these standards (49 FR 6866). After taking into account public comments, EPA published the final decision to retain these standards on June 19, 1985 (50 FR 25532).

On July 22, 1987, EPA announced that it was undertaking plans to revise the 1982 air quality criteria (52 FR 27580). In November 1991, EPA released an updated draft air quality criteria document for CASAC and public review and comment (56 FR 59285). The draft document provided a comprehensive assessment of the available scientific and technical information on health and welfare effects associated with NO<sub>2</sub> and other oxides of nitrogen. The

---

<sup>1</sup> 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.

CASAC reviewed the draft document at a meeting held on July 1, 1993 and concluded in a closure letter to the Administrator that the document “provides a scientifically balanced and defensible summary of current knowledge of the effects of this pollutant and provides an adequate basis for EPA to make a decision as to the appropriate NAAQS for NO<sub>2</sub>” (Wolff, 1993). The Air Quality Criteria Document for the Oxides of Nitrogen was then finalized (EPA, 1993).

The EPA also prepared a Staff Paper that summarized an air quality assessment for NO<sub>2</sub> conducted by the Agency (McCurdy, 1994), summarized and integrated the key studies and scientific evidence contained in the revised air quality criteria document, and identified the critical elements to be considered in the review of the NO<sub>2</sub> NAAQS. The CASAC reviewed two drafts of the Staff Paper and concluded in a closure letter to the Administrator (Wolff, 1995) that the document provided a “scientifically adequate basis for regulatory decisions on nitrogen dioxide.” In September of 1995, EPA finalized the Staff Paper entitled, “Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information” (EPA, 1995).

In October 1995, the Administrator announced her proposed decision not to revise either the primary or secondary NAAQS for NO<sub>2</sub> (60 FR 52874; October 11, 1995). A year later, the Administrator made a final determination not to revise the NAAQS for NO<sub>2</sub> after careful evaluation of the comments received on the proposal (61 FR 52852, October 8, 1996). The level for both the existing primary and secondary NAAQS for NO<sub>2</sub> is 0.053 parts per million (ppm) (100 micrograms per cubic meter of air [ $\mu\text{g}/\text{m}^3$ ]), annual arithmetic average, calculated as the arithmetic mean of the 1-hour NO<sub>2</sub> concentrations.

### **1.2.2 Health Evidence from Previous Review**

The prior Air Quality Criteria Document (AQCD) for Oxides of Nitrogen (EPA, 1993) concluded that there were two key health effects of greatest concern at ambient or near-ambient levels of NO<sub>2</sub>, increased airway responsiveness in asthmatic individuals after short-term exposures and increased occurrence of respiratory illness in children with longer-term exposures. Evidence also was found for increased risk of emphysema, but this was of major concern only with exposures to levels of NO<sub>2</sub> much higher than then-current ambient levels. The evidence regarding airway responsiveness was drawn largely from controlled human exposure studies.

The evidence for respiratory illness was drawn from epidemiologic studies that reported associations between respiratory symptoms and indoor exposures to NO<sub>2</sub> in people living in homes with gas stoves. The biological plausibility of the epidemiologic results was supported by toxicological studies that detected changes in lung host defenses following NO<sub>2</sub> exposure. Subpopulations considered potentially more susceptible to the effects of NO<sub>2</sub> included individuals with preexisting respiratory disease, children, and the elderly.

### **1.2.3 Assessment from Previous Review**

In the previous review of the NO<sub>2</sub> NAAQS, risks were characterized by comparing ambient monitoring data, which were used as a surrogate for exposure, with potential health benchmark levels identified from controlled human exposure studies. At the time of the review, a few studies indicated the possibility for adverse health effects due to short-term (e.g., 1-hour) exposures between 0.20 ppm and 0.30 ppm NO<sub>2</sub>. Therefore, the focus of the assessment was on the potential for short-term (i.e., 1-hour) exposures to NO<sub>2</sub> levels above potential health benchmarks in this range. The assessment used monitoring data from the years 1988-1992 and screened for sites with one or more hourly exceedances of potential short-term health effect benchmarks. Predictive models were then constructed to relate the frequency of hourly concentrations above short-term health effect benchmarks to a range of annual average concentrations, including the current standard. Based on the results of this analysis, both CASAC (Wolff, 1995) and the Administrator (60 FR 52874) concluded that the minimal occurrence of short-term peak concentrations at or above a potential health effect benchmark of 0.20 ppm (1-h average) indicated that the existing annual standard would provide adequate health protection against short-term exposures. This conclusion was a key element in the decision in the 1996 review to retain the existing annual standard.

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

NO<sub>x</sub>, for purposes of this document, include multiple gaseous (e.g., NO<sub>2</sub>, NO, HONO) and particulate (e.g., nitrate) species. As discussed in the integrated review plan (2007a), the current review of the NO<sub>2</sub> NAAQS will focus on the gaseous species of NO<sub>x</sub> and will not consider health effects directly associated with particulate species of NO<sub>x</sub>. Of the gaseous

species, EPA has historically determined it appropriate to specify the indicator of the standard in terms of NO<sub>2</sub> because the majority of the information regarding health effects and exposures is for NO<sub>2</sub>. In the current review, staff notes that no alternative to NO<sub>2</sub> has been advanced as being a more appropriate surrogate for ambient gaseous NO<sub>x</sub>. Controlled human exposure studies and animal toxicology studies provide specific evidence for health effects following exposure to NO<sub>2</sub>. Epidemiologic studies also typically report levels of NO<sub>2</sub>, as opposed to other gaseous NO<sub>x</sub>, though the degree to which monitored NO<sub>2</sub> reflects actual NO<sub>2</sub> levels, as opposed to NO<sub>2</sub> plus other gaseous NO<sub>x</sub>, can vary (e.g., see section 2.2.3 of this document). Therefore, NO<sub>2</sub> will be used as the indicator for the gaseous NO<sub>x</sub> in the risk and exposure assessments described in this document.

## **2. SOURCES, AMBIENT LEVELS, AND EXPOSURES**

### **2.1 SOURCES OF NO<sub>2</sub>**

Ambient levels of NO<sub>2</sub> are the product of both direct NO<sub>2</sub> emissions and emissions of other NO<sub>x</sub> (e.g., NO), which can then be converted to NO<sub>2</sub> (for a more detailed discussion see the ISA, section 2.2). Nationally, anthropogenic sources account for approximately 87% of total NO<sub>x</sub> emissions. Mobile sources (both on-road and off-road) account for about 60% of total anthropogenic emissions of NO<sub>x</sub>, 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<sub>x</sub> including microbial activity in soils, lightning, and wildfires (ISA, section 2.2.1 and annex section 2.6.2).

### **2.2 AMBIENT LEVELS OF NO<sub>2</sub>**

#### **2.2.1 Background on NO<sub>2</sub> monitoring network**

From the inception of the NO<sub>2</sub> monitoring network in the late 1970's through the present day, the number of monitoring sites has remained relatively stable (Watkins, 2008). As of October 2008, there were 409 NO<sub>x</sub> monitors within the United States actively reporting NO<sub>2</sub> data into EPA's Air Quality System (AQS). The NO<sub>2</sub> network was originally established for implementation of the NO<sub>2</sub> NAAQS promulgated in 1971. The first requirements for NO<sub>2</sub> monitoring to implement the 1971 NO<sub>2</sub> NAAQS were established in May of 1979. At that time, two NO<sub>2</sub> national ambient monitoring stations (NAMS) were required in areas of the country with populations greater than 1,000,000. 40 CFR Part 58, Appendix D, section 3.5. The regulations noted that within urban areas, two permanent monitors are sufficient, and with respect to those two monitors provided:

The first station (category (a), middle scale or neighborhood scale) would be to measure the photochemical production of NO<sub>2</sub> and would best be located in that part of the urban area where the emission density of NO<sub>x</sub> is the highest. The second station (category (b) urban scale), would be to measure the NO<sub>2</sub> produced

from the reaction of NO with O<sub>3</sub> and should be downwind of the area peak NO<sub>x</sub> emission areas.

40 CFR Part 58, Appendix D, section 3.5.

In October 2006, EPA revised the monitoring requirements for NO<sub>2</sub> in light of the fact that there are no NO<sub>2</sub> non-attainment areas under the current standards. The 2006 rule eliminated the minimum requirements for the number of NO<sub>2</sub> monitoring sites. 40 CFR Part 58, Appendix D, section 4.3. However, the rule requires continued operation of existing State and local monitoring stations (SLAMS) until discontinuation is approved by the EPA Regional Administrator. The revised rule further requires that where SLAMS NO<sub>2</sub> monitoring is ongoing, “at least one NO<sub>2</sub> site in the area must be located to measure the maximum concentration of NO<sub>2</sub>.”

As noted above, the size of the NO<sub>2</sub> network has remained fairly stable through time, even though no minimum monitoring sites were required under the 2006 rule. The maintenance of the NO<sub>2</sub> monitoring network has been driven by several factors, including the need to support ozone (O<sub>3</sub>) modeling and forecasting, the need to track PM precursors, and a general desire on the part of states to continue to understand trends in ambient NO<sub>2</sub>.

To characterize the current NO<sub>2</sub> network, staff has reviewed the NO<sub>2</sub> network meta-data. The data reviewed are those available from AQS in October 2008, for monitors reporting data in 2008. The meta-data fields are typically created by state and local agencies when a monitor site is opened, moved, or re-characterized. While these files are useful for characterizing specific monitors, there is some uncertainty surrounding this meta-data given that there is no routine or enforced process for updating or correcting meta-data fields. With this uncertainty in mind, staff has compiled information on the monitoring objectives and measurement scales for monitors in the NO<sub>2</sub> network.

The monitor objective meta-data field describes the purpose of the monitor. For example the purpose of a particular monitor could be to characterize health effects, photochemical activity, transport, and/or welfare effects. As of October 2008, there were 489 records of NO<sub>2</sub> monitor objective values (some monitors have multiple monitor objectives). Table 2-1 lists the distribution of monitoring objectives across the network. There are 12 categories of monitor objectives for NO<sub>2</sub> monitors within AQS. The “other” category is for sites likely addressing a

state or local need outside of the routine objectives, and the “unknown” category represents missing meta-data. The remaining categories stem directly from categorizations of site types within CFR. In 40 CFR Part 58 Appendix D, there are six examples of NO<sub>2</sub> site types:

1. Sites located to determine the highest concentration expected to occur in the area covered by the network (Highest Concentration).
2. Sites located to measure typical concentrations in areas of high population (Population Exposure).
3. Sites located to determine the impact of significant sources or source categories on air quality (Source Oriented).
4. Sites located to determine general background concentration levels (General Background).
5. Sites located to determine the extent of regional pollutant transport among populated areas; and in support of secondary standards (Regional Transport).
6. Sites located to measure air pollution impacts on visibility, vegetation damage, or other welfare-based impacts (Welfare Related Impacts).

The remaining four categories available are a result of updating the AQS database. In the more recent upgrade to AQS, the data handlers inserted the available site types for Photochemical Assessment Monitoring Stations (PAMS) network. These PAMS site types are spelled out in 40 CFR Part 58 Appendix D:

1. Type 1 sites are established to characterize upwind background and transported O<sub>3</sub> and its precursor concentrations entering the area and will identify those areas which are subjected to transport (Upwind Background).
2. Type 2 sites are established to monitor the magnitude and type of precursor emissions in the area where maximum precursor emissions are expected to impact and are suited for the monitoring of urban air toxic pollutants (Max. Precursor Impact).
3. Type 3 sites are intended to monitor maximum O<sub>3</sub> concentrations occurring downwind from the area of maximum precursor emissions (Max. O<sub>3</sub> Concentration).

4. Type 4 sites are established to characterize the downwind transported O<sub>3</sub> and its precursor concentrations exiting the area and will identify those areas which are potentially contributing to overwhelming transport in other areas (Extreme Downwind).

**Table 2-1. NO<sub>x</sub> Network Distribution of Monitor Objectives**

<b>NO<sub>x</sub> Monitor Objective</b>	<b>Number of Monitor Objective Records</b>	<b>Percent Distribution</b>
Population Exposure	177	36.20
Highest Concentration	58	11.86
General Background	51	10.43
Max. Precursor Impact (PAMS Type 2 Site)	21	4.29
Source Oriented	19	3.89
Upwind Background (PAMS Type 1 Site)	18	3.68
Regional Transport	12	2.45
Other	9	1.84
Max. O <sub>3</sub> Concentration (PAMS Type 3 Site)	8	1.64
Extreme Downwind (PAMS Type 4 Site)	3	0.61
Welfare Related Impacts	1	0.20
Unknown	112	22.90
<b>Totals:</b>	<b>489</b>	<b>100%</b>

The spatial measurement scales are laid out in 40 CFR Part 58, Appendix D, Section 1 “Monitoring Objectives and Spatial Scales.” This part of the regulation spells out what data from a monitor can represent in terms of air volumes associated with area dimensions:

- Microscale - 0 to 100 meters
- Middle Scale - 100 to 500 meters
- Neighborhood Scale - 500 meters to 4 kilometers
- Urban Scale - 4 to 50 kilometers
- Regional Scale - 50 kilometers up to 1000km

There are meta-data records for the NO<sub>2</sub> network to indicate what the measurement scale of a particular monitor represents. There are 386 NO<sub>2</sub> monitor records in AQS with available measurement scale information. Table 2-2 shows the measurement scale distribution across all NO<sub>2</sub> sites from the available data in AQS of monitors reporting data in 2008.

**Table 2-2. NO<sub>x</sub> Network Distribution across Measurement Scales.**

<b>Measurement Scale</b>	<b>Number of Measurement Scale Records</b>	<b>Percent Distribution</b>
Microscale	3	0.78
Middle Scale	23	5.96
Neighborhood	212	54.92
Urban Scale	119	30.83
Regional Scale	29	7.51
<b>Totals:</b>	<b>386</b>	<b>100%</b>

In summary, upon review of the known 409 monitors reporting data to AQS in 2008, and the distribution of the available data from the categories of monitor objective and measurement scale, we see the NO<sub>2</sub> network is primarily targeting public health and photochemical process monitoring objectives. We note that nearly half of the monitor objective records are directly targeting public health through the population exposure (36.2%) and highest concentration (11.8%) categories alone. The other categories serve to inform public health concerns, but also address photochemistry issues where NO<sub>x</sub> serves as a precursor to ozone. Further, it appears that approximately 10% of NO<sub>2</sub> monitors are in place to serve the PAMS network. In reality, a large majority of sites likely could serve both public health and photochemistry related objectives due to their proximity to urban areas. The exceptions would likely be categories such as upwind background, extreme downwind, regional transport, and possibly maximum O<sub>3</sub> concentration. These four categories only represent approximately 7% of the NO<sub>2</sub> network, and have a higher likelihood of being rural and likely regional in scale.

### **2.2.2 Trends in ambient concentrations of NO<sub>2</sub>**

As noted above, NO<sub>2</sub> is monitored largely in urban areas and, therefore, data from the NO<sub>2</sub> monitoring network is generally more representative of urban areas than rural areas. According to monitoring data, nationwide levels of ambient NO<sub>2</sub> (annual average) decreased 41% between 1980 and 2006 (ISA, Figure 2.4-15). Between 2003 and 2005, national mean concentrations of NO<sub>2</sub> were about 15 ppb for averaging periods ranging from a day to a year. The average daily maximum hourly NO<sub>2</sub> 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 (ISA, Figure 2.4-13). The highest levels of NO<sub>2</sub> in the United States can be found in and around Los Angeles, in the Midwest, and in the Northeast. 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), are estimated to range from only 0.1 ppb to 0.3 ppb (ISA, section 2.4.6).

Ambient levels of NO<sub>2</sub> exhibit both seasonal and diurnal variation. In southern cities, such as Atlanta, higher concentrations are found during winter, consistent with the lowest mixing layer heights being found during that time of the year. Lower concentrations are found during summer, consistent with higher mixing layer heights and increased rates of photochemical oxidation of NO<sub>2</sub>. For cities in the Midwest and Northeast, such as Chicago and New York City, higher levels tend to be found from late winter to early spring with lower levels occurring from summer through the fall. In Los Angeles the highest levels tend to occur from autumn through early winter and the lowest levels from spring through early summer. Mean and peak concentrations in winter can be up to a factor of two larger than in the summer at sites in Los Angeles. In terms of daily variability, NO<sub>2</sub> levels typically peak during the morning rush hours. Monitor siting plays a key role in evaluating diurnal variability as monitors located further away from traffic will show cycles that are less pronounced over the course of a day than monitors located closer to traffic.

### **2.2.3 Uncertainty Associated with the Ambient NO<sub>2</sub> Monitoring Method**

The method for estimating ambient NO<sub>2</sub> levels (i.e., subtraction of NO from a measure of total NO<sub>x</sub>) is subject to interference by NO<sub>x</sub> oxidation products (e.g., PAN, HNO<sub>3</sub>) (ISA, section 2.3). Limited evidence suggests that these compounds result in an overestimate of NO<sub>2</sub> levels by roughly 20 to 25% at typical ambient levels. Smaller relative errors are estimated to occur in measurements taken near strong NO<sub>x</sub> sources since most of the mass emitted as NO or NO<sub>2</sub> would not yet have been further oxidized. Relatively larger errors appear in locations more distant from strong local NO<sub>x</sub> sources. Additionally, many NO<sub>2</sub> monitors are elevated above ground level in the cores of large cities. Because most sources of NO<sub>2</sub> are near ground level (i.e., combustion emissions from traffic), there is a gradient of NO<sub>2</sub> with higher levels near ground level and lower levels being detected at the elevated monitor. One comparison has found an

average of a 2.5-fold higher NO<sub>2</sub> concentration measured at 4 meters above the ground compared to 15 meters above the ground. The ISA notes that levels are likely even higher at elevations below 4 meters (ISA, section 2.5.3.3). Another source of uncertainty in exposure estimates can result from monitor location. NO<sub>2</sub> monitors are sited for compliance with air quality standards rather than for capturing small-scale variability in NO<sub>2</sub> concentrations near sources such as roadway traffic. Significant gradients in NO<sub>2</sub> concentrations near roadways have been observed in several studies, and NO<sub>2</sub> concentrations have been found to be correlated with distance from roadway and traffic volume (ISA, section 2.5.3.2).

## **2.3 EXPOSURE TO NO<sub>2</sub>**

### **2.3.1 Overview**

Human exposure to an airborne pollutant can be characterized by contact between a person and the pollutant at a specific concentration for a specified period of time (ISA, section 2.5.1). The integrated exposure of a person to a given pollutant is the time-weighted average of the exposures over all time intervals for all microenvironments in which the individual spends time. Microenvironments in which people are exposed to air pollutants such as NO<sub>2</sub> typically include residential indoor environments and other indoor locations, near-traffic outdoor environments and other outdoor locations, and in vehicles (ISA, Figure 2.5-1).

There is a large amount of variability in the time that individuals spend in different microenvironments, but on average people spend the majority of their time (about 87%) indoors. Most of this time is spent at home with less time spent in an office/workplace or other indoor locations (ISA, Figure 2.5-1). On average in the U.S., people spend about 8% of their time outdoors and 6% of their time in vehicles. Significant variability surrounds each of these broad estimates, particularly when considering influential personal attributes such as age or gender; when accounting for daily, weekly, or seasonal factors influencing personal behavior; or when characterizing individual variability in time spent in various locations (McCurdy and Graham, 2003; Graham and McCurdy, 2004). Typically, the time spent outdoors or in vehicles could vary by 100% or more depending on which of these influential factors are considered. Exposure misclassification can result when the time spent in different microenvironments is not taken into consideration and may obscure the true relationship between ambient air pollutant exposures and health outcomes.

### **2.3.2 Uncertainty Associated with Ambient Levels as a Surrogate for Exposure**

Many epidemiologic studies rely on measures of ambient NO<sub>2</sub> concentrations as surrogates for personal exposure to ambient NO<sub>2</sub>. Results have been mixed regarding the appropriateness of using ambient levels of NO<sub>2</sub> as a surrogate for personal exposures to ambient NO<sub>2</sub>. Studies examining the association between ambient NO<sub>2</sub> and personal exposure to NO<sub>2</sub> have generated mixed results due to 1) the prevalence of indoor sources of NO<sub>2</sub>; 2) the spatial heterogeneity of NO<sub>2</sub> in study areas; 3) the seasonal and geographic variability in the infiltration of ambient NO<sub>2</sub>; 4) differences in the time spent in different microenvironments; and 5) differences in study design. As a result, some researchers have concluded that ambient NO<sub>2</sub> may be a reasonable proxy for personal exposure, while others have noted that caution must be exercised (ISA, section 2.5.9). However, the possible consequences of this exposure error do not bias conclusions in a positive direction (see chapter 4 of this document) since it generally tends to reduce, rather than increase, effect estimates (ISA, section 5.2.2).

## **3. AT RISK POPULATIONS**

### **3.1 OVERVIEW**

Specific groups within the general population are at increased risk for suffering adverse effects from NO<sub>2</sub> exposure. This could occur because they are affected by lower levels of NO<sub>2</sub> 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 NO<sub>2</sub> than the general population (vulnerability). The term susceptibility generally encompasses innate (e.g., genetic or developmental) and/or acquired (e.g., age or disease) factors that make individuals more likely to experience effects with exposure to pollutants. Given the likely heterogeneity of individual responses to air pollution, the severity of health effects experienced by a susceptible subgroup may be much greater than that experienced by the population at large. Factors that may influence susceptibility to the effects of air pollution include age (e.g., infants, children, elderly); gender; race/ethnicity; genetic factors; and pre-existing disease/condition (e.g., obesity, diabetes, respiratory disease, asthma, chronic obstructive pulmonary disease (COPD), cardiovascular disease, airway hyperresponsiveness, respiratory infection, adverse birth outcome) (ISA, sections 4.3.1, 4.3.5, and 5.3.2.8). In addition, some population groups are vulnerable to pollution-related effects because their air pollution exposures are higher than those of the general population. Factors that may influence vulnerability to the effects of air pollution include socioeconomic status, education level, air conditioning use, proximity to roadways, geographic location, level of physical activity, and work environment (e.g., indoor versus outdoor) (ISA, section 4.3.5). The ISA discusses factors that can confer susceptibility and/or vulnerability to air pollution with most of the discussion devoted to factors for which NO<sub>2</sub>-specific evidence exists (ISA, section 4.3). These factors are presented in table 3-1 below (from section 4.3.5 of the ISA) and are discussed in subsequent sections of this chapter (see ISA, chapter 4 for more detail).

**Table 3-1. Overview of Susceptibility and Vulnerability Factors**

**Susceptibility Factors**

- Age, Gender
- Adverse birth outcomes: e.g., preterm birth, low birth weight, growth restriction, birth defects
- Race/ethnicity
- Genetic factors
- Pre-existing disease, e.g., diabetes
- Obesity
- Respiratory diseases, e.g., asthma, COPD
- Cardiovascular diseases

**Vulnerability Factors**

- Socioeconomic status
- Education level
- Air conditioning Use
- Proximity to Roadways
- Geographic Location (West vs. East)
- Level of Exercise
- Work Environment (e.g., outdoor workers)

**3.2 SUSCEPTIBILITY: PRE-EXISTING DISEASE**

A number of health conditions have been found to put individuals at greater risk for adverse events following exposure to air pollution. In general, these include asthma, COPD, respiratory infection, conduction disorders, congestive heart failure (CHF), diabetes, past myocardial infarction (MI), obesity, coronary artery disease, low birth weight/prematurity, and hypertension (ISA, sections 4.3.1, 4.3.5, and 5.3.2.9). In addition to these conditions, epidemiologic evidence indicates that individuals with bronchial or airway hyperresponsiveness, as determined by methacholine provocation, may be at increased risk for experiencing respiratory symptoms (ISA, section 4.3.1). In considering NO<sub>2</sub> specifically, the ISA evaluated studies on asthmatics, individuals with cardiopulmonary disease, and diabetics (ISA, sections 4.3.1.1 and 4.3.1.2). These groups are discussed in more detail below.

Epidemiologic and controlled human exposure studies, supported by animal toxicology studies, have provided evidence for associations between NO<sub>2</sub> exposure and respiratory effects in asthmatics (ISA, section 4.3.1.1). The ISA found evidence from epidemiologic studies for an association between ambient NO<sub>2</sub> and children’s hospital admissions, emergency department (ED) visits, and calls to doctors for asthma. NO<sub>2</sub> levels were associated with aggravation of asthma effects that include symptoms, medication use, and lung function. Time-series studies also demonstrated a relationship in children between hospital admissions or ED visits for asthma and ambient NO<sub>2</sub> levels, even after adjusting for co-pollutants such as particulate matter (PM) and carbon monoxide (CO) (ISA, section 4.3.1.1). Important evidence was also available from

epidemiologic studies of indoor NO<sub>2</sub> exposures. Recent studies have shown associations with asthma attacks and severity of virus-induced asthma (ISA, section 4.3.1.1). In addition, in controlled human exposure studies, airway hyperresponsiveness in asthmatics occurred following exposure to lower NO<sub>2</sub> concentrations than the concentrations that caused effects on other endpoints (ISA, sections 5.3.2.1-5.3.2.6).

Compared to asthma, less evidence is available to support cardiovascular disease as a mediator of susceptibility to NO<sub>2</sub>. However, recent epidemiologic studies report that individuals with preexisting conditions (e.g., including diabetes, CHF, prior MI) may be at increased risk for adverse cardiac health events associated with ambient NO<sub>2</sub> concentrations (ISA, section 4.3.1.2). The small number of controlled human exposure and animal toxicological studies that have evaluated cardiovascular endpoints provide only limited supporting evidence for susceptibility to NO<sub>2</sub> in persons with cardiovascular disease (ISA, section 4.3.1.2).

### **3.3 SUSCEPTIBILITY: AGE**

The ISA identifies infants, 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 ambient NO<sub>2</sub> concentrations (ISA, section 4.3.2). The ISA found evidence that associations of NO<sub>2</sub> with respiratory ED visits and hospitalizations were stronger among children and older adults, though not all studies had comparable findings on this issue (ISA, section 4.3.2). In addition, long-term exposure studies suggest effects in children that include impaired lung function growth, increased respiratory symptoms and infections, and onset of asthma (ISA, section 3.4 and 4.3.2). In some studies, associations between NO<sub>2</sub> and hospitalizations or ED visits for CVD have been observed in elderly populations. Among studies that observed positive associations between NO<sub>2</sub> and mortality, a comparison indicated that, in general, the elderly population was more susceptible than the non-elderly population to NO<sub>2</sub> effects (ISA, section 4.3.2).

### **3.4 SUSCEPTIBILITY: GENETICS**

As noted in the ISA (section 4.3.4), genetic factors related to health outcomes and ambient pollutant exposures merit consideration. Several criteria must be satisfied in selecting and establishing useful links between polymorphisms in candidate genes and adverse respiratory

effects. First, the candidate gene must be significantly involved in the pathogenesis of the adverse effect of interest. Second, polymorphisms in the gene must produce a functional change in either the protein product or in the level of expression of the protein. Third, in epidemiologic studies, the issue of confounding by other environmental exposures must be carefully considered (ISA, section 4.3.4).

Investigation of genetic susceptibility to NO<sub>2</sub> effects has focused on the glutathione S-transferase (GST) gene. Several GST genes have common, functionally-important alleles that affect host defense in the lung (ISA, section 4.3.4). GST genes are inducible by electrophilic species (e.g., reactive oxygen species) and individuals with genotypes that result in enzymes with reduced or absent peroxidase activity are likely to have reduced defenses against oxidative insult. This could potentially result in increased susceptibility to inhaled oxidants and radicals. However, data on genetic susceptibility to NO<sub>2</sub> are only beginning to emerge and, while it remains plausible that there are genetic factors that can influence health responses to NO<sub>2</sub>, the few available studies do not provide specific support for genetic susceptibility to NO<sub>2</sub> exposure (ISA, section 4.3.4).

### **3.5 SUSCEPTIBILITY: GENDER**

As reported in the ISA, a limited number of NO<sub>2</sub> studies have stratified results by gender. The results of these studies were mixed, and the ISA does not draw conclusions regarding the potential for gender to confer susceptibility to the effects of NO<sub>2</sub> (ISA, section 4.3.3).

### **3.6 VULNERABILITY: PROXIMITY (ON OR NEAR) TO ROADWAYS**

The ISA includes discussion of vulnerable populations that experience increased NO<sub>2</sub> exposures on or near roadways (ISA, section 4.3.6). Large gradients in NO<sub>x</sub> concentrations near roadways lead to increased exposures for individuals residing, working, or attending school in the vicinity of roadways. Many studies find that indoor, personal, and outdoor NO<sub>2</sub> levels are strongly associated with proximity to traffic or to traffic density (ISA, section 4.3.6). Due to high air exchange rates, NO<sub>2</sub> levels inside a vehicle could rapidly approach levels outside the vehicle during commuting (ISA, section 4.3.6). Mean in-vehicle NO<sub>2</sub> levels are between 2 and 3 times ambient levels measured at fixed sites nearby (ISA, section 4.3.6). Therefore, individuals with occupations that require them to be in traffic or close to traffic (e.g., bus and taxi drivers,

highway patrol officers, toll collectors) and individuals with long commutes could be exposed to relatively high levels of NO<sub>2</sub> compared to ambient levels. Due to the high peak exposures while driving, total personal exposure could be underestimated if exposures while commuting are not considered.

### **3.7 VULNERABILITY: SOCIOECONOMIC STATUS**

The ISA discusses evidence that socioeconomic status (SES) modifies the effects of air pollution (section 4.3.6). Many recent studies examined modification by SES indicators on the association between mortality and PM or other indices such as traffic density, distance to roadway, or a general air pollution index (ISA, section 4.3.6). SES modification of NO<sub>2</sub> associations has been examined in fewer studies. For example, in a study conducted in Seoul, South Korea, community-level SES indicators modified the association of air pollution with ED visits for asthma. Of the five criteria air pollutants evaluated, NO<sub>2</sub> showed the strongest association in lower SES districts compared to high SES districts (Kim et al., 2007). In addition, Clougherty et al. (2007) evaluated exposure to violence (a potential surrogate for SES) as a modifier of the effect of traffic-related air pollutants, including NO<sub>2</sub>, on childhood asthma. The authors reported an elevated risk of asthma with an increase in NO<sub>2</sub> exposure solely among children with above-median exposure to violence in their neighborhoods (ISA, section 4.3.6). Although these recent studies have evaluated the impact of SES on vulnerability to NO<sub>2</sub>, they are too few in number to draw definitive conclusions (ISA, section 5.3.2.8).

### **3.8 CONCLUSIONS**

The population potentially affected by NO<sub>2</sub> is large. A considerable fraction of the population resides, works, or attends school near major roadways, and these individuals are likely to have increased exposure to NO<sub>2</sub> (ISA, section 4.4). Based on data from the American Housing Survey, approximately 36 million individuals live within 300 feet (~90 meters) of a four-lane highway, railroad, or airport (ISA, section 4.4). Furthermore, in California, 2.3% of schools with a total enrollment of more than 150,000 students were located within ~500 feet of high-traffic roads, with a higher proportion of non-white and economically disadvantaged students attending those schools (ISA, section 4.4). Of this population, asthmatics and members of other susceptible groups discussed above will have even greater risks of health effects related

to NO<sub>2</sub>. In the United States, approximately 10% of adults and 13% of children have been diagnosed with asthma, and 6% of adults have been diagnosed with COPD (ISA, section 4.4). The prevalence and severity of asthma is higher among certain ethnic or racial groups such as Puerto Ricans, American Indians, Alaskan Natives, and African Americans (ISA, section 4.4). Furthermore, a higher prevalence of asthma among persons of lower SES and an excess burden of asthma hospitalizations and mortality in minority and inner-city communities have been observed (ISA, section 4.4). In addition, population groups based on age also comprise substantial segments of the population that may be potentially at risk for NO<sub>2</sub>-related health impacts. Based on U.S. census data from 2000, about 72.3 million (26%) of the U.S. population are under 18 years of age, 18.3 million (7.4%) are under 5 years of age, and 35 million (12%) are 65 years of age or older. Hence, large proportions of the U.S. population are in age groups that are likely to have increased susceptibility and vulnerability for health effects from ambient NO<sub>2</sub> exposure. The considerable size of the population groups at risk indicates that exposure to ambient NO<sub>2</sub> could have a significant impact on public health in the United States.

## **4. HEALTH EFFECTS**

### **4.1 INTRODUCTION**

The ISA, along with its associated annexes, provides a comprehensive review and assessment of the scientific evidence related to the health effects associated with NO<sub>2</sub> exposures. For these health effects, the ISA characterizes judgments about causality with a hierarchy (for discussion see ISA, section 1.3) 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 criteria that are based on those set forth by Sir Austin Bradford Hill in 1965 (ISA, table 1.3-1). These criteria 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. A summary of each of the five levels of the hierarchy is provided in table 1.3-2 of the ISA, which has been included below (table 4-1).

**Table 4-1. Weight of Evidence for Causal Determination**

<p><b>Sufficient to infer a causal relationship</b></p>	<p>Evidence is sufficient to conclude that there is a causal relationship between relevant pollutant exposure and the outcome. Causality is supported when an association has been observed between the pollutant and the outcome in studies in which chance, bias, and confounding could be ruled out with reasonable confidence. That is, human clinical studies provide the strongest evidence for causality. Causality is also supported by findings from epidemiologic “natural experiments” or observational studies supported by other lines of evidence. Generally, determination is based on multiple studies from more than one research group.</p>
<p><b>Sufficient to infer a likely causal relationship (i.e., more likely than not).</b></p>	<p>Evidence is sufficient to conclude that there is a likely causal association between relevant pollutant exposures and the outcome. That is, an association has been observed between the pollutant and the outcome in studies in which chance, bias and confounding are minimized, but uncertainties remain. For example, observational studies show associations but confounding and other issues are difficult to address and/or other lines of evidence (human clinical, animal, or mechanism of action information) are limited or inconsistent. Generally, determination is based on multiple studies from more than one research group.</p>
<p><b>Suggestive, but not sufficient to infer a causal relationship</b></p>	<p>Evidence is suggestive of an association between relevant pollutant exposures and the outcome, but is weakened because chance, bias and confounding cannot be ruled out. For example, at least one high-quality study shows an association, while the results of other studies are inconsistent.</p>
<p><b>Inadequate to infer the presence or absence of a causal relationship</b></p>	<p>The available studies are inadequate to infer the presence or absence of a causal relationship. That is, studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of an association between relevant pollutant exposure and the outcome. For example, studies which fail to control for confounding or which have inadequate exposure assessment, fall into this category.</p>
<p><b>Suggestive of no causal relationship</b></p>	<p>The available studies are suggestive of no causal relationship. That is, several adequate studies, examining relationships between relevant population exposures and outcomes, and considering sensitive subpopulations, are mutually consistent in not showing an association between exposure and the outcome at any level of exposures. In addition, the possibility of a small elevation in risk at the levels of exposure studied can never be excluded.</p>

The judgments of the ISA, along with the rationale supporting those judgments, are summarized in tables 4-2 and 4-3 below (ISA, table 5.3-1) and are presented in more detail in subsequent sections of this chapter.

**Table 4-2. Causality judgments made in the ISA for endpoints associated with short-term NO<sub>2</sub> exposures**

HEALTH OUTCOME	CONCLUSION FROM PREVIOUS NAAQS REVIEW	CONCLUSION FROM 2008 ISA
<b>SHORT-TERM EXPOSURE TO NO<sub>2</sub></b>		
<b>Respiratory Morbidity</b>	<b>No Overall Conclusion</b>	<b>“sufficient to infer a likely causal relationship”</b>
Lung Host Defense	Human clinical studies suggest NO <sub>2</sub> effects; Animal toxicological studies indicate that alveolar macrophages and humoral and cell-mediated immune systems are affected and show that exposure can impair the respiratory host defense system resulting in susceptibility to infection.	Impaired host-defense systems and increased risk of susceptibility to both viral and bacterial infections after NO <sub>2</sub> exposures have been observed in epidemiologic, human clinical, and animal toxicological studies.
Airway Inflammation	No Studies	Human clinical studies report effects of NO <sub>2</sub> (1-2 ppm) on airway inflammation in healthy humans. Animal toxicological studies and limited available epidemiologic studies on children support these findings.
Airway Hyperresponsiveness	An increase in responsiveness to bronchoconstrictors was found in asthmatics and healthy individuals exposed to NO <sub>2</sub> at rest.	Human clinical studies of allergen and nonspecific bronchial challenges in asthmatics observed increased airway hyperresponsiveness at near ambient concentrations (0.1-0.3 ppm). Increased responsiveness to nonspecific challenges was also observed in animals at higher NO <sub>2</sub> levels (e.g., 0.5 ppm).
Respiratory Symptoms	Children living in homes with gas stoves are at increased risk for developing respiratory diseases and illnesses compared to children living in homes without gas stoves.	Epidemiologic studies provide consistent evidence of an association of respiratory effects with indoor and personal NO <sub>2</sub> exposures in children. Multicity studies provide further support for associations between ambient NO <sub>2</sub> concentrations (means of 7-70 ppb) and respiratory symptoms in asthmatic children.
Lung Function	Lung function changes in asthmatics reported at low (0.2 to 0.5 ppm), but not higher (up to 4 ppm), NO <sub>2</sub> concentrations. No convincing evidence of lung function decrements in healthy individuals below 1.0 ppm.	The association between ambient NO <sub>2</sub> concentrations and lung function in epidemiologic studies were generally inconsistent. Recent clinical evidence generally confirms prior findings.
ED Visits / Hospital Admissions	No Studies	Positive and generally robust associations observed between ambient NO <sub>2</sub> levels (means of 3-50 ppb) and increased ED visits and hospital admissions for respiratory causes, especially asthma.
<b>Cardiovascular Morbidity</b>	<b>No Studies</b>	<b>“inadequate to infer the presence or absence of a causal relationship”</b>
Cardiovascular Effects	No Studies	Evidence from epidemiologic studies of heart rate variability, repolarization changes, and cardiac rhythm disorders among heart patients with ischemic cardiac disease are inconsistent.
ED Visits / Hospital Admissions	No Studies	Generally positive associations between ambient NO <sub>2</sub> concentrations and hospital admissions or ED visits for cardiovascular disease; however, the effects were not robust to adjustment for copollutants.
<b>Mortality</b>	<b>No Studies</b>	<b>“suggestive but not sufficient to infer a causal relationship”</b>
All Cause and Cardiopulmonary Mortality	No Studies	Positive and generally robust associations between ambient NO <sub>2</sub> concentrations and risk of nonaccidental and cardiopulmonary mortality.

**Table 4-3. Causality judgments made in the ISA for endpoints associated with long-term NO<sub>2</sub> exposures**

HEALTH OUTCOME	CONCLUSION FROM PREVIOUS NAAQS REVIEW	CONCLUSION FROM 2008 ISA
<b>LONG-TERM EXPOSURE TO NO<sub>2</sub></b>		
Respiratory Morbidity	No Overall Conclusion	“suggestive but not sufficient to infer a causal relationship”
Respiratory Effects	NO <sub>2</sub> can cause emphysema (meeting the human definition criteria) in animals at high concentrations of NO <sub>2</sub> .	Epidemiologic studies observed decrements in lung function growth associated with long-term exposure to NO <sub>2</sub> .
Other Morbidity	No Studies	“inadequate to infer the presence or absence of a causal relationship”
Cancer	No Studies	Limited epidemiologic studies observed an association between long-term NO <sub>2</sub> exposure and cancer; animal toxicological studies have not provided clear evidence that NO <sub>2</sub> acts as a carcinogen.
Cardiovascular Effects	No Studies	Very limited epidemiologic and toxicological evidence does not suggest that long-term exposure to NO <sub>2</sub> has cardiovascular effects.
Birth Outcomes	No Studies	The epidemiologic evidence for an association between long-term exposure to NO <sub>2</sub> and birth outcomes is generally inconsistent, with limited support from animal toxicological studies.
Mortality	No Studies	“inadequate to infer the presence or absence of a causal relationship”
All Cause and Cardiopulmonary Mortality	No Studies	The results of epidemiologic studies examining the association between long-term exposure to NO <sub>2</sub> and mortality were generally inconsistent.

## 4.2 ADVERSE RESPIRATORY EFFECTS FOLLOWING SHORT-TERM EXPOSURES

### 4.2.1 Overview

The ISA concludes that, taken together, recent studies provide scientific evidence that is sufficient to infer a likely causal relationship between short-term NO<sub>2</sub> exposure and adverse effects on the respiratory system (ISA, section 5.3.2.1). This finding is supported by the large body of recent epidemiologic evidence as well as findings from human and animal experimental studies. These epidemiologic and experimental studies encompass a number of endpoints including ED visits and hospitalizations, respiratory symptoms, airway hyperresponsiveness, airway inflammation, and lung function. Effect estimates from epidemiologic studies conducted in the United States and Canada generally indicate a 2-20%<sup>2</sup> increase in risks for ED visits and hospital admissions and higher risks for respiratory symptoms (ISA, section 5.4). The findings

<sup>2</sup> Effect estimates in the ISA were standardized to a 30 ppb increase in NO<sub>2</sub> for studies that evaluated 1-h daily maximum NO<sub>2</sub> concentrations and to a 20 ppb increase for studies that evaluated 24-h average concentrations.

relevant to these endpoints, which provide the rationale to support the judgment of a likely causal relationship, are described in more detail below.

#### **4.2.2 Respiratory Emergency Department Visits and Hospitalizations**

Epidemiologic evidence exists for positive associations of short-term ambient NO<sub>2</sub> concentrations below the current NAAQS with increased numbers of ED visits and hospital admissions for respiratory causes, especially asthma (ISA, section 5.3.2.1). Total respiratory causes for ED visits and hospitalizations typically include asthma, bronchitis and emphysema (collectively referred to as COPD), pneumonia, upper and lower respiratory infections, and other minor categories. Temporal associations between ED visits or hospital admissions for respiratory diseases and ambient levels of NO<sub>2</sub> have been the subject of over 50 peer-reviewed research publications since the review of the NO<sub>2</sub> NAAQS that was completed in 1996. These studies have examined morbidity in different age groups and have often utilized multi-pollutant models to evaluate potential confounding effects of co-pollutants. Associations are particularly consistent among children (< 14 years) and older adults (> 65 years) when all respiratory outcomes are analyzed together (ISA, figures 3.1-8 and 3.1-9) and among children and subjects of all ages for asthma admissions (ISA, figures 3.1-12 and 3.1-13). When examined with co-pollutant models, associations of NO<sub>2</sub> with respiratory ED visits and hospital admissions were generally robust and independent of the effects of co-pollutants (ISA, figures 3.1-10 and 3.1-11). The plausibility and coherence of these effects are supported by experimental (i.e., toxicologic and controlled human exposure) studies that evaluate host defense and immune system changes, airway inflammation, and airway responsiveness (see subsequent sections of this document and ISA, section 5.3.2.1).

Of the ED visit and hospital admission studies reviewed in the ISA, 6 key studies were conducted in the United States (ISA, table 5.4-1). Of these 6 studies, 4 evaluated associations with NO<sub>2</sub> using multi-pollutant models (Peel et al., 2005 and Tolbert et al., 2007 in Atlanta; New York Department of Health (NYDOH), 2006 and Ito et al., 2007 in New York City) while 2 studies used only single pollutant models (Linn et al., 2000; Jaffe et al., 2003). In the study by Peel and colleagues, investigators evaluated ED visits among all ages in Atlanta, GA during the period of 1993 to 2000. Using single pollutant models, the authors reported a 2.4% (95% CI: 0.9, 4.1) increase in respiratory ED visits associated with a 30-ppb increase in 1-h max NO<sub>2</sub>

concentrations. For asthma visits, a 4.1% (95% CI: 0.8%, 7.6%) increase was estimated in individuals 2 to 18 years of age. Tolbert and colleagues reanalyzed these data with 4 additional years of information and found essentially similar results in single pollutant models (2.0% increase, 95% CI: 0.5, 3.3). This same study found that the associations were positive, but not statistically-significant, in multi-pollutant models that included PM<sub>10</sub> or O<sub>3</sub>. In the study conducted by the New York Department of Health, investigators evaluated asthma ED visits in Bronx and Manhattan, New York over the period of January, 1999 to November, 2000. In Bronx, the authors estimated a 6% (95% CI: 1%-10%) increase in visits per 20 ppb increase in 24-h average concentrations of NO<sub>2</sub> and a 7% increase in visits per 30 ppb increase in daily 1-h maximum concentrations. These effects were not statistically-significant in 2-pollutant models that included PM<sub>2.5</sub> or SO<sub>2</sub>. In Manhattan, the authors found non-significant decreases (3% for 24-h and a 2% for daily 1-h maximum) in ED visits associated with increasing NO<sub>2</sub>. In the study by Ito and colleagues, investigators evaluated ED visits for asthma in New York City during the years 1999 to 2002. The authors estimated a 12% (95% CI: 7%, 15%) increase in risk per 20 ppb increase in 24-h ambient NO<sub>2</sub>. Risk estimates were robust and remained statistically significant in multi-pollutant models that included PM<sub>2.5</sub>, O<sub>3</sub>, CO, and SO<sub>2</sub>. With regard to the studies that evaluated only single pollutant models, Linn et al. (2000) detected a statistically-significant increase in hospital admissions and Jaffee et al. (2003) detected a positive, but statistically-nonsignificant, increase in ED visits associated with 24-h NO<sub>2</sub> concentrations.

#### **4.2.3 Respiratory Symptoms**

Evidence for associations between NO<sub>2</sub> and respiratory symptoms is derived primarily from the epidemiologic literature, although the experimental evidence for airway inflammation and immune system effects (described in the ISA, section 3.1 and summarized in subsequent sections of this document) does provide some plausibility and coherence for the epidemiologic results (ISA, section 5.3.2.1). Consistent evidence has been observed for an association of respiratory effects with indoor and personal NO<sub>2</sub> exposures in children (ISA, sections 3.1.5.1 and 5.3.2.1) and with ambient levels of NO<sub>2</sub> as measured by community monitors (ISA, sections 3.1.4.2 and 5.3.2.1, see Figure 3.1-6). In the results of multi-pollutant models, NO<sub>2</sub> associations in multicity studies are generally robust to adjustment for co-pollutants including O<sub>3</sub>, CO, and

PM<sub>10</sub> (ISA, sections 3.1.4.3, 5.3.2.1 and Figure 3.1-7). Specific studies of respiratory symptoms are discussed in more detail below.

### *Studies of Ambient NO<sub>2</sub>*

Epidemiologic studies using community ambient monitors have found associations between ambient NO<sub>2</sub> concentrations and respiratory symptoms (ISA, sections 3.1.4.2 and 5.3.2.1, Figure 3.1-6) in cities where NO<sub>2</sub> concentrations were within the range of 24-h average concentrations observed in recent years. Several studies have been published since the 1996 review of the NO<sub>2</sub> NAAQS including single-city studies (e.g., Ostro et al., 2001; Delfino et al., 2002) and multi-city studies in urban areas covering the continental United States and southern Ontario (Schwartz et al., 1994; Mortimer et al., 2002; Schildcrout et al., 2006). The multi-city studies are discussed in more detail below.

Schwartz et al. (1994) studied 1,844 schoolchildren, followed for 1 year, as part of the Six Cities Study that included the cities of Watertown, MA, Baltimore, MD, Kingston-Harriman, TN, Steubenville, OH, Topeka, KS, and Portage, WI. Respiratory symptoms were recorded daily. The authors reported a significant association between 4-day mean NO<sub>2</sub> levels and incidence of cough among all children in single-pollutant models, with an odds ratio (OR) of 1.61 (95% CI: 1.08, 2.43) standardized to a 20-ppb increase in NO<sub>2</sub>. The incidence of cough increased up to approximately mean NO<sub>2</sub> levels (~13 ppb) (p = 0.01), after which no further increase was observed. The significant association between cough and 4-day mean NO<sub>2</sub> level remained unchanged in models that included O<sub>3</sub> but lost statistical significance in two-pollutant models that included PM<sub>10</sub> (OR = 1.37 [95% CI: 0.88, 2.13]) or SO<sub>2</sub> (OR = 1.42 [95% CI: 0.90, 2.28]).

Mortimer et al. (2002) studied the risk of asthma symptoms among 864 asthmatic children in New York City, NY, Baltimore, MD, Washington, DC, Cleveland, OH, Detroit, MI, St Louis, MO, and Chicago, IL. Subjects were followed daily for four 2-week periods over the course of nine months with morning and evening asthma symptoms and peak flow recorded. The greatest effect was observed for morning symptoms using a 6-day moving average, with a reported OR of 1.48 (95% CI: 1.02, 2.16) per 20 ppb increase in NO<sub>2</sub>. Although the magnitudes of effect estimates were generally robust in multi-pollutant models that included O<sub>3</sub> (OR for 20-ppb increase in NO<sub>2</sub> = 1.40 [95% CI: 0.93, 2.09]), O<sub>3</sub> and SO<sub>2</sub> (OR for NO<sub>2</sub> = 1.31 [95% CI:

0.87, 2.09]), or O<sub>3</sub>, SO<sub>2</sub>, and PM<sub>10</sub> (OR for NO<sub>2</sub> = 1.45 [95% CI: 0.63, 3.34]), they were not statistically-significant.

Schildcrout et al. (2006) investigated the association between ambient NO<sub>2</sub> and respiratory symptoms and rescue inhaler use as part of the Childhood Asthma Management Program (CAMP) study. The study reported on 990 asthmatic children living within 50 miles of an NO<sub>2</sub> monitor in Boston, MA, Baltimore, MD, Toronto, ON, St. Louis, MO, Denver, CO, Albuquerque, NM, or San Diego, CA. Symptoms and use of rescue medication were recorded daily, resulting in each subject having an average of approximately two months of data. The authors reported the strongest association between NO<sub>2</sub> and 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 increase in NO<sub>2</sub> occurring 2 days before measurement. Multi-pollutant models that included CO, PM<sub>10</sub>, or SO<sub>2</sub> produced similar results (ISA, Figure 3.1-5, panel A). Additionally, increased NO<sub>2</sub> exposure was associated with increased use of rescue medication, with the strongest association for a 2-day lag, both for single- and multi-pollutant models (e.g., for an increase of 20-ppb NO<sub>2</sub> in the single-pollutant model, the RR for increased inhaler usage was 1.05 (95% CI: 1.01, 1.09).

### ***Studies of Indoor NO<sub>2</sub>***

Evidence supporting increased respiratory morbidity following NO<sub>2</sub> exposures is also found in studies of indoor NO<sub>2</sub> (ISA, section 3.1.4.1). For example, in a randomized intervention study in Australia (Pilotto et al., 2004), students attending schools that switched out unvented gas heaters, a major source of indoor NO<sub>2</sub>, experienced a decrease in both levels of NO<sub>2</sub> and in respiratory symptoms (e.g., difficulty breathing, chest tightness, and asthma attacks) compared to students in schools that did not switch out unvented gas heaters (ISA, section 3.1.4.1). An earlier indoor study by Pilotto and colleagues (1997) also found that students in classrooms with higher levels of NO<sub>2</sub> had higher rates of respiratory symptoms (e.g., sore throat, cold) and absenteeism than students in classrooms with lower levels of NO<sub>2</sub>. This study detected a significant concentration-response relationship, strengthening the argument that NO<sub>2</sub> is causally related to respiratory morbidity. A number of other indoor studies conducted in homes have also detected significant associations between indoor NO<sub>2</sub> and respiratory symptoms (ISA, section 3.1.4.1).

#### **4.2.4 Lung Host Defenses and Immunity**

Impaired host-defense systems and increased risk of susceptibility to both viral and bacterial infections after NO<sub>2</sub> exposures have been observed in epidemiologic, controlled human exposure, and animal toxicological studies (ISA, section 3.1.1 and 5.3.2.1). A recent epidemiologic study (Chauhan et al., 2003) provides evidence that increased personal exposure to NO<sub>2</sub> worsened virus-associated symptoms and decreased lung function in children with asthma. The limited evidence from controlled human exposure studies indicates that NO<sub>2</sub> may increase susceptibility to injury by subsequent viral challenge at exposures of as low as 0.6 ppm for 3 hours in healthy adults (Frampton et al., 2002). Toxicological studies have shown that lung host defenses, including mucociliary clearance and immune cell function, are sensitive to NO<sub>2</sub> exposure, with effects observed at concentrations of less than 1 ppm (ISA, section 3.1.7). When taken together, epidemiologic and experimental studies linking NO<sub>2</sub> exposure with viral illnesses provide coherent and consistent evidence that NO<sub>2</sub> exposure can result in lung host defense or immune system effects (ISA, sections 3.1.7 and 5.3.2.1). This group of outcomes also provides some plausibility for other respiratory system effects. For example, effects on ciliary action (clearance) or immune cell function (i.e. macrophage phagocytosis) could be the basis for the effects observed in epidemiologic studies, including increased respiratory illness or respiratory symptoms (ISA, section 5.3.2.1). Proposed mechanisms by which NO<sub>2</sub>, in conjunction with viral infections, may exacerbate airway symptoms are summarized in table 4-4 below (ISA, table 3.1-1).

**Table 4-4. Proposed Mechanisms Whereby NO<sub>2</sub> and Respiratory Virus Infections May Exacerbate Upper and Lower Airway Symptoms**

	PROPOSED MECHANISMS
<b>Upper Airway</b>	
Epithelium	↓ Ciliary beat frequency
	↑ Epithelial permeability
<b>Lower Airway</b>	
Epithelium	(as in upper airway)
Cytokines	↓ Epithelial-derived IL-8, GM-CSF, TNF-α
	↑ Macrophage-derived IL-1b, IL-6, IL-8, TNF-α
Inflammatory cells	↑ Mast cell tryptase
	↑ Neutrophils
	↑ Total lymphocytes
	↑ NK lymphocytes
	↓ T-helper/T-cytotoxic cell ratio
Inflammatory mediators	↑ Free radicals, proteases, TXA <sub>2</sub> , TXB <sub>2</sub> , LTB <sub>4</sub>
Allergens	↑ Penetrance due to ciliostasis
	↓ PD20-FEV <sub>1</sub>
	↑ Antigen-specific IgE
	↑ Epithelial permeability
<b>Peripheral Blood</b>	
	↓ B and NK lymphocytes
	↓ Total lymphocytes

#### 4.2.5 Airway Response

In acute exacerbations of asthma, bronchial smooth muscle contraction occurs quickly to narrow the airway in response to exposure to various stimuli including allergens or irritants. Bronchoconstriction is the dominant physiological event leading to clinical symptoms and interference with airflow (National Heart, Lung, and Blood Institute, 2007). Inhaled pollutants such as NO<sub>2</sub> may enhance the inherent responsiveness of the airway to a challenge by allergens and nonspecific agents (ISA, section 3.1.3). In the laboratory, airway responses can be measured by assessing changes in pulmonary function (e.g., decline in FEV<sub>1</sub>) or changes in the inflammatory response (e.g., using markers in bronchoalveolar lavage (BAL) fluid or induced sputum) (ISA, section 3.1.3).

The ISA (section 5.3.2.1) draws two broad conclusions regarding the airway response following NO<sub>2</sub> exposure. First, the ISA concludes that NO<sub>2</sub> exposure may enhance the

sensitivity to allergen-induced decrements in lung function and increase the allergen-induced airway inflammatory response at exposures as low as 0.26 ppm NO<sub>2</sub> for 30 minutes (ISA, section 5.3.2.1 and Figure 3.1-2). Second, exposure to NO<sub>2</sub> has been found to enhance the inherent responsiveness of the airway to subsequent nonspecific challenges in controlled human exposure studies (section 3.1.4.2). In general, small but significant increases in nonspecific airway responsiveness were observed in the range of 0.2 to 0.3 ppm NO<sub>2</sub> for 30-minute exposures and at 0.1 ppm NO<sub>2</sub> for 60-minute exposures in asthmatics. These conclusions are consistent with results from animal toxicological studies which have detected 1) increased immune-mediated pulmonary inflammation in rats exposed to house dust mite allergen following exposure to 5 ppm NO<sub>2</sub> for 3-h and 2) increased responsiveness to non-specific challenges following sub-chronic (6-12 weeks) exposure to 1 to 4 ppm NO<sub>2</sub> (ISA, section 5.3.2.1). Enhanced airway responsiveness could have important clinical implications for asthmatics since transient increases in airway responsiveness following NO<sub>2</sub> exposure have the potential to increase symptoms and worsen asthma control (ISA, section 5.4). In addition, the ISA cites the controlled human exposure literature on the NO<sub>2</sub> airway response as being supportive of the epidemiologic evidence on respiratory morbidity (ISA, section 5.4). Because studies on airway responsiveness have been used to identify potential health effect benchmark values and to inform the identification of potential alternative standards for evaluation (see sections 4.5 and 5 of this document), more detail is provided below on the specific studies that form the basis for the conclusions in the ISA regarding this endpoint.

Folinsbee (1992) conducted a meta-analysis using individual level data from 19 clinical NO<sub>2</sub> exposure studies measuring airway responsiveness in asthmatics (ISA, section 3.1.3.2). These studies included NO<sub>2</sub> exposure levels between 0.1 ppm and 1.0 ppm and most of them used nonspecific bronchoconstricting agents such as methacholine, carbachol, histamine, or cold air. The largest effects were observed for subjects at rest. Among subjects exposed at rest, 76% experienced increased airway responsiveness following exposure to NO<sub>2</sub> levels between 0.2 and 0.3 ppm. Results from an update of this meta-analysis (results combined only from nonspecific responsiveness studies) are presented in the ISA (Table 3.1-3) and in Table 4-5 below.

**Table 4-5. Fraction of nitrogen dioxide-exposed asthmatics with increased nonspecific airway hyperresponsiveness<sup>3</sup>**

NO <sub>2</sub> ppm	ALL EXPOSURES	EXPOSURE WITH EXERCISE	EXPOSURE AT REST
0.1	0.66 (50) <sup>B</sup>	—	0.66 (50) <sup>B</sup>
0.1 - 0.15	0.66 (87) <sup>C</sup>	0.59 (17)	0.67 (70) <sup>C</sup>
0.2 - 0.3	0.58 (187) <sup>B</sup>	0.52 (136)	0.75 (51) <sup>C</sup>
>0.3	0.59 (81)	0.49 (48)	0.73 (33) <sup>B</sup>
0.1 - 0.6	0.60 (355) <sup>C</sup>	0.52 (201)	0.71 (154) <sup>C</sup>

As noted in Table 4-5, when exposed at rest 66% of subjects experienced an increase in airway responsiveness following exposure to 0.1 ppm NO<sub>2</sub>, 67% of subjects experienced an increase in airway responsiveness following exposure to NO<sub>2</sub> concentrations between 0.1 and 0.15 ppm (inclusively), 75% of subjects experienced an increase in airway responsiveness following exposure to NO<sub>2</sub> concentrations between 0.2 and 0.3 ppm (inclusively), and 73% of subjects experienced an increase in airway responsiveness following exposure to NO<sub>2</sub> concentrations above 0.3 ppm. Effects of NO<sub>2</sub> exposure on the direction of airway responsiveness are statistically-significant at all of these levels. Because this meta-analysis evaluates only the direction of the change in airway responsiveness, it is not possible to discern the magnitude of the change from these data. However, the results do suggest that short-term exposures to NO<sub>2</sub> at near-ambient levels (<0.3 ppm) can alter airway responsiveness in people with mild asthma (ISA, section 3.1.3.2).

Several studies published since the 1996 review address the question of whether low-level exposures to NO<sub>2</sub> enhance the response to specific allergen challenge in mild asthmatics (ISA, section 3.1.3.1). These recent studies suggest that NO<sub>2</sub> may enhance the sensitivity to allergen-induced decrements in lung function and increase the allergen-induced airway inflammatory response. Strand et al. (1997) demonstrated that single 30-minute exposures to 0.26-ppm NO<sub>2</sub> increased the late phase response to allergen challenge 4 hours after exposure, as measured by changes in lung function. In a separate study (Strand et al., 1998), 4 daily repeated exposures to 0.26-ppm NO<sub>2</sub> for 30 minutes increased both the early and late-phase responses to allergen, as measured by changes in lung function. Barck et al. (2002) used the same exposure and challenge protocol in the earlier Strand study (0.26 ppm for 30 min, with allergen challenge

<sup>3</sup> Values are the fraction of asthmatics (out of the total number of individuals in parenthesis) having an increase in airway responsiveness following NO<sub>2</sub> versus air exposure. See table 3.1-3 in the ISA for more detail. <sup>B</sup> indicates  $p \leq 0.05$  and <sup>C</sup> indicates  $p \leq 0.01$ .

4 hours after exposure), and performed BAL 19 hours after the allergen challenge to determine NO<sub>2</sub> effects on the allergen-induced inflammatory response. Compared with air followed by allergen, NO<sub>2</sub> followed by allergen caused an increase in the BAL recovery of polymorphonuclear (PMN) cells and eosinophil cationic protein (ECP) as well as a reduction in total BAL fluid volume and cell viability. ECP is released by degranulating eosinophils, is toxic to respiratory epithelial cells, and is thought to play a role in the pathogenesis of airway injury in asthma. Subsequently, Barck et al. (2005) exposed 18 mild asthmatics to air or 0.26 ppm NO<sub>2</sub> for 15 minutes on day 1, followed by two 15 minute exposures separated by 1 hour on day 2, with allergen challenge after exposures on both days 1 and 2. Sputum was induced before exposure on day 1 and after exposures (morning of day 3). Compared to air plus allergen, NO<sub>2</sub> plus allergen resulted in increased levels of ECP in both sputum and blood and increased myeloperoxidase levels in blood. All exposures in these studies (Barck et al., 2002, 2005; Strand et al., 1997, 1998) used subjects at rest. They used an adequate number of subjects, included air control exposures, randomized exposure order, and separated exposures by at least 2 weeks. Together, they indicate the possibility for effects on allergen responsiveness in some asthmatics following brief exposures to 0.26 ppm NO<sub>2</sub>. However, other recent studies have failed to find effects using similar, but not identical, approaches (ISA, section 3.1.3.1). The differing findings may relate in part to differences in timing of the allergen challenge, the use of multiple versus single-dose allergen challenge, the use of BAL versus sputum induction, exercise versus rest during exposure, and differences in subject susceptibility (ISA, section 3.1.3.1).

#### **4.2.6 Airway Inflammation**

Effects of NO<sub>2</sub> on airway inflammation have been observed in controlled human exposure and animal toxicological studies at higher than ambient levels (0.4-5 ppm). The few available epidemiologic studies were suggestive of an association between ambient NO<sub>2</sub> concentrations and inflammatory response in the airway in children, though the associations were inconsistent in the adult populations examined (ISA, section 3.1.2 and 5.3.2.1). Controlled human exposure studies provide evidence for increased airway inflammation at NO<sub>2</sub> concentrations of <2.0 ppm. The onset of inflammatory responses in healthy subjects appears to be between 100 and 200 ppm-minutes, i.e., 1 ppm for 2 to 3 hours (ISA, Figure 3.1-1). Increases in biological markers of inflammation were not observed consistently in healthy animals at levels

of less than 5 ppm; however, increased susceptibility to NO<sub>2</sub> concentrations of as low as 0.4 ppm was observed when lung vitamin C was reduced (by diet) to levels that were <50% of normal. These data provide some evidence for biological plausibility and one potential mechanism for other respiratory effects, such as exacerbation of asthma symptoms and increased ED visits for asthma (ISA, section 5.3.2.1).

#### **4.2.7 Lung Function**

Recent epidemiologic studies that examined the association between ambient NO<sub>2</sub> concentrations and lung function in children and adults generally produced inconsistent results (ISA, sections 3.1.5.1 and 5.3.2.1). Controlled human exposure studies generally did not find direct effects of NO<sub>2</sub> on lung function in healthy adults at levels as high as 4.0 ppm (ISA, section 5.3.2.1). For asthmatics, the direct effects of NO<sub>2</sub> on lung function also have been inconsistent at exposure concentrations of less than 1 ppm NO<sub>2</sub>.

#### **4.2.8 Conclusions and Coherence of Evidence for Short-Term Respiratory Effects**

As noted previously, the ISA concludes that the findings of epidemiologic, controlled human exposure, and animal toxicological studies provide evidence that is sufficient to infer a likely causal relationship for respiratory effects following short-term NO<sub>2</sub> exposure (ISA, sections 3.1.7 and 5.3.2.1). The ISA (section 5.4) concludes that the strongest evidence for an association between NO<sub>2</sub> exposure and adverse human health effects comes from epidemiologic studies of respiratory symptoms, ED visits, and hospital admissions. These studies include panel and field studies, studies that control for the effects of co-occurring pollutants, and studies conducted in areas where the whole distribution of ambient 24-h average NO<sub>2</sub> concentrations was below the current NAAQS level of 0.053 ppm (53 ppb) (annual average). The effect estimates from the U.S. and Canadian studies generally indicate a 2-20% (see footnote 2 above) increase in risks for ED visits and hospital admissions. Risks associated with respiratory symptoms are generally higher (ISA, section 5.4).

Overall, the epidemiologic evidence for respiratory effects can be characterized as consistent, in that associations are reported in studies conducted in numerous locations with a variety of methodological approaches. Considering this large body of epidemiologic studies alone, the findings are also coherent in the sense that the studies report associations with respiratory health outcomes that are logically linked together. In addition, a number of these

associations are statistically-significant, particularly the more precise effect estimates (ISA, section 5.3.2.1). These epidemiologic studies are supported by evidence from toxicological and controlled human exposure studies, particularly by controlled human exposure studies that evaluate airway hyperresponsiveness in asthmatic individuals (ISA, section 5.4). Together, the epidemiologic and experimental data sets form a plausible, consistent, and coherent description of a relationship between NO<sub>2</sub> exposures and an array of adverse respiratory health effects that range from the onset of respiratory symptoms to hospital admission.

However, as noted in the ISA (section 5.4), it is difficult to determine “the extent to which NO<sub>2</sub> is independently associated with respiratory effects or if NO<sub>2</sub> is a marker for the effects of another traffic-related pollutant or mix of pollutants.” On-road vehicle exhaust emissions are a nearly ubiquitous source of combustion pollutant mixtures that include NO<sub>x</sub> and can be an important contributor to NO<sub>2</sub> levels in near-road locations. Although this complicates the efforts to quantify specific NO<sub>2</sub>-related health effects, the evidence summarized in the ISA indicates that NO<sub>2</sub> associations generally remain robust in multi-pollutant models and supports a direct effect of short-term NO<sub>2</sub> exposure on respiratory morbidity at ambient concentrations below the current NAAQS level. The robustness of epidemiologic findings to adjustment for co-pollutants, coupled with data from animal and human experimental studies, support the determination that the relationship between NO<sub>2</sub> and respiratory morbidity is likely causal, while still recognizing the relationship between NO<sub>2</sub> and other traffic related pollutants and the potential for confounding.

### **4.3 OTHER ADVERSE EFFECTS FOLLOWING SHORT-TERM EXPOSURES**

The ISA concludes that the epidemiologic evidence is suggestive but not sufficient to infer a causal relationship between short-term exposure to NO<sub>2</sub> and all-cause and cardiopulmonary-related mortality (ISA, section 5.3.2.3). Results from several large U.S. and European multi-city studies and a meta-analysis study indicate positive associations between ambient NO<sub>2</sub> concentrations and the risk of all-cause (nonaccidental) mortality, with effect estimates ranging from 0.5 to 3.6% excess risk in mortality per standardized increment (20 ppb for 24-h averaging time, 30 ppb for 1-h averaging time) (ISA, section 3.3.1, Figure 3.3-2, section 5.3.2.3). In general, the NO<sub>2</sub> effect estimates were robust to adjustment for co-pollutants. Both

cardiovascular and respiratory mortality have been associated with increased NO<sub>2</sub> concentrations in epidemiologic studies (ISA, Figure 3.3-3); however, similar associations were observed for other pollutants, including PM and SO<sub>2</sub>. The range of risk estimates for excess mortality is generally smaller than that for other pollutants such as PM. In addition, while NO<sub>2</sub> exposure, alone or in conjunction with other pollutants, may contribute to increased mortality, evaluation of the specificity of this effect is difficult. Clinical studies showing hematologic effects and animal toxicological studies showing biochemical, lung host defense, permeability, and inflammation changes with short-term exposures to NO<sub>2</sub> provide limited evidence of plausible pathways by which risks of mortality may be increased, but no coherent picture is evident at this time (ISA, section 5.3.2.3).

The ISA concludes that the available evidence on cardiovascular health effects following short-term exposure to NO<sub>2</sub> is inadequate to infer the presence or absence of a causal relationship at this time (ISA, section 5.3.2.2). Evidence from epidemiologic studies of heart rate variability, repolarization changes, and cardiac rhythm disorders among heart patients with ischemic cardiac disease are inconsistent (ISA, section 5.3.2.2). In most studies, associations with PM were found to be similar or stronger than associations with NO<sub>2</sub>. Generally positive associations between ambient NO<sub>2</sub> concentrations and hospital admissions or ED visits for cardiovascular disease have been reported in single-pollutant models (ISA, section 5.3.2.2); however, most of these effect estimate values were diminished in multi-pollutant models that also contained CO and PM indices (ISA, section 5.3.2.2). Mechanistic evidence of a role for NO<sub>2</sub> in the development of cardiovascular diseases from studies of biomarkers of inflammation, cell adhesion, coagulation, and thrombosis is lacking (ISA, section 5.3.2.2). Furthermore, the effects of NO<sub>2</sub> on various hematological parameters in animals are inconsistent and, thus, provide little biological plausibility for effects of NO<sub>2</sub> on the cardiovascular system (ISA, section 5.3.2.2).

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

### **4.4.1 Respiratory Morbidity**

The ISA concludes that overall, the epidemiologic and experimental evidence is suggestive but not sufficient to infer a causal relationship between long-term NO<sub>2</sub> exposure and respiratory morbidity (ISA, section 5.3.2.4). The available database evaluating the relationship between respiratory illness in children and long-term exposures to NO<sub>2</sub> has increased since the

1996 review of the NO<sub>2</sub> NAAQS. A number of epidemiologic studies have examined the effects of long-term exposure to NO<sub>2</sub> and reported positive associations with decrements in lung function and partially irreversible decrements in lung function growth (ISA, section 3.4.1, figures 3.4-1 and 3.4-2). Specifically, results from the California-based Children's Health Study, which evaluated NO<sub>2</sub> exposures in children over an 8-year period, demonstrated deficits in lung function growth (Gauderman et al., 2004). This effect has also been observed in Mexico City, Mexico (Rojas-Martinez et al., 2007a,b) and in Oslo, Norway (Ofstedal et al., 2008), with decrements ranging from 1 to 17.5 ml per 20- ppb increase in annual NO<sub>2</sub> concentration. Similar associations have been found for PM, O<sub>3</sub>, and proximity to traffic (<500 m), though these studies did not report the results of co-pollutant models. The high correlation among traffic-related pollutants makes it difficult to accurately estimate independent effects in these long-term exposure studies (ISA, section 5.3.2.4). With regard to asthma incidence and long-term NO<sub>2</sub>, two major cohort studies, the Children's Health Study (Gauderman et al., 2005) and a birth cohort study in the Netherlands (Brauer et al., 2007), observed significant associations. However, several other studies failed to find consistent associations between long-term NO<sub>2</sub> exposure and asthma outcomes (ISA, section 5.3.2.4). Similarly, epidemiologic studies conducted in the United States and Europe have produced inconsistent results regarding an association between long-term exposure to NO<sub>2</sub> and respiratory symptoms (ISA, sections 3.4.3 and 5.3.2.4). While some positive associations were noted, a large number of symptom outcomes were examined and the results across specific outcomes were inconsistent (ISA, section 5.3.2.4).

Animal toxicological studies may provide biological plausibility for the chronic effects of NO<sub>2</sub> that have been observed in epidemiologic studies (ISA, sections 3.4.5 and 5.3.2.4). The main biochemical targets of NO<sub>2</sub> exposure appear to be antioxidants, membrane polyunsaturated fatty acids, and thiol groups. NO<sub>2</sub> effects include changes in oxidant/antioxidant homeostasis and chemical alterations of lipids and proteins. Lipid peroxidation has been observed at NO<sub>2</sub> exposures as low as 0.04 ppm for 9 months and at exposures of 1.2 ppm for 1 week, suggesting lower effect thresholds with longer durations of exposure. Other studies showed decreases in formation of key arachidonic acid metabolites in AMs following NO<sub>2</sub> exposures of 0.5 ppm. NO<sub>2</sub> has been shown to increase collagen synthesis rates at concentrations as low as 0.5 ppm. This could indicate increased total lung collagen, which is associated with pulmonary fibrosis, or

increased collagen turnover, which is associated with remodeling of lung connective tissue. Morphological effects following chronic NO<sub>2</sub> exposures have been identified in animal studies that link to these increases in collagen synthesis and may provide plausibility for the deficits in lung function growth described in epidemiologic studies (ISA, section 3.4.5).

#### **4.4.2 Mortality**

The ISA concludes that the epidemiologic evidence is inadequate to infer the presence or absence of a causal relationship between long-term exposure to NO<sub>2</sub> and mortality (ISA, section 5.3.2.6). In the United States and European cohort studies examining the relationship between long-term exposure to NO<sub>2</sub> and mortality, results have been inconsistent (ISA, section 5.3.2.6). Further, when associations were suggested, they were not specific to NO<sub>2</sub> but also implicated PM and other traffic indicators. The relatively high correlations reported between NO<sub>2</sub> and PM indices make it difficult to interpret these observed associations at this time (ISA, section 5.3.2.6).

#### **4.4.3 Other Long-Term Effects**

The ISA concludes that the available epidemiologic and toxicological evidence is inadequate to infer the presence or absence of a causal relationship for carcinogenic, cardiovascular, and reproductive and developmental effects related to long-term NO<sub>2</sub> exposure (ISA, section 5.3.2.5). Epidemiologic studies conducted in Europe have shown an association between long-term NO<sub>2</sub> exposure and increased incidence of cancer (ISA, section 5.3.2.5). However, the animal toxicological studies have provided no clear evidence that NO<sub>2</sub> acts as a carcinogen (ISA, section 5.3.2.5). The very limited epidemiologic and toxicological evidence does not suggest that long-term exposure to NO<sub>2</sub> has cardiovascular effects (ISA, section 5.3.2.5). The epidemiologic evidence is not consistent for associations between NO<sub>2</sub> exposure and fetal growth retardation; however, some evidence is accumulating for effects on preterm delivery (ISA, section 5.3.2.5). Scant animal evidence supports a weak association between NO<sub>2</sub> exposure and adverse birth outcomes and provides little mechanistic information or biological plausibility for the epidemiologic findings.

## **4.5 RELEVANCE OF SPECIFIC HEALTH EFFECTS TO THE NO<sub>2</sub> RISK CHARACTERIZATION**

### **4.5.1 Overview**

As described previously, the ISA characterizes judgments about causality with a hierarchy (for discussion see ISA, section 1.3) 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

In order to be judged sufficient to infer a causal relationship, an association must have been observed between the pollutant and the outcome in studies where chance, bias, and confounding can be ruled out with reasonable confidence. Human clinical studies provide the strongest evidence for causality while other lines of evidence (e.g., epidemiologic studies) provide support for this determination. An inference of a causal relationship is generally based on multiple studies from more than one research group. In order to be judged sufficient to infer a likely causal relationship, an association must have been observed between the pollutant and the outcome in studies where chance, bias, and confounding are minimized even though uncertainties remain. These uncertainties could be due to the difficulty associated with addressing chance, bias, and confounding and/or due to the fact that other lines of evidence are limited or inconsistent. An inference of a likely causal relationship is generally based on multiple studies from more than one research group. In order to be judged suggestive, but not sufficient to infer a causal relationship, existing evidence must suggest an association between the pollutant and the outcome but that evidence is weakened because chance, bias, and confounding cannot be ruled out (see table 4-1). For example, this determination might apply if at least one high-quality study shows an association, but the results of other studies are inconsistent (ISA, Table 1.3-2). For purposes of the quantitative characterization of NO<sub>2</sub> health risks, staff has judged it appropriate to focus on endpoints for which the ISA concludes that the available evidence is sufficient to infer either a causal or a likely causal relationship. This is consistent with judgments that have been made in other recent NAAQS reviews (e.g., see EPA,

2005) and it will help ensure that our risk characterization is based on endpoints for which a causal relationship with NO<sub>2</sub> is judged to be more likely than not (see table 4-1 above and ISA, table 1.3-2).

The only endpoint evidence for which the evidence is judged to be sufficient to infer either a causal or a likely causal relationship is respiratory morbidity following short-term NO<sub>2</sub> exposure. Therefore, for purposes of characterizing health risks associated with NO<sub>2</sub>, we have focused on respiratory morbidity endpoints that have been associated with short-term NO<sub>2</sub> exposures. Other endpoints (e.g., long-term effects) will be considered as part of the evidence-based evaluation of potential alternative standards during the rulemaking stage of the NAAQS review. In evaluating the appropriateness of specific endpoints for use in the NO<sub>2</sub> risk characterization, we have considered both epidemiologic and controlled human exposure studies.

#### **4.5.2 Epidemiology**

The ISA characterizes the epidemiologic evidence for respiratory effects as consistent, in that associations are reported in studies conducted in numerous locations and with a variety of methodological approaches (ISA, section 5.3.2.1). The findings are also coherent in the sense that the studies report associations with respiratory health outcomes that are logically linked together (ISA, section 5.3.2.1). When the epidemiologic literature is considered as a whole, there are generally positive associations between NO<sub>2</sub> and respiratory symptoms, hospitalization, and ED visits. A number of these associations are statistically significant, particularly the more precise effect estimates (ISA, section 5.3.2.1). However, the ISA (section 5.4) offers the following caveat to consider when interpreting the epidemiologic results: “It is difficult to determine from these new studies the extent to which NO<sub>2</sub> is independently associated with respiratory effects or if NO<sub>2</sub> is a marker for the effects of another traffic-related pollutant or mix of pollutants (see Section 5.2.2 for more details on exposure issues). A factor contributing to uncertainty in estimating the NO<sub>2</sub>-related effect from epidemiologic studies is that NO<sub>2</sub> is a component of a complex air pollution mixture from traffic related sources that include CO and various forms of PM.” These caveats should be considered when interpreting a quantitative NO<sub>2</sub> risk estimate based on the epidemiology literature. Despite these uncertainties, the ISA (section 5.4) concludes that, “Although this complicates the efforts to disentangle specific NO<sub>2</sub>-related health effects, the evidence summarized in this assessment indicates that NO<sub>2</sub> associations

generally remain robust in multi-pollutant models and supports a direct effect of short-term NO<sub>2</sub> exposure on respiratory morbidity at ambient concentrations below the current NAAQS. The robustness of epidemiologic findings to adjustment for copollutants, coupled with data from animal and human experimental studies, support a determination that the relationship between NO<sub>2</sub> and respiratory morbidity is likely causal, while still recognizing the relationship between NO<sub>2</sub> and other traffic related pollutants.”

When evaluating epidemiologic studies as to their appropriateness for use as the basis for a quantitative risk assessment, staff has considered several factors. First, we have judged that studies conducted in the United States are preferable to those conducted outside the United States given the potential for effect estimates to be impacted by factors such as the ambient pollutant mix, the placement of monitors, activity patterns of the population, and characteristics of the healthcare system. Second, we judged that studies of ambient NO<sub>2</sub> are preferable to those of indoor NO<sub>2</sub>. This does not suggest that indoor studies are uninformative in the review of an ambient standard. In fact, indoor studies provide a large part of the evidence base used in the ISA to reach conclusions regarding causality. However, studies of indoor NO<sub>2</sub> focus on individuals exposed to NO<sub>2</sub> from indoor sources. These indoor sources can result in exposure patterns, NO<sub>2</sub> levels, and co-pollutants that are different from those typically associated with ambient NO<sub>2</sub>. Because the purpose of a quantitative risk assessment based on the epidemiological literature would be to inform decisions regarding an ambient NO<sub>2</sub> standard, the preferred approach would be to consider studies of ambient NO<sub>2</sub>. Third, we judged it appropriate to focus on studies of ED visits and hospital admissions. When compared to studies of respiratory symptoms, the public health significance of ED visits and hospital admissions are less ambiguous (e.g., because of the potential disconnect between health outcomes and subjective symptom ratings). In addition, baseline incidence data are more readily available for these endpoints. Finally, we judged it appropriate to focus on studies that evaluated NO<sub>2</sub> health effect associations using both single- and multi-pollutant models. Taking these factors into consideration, we have chosen to focus on the studies by Peel and colleagues (2005) and by Tolbert and colleagues (2007) in Atlanta, Georgia. The epidemiology-based risk assessment is described in more detail in subsequent sections of this document.

### 4.5.3 Controlled Human Exposure Studies

Controlled human exposure studies have addressed the consequences of short-term (e.g., 30-minutes to several hours) NO<sub>2</sub> exposures for a number of health endpoints including airway responsiveness, host defense and immunity, inflammation, and lung function (ISA, section 3.1). In identifying health endpoints from controlled human exposure studies on which to focus the characterization of NO<sub>2</sub> health risks, staff judges it appropriate to focus on endpoints that occur at or near ambient levels of NO<sub>2</sub> and endpoints that are of clinical significance. With regard to the NO<sub>2</sub> levels at which different effects have been documented, the ISA concludes that 1) in asthmatics NO<sub>2</sub> may increase the allergen-induced airway inflammatory response at exposures as low as 0.26-ppm for 30 min (ISA, Figure 3.1-2) and NO<sub>2</sub> exposures between 0.2 and 0.3 ppm for 30 minutes or 0.1 ppm for 60-minutes can result in small but significant increases in nonspecific airway responsiveness (ISA, section 5.3.2.1); 2) limited evidence indicates that NO<sub>2</sub> may increase susceptibility to injury by subsequent viral challenge following exposures of 0.6-1.5 ppm for 3 hours; 3) evidence exists for increased airway inflammation at NO<sub>2</sub> concentrations less than 2.0 ppm; and 4) the direct effects of NO<sub>2</sub> on lung function in asthmatics have been inconsistent at exposure concentrations below 1 ppm (ISA, section 5.3.2.1). The ISA notes that epidemiologic studies have reported health effects associations in areas reporting maximum ambient concentrations from 100 to 300 ppb (ISA, Tables 5.3-2 and 5.3-3). Therefore, of the health effects caused by NO<sub>2</sub> in controlled human exposure studies, the only effect identified by the ISA to occur at or near ambient levels is increased airway responsiveness in asthmatics.

Staff judges that airway responsiveness in the asthmatic population is an appropriate focus for the risk characterization for several reasons. First, the ISA concludes that “persons with preexisting pulmonary conditions are likely at greater risk from ambient NO<sub>2</sub> exposures than the general public, with the most extensive evidence available for asthmatics as a potentially susceptible group” (ISA, section 5.3.2.8). Second, when discussing the clinical significance of NO<sub>2</sub>-related airway hyperresponsiveness in asthmatics, the ISA concludes that “transient increases in airway responsiveness following NO<sub>2</sub> exposure have the potential to increase symptoms and worsen asthma control” (ISA, sections 3.1.3 and 5.4). That this effect could have public health implications is suggested by the large size of the asthmatic population in the United States (see above and ISA, Table 4.4-1). Third, NO<sub>2</sub> effects on airway responsiveness in asthmatics are part of the body of experimental evidence that provides plausibility and coherence

for the effects observed on hospital admissions and ED visits in epidemiologic studies (ISA, section 5.3.2.1). As a result of these considerations, although studies on other endpoints evaluated in controlled human exposure studies provide qualitative support for the ability of NO<sub>2</sub> to cause adverse effects on respiratory health, the focus for purpose of quantifying risks associated with ambient NO<sub>2</sub> is airway responsiveness in asthmatics (see below).

Because many of the studies of airway responsiveness evaluate only a single level of NO<sub>2</sub> and because of methodological differences between the studies, staff has judged that the data are not sufficient to derive an exposure-response relationship in the range of interest. Therefore, the most appropriate approach to characterizing risks based on the controlled human exposure evidence for airway responsiveness is to compare estimated NO<sub>2</sub> air quality and exposure levels with potential health effect benchmark levels. Estimates of hourly peak air quality concentrations and personal exposures to ambient NO<sub>2</sub> concentrations at and above specified potential health effect benchmark levels provide some perspective on the potential health impacts of NO<sub>2</sub> exposure. Staff recognizes that there is high inter-individual variability in NO<sub>2</sub>-induced effects on airway responsiveness such that only a subset of asthmatic individuals exposed at and above a given benchmark level may actually be expected to experience an adverse effect. Potential health benchmark levels and the approach to using these benchmarks to characterize health risks are described in more detail in chapter 6.

## **5. IDENTIFICATION OF POTENTIAL ALTERNATIVE STANDARDS FOR ANALYSIS**

### **5.1 INTRODUCTION**

The primary goals of the NO<sub>2</sub> risk and exposure assessment described in this document are to estimate short-term exposures and potential human health risks associated with 1) recent levels of ambient NO<sub>2</sub>; 2) NO<sub>2</sub> levels associated with just meeting the current standard; and 3) NO<sub>2</sub> levels associated with just meeting potential alternative standards. This section identifies potential alternative standards in terms of indicator, averaging time, form, and level and provides the rationale that was used to select them.

### **5.2 INDICATOR**

The NO<sub>x</sub>, for purposes of this document, include multiple gaseous (e.g., NO<sub>2</sub>, NO) and particulate (e.g., nitrate) species. In considering the appropriateness of different indicators, we note that the health effects associated with particulate species of NO<sub>x</sub> have been considered within the context of the health effects of ambient particles in the Agency's review of the NAAQS for PM. Thus, as discussed in the integrated review plan (2007a), the current review of the NO<sub>2</sub> NAAQS is focused on the gaseous species of NO<sub>x</sub> and will not consider health effects directly associated with particulate species of NO<sub>x</sub>. Of the gaseous species, EPA has historically determined it appropriate to specify the indicator of the standard in terms of NO<sub>2</sub> because the majority of the information regarding health effects and exposures is for NO<sub>2</sub>. The final ISA has found that this continues to be the case and, therefore, staff believes that NO<sub>2</sub> remains the most appropriate indicator.

### **5.3 AVERAGING TIME**

The current annual standard for NO<sub>2</sub> was originally set in 1971 based on epidemiologic studies that supported a link between adverse respiratory effects and long-term exposure to low-levels of NO<sub>2</sub>. Although the quantitative basis for the annual averaging time was later called into question (60 FR 52876), the annual standard was retained in the most recent review (60 FR 52876) for two key reasons. First, the evidence showing the most serious health effects associated with long-term exposures (e.g., emphysematous-like alterations in the lung and

increased susceptibility to infection) came from animal studies conducted at concentrations well above those permitted in the ambient air by the annual standard. Second, an air quality assessment conducted by EPA concluded that areas that meet the annual standard would be unlikely to experience short-term peaks above levels that had been shown in controlled human exposure studies to impact endpoints of potential concern (i.e., airway responsiveness).

The issue of averaging time will be reconsidered in the current review. As described above, the ISA concludes that, when taken together, “recent studies provide scientific evidence that NO<sub>2</sub> is associated with a range of respiratory effects and is sufficient to infer a likely causal relationship between short-term NO<sub>2</sub> exposure and adverse effects on the respiratory system” (ISA, section 5.3.2.1). This conclusion is based, in part, on the observation that a number of epidemiologic studies have detected positive associations between short-term (e.g., 1-h, 24-h) NO<sub>2</sub> concentrations and health effects. Many of these studies have been conducted in locations where long-term ambient levels of NO<sub>2</sub> are well below the current annual standard. As a result, staff has concluded that it is appropriate to consider alternative averaging times for their ability to protect against health effects associated with short-term NO<sub>2</sub> levels and/or exposures.

In contrast to the conclusion in the ISA concerning respiratory morbidity associated with short-term exposures to NO<sub>2</sub>, the ISA concludes that the “evidence examining the effect of long-term exposure to NO<sub>2</sub> on respiratory morbidity is suggestive but not sufficient to infer a causal relationship” (ISA, section 5.3.2.4). In addition, the ISA concludes that the available evidence for the effect of long-term exposure to NO<sub>2</sub> on other health outcomes (i.e., mortality, cancer, cardiovascular effects, reproductive and developmental effects) is “inadequate to infer the presence or absence of a causal relationship” (ISA, sections 5.3.2.5 and 5.3.2.6). As a result, staff has not considered alternative long-term standards in the current assessment.

In considering appropriate short-term averaging times, staff has considered evidence from both experimental and epidemiologic studies. New evidence from controlled human exposure studies generally evaluates exposures between 30 minutes and 3 hours while epidemiologic studies have used different short-term averaging periods, most commonly 1-h and 24-h (ISA, section 3.1). A few epidemiologic studies have considered both 1-h and 24-h averaging times, allowing comparisons to be made. The ISA reports that such comparisons failed to reveal differences between effect estimates based on a 1-h averaging time versus those based on a 24-h averaging time (ISA, section 5.3.2.7). Therefore, the ISA concludes that it is not possible to

discern whether effects observed in epidemiologic studies are attributable to average daily (or multiday) concentrations (24-h avg) or high, peak exposures (1-h max) (ISA, section 5.3.2.7). In addition, the ISA concludes that experimental studies in both animals and humans provide evidence that NO<sub>2</sub> exposures from less than 1 hour up to 3 hours can result in respiratory effects (section 5.3.2.7). Given that the epidemiologic evidence does not provide clear guidance in choosing between 1-h and 24-h averaging times, and given that the experimental literature provides support for the occurrence of effects following exposures of shorter duration than 24 hours (e.g., 1-h), staff has chosen to evaluate standards with a 1-h averaging time.

## 5.4 FORM

In evaluating alternative forms for the primary standard, staff recognizes that it is important to have a form that 1) reflects the health risks posed by elevated NO<sub>2</sub> concentrations and 2) achieves a balance between limiting the occurrence of peak concentrations and providing a stable and robust regulatory target. Consistent with judgments made in recent reviews of the PM (71 FR 61144) and O<sub>3</sub> (73 FR 16436) NAAQS, staff judges that a concentration-based form averaged over 3 years for the NO<sub>2</sub> standard would better reflect health risks and would provide greater stability than a form based on expected exceedances. A concentration-based form would give proportionally greater weight to hours when NO<sub>2</sub> concentrations are well above the level of the standard than to hours when concentrations are just above the standard, while an expected exceedance form would give the same weight to hours that just exceed the standard as to hours that greatly exceed the standard. Therefore, a concentration-based form averaged over 3 years better reflects the health risks posed by elevated NO<sub>2</sub> concentrations and, in developing potential alternative standards for consideration, we have focused on standards with this concentration-based form. The most recent review of the PM NAAQS (completed in 2006) judged that using a 98<sup>th</sup> percentile form averaged over 3 years provides an appropriate balance between limiting the occurrence of peak concentrations and providing a stable regulatory target (71 FR 61144). In consideration of this balance, we have determined it appropriate in the current review to evaluate both the 98<sup>th</sup> and 99<sup>th</sup> percentile NO<sub>2</sub> concentrations averaged over 3 years.<sup>4</sup> We have judged that these percentiles, when combined with the range of alternatives identified for the level of the

---

<sup>4</sup> 98<sup>th</sup> or 99<sup>th</sup> percentiles of the 1-h daily maximum NO<sub>2</sub> concentrations would be calculated for each of 3 consecutive years. The 98<sup>th</sup> or 99<sup>th</sup> percentile concentrations for each of these 3 years would then be averaged together.

standard (see below), offer a sufficient range of options to balance the objective of providing a stable regulatory target against the objective of limiting the occurrence of peak concentrations.

## **5.5 LEVEL**

In developing an approach to formulating an appropriate range of NO<sub>2</sub> levels for analysis, staff has taken into account several considerations including the following. First, since the review of the NO<sub>2</sub> NAAQS that was completed in 1996, a large number of published epidemiologic studies have evaluated associations between respiratory morbidity and short-term levels of ambient NO<sub>2</sub>. In general, these studies report positive associations and a number of these associations are statistically-significant. The ISA notes that many of these studies have been conducted in locations where ambient levels of NO<sub>2</sub> are well below the level of the current NAAQS (ISA, section 5.3.2.1). Second, controlled human exposure studies have detected effects of NO<sub>2</sub> exposure on several health endpoints. Of these, only airway hyperresponsiveness is associated with exposures to NO<sub>2</sub> concentrations at or near ambient levels. In fact, the NO<sub>2</sub> exposure levels associated with increased airway responsiveness overlap the maximum ambient NO<sub>2</sub> concentrations in some locations where associations with respiratory effects have been detected. Third, limitations in both epidemiologic studies (e.g., confounding by co-pollutants) and controlled human exposure studies (e.g., most sensitive populations likely not evaluated) suggest that an appropriate approach to identifying levels for potential alternative standards is to consider both types of studies.

In considering both types of studies, we note that NO<sub>2</sub> concentrations represent different metrics when reported in epidemiologic studies versus controlled human exposure studies. Concentrations of NO<sub>2</sub> reported in epidemiologic studies are typically based on ambient monitoring data while NO<sub>2</sub> levels reported in controlled human exposure studies represent the concentration of NO<sub>2</sub> in the breathing zone of the individual. Therefore, consideration of NO<sub>2</sub> levels from controlled human exposure studies when identifying alternative levels for an ambient standard introduces some uncertainty. For example, elevated NO<sub>2</sub> monitors, particularly in inner cities, likely underestimate personal exposures that occur at lower elevations closer to traffic (ISA, section 5.2.2). In situations where personal exposure to ambient NO<sub>2</sub> is higher than ambient levels measured at a monitor, ambient standard levels based on controlled exposure studies could be less health-protective than levels based on concentrations reported in

epidemiologic studies at ambient monitors. However, in studies where it has been determined, the ratio of personal exposure to NO<sub>2</sub> of ambient origin to the ambient NO<sub>2</sub> concentration ranges from approximately 0.3 to 0.6 (ISA, section 5.2.2). This suggests that in some cases personal exposure concentrations for ambient NO<sub>2</sub> are lower than the levels of NO<sub>2</sub> measured at ambient monitors. In situations where personal exposure concentrations for ambient NO<sub>2</sub> are lower than the levels measured at ambient monitors, an ambient standard level based on controlled exposure studies could be more health-protective than a level based on concentrations reported in epidemiologic studies at ambient monitors. Overall, because some individuals are likely exposed to NO<sub>2</sub> levels higher than those measured at ambient monitors (ISA, section 5.2.2) while others are likely exposed to NO<sub>2</sub> levels lower than those measured at ambient monitors (see ISA, tables 2.5-4 and 2.5-5), we have chosen to use NO<sub>2</sub> concentrations associated with both epidemiologic studies and controlled human exposure studies for purposes of selecting alternative levels for analysis.

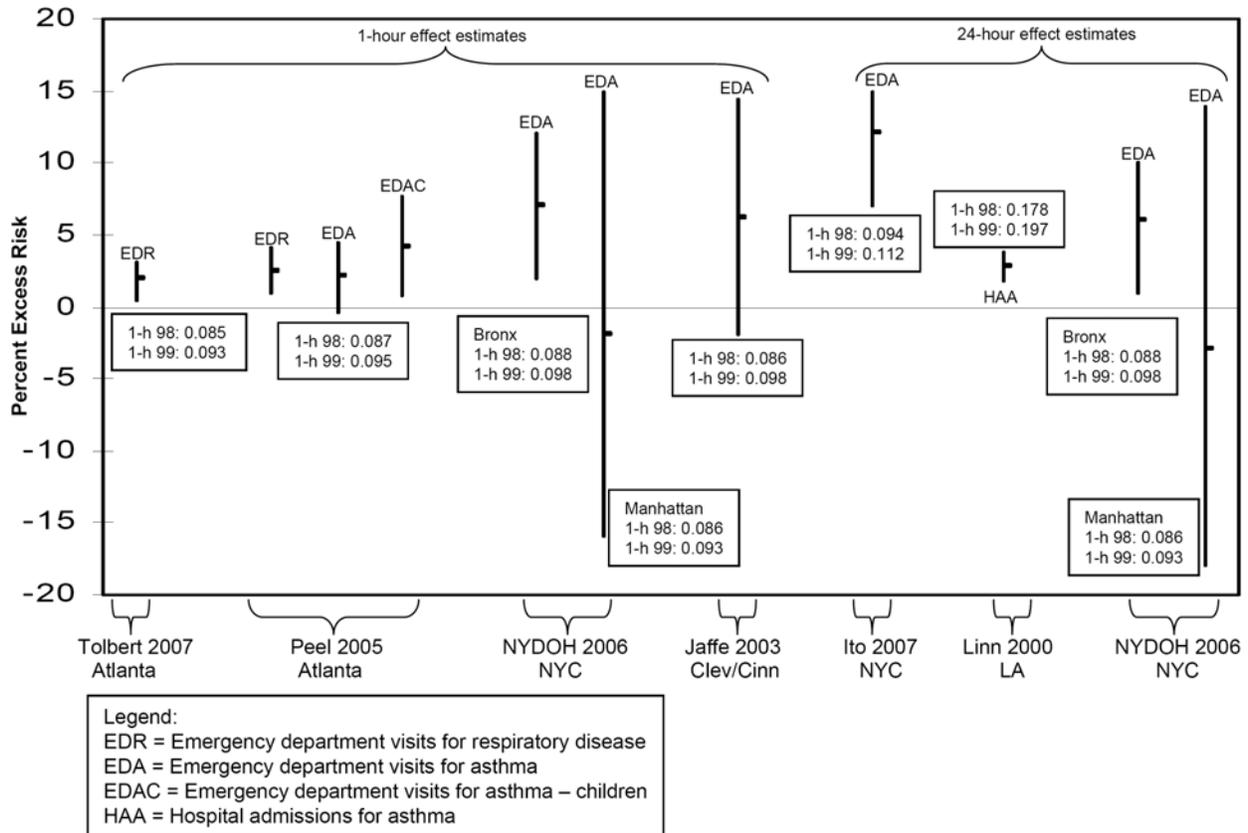
As a result of the above considerations, to determine the levels that should be evaluated staff has relied on both key epidemiologic studies conducted in the United States that evaluate associations between short-term levels of NO<sub>2</sub> and respiratory morbidity (symptoms, hospital admissions, ED visits) and on controlled human exposure studies that evaluate airway hyperresponsiveness following NO<sub>2</sub> exposure. Figures 5-1 and 5-2 below show standardized effect estimates<sup>5</sup> and the 98<sup>th</sup> and 99<sup>th</sup> percentile concentrations of daily 1-h maximum NO<sub>2</sub> for locations and time periods that correspond to key U.S. epidemiologic studies identified in the ISA (see table 5.4-1 in ISA for a list of key studies; Thompson and Jenkins, 2008).

Of the key U.S. epidemiologic studies included in figures 5-1 and 5-2, the highest 1-h NO<sub>2</sub> concentrations were detected in the two studies conducted in Los Angeles (Linn et al., 2000; Ostro et al., 2001). For these studies, the 98<sup>th</sup> and 99<sup>th</sup> percentile 1-h daily maximum concentrations of NO<sub>2</sub> overlap levels that the ISA concludes are associated with increased airway responsiveness in controlled human exposure studies (ISA, section 5.3.2.1). Therefore, staff judges that the combination of the epidemiologic studies by Linn et al. (2000) and Ostro et al. (2001), as well as the meta-analysis (Folinsbee, 1992; ISA, table 3.1-3; table 4-2 of this document) of controlled human exposure studies on airway responsiveness, provide an

---

<sup>5</sup> The effect estimates presented in figures 5-1 and 5-2 are for those endpoints included in figure 5.3-1 and table 5.4-1 of the ISA.

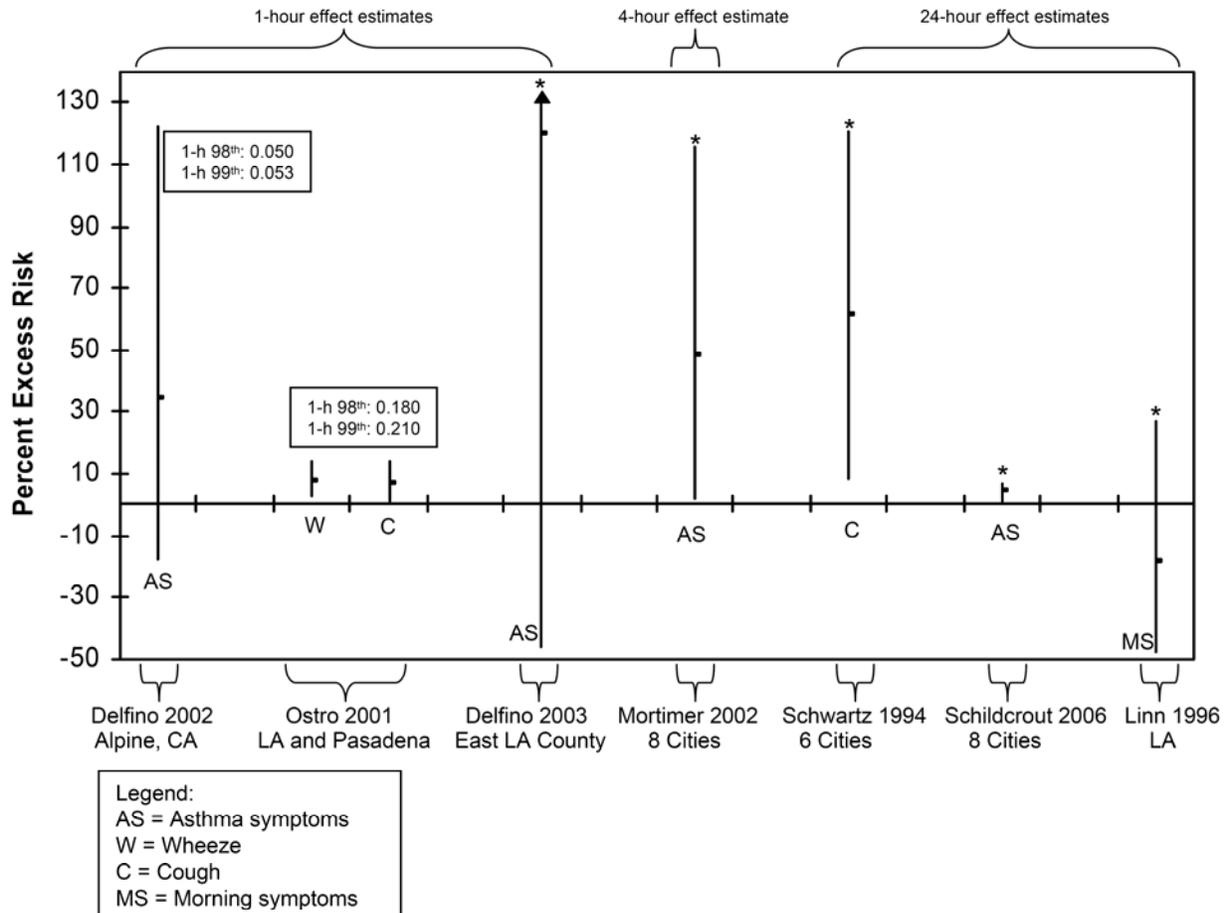
appropriate basis for identifying the upper end of the range of standard levels to be considered. Given that the ISA concludes that significant increases in airway responsiveness are associated with short-term exposures to NO<sub>2</sub> at 0.2 to 0.3 ppm and given that the epidemiologic studies by Linn et al. (2000) and Ostro et al. (2001) are associated with 98<sup>th</sup> and 99<sup>th</sup> percentile 1-h daily maximum NO<sub>2</sub> levels that are just below (Linn et al., 2000) and just above (99<sup>th</sup> percentile level for Ostro et al., 2001) 0.2 ppm (see figures 1 and 2 below), staff judges that an appropriate upper end of the range of potential standard levels is a daily maximum 1-h NO<sub>2</sub> concentration of 0.20 ppm.



**Figure 5-1. NO<sub>2</sub> effect estimates<sup>6</sup> (95% CI) for ED visits/HA and associated 1-h daily maximum NO<sub>2</sub> levels (98<sup>th</sup> and 99<sup>th</sup> percentile values in boxes<sup>7</sup>)**

<sup>6</sup>Effect estimates presented in figures 5-1 and 5-2 are from single pollutant models only. The studies by Tolbert et al., (2007); Peel et al., (2005); NYDOH (2006); Ito et al., (2007); and Delfino et al. (2002) also evaluated multi-pollutant models. NO<sub>2</sub> effect estimates retained statistical-significance in the study by Ito, but not in the other studies.

<sup>7</sup> Authors of relevant U.S. and Canadian studies were contacted and air quality statistics from the study monitor that recorded the highest NO<sub>2</sub> levels were requested. In cases where authors provided 1-hour daily maximum air quality statistics, this information is presented in figures 1 and 2 (studies by Tolbert, Peel, NYDOH, Delfino). In one case (study by Ito) authors provided 24-hour air quality data, but identified a specific monitor in AQS. We used AQS to reconstruct the 1-hour daily maximum air quality for that monitor during the time period of the study. In three cases (studies by Jaffe, Linn, Ostro), we were not able to identify appropriate statistics from the information provided by the authors and the authors did not provide monitor identification information. In these cases, we attempted to reconstruct the air quality data set for the location and time of the study using EPA's Air Quality System (AQS). Prior to identifying potential alternative standards, we did not receive air quality information from any of the Canadian authors contacted and we were unable to reconstruct the air quality data sets for the Canadian studies. Therefore, for purposes of identifying levels of potential alternative standards, our analysis was based on these key U.S. studies. Note that the NO<sub>2</sub> concentrations reported in table 1 of the study by Jaffe are labeled as 24-hour concentrations, but the author indicated in a personal communication (Jaffe, 2008) that they actually represent 1-hour daily maximum concentrations.



\*We do not have 1-h 98<sup>th</sup> and 99<sup>th</sup> percentile NO<sub>2</sub> levels for several of the U.S. respiratory symptom studies identified in table 5.4-1 of the ISA. Comparison of averages (see ISA, table 5.4-1) suggests that 24-h NO<sub>2</sub> levels in the studies by studies by Schildcrout and Schwartz are somewhat lower than the 24-hour levels reported in other U.S. studies, 24-h levels in the study by Linn are similar to 24-h levels reported in other U.S. studies, and 1-h maximum levels in the study by Delfino are lower than 1-h maximum levels reported in other U.S. studies. Such comparisons have not been made for the study by Mortimer because it is the only study that reports 4-hour NO<sub>2</sub> levels.

**Figure 5-2. NO<sub>2</sub> effect estimates for respiratory symptoms and associated 1-h daily maximum NO<sub>2</sub> levels (98<sup>th</sup> and 99<sup>th</sup> percentile values in boxes)**

In identifying additional standard levels that should be analyzed, staff has considered that

- 1) health effect associations in epidemiologic studies are observed in locations with 1-h daily maximum levels of NO<sub>2</sub> below 0.2 ppm (i.e., 99<sup>th</sup> percentile levels in several studies are close to 0.1 ppm);
- 2) controlled human exposure studies that evaluate the ability of NO<sub>2</sub> to elicit airway hyperresponsiveness have assessed mild asthmatics and more severely affected asthmatics could experience increased airway responsiveness at lower levels of NO<sub>2</sub> than observed in these

studies; and 3) a meta-analysis presented in the ISA (see Table 4-2) detects statistically-significant effects on the direction of airway responsiveness following short-term NO<sub>2</sub> exposures as low as 0.1 ppm. As a result of these considerations, staff judges that it would be appropriate to consider additional standard levels that provide a margin of safety relative to 0.20 ppm. Therefore, we will also consider daily maximum 1-h NO<sub>2</sub> standard levels of 0.10 ppm and 0.15 ppm.

In identifying the lower end of the range of standards that will be analyzed, staff has considered the fact that the study by Delfino et al., (2002) provides evidence for associations between short-term ambient NO<sub>2</sub> concentrations and respiratory morbidity in a location where the 98<sup>th</sup> and 99<sup>th</sup> percentile concentrations of the 1-h daily maximum levels of NO<sub>2</sub> were well below 0.1 ppm (Delfino et al., 2002). This study detects associations between 1-h and 8-h (only 8-h associations were statistically-significant) levels of NO<sub>2</sub> and asthma symptoms in a location where the 98<sup>th</sup> and 99<sup>th</sup> percentile 1-h daily maximum NO<sub>2</sub> concentrations were 0.050 and 0.053 ppm, respectively. The 8-h effect estimate in this study remained positive, but became statistically non-significant, in a two-pollutant model that also included PM<sub>10</sub>. Staff judges that it is appropriate to base the lower end of the range of alternative standard levels on this study by Delfino et al. (2002). Therefore, we will also consider a 1-h daily maximum standard level of 0.050 ppm.

## **6. OVERVIEW OF APPROACHES TO ASSESSING EXPOSURES AND RISKS**

### **6.1 INTRODUCTION**

The purpose of the assessments described in this document is to characterize exposures and risks associated with recent ambient levels of NO<sub>2</sub>, with levels associated with just meeting the current NO<sub>2</sub> NAAQS, and with levels associated with just meeting potential alternative standards (see chapter 5 of this document for discussion of potential alternative standards). To characterize health risks, we have employed three approaches. With each approach, we have characterized health risks associated with the air quality scenarios of interest (i.e., recent air quality unadjusted, air quality adjusted to simulate just meeting the current standard, and air quality adjusted to simulate just meeting potential alternative standards). In the first approach, NO<sub>2</sub> air quality levels have been compared to potential health effect benchmark values derived from the controlled human exposure literature (see section 6.2 below for discussion of benchmark levels). In the second approach, modeled estimates of actual exposures have been compared to potential health effect benchmarks. In the third approach, exposure-response relationships from epidemiologic studies have been used to estimate health impacts. An overview of the approaches to characterizing health risks is provided below and each approach is described in more detail in subsequent sections of this document and the associated appendices.

In the first approach, we have compared NO<sub>2</sub> air quality with potential health effect benchmark levels for NO<sub>2</sub>. Scenario-driven air quality analyses have been performed using ambient NO<sub>2</sub> concentrations for the years 1995 through 2006. With this approach, NO<sub>2</sub> air quality serves as a surrogate for exposure. All U.S. monitoring sites where NO<sub>2</sub> data have been collected are represented by this analysis and, as such, the results generated are considered a broad characterization of national air quality and human exposures that might be associated with these concentrations. An advantage of this approach is its relative simplicity; however, there is uncertainty associated with the assumption that NO<sub>2</sub> air quality can serve as an adequate surrogate for exposure to ambient NO<sub>2</sub>. Actual exposures might be influenced by factors not considered by this approach, such as the spatial and temporal variability in human activities.

In the second approach, we have used an inhalation exposure model to generate more realistic estimates of personal exposures. Estimates of personal exposure have been compared to potential NO<sub>2</sub> health benchmark levels. For this exposure analysis, a probabilistic approach was used to model individual exposures considering the time people spend in different microenvironments and the variable NO<sub>2</sub> concentrations that occur within these microenvironments across time, space, and microenvironment type. This approach to assessing exposures was more resource intensive than using ambient levels as a surrogate for exposure; therefore, staff has included the analysis of only one specific location in the U.S. (Atlanta MSA)<sup>8</sup>. Although the geographic scope of this analysis is restricted, the approach provides realistic estimates of NO<sub>2</sub> exposures, particularly those exposures associated with important emission sources of NO<sub>x</sub> and NO<sub>2</sub>, and serves to complement the broad air quality characterization.

For the characterization of risks in both the air quality analysis and the exposure modeling analysis described above, staff has used a range of short-term potential health effect benchmarks. The levels of potential benchmarks are based on NO<sub>2</sub> exposure levels that have been associated with increased airway responsiveness in asthmatics in controlled human exposure studies (ISA, section 5.3.2.1; see above for discussion). Benchmark values of 100, 150, 200, 250, and 300 ppb have been compared to both NO<sub>2</sub> air quality levels and to estimates of NO<sub>2</sub> exposure. When NO<sub>2</sub> air quality is used as a surrogate for exposure, the output of the analysis is an estimate of the number of times per year specific locations experience 1-h levels of NO<sub>2</sub> that exceed a particular benchmark. When personal exposures are simulated, the output of the analysis is an estimate of the number of individuals at risk for experiencing daily maximum 1-h levels of NO<sub>2</sub> of ambient origin that exceed a particular benchmark. An advantage of using potential health effect benchmark levels to characterize health risks is that the effects observed in controlled human exposure studies clearly result from NO<sub>2</sub> exposure. This is in contrast to health effects associated with NO<sub>2</sub> in epidemiologic studies, which may also be associated with pollutants that co-occur with NO<sub>2</sub> in the ambient air. Thus, when using epidemiologic studies as the basis for risk characterization, the unique contribution of NO<sub>2</sub> to a particular health effect

---

<sup>8</sup> In the document titled *Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard: First Draft*, we have presented the results of an exposure analysis for Philadelphia. Based on CASAC comments received on that exposure analysis, we have refined our approach and applied those refinements to the Atlanta analysis presented in this document. The original Philadelphia analysis is presented in the appendix to this document, but has not been modified since the first draft.

may be difficult to quantify. A disadvantage of the potential benchmark approach is that the magnitude of the NO<sub>2</sub> effect on airway responsiveness can vary considerably from individual to individual and not all asthmatics would be expected to respond to the same levels of NO<sub>2</sub> exposure. Therefore, the public health impacts of NO<sub>2</sub>-induced airway hyperresponsiveness are difficult to quantify.

In the third approach, we have estimated respiratory ED visits as a function of ambient levels of NO<sub>2</sub> measured at a fixed-site monitor representing ambient air quality for an urban area. In this approach, concentration-response functions are derived from NO<sub>2</sub> epidemiologic studies and are used to estimate the impact of ambient levels of NO<sub>2</sub>, as measured at a fixed-site monitor, on ED visits. By focusing on a different health endpoint from the first two approaches described above, this epidemiology-based approach provides additional perspective on the potential public health impacts of NO<sub>2</sub>. Relative to the approaches that use controlled human exposure studies, this approach to characterizing health risks has several advantages. For example, the public health significance of the effect in question (i.e., ED visits) is less ambiguous in terms of its impact on individuals than is an increase in the airway response measured in a controlled human exposure study. In addition, the concentration-response relationship reflects real-world levels of NO<sub>2</sub> and co-pollutants present in ambient air. However, a disadvantage of this approach is the ambiguity and complexity associated with quantifying the contribution of NO<sub>2</sub> to the reported health impacts relative to the contributions of co-occurring pollutants.

## **6.2 POTENTIAL HEALTH BENCHMARK LEVELS**

As noted above (section 4.5.3 and 6.1), staff has developed potential health benchmark levels that are based on results from controlled human exposure studies of airway responsiveness. The purpose of these potential health benchmark levels is to provide a basis for comparing NO<sub>2</sub> concentrations shown to increase airway responsiveness in asthmatics (from controlled human exposure studies) to estimates of NO<sub>2</sub> exposures derived from our air quality and exposure analyses. Because the purpose of the benchmarks is to provide a way to link estimates of NO<sub>2</sub> exposure with levels known to produce respiratory effects in individual asthmatics, benchmark levels have not been developed from the epidemiologic literature.

To identify potential health effect benchmarks, staff has relied on the ISA's evaluation of the NO<sub>2</sub> human exposures studies. Controlled human exposure studies involving allergen challenge in asthmatics suggest that NO<sub>2</sub> exposure may enhance the sensitivity to allergen-induced decrements in lung function and increase the allergen-induced airway inflammatory response at exposures as low as 0.26-ppm NO<sub>2</sub> for 30 min (ISA, Figure 3.1-2 and section 5.3.2.1). Exposure to NO<sub>2</sub> also has been found to enhance the inherent responsiveness of the airway to subsequent nonspecific challenges (ISA, section 5.3.2.1). In asthmatics, small but significant increases in nonspecific airway responsiveness have been observed in the range of 0.2 to 0.3 ppm NO<sub>2</sub> for 30 minute exposures and at 0.1 ppm NO<sub>2</sub> for 1-h exposures (ISA, section 5.3.2.1). Therefore, for the risk characterization, staff judges that 1-h NO<sub>2</sub> levels in this range are appropriate to consider as potential health benchmarks for comparison to air quality levels and exposure estimates. To characterize health risks with respect to this range, potential health effect benchmark values of 0.10 ppm, 0.20 ppm, 0.25 ppm, and 0.30 ppm have been employed to reflect the lower- middle- and upper-end of the range identified in the ISA as levels at which controlled human exposure studies have provided evidence for the occurrence of NO<sub>2</sub>-induced airway hyperresponsiveness.

In choosing this range, we recognize that uncertainties exist regarding the percentage of asthmatics expected to experience an increase in responsiveness following NO<sub>2</sub> exposure and in the clinical implications of such an increase. A meta-analysis presented in the ISA (see Table 4-2 above) suggests that between 66% and 75% of asthmatics may experience an increase in airway responsiveness following short-term NO<sub>2</sub> exposures in the range of 0.1 to 0.3 ppm. However, this meta-analysis provides information only on the direction of the NO<sub>2</sub> effect and not on its magnitude. In addition, the NO<sub>2</sub> controlled human exposure studies of airway responsiveness have focused primarily on mild asthmatics. It is possible that more severely affected asthmatics could experience a more severe response following NO<sub>2</sub> exposures in this range. It is also possible that they could experience a response at lower levels of NO<sub>2</sub> than the mild asthmatics included in these studies. However, even considering these uncertainties, staff judges that the identified range of concentrations is sufficient to provide some perspective on the potential public health impacts of NO<sub>2</sub> exposures, especially when the results of the risk characterization based on airway responsiveness are considered in conjunction with the risk assessment based on the epidemiology literature.

## **6.3 SIMULATING THE CURRENT AND ALTERNATIVE STANDARDS**

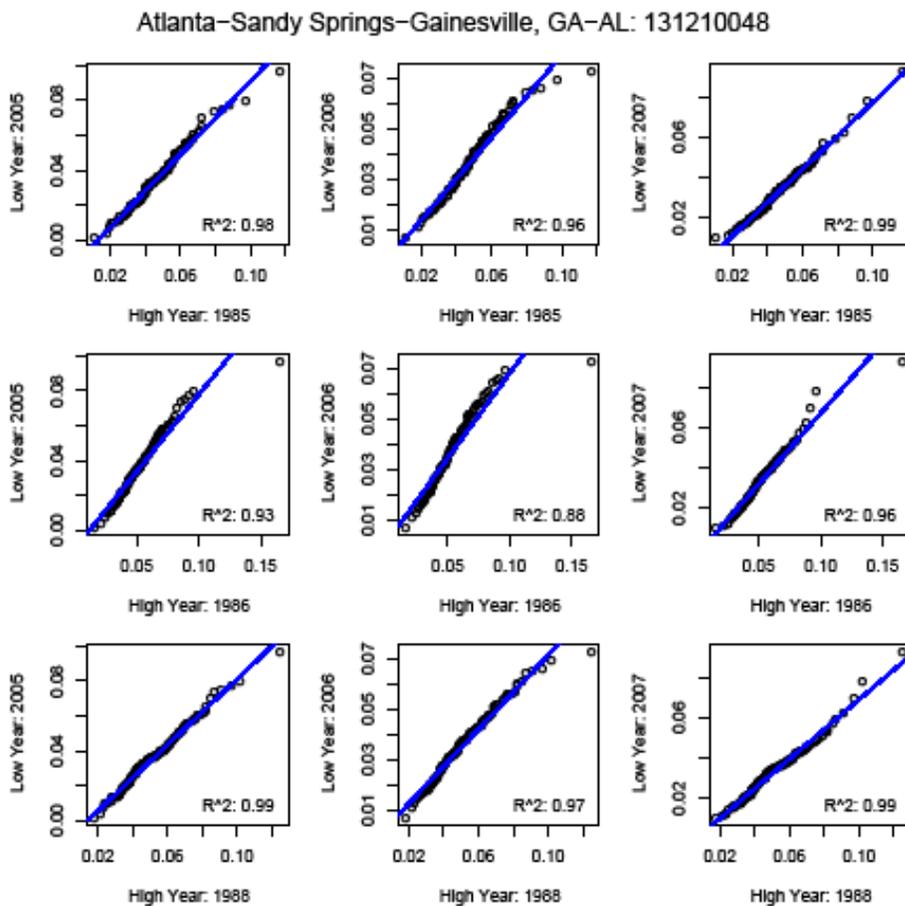
A primary goal of these risk and exposure assessments is to evaluate the ability of the current NO<sub>2</sub> standard (0.053 ppm annual average) and potential alternative standards (0.05, 0.10, 0.15, and 0.20 ppm; see chapter 5 of this document) to protect public health. In order to evaluate the ability of a specific standard to protect public health, ambient NO<sub>2</sub> concentrations need to be adjusted such that they simulate levels of NO<sub>2</sub> that just meet that standard. Such adjustments allow comparisons of the level of public health protection that could be associated with just meeting the current and potential alternative standards. All areas of the United States currently have ambient NO<sub>2</sub> levels below the current annual standard. Therefore, to simulate just meeting the current annual standard, NO<sub>2</sub> air quality levels must be adjusted upward. Similarly, to simulate a potential standard that is below current air quality levels, those current levels must be adjusted downward. This process of adjusting air quality to simulate just meeting a specific standard is described in more detail below.

### **6.3.1 Adjustment of Ambient Air Quality**

Based on the level of U.S. policy-relevant background (PRB) and observed trends in ambient monitoring, ambient NO<sub>2</sub> concentrations were proportionally adjusted at each location using the maximum monitored concentration that occurred in each year. Policy-relevant background is defined as the distribution of NO<sub>2</sub> concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of NO<sub>2</sub> precursors in the U.S., Canada, and Mexico. Policy-relevant background for most of the continental U.S. is estimated to be less than 300 parts per trillion (ppt) (ISA, Section 2.4.6). In the Northeastern U.S. where present-day NO<sub>2</sub> concentrations are highest, this amounts to a contribution of less than 1% percent of the total observed ambient NO<sub>2</sub> concentration. This low contribution of PRB to NO<sub>2</sub> concentrations provides support for a proportional method to adjust air quality, i.e., an equal adjustment of air quality values across the entire air quality distribution to just meet a target value.

While annual average concentrations have declined significantly over the time period of analysis, the variability in the concentrations, both the annual average and 1-hour concentrations, have remained relatively constant. This trend is apparent when considering the air quality data collectively (Appendix A, section 7) and when considering individual locations (Rizzo, 2008).

As an example, Figure 6-1 compares the trends in daily maximum NO<sub>2</sub> 1-hour concentration percentiles at the one ambient monitor in Atlanta that was in operation as far back as 1985 and is currently part of the monitoring network. Three recent years of data (2005-2007) were selected to constitute a series of low concentration year data along with three historical years of data (1985, 1986, and 1988) constituting a series of high concentration year data. As shown in the figure, the relationships between the low and high concentration years at each of the daily maximum concentration percentiles are mostly linear, with R<sup>2</sup> values ranging from 0.88 to 0.99. Where deviation from linearity did occur, it occurred primarily at a single point, either at the maximum daily maximum or the minimum daily maximum 1-hour concentration. This indicates that the rate of decrease in ambient air quality concentrations at the mean value is consistent with the rate of change at the lower and upper daily maximum 1-hour concentration percentiles. This trend provides support for the use of a proportional approach to adjust current ambient concentrations to represent air quality under both the current and alternative standard scenarios.



**Figure 6-1. Comparison of measured daily maximum NO<sub>2</sub> concentration percentiles in Atlanta for three high concentration years (1985, 1986, 1988) versus three low concentration years (2005-2007) at one ambient monitor.**

To adjust concentrations that simulate just meeting the current annual average standard of 0.053 ppm, proportional adjustment factors  $F$  for each location ( $i$ ) and year ( $j$ ) were derived by the following:

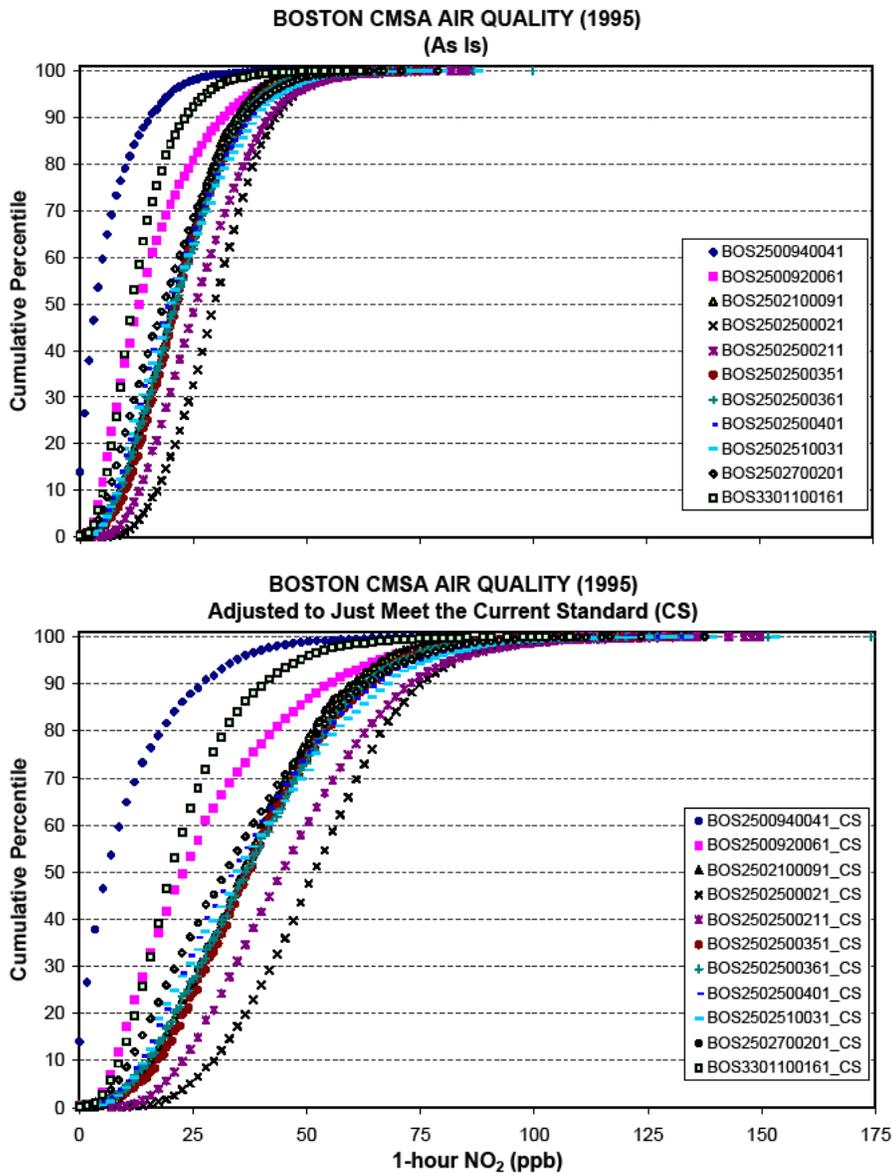
$$F_{ij} = S / C_{\max,ij} \quad \text{equation (6-1)}$$

where,

- $F_{ij}$  = NO<sub>2</sub> concentration adjustment factor (unitless) in location  $i$  given the annual average standard and for each year  $j$
- $S$  = Current standard level (i.e., 53 ppb, annual average NO<sub>2</sub> concentration)
- $C_{\max,ij}$  = The maximum annual average NO<sub>2</sub> concentration at a monitor in each location  $i$  and for each year  $j$  (ppb)

In these cases where staff simulated a proportional adjustment in ambient NO<sub>2</sub> concentrations using equation (6-2), it was assumed that the current temporal and spatial distribution of air concentrations (as characterized by the current air quality data) is maintained and increased NO<sub>x</sub> emissions contribute to increased NO<sub>2</sub> concentrations, with the highest monitor (in terms of annual averages) being adjusted so that it just meets the current 0.053 ppm annual average standard. Values for each air quality adjustment factor used for each location evaluated in the air quality and risk characterization are given in Appendix A (section 7.2). For each location and calendar year, all the hourly NO<sub>2</sub> concentrations in a location were multiplied by the same constant value  $F$  to make the highest annual mean equal to 53 ppb for that location and year. For example, of twelve monitors measuring NO<sub>2</sub> in Boston for year 1995 (Figure 6-2, top), the maximum annual average concentration was 30.5 ppb, giving an adjustment factor of  $F = 53/30.5 = 1.74$  for that year. All hourly concentrations measured at all monitoring sites in that location are then multiplied by 1.74, resulting in an upward scaling of hourly NO<sub>2</sub> concentrations for that year. Therefore, one monitoring site in Boston for year 1995 would have an annual average concentration of 0.053 ppm, while all other monitoring sites would have an annual

average concentration below that value, although still proportionally scaled up by 1.74 (Figure 6-2, bottom). Then, using the adjusted hourly concentrations to simulate just meeting the current standard, the metrics of interest (e.g., annual mean NO<sub>2</sub> concentration, the number of potential health effect benchmark exceedances) were estimated for each site-year.



**Figure 6-2. Distributions of hourly NO<sub>2</sub> concentrations at twelve ambient monitors in the Boston CMSA, as is (top) and air quality adjusted to just meet the current standard (bottom), Year 1995.**

Proportional adjustment factors were also derived considering the form, averaging time, and levels of the potential alternative standards under consideration. Discussion regarding the staff selection of each of these components is provided in chapter 5 of this document. The 98<sup>th</sup> and 99<sup>th</sup> percentile 1-hour NO<sub>2</sub> daily maximum concentrations averaged across three years of monitoring were used in calculating the adjustment factors at each of four standard levels as follows:

$$F_{ikl} = S_l / \left( \frac{\sum_{j=1}^3 C_{ijk}}{3} \right)_{\max,i} \quad \text{equation (6-2)}$$

where,

- $F_{ikl}$  = NO<sub>2</sub> concentration adjustment factor (unitless) in location  $i$  given alternative standard percentile form  $k$  and standard level  $l$  across a 3-year period
- $S_l$  = Standard level  $l$  (i.e., 50, 100, 150, 200 ppb 1-hour NO<sub>2</sub> concentration (ppb))
- $C_{ijk}$  = Selected percentile  $k$  (i.e., 98<sup>th</sup> or 99<sup>th</sup>) 1-hour daily maximum NO<sub>2</sub> concentration at a monitor in location  $i$  (ppb) for each year  $j$

As described above for adjustments made in simulating just meeting the current standard, it was assumed that the current temporal and spatial distribution of air concentrations (as characterized by the current NO<sub>2</sub> air quality data) is maintained and increased NO<sub>x</sub> emissions contribute to increased NO<sub>2</sub> concentrations, with the highest monitor (in terms of the 3-year average at the 98<sup>th</sup> or 99<sup>th</sup> percentile) being adjusted so that it just meets the level of the particular 1-hour alternative standard. Since the alternative standard levels range from 50 ppb through 200 ppb, both proportional upward and downward adjustments were made to the 1-hour ambient NO<sub>2</sub> concentrations. The values for each air quality adjustment factor used for each location evaluated in the air quality and risk characterization are given in Appendix A, section 7.2. Due to the form of the alternative standards, the expected utility of such an analysis, and the limited time available to conduct the analysis, only the more recent air quality data were used (i.e., years 2001-2006) and separated into two 3-year periods, 2001-2003 and 2004-2006. The 1-hour ambient NO<sub>2</sub> concentrations were adjusted in a similar manner described above for just meeting the current standard, however, due to the form of these standards, only one factor was

derived for each 3-year period, rather than one factor for each calendar year as was done with just meeting the current standard.

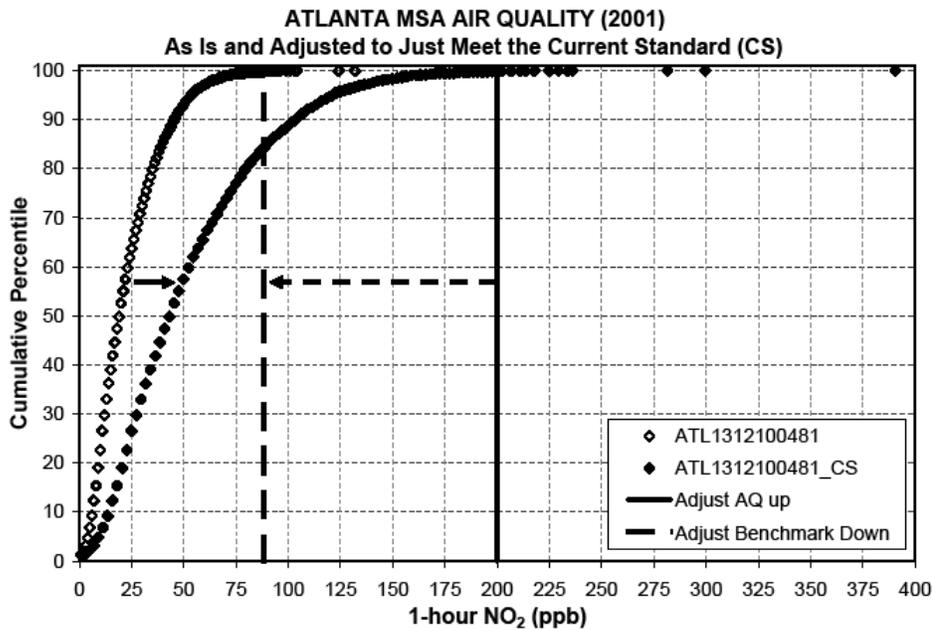
### **6.3.2 Adjustment of Potential Health Effect Benchmark Levels**

Rather than proportionally modify the air quality concentrations used for input to the exposure model, a proportional adjustment of the potential health effect benchmark level was performed. This was done to reduce the processing time associated with the exposure modeling simulations since there were several thousands of receptors modeled in the Atlanta exposure assessment. In addition, because the adjustment procedure is proportional, the application of a downward adjustment of the selected benchmark level is mathematically equivalent to a proportional upward adjustment of the air quality concentrations. The same approach used in the air quality adjustment described above was used in the exposure modeling to scale the benchmark levels to simulate just meeting the current and potential alternative standards. For example, an adjustment factor of 2.27 was determined for Atlanta for year 2001 to simulate ambient concentrations just meeting the current standard, based on the maximum annual average NO<sub>2</sub> concentration of 23.3 ppb observed at an ambient monitor for that year (see Appendix A, section 7.2). Therefore, the 1-hour potential health effect benchmark levels of 100, 200, and 300 ppb were proportionally adjusted to 44, 88, and 132 ppb, respectively for year 2001.

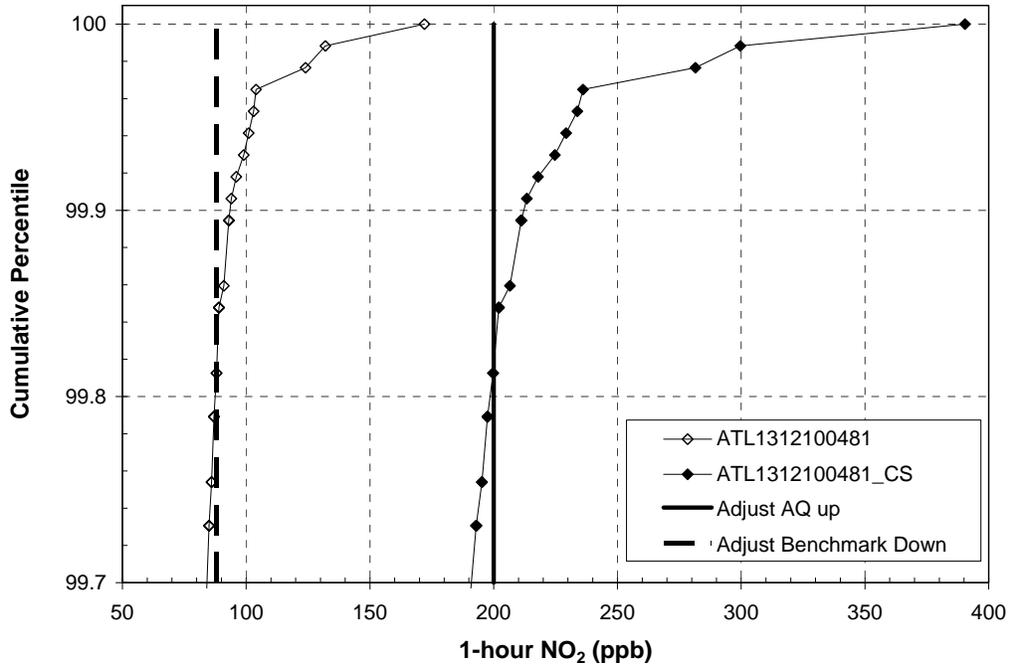
A comparison of the two procedures is presented in Figure 6-3 where air quality is adjusted to simulate just meeting the current annual standard and where the benchmark is adjusted to simulate air quality that just meets the current standard with using the *as is* air quality. This example uses the distribution of hourly NO<sub>2</sub> concentrations measured at the maximum ambient monitor (ID 1312100481) within the Atlanta modeling domain for year 2001. If we were interested in the number of exceedances of 200 ppb 1-hour under the current standard scenario for example, this would be equivalent to counting the number of exceedances of 88 ppb using the *as is* air quality.

For additional clarity, the same ambient air quality data are presented in Figure 6-4, only with expansion of the highest percentiles on the graph to allow for the counting of the number of exceedances. In using the air quality adjusted to just meet the current standard, i.e., the *as is* air quality was adjusted upwards by a factor 2.27, there are twelve exceedances of 200 ppb 1-hour. When considering the *as is* air quality without adjustment but with a downward adjustment of

the benchmark by the same factor of 2.27, there are the same number of exceedances of 200 ppb 1-hour. This benchmark adjustment procedure was applied in Atlanta where exposure modeling was performed to simulate just meeting the current and alternative standards. Additional details regarding derivation of the adjusted benchmark levels used in the exposure modeling are provided in chapter 8 of this document.



**Figure 6-3. Comparison of adjusted ambient monitoring concentrations (CS) or adjusted benchmark level (dashed line) to simulate just meeting the current annual average standard in Atlanta for year 2001.**



**Figure 6-4. Comparison of the upper percentiles for where ambient monitoring NO<sub>2</sub> concentrations (CS) and the benchmark level (dashed line) were adjusted to simulate just meeting the current annual standard in Atlanta for year 2001. The hourly NO<sub>2</sub> concentration distributions are provided in Figure 6-3.**

## 7. AMBIENT AIR QUALITY ASSESSMENT AND HEALTH RISK CHARACTERIZATION

### 7.1 OVERVIEW

Ambient monitoring data for each of the years 1995 through 2006 were used in this analysis to characterize NO<sub>2</sub> air quality across the U.S. This air quality data, as well as other NO<sub>2</sub> concentrations derived from ambient levels, were used as a surrogate to estimate potential human exposure. While an ambient monitor measures NO<sub>2</sub> concentrations at a stationary location, the monitor may well represent the concentrations that persons residing nearby are exposed to. The quality of the extrapolation of ambient monitor concentration to personal exposure will be dependent upon the spatial distribution of important emission sources, the siting of the ambient monitors, and consideration of places that persons visit. It is within this context that the approach for characterizing the ambient NO<sub>2</sub> air quality was designed.

Based on the health effects information from the human clinical and epidemiological studies, the averaging time of interest for the air quality characterization was 1-hour, with concentration levels ranging from between 100 and 300 ppb. Since the current standard is based on annual average levels of NO<sub>2</sub> while the most definitive health effects evidence is associated with short-term exposures (i.e., 30-minute to 1-hour, or one to several day), the air quality analysis required the development of a model that relates annual average and short-term levels of NO<sub>2</sub>. To characterize this relationship and to estimate the number of exceedances of the potential health effect benchmarks in specific locations, 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 observed air quality.

The available NO<sub>2</sub> air quality were first divided into two six-year 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 NO<sub>2</sub> 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, with the results within each year-group separated by monitor distance

from a major road.<sup>1</sup> The ambient monitor data were categorized in this manner to account for the potential influence of vehicle emissions on concentrations measured at the monitors within close proximity to roadways. There is potential for different concentration levels measured at each of these locations (i.e., near-road versus away from road) and thus potentially different exposure concentrations experienced by those persons spending time in these locations. A second scenario used the *as is* ambient monitoring data obtained from monitors sited  $\geq 100$  m from a major road and a simplified on-road simulation approach (described below in section 7.2.4) to estimate on-road NO<sub>2</sub> concentrations for each of the year-groups. This scenario was developed by recognizing that vehicles are important emission sources of NO<sub>x</sub> and NO<sub>2</sub> and that people spend time inside vehicles on roads.

Two additional scenarios followed in similar fashion to the *as is* air quality analysis, however these scenarios considered the ambient NO<sub>2</sub> concentrations simulated to just meeting the current standard of 0.053 ppm annual average and each of the alternative 1-hour standards of 50, 100, 150, and 200 ppb.<sup>2</sup> Due to the form of the alternative standards considered here (98<sup>th</sup> and 99<sup>th</sup> percentiles of the daily maximum concentrations averaged over 3 years), the recent ambient monitoring data set was divided into two three-year groups, 2001-2003 and 2004-2006.

Thus, the air quality characterization results are separated into two broad analyses, one using air quality as is and the other where air quality was adjusted to just meeting the current and alternative standards. Within both of these analyses, an additional simulation was performed to estimate NO<sub>2</sub> concentrations on roads. The first scenario described above is the only scenario that uses purely measurement data. Each of the other scenarios either uses a simulation procedure to estimate on-road concentrations (scenario 2), concentrations that just meet a particular standard level (scenario 3), or both (scenario 4).

Because many of the NO<sub>2</sub> ambient monitoring sites used in this analysis are primarily targeting public health monitoring objectives, the results are considered a broad characterization of national air quality and potential human exposures that might be associated with these scenario-driven concentrations. The output of this air quality characterization is an estimate the

---

<sup>1</sup> As part of our earlier analysis reported in the 2<sup>nd</sup> draft REA, the historical data were separated into two-road distance categories, <100 m and  $\geq 100$  m from a major road. The recent data were separated into both the two- and three-road distance categories.

<sup>2</sup> As part of our earlier analysis reported in the 1<sup>st</sup> draft REA, the historical data were evaluated using concentrations *as is* and for air quality adjusted to just meet the current standard. Only the recent data (2001-2006) were evaluated using air quality adjusted to just meet potential alternative standards and divided into two three-year groups.

number of times per year specific locations experience daily maximum levels<sup>3</sup> of NO<sub>2</sub> that may cause adverse health effects in susceptible individuals. Each location that was evaluated contained one to several monitors operating for a few to several years, generating a number of site-years of data. The number of site-years in a location were used to generate a distribution of two exposure and risk characterization metrics; the annual average concentrations and the number of daily maximum exceedances that did (observed data) or could occur (simulated data) in a year for that location. The mean and median values were reported to represent the central tendency of each metric for the four scenarios in each air quality year-group. For example, the mean annual average concentration for a location is the arithmetic average of all site-year annual average concentrations in that location given the particular year-group. The minimum annual average concentration served to represent a lower bound and could be a single or multiple site-year(s) of data in the location given the particular year-group, dependent on the distribution of annual average concentrations for each site-year. Since there were either multiple site-years of monitoring or numerous simulations performed for each location using all available site-years of data, results for the upper percentiles generally included the 95<sup>th</sup>, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution. As described for the minimum value, these upper percentile estimates could also represent either a single or multiple site-year(s) of data at a location given the particular year-group.

## 7.2 APPROACH

There were five broad steps to allow for the characterization of the air quality. The first step involved collecting, compiling, and screening the ambient air quality data collected since the prior review in 1995 to ensure consistency with the NO<sub>2</sub> NAAQS requirements. Then, criteria based on the current standard and the potential health effect benchmark levels were used to identify specific locations for analysis using descriptive statistical analysis of the screened data set. All remaining monitoring data not identified by the selection criteria were grouped into one of two non-specific categories.

---

<sup>3</sup> The historical data (1995-2000) were only evaluated for total number of exceedances, not for daily maximum exceedances as done for the recent air quality data (2001-2006). Because of the differences in benchmarks levels, differences in the number of road-to-monitor categories used, and different exceedance levels, the historical data analyses are presented in Appendix A section A-9.1 to A-9.3, along with the comparable metrics using the recent (2001-2006) air quality.

The locations (both the specific and non-specific) served as the geographic centers of the analysis, where application of the empirical model was done to estimate concentrations and the number of exceedances of potential health effect benchmark levels. Next, due to expected variable influence of road emissions on ambient monitor concentrations, the monitors within each of the named locations were categorized according to particular attributes, including land use characteristics, location type, monitoring objective, monitoring height above ground, and distance to major roadways. In addition to the use of the ambient concentrations (*as is*) and ambient concentrations just meeting the current and alternative standard levels, on-road NO<sub>2</sub> concentrations were estimated in each location to approximate the potential exposure and risk metrics associated with these concentrations. And finally, air quality metrics of interest were calculated using air quality data from each scenario.

### **7.2.1 Air Quality Data Screen**

NO<sub>2</sub> 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 NO<sub>2</sub> 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 ambient monitoring data used in the air quality characterization.

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 NO<sub>2</sub> 1-hour concentration database, 1,039 site-years did not meet the above completeness 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 7-1 provides a summary of the site-years included in the analysis,

relative to those excluded, by location and by two site-year groups.<sup>4</sup> The air quality data from AQS were separated into these two groups, one representing *historical* data (1995-2000) and the other representing *recent* ambient monitoring data (2001-2006) to account for anticipated long-term temporal variability in NO<sub>2</sub> concentrations within each location. The selection of specific locations was a companion analysis to this data screening, and is discussed in section 7.2.2.

**Table 7-1. Counts of complete and incomplete site-years of NO<sub>2</sub> ambient monitoring data.**

Location	Number of Site-Years <sup>1</sup>				Site-Years % Complete	
	Complete		Incomplete		1995-2000	2001-2006
	1995-2000	2001-2006	1995-2000	2001-2006		
Atlanta	24	29	5	1	83%	97%
Boston	58	47	16	34	78%	58%
Chicago	47	36	20	22	70%	62%
Cleveland	11	11	2	2	85%	85%
Colorado Springs	26	ND	4	4	87%	ND
Denver	26	10	10	4	72%	71%
Detroit	12	12	4	1	75%	92%
El Paso	14	30	11	0	56%	100%
Jacksonville	6	4	0	2	100%	67%
Las Vegas	16	35	4	9	80%	80%
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%
Phoenix	22	27	8	25	73%	52%
Provo	6	6	0	0	100%	100%
St. Louis	56	43	3	9	95%	83%
Washington DC	69	66	21	18	77%	79%
Other MSA	1135	1177	249	235	82%	83%
Other Not MSA	200	243	112	141	64%	63%
<b>Total</b>	4177		1066		80%	
<b>Notes:</b>						
<sup>1</sup> The average number of monitors operating per year within the six-year group can be estimated by dividing the number of site-years by 6 (the total would be divided by 12). The actual number of monitors operating in any specific year is variable. See Appendix A-4, Table A-1 as an example.						
ND no available monitoring data.						

### 7.2.2 Selection of Locations for Air Quality Analysis

Criteria were established for selecting monitoring sites with high annual means and/or frequent exceedances of potential health effect benchmarks. Selected locations were those that

<sup>4</sup> At the time data were downloaded from AQS, 14 of 18 named locations and the 2 grouped locations contained enough data to be considered valid for year 2006.

had a maximum annual mean NO<sub>2</sub> level at a particular monitor greater than or equal to 25.7 ppb, which represents the 90<sup>th</sup> percentile across all locations and site-years, and/or had at least one reported 1-hour NO<sub>2</sub> level greater than or equal to 200 ppb, the lowest level of the potential health effect benchmarks.<sup>5</sup> 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 ambient 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 NO<sub>2</sub> air quality data for each site within a location.

Fourteen locations met both selection criteria and an additional four met at least one of the criteria (see Table 7-2).<sup>6</sup> In addition to these 18 specific locations, the remaining sites were grouped into two broad location groupings. The *Other CMSA* location contains all the other sites that are in MSAs or CMSAs but are not in any of the 18 specified locations. The *Other Not MSA* location contains all the sites that are not in an MSA or CMSA. The final database for analysis included air quality data from a total of 204 monitors within the named locations, 331 monitors in the *Other CMSA* group, and 92 monitors in the *Other Not MSA* group.

---

<sup>5</sup> At the time the locations were selected for analysis, the ISA identified 200 ppb as a lower level where health effects were observed in human clinical studies.

<sup>6</sup> 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 this location was not separated out as a specific location for analysis and was grouped within a non-specific category.

**Table 7-2. Locations selected for NO<sub>2</sub> Air Quality Characterization, associated abbreviations, and values of selection criteria.**

Location				Maximum # of Exceedances of 200 ppb	Maximum Annual Mean (ppb)
Type <sup>1</sup>	Code	Description	Abbreviation		
MSA	0520	Atlanta, GA	Atlanta*	1	26.6
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
MSA	1720	Colorado Springs, CO	Colorado Springs*	69	34.8
CMSA	2082	Denver-Boulder-Greeley, CO	Denver*	2	36.8
CMSA	2162	Detroit-Ann Arbor-Flint, MI	Detroit*	12	25.9
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
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
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
CMSA	8872	Washington-Baltimore, DC-MD-VA-WV	Washington DC*	2	27.2
MSA/CMSA	-	Other MSA/CMSA	Other MSA	10	31.9
-	-	Other Not MSA	Other Not MSA	2	19.7

**Notes:**  
<sup>1</sup> 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.

### 7.2.3 Site Characteristics of Ambient NO<sub>2</sub> Monitors

The siting of the ambient monitors is of particular importance, recognizing that the purpose of the monitoring could have an influence on the measured NO<sub>2</sub> concentrations and subsequent interpretation in the air quality characterization. Specific monitoring site characteristics provided in AQS were obtained, including the monitoring objective, measurement

scale, and predominant land-use. Additional features such as proximity to NO<sub>x</sub> emission sources, including mobile and stationary sources, were estimated using each monitoring site and emission source geographic coordinates. Each of these attributes is summarized here to provide perspective on the representation the ambient NO<sub>2</sub> monitoring network within each location. A more thorough discussion of the ambient monitoring network is provided in Chapter 2. Individual monitor site characteristics are given in Appendix A-4.

The monitor objective meta-data field describes the nature of the network in terms of its attempt to generally characterize health effects, photochemical activity, transport, or welfare effects. In recognizing that there were variable numbers of ambient monitors in operation or with valid data in a given year, and that the air quality characterization was performed for particular year-groups of data, the monitoring objectives were weighted by the number of site-years. In addition, the monitors can have more than one objective. To evaluate the representation of the monitors used for the purposes of the REA, the four objective categories were used in the following order (i.e., population exposure, high concentration, general/background, unknown) to characterize the monitor with one objective. All other objectives were grouped into an “Other” category. Tables 7-3 and 7-4 summarize the monitoring objectives for each location using the historical and recent air quality data, respectively. Most locations contained monitoring site-years of data that would target public health through the population exposure and highest concentration categories. Where these categories were not the predominant objective, the monitoring objective was mainly unknown.

**Table 7-3. Percent of ambient NO<sub>2</sub> monitors with selected monitoring objectives, using all valid site-years of historical air quality (1995-2000).**

Location	Population Exposure	High Concentration	General /Background	Unknown	Other
Atlanta	88			13	
Boston	43	21		33	3
Chicago	55	32	11	2	
Cleveland	100				
Colorado Springs				100	
Denver	23	19	27	31	
Detroit	42	42		17	
El Paso	50	14	29		7
Jacksonville				100	
Las Vegas	50	13	13	13	13
Los Angeles	23	9	3	64	2
Miami	50	50			
New York	26	40	2	32	
Philadelphia	85	13			2
Phoenix	73	27			
Provo				100	
St. Louis	21	11		68	
Washington DC	38	19		39	4
Other MSA	45	14	4	32	5
Other Not MSA	22	7	17	35	19

**Table 7-4. Percent of ambient NO<sub>2</sub> monitors with selected monitoring objectives, using all valid site-years of recent air quality (2001-2006).**

Location	Population Exposure	High Concentration	General /Background	Unknown	Other
Atlanta	79			21	
Boston	66	15		15	4
Chicago	53	47			
Cleveland	100				
Denver	50	50			
Detroit	50	50			
El Paso	80	20			
Jacksonville				100	
Las Vegas	54	14	14		17
Los Angeles	20	10	2	64	5
Miami	50	50			
New York	40	23	1	36	
Philadelphia	79	10		10	
Phoenix	67	22		4	7
Provo				100	
St. Louis	28	21		51	
Washington DC	48	17		27	8
Other MSA	45	16	9	23	6
Other Not MSA	22	5	25	21	27

Similarly, the overall measurement scale of the monitors used for the air quality characterization in each location was evaluated based on the valid site-years of air quality data. The measurement scale represents the air volumes associated with the monitoring area dimensions. While a monitor can have multiple objectives, each monitor has only one measurement scale. Tables 7-5 and 7-6 summarize the measurement scales of the monitors in each location using the historical and recent air quality data, respectively. Most locations contained monitoring site-years of data with measurement scales of urban (4 to 50 km) or neighborhood (500 m to 4 km). Where these categories were not the predominant objective, the measurement scale was commonly not indicated.

The land use meta-data indicate the prevalent land use within ¼ mile of the monitoring site. Tables 7-7 and 7-8 summarize the land use surrounding the monitors in each location using the historical and recent air quality data, respectively. Most locations contained monitoring site-years of data from areas within residential and commercial areas, but were generally dominated by residential land use. Two locations however were characterized with site-years of data associated entirely with commercial land use (i.e., Jacksonville and Provo).

**Table 7-5. Percent of ambient NO<sub>2</sub> monitors with selected measurement scales, using all valid site-years of historical air quality (1995-2000).**

Location	Regional	Urban	Neighborhood	Middle	Micro	None
Atlanta		63	38			
Boston	3	26	28		12	31
Chicago	11	6	55	26		2
Cleveland		45	55			
Colorado Springs						100
Denver		23	65			12
Detroit		42	42			17
El Paso		29	50		7	14
Jacksonville						100
Las Vegas		13	75			13
Los Angeles		17	14	14		55
Miami		38	63			
New York		6	53	6		34
Philadelphia		13	74			13
Phoenix		23	55	14		9
Provo						100
St. Louis		11	21			68
Washington DC	1	26	42			30
Other MSA	4	21	42	2	1	30
Other Not MSA	8	22	26			45

**Table 7-6. Percent of ambient NO<sub>2</sub> monitors with selected measurement scales, using all valid site-years of recent air quality (2001-2006).**

Location	Regional	Urban	Neighborhood	Middle	Micro	None
Atlanta		59	41			
Boston	4	23	57		11	4
Chicago		14	53	33		
Cleveland		27	73			
Denver		50	50			
Detroit		50	50			
El Paso			60			40
Jacksonville						100
Las Vegas	3	14	83			
Los Angeles		18	15	11		56
Miami		50	50			
New York		6	46	5		43
Philadelphia		13	77			10
Phoenix		26	37	22		15
Provo						100
St. Louis		14	35			51
Washington DC		27	48			24
Other MSA	4	22	44	4	1	25
Other Not MSA	19	20	27	1		33

**Table 7-7. Percent of ambient NO<sub>2</sub> monitors with selected land use, using all valid site-years of historical air quality (1995-2000).**

Location	Residential	Commercial	Industrial	Mobile	Agricultural	Other
Atlanta	33	25			42	
Boston	41	45	10		3	
Chicago	57		6	26	11	
Cleveland	45	55				
Colorado Springs	54	15	27		4	
Denver		19	38		42	
Detroit	83	17				
El Paso	14	86				
Jacksonville		100				
Las Vegas	27	33	13	20		7
Los Angeles	55	32	4	5	3	1
Miami	63	25	13			
New York	36	36	13		13	1
Philadelphia	72	13	13		2	
Phoenix	100					
Provo		100				
St. Louis	41	32	11		16	
Washington DC	57	36			7	
Other MSA	42	33	7	4	14	
Other Not MSA	21	20	15	7	35	2

**Table 7-8. Percent of ambient NO<sub>2</sub> monitors with selected land use, using all valid site-years of recent air quality (2001-2006).**

Location	Residential	Commercial	Industrial	Mobile	Agricultural	Other
Atlanta	38	21			41	
Boston	28	62	11			
Chicago	53		14	33		
Cleveland	45	55				
Denver		50			50	
Detroit	100					
El Paso	40	60				
Jacksonville		100				
Las Vegas	40	23	17	17		3
Los Angeles	58	34	3	3	1	1
Miami	75	25				
New York	43	35	8		13	1
Philadelphia	64	21	15			
Phoenix	93				7	
Provo		100				
St. Louis	42	30	14		14	
Washington DC	64	27			9	
Other MSA	42	34	8	2	13	
Other Not MSA	23	13	14	9	39	1

Mobile and stationary sources (i.e., primarily power generating utilities using fossil fuels) are the most significant contributors to nitrogen oxides (NO<sub>x</sub>) emissions in the U.S. (ISA, section 2.2.1). Therefore, the distances of each ambient monitor in the named locations to major roads and stationary sources were calculated.<sup>7</sup> The estimated distances of the monitors to major roads ranged from a few meters to several hundred meters (Table 7-9). On average, most of the ambient monitors are placed at a distance of 100 meters or greater from a major road, however in locations with a large monitoring network such as Boston, Chicago, or New York, there were a few monitors sited within close proximity (<20 meters) of a road. Most of the monitors were sited at much greater distances to NO<sub>x</sub> stationary sources than major roads. In general, monitors were located at least 1 km from NO<sub>x</sub> stationary sources, with over half of the monitors located at distances > 5 km.

Because there is potential for roadway emissions to affect concentrations at monitors sited close to major roads, each of the ambient monitors were further categorized based on the monitor distance from major roads. Three categories were identified, the first containing those

<sup>7</sup> Major road types were defined as: primary limited access or interstate, primary US and State highways, secondary State and County, freeway ramp, or other ramps. Distances were estimated for stationary sources within 10 km having emissions greater than 5 tons per year (tpy). See Appendix A-4 for details for the approach used.

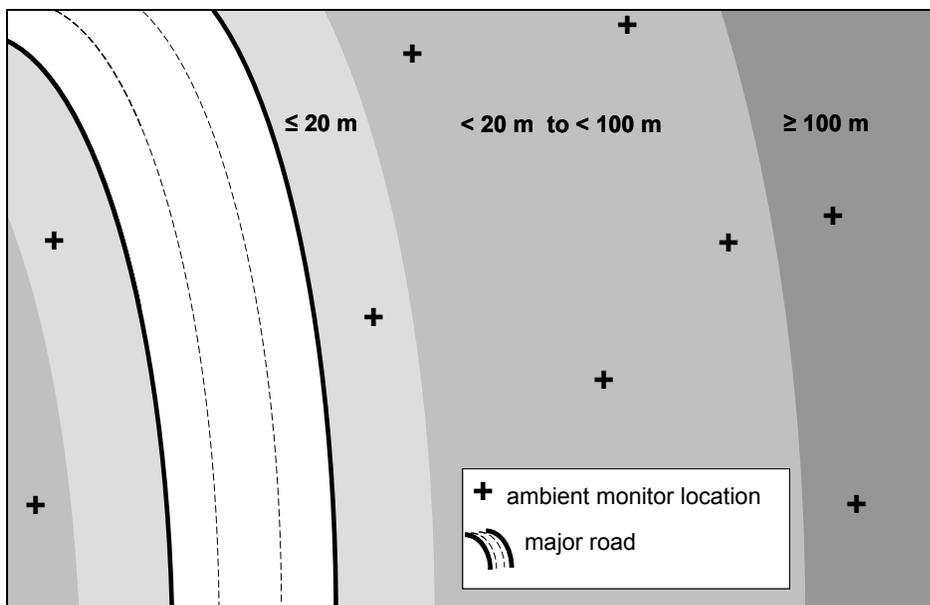
monitors sited at or within 20 meters, ( $\leq 20$  m), those between 20 meter and 100 meters ( $>20$  m,  $<100$  m), and those located at least 100 meters from a major road ( $\geq 100$  m) (Figure 7-1).

**Table 7-9. Distance of ambient monitors to the nearest major sources in selected locations.**

Location	n <sup>1</sup>	Distance (m) of monitors to nearest major road			Distance (m) of monitor to nearest 5 ton per year (tpy) NO <sub>x</sub> stationary source		
		min	med	max	min	med	max
Atlanta	4	134	505	809	656	7327	9847
Boston	21	7	70	337	142	5363	9988
Chicago	12	2	93	738	411	7277	9994
Cleveland	4	2	134	187	956	7278	9884
Colorado Springs	6	79	180	386	782	6340	9933
Denver	7	18	65	748	910	5904	9979
Detroit	3	339	393	415	321	7549	9997
El Paso	7	33	128	718	119	6085	9991
Jacksonville	1	144			708	5720	9558
Las Vegas	10	1	181	914	3837	7237	9950
Los Angeles	43	1	89	570	140	6165	9991
Miami	4	15	55	103	1323	7611	9117
New York	26	6	119	508	103	6467	9983
Philadelphia	10	45	167	630	231	5689	9982
Phoenix	7	7	141	433	833	6355	9890
Provo	1	353			1214	8178	9433
St. Louis	13	5	97	421	396	7120	9990
Washington DC	16	14	83	338	288	6254	9973

**Notes:**

<sup>1</sup> n is the number of monitors operating in a particular location between 1995 and 2006. The min, med, and max represent the minimum, median, and maximum percentiles of the distribution for the distance in meters (m) of the monitor to the nearby sources. Monitors >1km from a major road and monitors having no stationary sources within 10 km are not included in this distribution. Individual monitor distances and stationary source emissions within 10 km is provided in Appendix A-4.



**Figure 7-1. Illustration of three roadway distance categories used to characterize ambient monitors in the Air Quality Characterization.**

#### 7.2.4 Estimation of On-Road Concentrations using Ambient Concentrations

Since mobile sources can account for a large part of personal exposures to ambient NO<sub>2</sub> in some individuals, the potential impact of roadway levels of NO<sub>2</sub> was evaluated. A strong relationship has been reported between NO<sub>2</sub> levels measured on roadways and NO<sub>2</sub> measured at increasing distance from the road. This relationship has been described previously (e.g., Cape et al., 2004) using an exponential decay equation of the form:

$$C_x = C_b + C_v e^{-kx} \quad \text{equation (7-1)}$$

where,

- $C_x$  = NO<sub>2</sub> concentration at a given distance ( $x$ ) from a roadway (ppb)
- $C_b$  = NO<sub>2</sub> concentration (ppb) at a distance from a roadway, not directly influenced by road or non-road source emissions
- $C_v$  = NO<sub>2</sub> concentration contribution from vehicles on a roadway (ppb)
- $k$  = Combined formation/decay constant describing NO<sub>2</sub> with perpendicular distance from roadway (meters<sup>-1</sup>)
- $x$  = Distance from roadway (meters)

Based on the findings of several researchers, much of the decline in NO<sub>2</sub> concentrations with distance from the road has been shown to occur within the first few meters (approximately 90% within a 10 meter distance), returning to near ambient levels between 200 to 500 meters (Rodes and Holland, 1981; Bell and Ashenden, 1997; Gilbert et al., 2003; Pleijel et al., 2004). At a distance of 0 meter, referred to here as *on-road*, the equation reduces to the sum of the non-source influenced NO<sub>2</sub> concentration and the concentration contribution expected from vehicle emissions on the roadway using

$$C_r = C_a (1 + m) \quad \text{equation (7-2)}$$

where,

- $C_r$  = 1-hour on-road NO<sub>2</sub> concentration (ppb)
- $C_a$  = 1-hour ambient monitoring NO<sub>2</sub> concentration (ppb) either *as is* or adjusted to just meet the current or alternative standards
- $m$  = Ratio derived from estimates of  $C_v/C_b$  (from equation (7-1))

and assuming that  $C_a = C_b$ .<sup>8</sup>

To estimate on-road NO<sub>2</sub> levels as a function of the level recorded at ambient monitors at a distance from a roadway, empirical data from published scientific literature were used. A literature review was conducted to identify published studies containing NO<sub>2</sub> concentrations on roadways and at varying distances from roadways. Relevant data identified from this literature review were used to estimate the ratio ( $m$ ) of the on-road vehicle concentrations ( $C_v$ ) to NO<sub>2</sub> concentration at a distance from a roadway ( $C_b$ ) (equation 7-1), generating a distribution of values for use in estimating on-road concentrations from the ambient monitor concentrations (Table 7-10). The distribution of derived  $m$  ratios were evaluated for possible stratification using potential influential factors reported in the collection of studies, the number of values of  $m$  available for potential groupings, and how the data were to be applied to the ambient monitoring

---

<sup>8</sup> Note that  $C_a$  may differ from  $C_b$  since  $C_a$  could include the influence of on-road as well as non-road sources. However, it is expected that for most monitors sited at a distance of  $\geq 100$  from a major road, the influence of on-road emissions would be negligible so that  $C_a \cong C_b$ .

data. In general, categorizing the data based on *summer* and *not summer* seasons were determined appropriate, containing 21 and 19 samples, respectively. Then, on-road NO<sub>2</sub> concentrations are estimated by probabilistically applying the distribution of adjustment factors (1+m) to the ambient monitor concentrations used in this study that were sited at distances ≥ 100 m of a major road. See Appendix A, section 8 for a detailed explanation of the *m* ratio derivation and the literature sources used.

**Table 7-10. Derived  $C_v/C_b$  ratios (*m*) for two season groups used for adjusting ambient NO<sub>2</sub> concentrations to simulate on-road NO<sub>2</sub> concentrations.**

<b><math>C_v/C_b</math> ratios (<i>m</i>)</b>	
<b>Summer</b>	<b>Not Summer</b>
0.49	0.22
0.51	0.25
0.52	0.36
0.67	0.36
0.70	0.42
0.74	0.47
0.75	0.58
0.78	0.59
0.78	0.64
0.79	0.75
0.90	0.78
0.92	0.79
0.93	0.79
0.94	0.82
1.13	0.86
1.19	1.08
1.21	1.14
1.32	1.50
1.95	1.54
2.43	
2.45	
2.70	

Theoretically, NO<sub>2</sub> concentrations can increase at a distance from the road due to chemical interaction of NO<sub>x</sub> with O<sub>3</sub>, the magnitude of which can be driven by certain meteorological conditions (e.g., wind direction). As such, the maximum NO<sub>2</sub> concentration may not occur on the road but at a distance from the road. However, there are two important components of this estimation procedure that need consideration. First, the relationship developed from the peer-reviewed NO<sub>2</sub> roadway and near-road measurement studies was used to

estimate NO<sub>2</sub> concentrations that occur on the road and not used to estimate NO<sub>2</sub> concentrations that could occur at a distance from the road. If there are peak concentrations at a given distance from a roadway that occur frequently in a location, the ambient monitors located within 20 m or 100 m of a road would capture these concentrations, where such monitors are available in a location. Second, since there is potential for monitors that are sited near roadways to be influenced by vehicle emissions and equation (7-2) assumes the ambient concentration is approximating NO<sub>2</sub> concentrations not directly influenced by the roadway, the ambient monitors <100 m were not used for estimating the on-road NO<sub>2</sub> concentrations in this analysis. The uncertainty regarding any additional issues or assumptions and the potential effect on exposure estimates are discussed in section 7.4.

To estimate on-road NO<sub>2</sub> concentrations, each monitoring site was randomly assigned one on-road adjustment factor ( $1+m$ ) for summer months and one for non-summer months from the derived empirical distribution. On-road adjustment factors were assigned randomly because we expect the empirical relationship between  $C_v$  and  $C_b$  to vary from place to place and we do not have sufficient information to match specific ratios with any of the locations simulated in this assessment. Hourly NO<sub>2</sub> levels were estimated for each site-year of data in a location using equation (7-2) and the randomly assigned on-road adjustment factors. The process was simulated 100 times for each site-year of hourly data. For example, the Boston CMSA location had 210 random selections from the on-road distributions applied independently to the total site-years of data (105). Following 100 simulations, a total of 10,500 site-years of data were generated using this procedure (along with 21,000 randomly assigned on-road values selected from the appropriate empirical distribution).

### **7.2.5 Air Quality Concentration Metrics**

For each of the four air quality characterization scenarios considered, two concentration metrics were calculated, including the annual average NO<sub>2</sub> concentrations for each site-year and the number of exceedances of the potential health effect benchmark levels. 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 NO<sub>2</sub> concentrations, was chosen to avoid some

of the difficulties in extrapolating outside the range of the measurement data. In addition, an empirical model could be used for any averaging time of interest. A detailed discussion justifying the selection of the empirical approach rather than using a regression approach is provided in Appendix A, section 6.

Using Figure 7-1 as an illustrative example of a location, assume that for a recent air quality monitoring year-group (2001-2003) there are an equal number of valid monitoring years (i.e., 3 years in this example) at each monitoring site indicated in the figure. In total there would be 27 site-years of data (9 monitors by 3 years) and, when separated into the major road categories, there would be 6, 15, and 6 site-years of ambient concentration data at  $\leq 20$  m,  $< 20$  and  $< 100$  m,  $\geq 100$  m from a major road, respectively. In the first scenario, the air quality is analyzed without adjustment, giving mean annual average concentrations based on averaging the 6, 15, and 6 annual average NO<sub>2</sub> concentrations within each respective monitor-to-road distance category. Median annual average concentrations are also provided to represent the central tendency and are the median concentration values of the 6, 15, and 6 site-years of data. Due to the limited number of site-years of data for each of the road distance categories, the 98<sup>th</sup> and 99<sup>th</sup> percentile values are essentially the same, i.e., the maximum site-year annual average concentration. The same approach was used for the counts of exceedances in a year, reporting each the central tendency values and the lower and upper percentiles for each location.

In the second scenario, air quality metrics for the estimated on-road NO<sub>2</sub> concentrations were generated in a similar manner. The concentration distributions for the annual average concentrations and the distributions for the number of exceedances of short-term potential health effect benchmark levels were calculated for each location and year-group. In using the hypothetical location described above (Figure 7-1) for years 2001-2003, 600 site-years of hourly on-road concentrations were simulated (6 site-years by 100 simulations each). Mean (average of the 600 site-year values) and median (the average of the 300<sup>th</sup> and 301<sup>st</sup> site-year values) are reported to represent the central tendency of each air quality metric. Since there were multiple site-years and numerous simulations performed at each location using all valid site-years of data, results for the upper percentiles also included the 98<sup>th</sup> and 99<sup>th</sup> percentiles of the distribution. The 98<sup>th</sup> and 99<sup>th</sup> percentiles were the 588<sup>th</sup> and the 594<sup>th</sup> highest site-year values, respectively, of the 600 calculated and ranked values. Roadways with high vehicle densities are likely better represented by on-road concentration estimates at these upper tails of the distribution.

## 7.3 AIR QUALITY AND HEALTH RISK CHARACTERIZATION RESULTS

### 7.3.1 Ambient Air Quality (As Is)

As described earlier, this first scenario analyzing the *as is* air quality is based purely on the measurement data. The air quality data obtained from AQS were first separated into two six-year groups, one representing historical data (1995-2000) and the other representing more recent data (2001-2006). The two broad six-year groups of data were compared using each location's distribution of annual average concentrations and the *total number* of 1-hour exceedances of the potential health effect benchmark levels. Briefly, annual average concentrations were about 15% higher for the historical data when compared with the more recent data using corresponding locations. In general, concentrations were about 20-25% higher at monitors sited <100 m from a major road considering either six-year group of data. While the exceedances of potential health effect benchmark levels were limited to a few locations, in general, a greater number of exceedances were observed using the historical data set and at locations sited <100 m from a major road when compared with the recent air quality. Detailed descriptive statistics regarding concentration distributions for all locations, monitoring sites, and all monitoring years are provided in the Appendix A, section 5.

Detailed analyses were performed using the recent *as is* ambient monitoring data. As described in section 6.2.1, to remain consistent with the planned analysis of alternative standards (sections 7.3.3 and 7.3.4), the recent ambient monitoring data were separated into two three-year groups, 2001-2003 and 2004-2006. A summary of the descriptive statistics for the annual average ambient NO<sub>2</sub> concentrations at each selected location for years 2001-2003 and 2004-2006 are provided in Tables 7-11, 7-12 and 7-13 for monitors sited ≥100 m, 20 m to <100 m, and ≤20 m from a major road, respectively. None of the locations contained a measured exceedance of the current annual average standard of 0.053 ppm at any monitor, although Los Angeles and New York had at least one annual average concentration ≥40 ppb during 2001-2003. There were a fewer number of locations with monitors sited <100 m of a major road, however in most of the locations where comparative monitoring data were available, the annual average concentrations were greater at the monitors within 100 m of a major road (in 33 of 42 possible location/year-group combinations) when compared with monitors ≥ 100 m of a major road. Annual average

concentrations measured at monitors located  $\leq 20$  m from a major road were also frequently greater than those measured at monitors sited  $< 20$  m and  $> 100$  m for most location/year combinations (68%). Where concentrations were greater at the monitors  $< 100$  m from a major road, the concentrations were on average about 40-65% higher when compared with the more distant monitors in each corresponding location. A comparison of the three-year groups of data within each monitor site-group indicates that the more recent monitoring concentrations (i.e., 2004-2006) were frequently lower, on average by about 13-19%. These average trends in concentration across year-group and monitor site group were generally observed across all percentiles of the distribution.

**Table 7-11. Monitoring site-years and annual average NO<sub>2</sub> concentrations, using recent air quality data (*as is*) and monitors sited  $\geq 100$  m of a major road.**

Location	2001-2003							2004-2006						
	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>						Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					
		mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99
Atlanta	14	12	4	15	23	23	23	15	11	3	14	18	18	18
Boston	6	10	5	11	12	12	12	8	9	7	9	10	10	10
Chicago	9	22	17	20	28	28	28	8	19	16	18	24	24	24
Cleveland	3	18	17	17	19	19	19	ND						
Denver	2	24	21	24	26	26	26	3	20	18	20	21	21	21
Detroit	6	21	19	20	23	23	23	6	17	14	17	20	20	20
El Paso	12	15	10	16	18	18	18	12	14	8	15	18	18	18
Jacksonville	2	14	14	14	15	15	15	2	14	13	14	14	14	14
Las Vegas	16	10	2	7	22	22	22	11	9	1	6	20	20	20
Los Angeles	51	22	5	24	34	36	37	54	18	5	18	27	31	31
Miami	6	9	7	9	10	10	10	4	8	7	8	8	8	8
New York	26	20	11	18	28	31	31	22	19	10	20	25	27	27
Philadelphia	14	20	15	18	28	28	28	12	17	14	16	25	25	25
Phoenix	5	27	22	29	29	29	29	9	24	21	24	26	26	26
Provo	3	24	22	24	25	25	25	3	24	21	22	29	29	29
St. Louis	9	17	14	17	21	21	21	4	15	12	14	18	18	18
Washington DC	18	18	9	21	25	25	25	17	15	7	16	22	22	22
Other MSA	612	13	1	13	21	22	24	565	11	1	11	19	21	23
Other Not MSA	127	7	1	6	14	15	16	116	7	1	6	15	16	16

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group can be estimated by dividing the number of site-years by 3.

<sup>2</sup> Annual means for each monitor were first calculated based on all hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means 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, 95<sup>th</sup>, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual means in the selected three-year group.

ND no available monitoring data.

**Table 7-12. Monitoring site-years and annual average NO<sub>2</sub> concentrations, using recent air quality data (*as is*) and monitors sited >20 m and <100 m of a major road.**

Location	2001-2003							2004-2006						
	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>						Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					
		mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99
Atlanta	ND							ND						
Boston	14	17	9	19	25	25	25	11	15	10	16	19	19	19
Chicago	6	31	28	31	32	32	32	6	29	28	29	31	31	31
Cleveland	ND							2	15	14	15	17	17	17
Denver	ND							ND						
Detroit	ND							ND						
El Paso	3	21	20	21	22	22	22	3	15	13	13	18	18	18
Jacksonville	ND							ND						
Las Vegas	3	6	3	6	9	9	9	ND						
Los Angeles	35	24	4	24	40	41	41	22	25	9	27	33	34	34
Miami	3	14	13	14	16	16	16	2	13	13	13	14	14	14
New York	13	31	21	30	40	40	40	11	28	18	29	36	36	36
Philadelphia	7	24	19	24	30	30	30	6	22	18	22	26	26	26
Phoenix	2	23	22	23	24	24	24	3	19	19	19	20	20	20
Provo	ND							ND						
St. Louis	11	14	9	12	25	25	25	8	12	8	10	22	22	22
Washington DC	10	20	14	22	26	26	26	12	18	13	18	24	24	24
Other MSA	ND							ND						
Other Not MSA	ND							ND						

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group can be estimated by dividing the number of site-years by 3.

<sup>2</sup> Annual means for each monitor were first calculated based on all hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means 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, 95<sup>th</sup>, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual means.

ND no available monitoring data.

**Table 7-13. Monitoring site-years and annual average NO<sub>2</sub> concentrations, using recent air quality data (*as is*) and monitors sited ≤20 m of a major road.**

Location	2001-2003							2004-2006						
	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>						Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					
		mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99
Atlanta	ND							ND						
Boston	5	21	7	23	30	30	30	3	24	23	23	25	25	25
Chicago	4	22	22	22	24	24	24	3	19	18	20	20	20	20
Cleveland	3	23	22	22	24	24	24	3	21	18	22	22	22	22
Denver	2	36	35	36	37	37	37	3	28	27	28	29	29	29
Detroit	ND							ND						
El Paso	ND							ND						
Jacksonville	ND							ND						
Las Vegas	3	22	21	22	23	23	23	2	19	19	19	20	20	20
Los Angeles	9	30	23	29	37	37	37	6	27	20	29	31	31	31
Miami	3	6	6	6	7	7	7	2	6	6	6	6	6	6
New York	7	28	25	28	30	30	30	2	28	27	28	28	28	28
Philadelphia	ND							ND						
Phoenix	3	35	34	35	37	37	37	5	23	11	31	32	32	32
Provo	ND							ND						
St. Louis	6	18	16	19	20	20	20	5	16	15	16	17	17	17
Washington DC	4	23	20	24	26	26	26	5	19	14	18	23	23	23
Other MSA	ND							ND						
Other Not MSA	ND							ND						

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group can be estimated by dividing the number of site-years by 3.

<sup>2</sup> Annual means for each monitor were first calculated based on all hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means 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, 95<sup>th</sup>, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual means.

ND no available monitoring data.

The number of daily maximum exceedances of the potential health effect benchmark levels (100, 150, 200, 250, and 300 ppb NO<sub>2</sub> for 1-hour) is shown in Tables 7-14 through 7-19 using recent *as is* ambient monitoring data and considering the three road distance categories. As a reminder, these exceedance data are based on whether the daily maximum concentration at a monitor was above the benchmark level; a single monitor value of 10 would represent ten days in the year where there was at least one 1-hour exceedance of the benchmark level. Since there are multiple monitors and monitoring years in a location, a distribution of these values can be generated. The mean represents the central tendency value for each location; it is the average number of days per year that there were daily maximum 1-hour NO<sub>2</sub> concentration exceedances observed over the three year period in a location. The upper percentiles (98<sup>th</sup> and 99<sup>th</sup>) represent

an upper-level estimate of the number of days in one year there were daily maximum 1-hour concentration exceedances at a particular monitor (or possibly more than one) in a location.

In general, the number of daily maximum 1-hour benchmark exceedances was low across all locations and considering both three-year groups of the *as is* air quality and monitors located  $\geq 100$  m from a major road (Tables 7-14 and 7-15). The average number of exceedances of the 100 ppb 1-hour NO<sub>2</sub> concentration across each location was typically zero to a few, with one location (Provo) containing an average of fourteen exceedances. Considering that there are 365 days in a year, this many exceedances amounts to a small fraction of the year (at most 4%) with an exceedance of the lowest potential health effect benchmark level at a few locations. For the locations with greater than one annual average exceedance, the numbers were primarily driven by a single site-year of data. For example, Detroit contained a largest number of exceedances of 200 ppb (a maximum of 4 days in the year) for *as is* air quality data from years 2001-2003 (Table 7-14). All of these exceedances occurred at one monitor (ID 2616300192) during one year (2002). Provo contained that greatest number of daily maximum exceedances of both the 100 and 150 ppb benchmark level (Table 7-15), associated with measurements from one monitor (ID 4904900021) in 2006. However, when considering the collective locations (both the named locations and aggregated), daily maximum exceedances of 150 and above were rarely occurred.

When considering monitors sited  $< 20$  m and  $> 100$  m of a major road (Table 7-16 and 7-17), only a few locations contained exceedances of the potential health effect benchmark levels, driven mainly by observations from one or two monitors. For example, in Los Angeles a single year (2001) for two monitors (IDs 060370030 and 060371103) were responsible for many of the observed exceedances of 100 ppb in the 2001-2003 year-group (each had 31 exceedances in a year). Each of these monitors are located about 50 m from a major road along with around 40 stationary sources located within 10 km of this monitor, over half of which contained estimated emissions of less than 15 tons per year (tpy) (Appendix A, Table A-8). When considering the higher benchmark levels, nearly all locations had no daily maximum 1-hour NO<sub>2</sub> concentrations  $\geq 150$  ppb.

At monitoring locations  $\leq 20$  m to major roads, about half of the locations contained a non-zero average number of daily maximum exceedances of 100 ppb (Tables 7-18 and 7-19). Of those, Denver, Los Angeles, and Phoenix contained the highest average number of exceedances given either the 2001-2003 or 2004-2006 year-groups. On average, the percent of days in a year

with a daily maximum exceedance at these locations ranged from about 1-2%. As far as the higher benchmark levels, nearly all locations had no daily maximum 1-hour NO<sub>2</sub> concentrations  $\geq 150$  ppb.

**Table 7-14. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO<sub>2</sub> air quality (*as is*) and monitors sited ≥100 m of a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>									
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99					
Atlanta	0	0	0	3	3	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Boston	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	0	0	0	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Denver	2	1	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Detroit	3	0	2	7	7	1	0	1	5	5	1	0	0	4	4	1	0	0	4	4	0	0	0	2	2	0	0	0	0	0
El Paso	0	0	0	1	1	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	1	0	1	1	1	1	0	1	1	1	1	0	1	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0
Las Vegas	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Los Angeles	4	0	1	17	18	0	0	0	1	8	0	0	0	0	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miami	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New York	0	0	0	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	0	0	0	1	1	0	0	0	1	1	0	0	0	1	1	0	0	0	1	1	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	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	0	0	0	0	0	0	0	0	0	0	0	0
St. Louis	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA	0	0	0	1	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other Not MSA	0	0	0	5	5	0	0	0	1	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

**Notes:**

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

**Table 7-15. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO<sub>2</sub> air quality (*as is*) and monitors sited ≥100 m of a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>									
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99					
Atlanta	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Boston	0	0	0	0	0	0	0	0	0	0	0	0	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	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	ND																													
Denver	1	0	0	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Detroit	0	0	0	0	0	0	0	0	0	0	0	0	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	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	2	0	2	3	3	1	0	1	2	2	1	0	1	1	1	0	0	0	0	0	0	0	0	0	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	0	0	0	0	0	0	0	0	0	0	0	0
Los Angeles	0	0	0	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miami	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New York	0	0	0	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Phoenix	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Provo	14	0	0	43	43	7	0	0	20	20	0	0	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	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other Not MSA	0	0	0	2	3	0	0	0	0	2	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0

**Notes:**  
<sup>1</sup> The mean number of exceedances represents the sum of the daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-16. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO<sub>2</sub> air quality (*as is*) and monitors sited >20 m and <100 m of a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	ND																								
Boston	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	2	0	1	6	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	ND																								
Denver	ND																								
Detroit	ND																								
El Paso	2	0	2	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	ND																								
Las Vegas	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Los Angeles	6	0	1	31	31	0	0	0	2	2	0	0	0	1	1	0	0	0	1	1	0	0	0	0	0
Miami	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New York	1	0	0	8	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	0	0	0	1	1	0	0	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	0	0	0	0	0	0	0
Provo	ND																								
St. Louis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA	ND																								
Other Not MSA	ND																								

**Notes:**  
<sup>1</sup> The mean number of daily maximum exceedances represents the sum of the daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-17. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO<sub>2</sub> air quality (*as is*) and monitors sited >20 m and <100 m of a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	ND																								
Boston	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	1	0	1	5	5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cleveland	1	0	1	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Denver	ND																								
Detroit	ND																								
El Paso	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Jacksonville	ND																								
Las Vegas	ND																								
Los Angeles	1	0	1	9	9	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miami	1	0	1	2	2	1	0	1	1	1	1	0	1	1	1	1	0	1	1	1	1	0	1	1	1
New York	1	0	1	3	3	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	1	0	1	1	1	0	0	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	0	0	0	0	0	0	0
Provo	ND																								
St. Louis	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA	ND																								
Other Not MSA	ND																								

**Notes:**  
<sup>1</sup> The mean number of exceedances represents the sum of the daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-18. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 recent NO<sub>2</sub> air quality (*as is*) and monitors sited ≤20 m from a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	ND																								
Boston	0	0	0	1	1	0	0	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	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	0	0	0	0	0	0	0
Denver	7	3	7	10	10	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Detroit	ND																								
El Paso	ND																								
Jacksonville	ND																								
Las Vegas	1	0	0	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Los Angeles	6	0	6	9	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miami	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New York	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	ND																								
Phoenix	2	1	1	4	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Provo	ND																								
St. Louis	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA	ND																								
Other Not MSA	ND																								

**Notes:**  
<sup>1</sup> The mean number of exceedances represents the sum of the daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-19. Number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2004-2006 recent NO<sub>2</sub> air quality (*as is*) and monitors sited ≤20 m from a major road.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	ND																								
Boston	0	0	0	0	0	0	0	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	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	0	0	0	0	0	0	0
Denver	2	1	1	3	3	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Detroit	ND																								
El Paso	ND																								
Jacksonville	ND																								
Las Vegas	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Los Angeles	3	0	2	8	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Miami	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New York	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Philadelphia	ND																								
Phoenix	2	0	2	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Provo	ND																								
St. Louis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Washington DC	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other MSA/CMSA	ND																								
Other Not MSA	ND																								

**Notes:**  
<sup>1</sup> The mean number of exceedances represents the sum of the daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

### 7.3.2 On-Road Concentrations Derived From Ambient Air Quality (As Is)

Descriptive statistics for estimated on-road NO<sub>2</sub> concentrations for each recent three-year group are presented in Table 7-20. These estimated on-road concentrations were generated using the simulation procedure described above (section 7.2.3) and represent the second air quality scenario. For the 17 named locations, the calculation only used monitors sited at a distance  $\geq 100$  m of a major road. The two aggregate locations (i.e., *Other CMSA* and *Not MSA*) did not have estimated monitor distances to major roads therefore all monitoring data available were used to estimate the distribution of on-road NO<sub>2</sub> concentrations.

The simulated on-road annual average NO<sub>2</sub> concentrations are, on average, 80% higher than the respective ambient levels at distances  $\geq 100$  m from a road. This falls within the range of on-road to distant monitor concentration ratios reported in the ISA (about 2-fold higher concentrations on-roads) (ISA, section 2.5.4). Denver, Los Angeles, and Phoenix were predicted to have the highest on-road annual average NO<sub>2</sub> levels. This is a direct result of these locations already containing some of the highest *as-is* NO<sub>2</sub> concentrations prior to the on-road simulation (see Table 7-11). Estimated on-road annual average concentrations were greater by about 10% when using the 2001-2003 monitoring data compared with the 2004-2006 monitoring data.

The median of the simulated concentration estimates for Los Angeles were compared with NO<sub>2</sub> measurements provided by Westerdahl et al. (2005) for arterial roads and freeways in the same general location during spring 2003. Although the averaging time is not exactly the same, comparison of the medians is judged to be appropriate.<sup>9</sup> The estimated median on-road concentration for 2001-2003 is 41 ppb which falls within the range of 31 ppb to 55 ppb identified by Westerdahl et al. (2005).

On average, most locations are predicted to have fewer than 3 daily maximum exceedances of 1-hour NO<sub>2</sub> concentrations per year given the 200 ppb potential health effect benchmark, while the median frequency of exceedances in most locations is estimated to be 1 or less per year (Tables 7-21 and 7-22). When considering the lowest 1-hour benchmark of 100 ppb, most locations (30 out of 38 location/year-groups) were estimated to have fewer than 50 daily maximum exceedances per year, on average. There are generally fewer predicted mean

---

<sup>9</sup> Table 7-12 considers annual average of hourly measurements, while Westerdahl et al. (2005) reported time-averaged concentrations of 2-4 hours. 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).

exceedances of the potential health effect benchmark levels when considering the 2004-2006 recent air quality compared with the 2001-2003 air quality. Areas with a relatively high number of estimated exceedances (e.g., Provo, as described in section 7.3.1) are likely influenced by the presence of a small number of monitors and one or a few exceptional site-years where there were unusually high concentrations at the upper percentiles of the concentration distribution. When considering higher benchmark levels (i.e., 250 ppb), the mean number of estimated daily maximum exceedances was none or one for most locations.

The upper percentiles (98<sup>th</sup> and 99<sup>th</sup>) for estimated number of daily maximum exceedances of 100 ppb in most locations were under 200 per year, outside of Denver, Los Angeles, Phoenix, and Provo. In general, there were fewer estimated exceedances using the 2001-2003 air quality compared with the 2004-2006 data. Most locations had >100 estimated daily maximum 1-hour exceedances of 150 ppb per year at the 98<sup>th</sup> and 99<sup>th</sup> percentiles and as expected, the frequency of benchmark exceedances at all locations was lower when considering any of the higher benchmark levels (i.e., 200, 250, 300 ppb, 1-hour average) compared with the lower benchmarks. For example, it is estimated that between about 10 and 30 daily maximum exceedances of 200 ppb could occur in a year in most locations as an upper bound estimate. The estimated upper percentile number of exceedances of 250 and 300 ppb 1-hour were generally less than 10 in most locations.

**Table 7-20. Estimated annual average NO<sub>2</sub> concentrations on-roads, using recent air quality data (as is) and an on-road adjustment factor.**

Location	2001-2003							2004-2006						
	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>						Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					
		mean	min	med	p95	p98	p99		mean	min	med	p95	p98	p99
Atlanta	1400	22	5	24	41	47	53	1500	20	4	22	36	40	42
Boston	600	17	7	18	27	29	30	800	16	9	15	22	24	24
Chicago	900	39	21	37	60	65	68	800	35	20	33	51	57	60
Cleveland	300	32	22	32	42	43	45	ND						
Denver	200	42	27	40	61	63	64	300	36	23	36	50	51	53
Detroit	600	37	24	36	51	54	57	600	31	18	30	43	45	47
El Paso	1200	27	13	27	40	43	44	1200	25	10	25	38	42	43
Jacksonville	200	26	18	26	34	36	37	200	24	17	23	33	36	37
Las Vegas	1600	19	3	14	44	48	51	1100	16	2	11	40	44	46
Los Angeles	5100	41	6	40	69	77	82	5400	33	6	32	55	60	65
Miami	600	16	9	15	23	24	25	400	14	9	13	19	19	20
New York	2600	36	14	34	58	65	73	2200	35	12	35	54	58	61
Philadelphia	1400	36	18	33	56	64	66	1200	31	18	30	45	51	59
Phoenix	500	49	28	47	70	72	77	900	43	26	42	59	64	65
Provo	300	43	28	41	58	61	64	300	43	26	41	68	70	71
St. Louis	900	31	18	30	43	48	50	400	27	16	26	38	41	42
Washington DC	1800	33	11	34	54	58	63	1700	28	9	28	46	51	52
Other MSA	61200	23	1	22	41	47	50	56500	20	1	20	36	41	45
Other Not MSA	12700	12	1	11	27	31	33	11600	12	1	10	27	31	33

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group can be estimated by dividing the number of site-years by 300.

<sup>2</sup> Annual means for each monitor were first calculated based on all simulated hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p95, p98, p99 represent the minimum, median, 95<sup>th</sup>, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual means.

ND no available monitoring data.

**Table 7-21. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using 2001-2003 recent NO<sub>2</sub> air quality (*as is*) and an on-road adjustment factor.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	23	0	5	130	169	4	0	0	36	51	1	0	0	8	13	0	0	0	3	4	0	0	0	1	1
Boston	5	0	1	36	40	0	0	0	3	5	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0
Chicago	52	0	35	180	191	9	0	2	62	68	2	0	0	24	29	0	0	0	5	12	0	0	0	1	3
Cleveland	31	0	21	83	102	5	0	1	30	30	1	0	0	10	10	0	0	0	3	3	0	0	0	1	1
Denver	89	8	74	242	259	17	0	5	80	94	3	0	1	25	26	1	0	0	5	5	0	0	0	1	1
Detroit	41	1	30	130	141	9	0	5	44	46	3	0	2	16	18	2	0	1	7	7	1	0	1	7	7
El Paso	32	0	19	136	145	4	0	1	24	26	0	0	0	5	6	0	0	0	1	1	0	0	0	0	1
Jacksonville	13	0	7	55	56	1	0	1	6	7	1	0	1	1	1	1	0	1	1	1	1	0	1	1	1
Las Vegas	23	0	4	171	194	4	0	0	54	62	0	0	0	8	9	0	0	0	1	1	0	0	0	0	0
Los Angeles	71	0	57	231	251	17	0	6	94	108	5	0	0	41	48	1	0	0	16	23	0	0	0	7	10
Miami	7	0	2	50	64	1	0	0	6	10	0	0	0	1	2	0	0	0	0	0	0	0	0	0	0
New York	42	0	28	177	201	7	0	1	55	63	2	0	0	24	24	0	0	0	6	9	0	0	0	1	2
Philadelphia	37	0	19	149	172	6	0	1	49	62	1	0	0	11	24	0	0	0	3	5	0	0	0	1	1
Phoenix	101	1	83	280	315	16	0	2	113	124	2	0	0	17	20	0	0	0	4	5	0	0	0	0	0
Provo	61	1	38	248	289	9	0	0	62	64	1	0	0	11	11	0	0	0	2	2	0	0	0	0	0
St. Louis	25	0	12	128	139	3	0	0	28	37	0	0	0	5	6	0	0	0	1	1	0	0	0	1	1
Washington DC	36	0	17	169	205	6	0	0	46	54	1	0	0	9	14	0	0	0	1	1	0	0	0	0	0
Other MSA	16	0	3	105	129	2	0	0	22	32	0	0	0	4	7	0	0	0	1	2	0	0	0	0	0
Other Not MSA	4	0	0	43	62	1	0	0	9	15	0	0	0	2	5	0	0	0	1	2	0	0	0	1	1

**Notes:**

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

**Table 7-22. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using 2004-2006 recent NO<sub>2</sub> air quality (*as is*) and an on-road adjustment factor.**

Location	Exceedances of 100 ppb <sup>1</sup>					Exceedances of 150 ppb <sup>1</sup>					Exceedances of 200 ppb <sup>1</sup>					Exceedances of 250 ppb <sup>1</sup>					Exceedances of 300 ppb <sup>1</sup>				
	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99	mean	min	med	p98	p99
Atlanta	17	0	2	114	120	2	0	0	26	27	0	0	0	6	7	0	0	0	1	1	0	0	0	0	0
Boston	2	0	0	18	21	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chicago	36	0	20	148	161	5	0	0	41	53	1	0	0	7	18	0	0	0	1	2	0	0	0	0	0
Cleveland	ND																								
Denver	63	2	49	190	195	10	0	4	47	52	2	0	0	11	14	0	0	0	3	3	0	0	0	0	0
Detroit	20	0	9	90	103	2	0	0	20	24	0	0	0	3	6	0	0	0	0	0	0	0	0	0	0
El Paso	24	0	12	114	143	3	0	0	20	23	0	0	0	4	4	0	0	0	1	1	0	0	0	0	0
Jacksonville	11	0	5	48	59	2	0	2	5	6	1	0	1	4	4	1	0	0	3	3	1	0	0	3	3
Las Vegas	15	0	0	133	148	2	0	0	54	64	0	0	0	6	8	0	0	0	0	1	0	0	0	0	0
Los Angeles	38	0	25	150	169	6	0	1	43	55	1	0	0	11	14	0	0	0	2	3	0	0	0	1	1
Miami	6	0	1	46	46	0	0	0	6	6	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0
New York	35	0	23	149	171	5	0	1	40	45	1	0	0	13	15	0	0	0	3	4	0	0	0	1	1
Philadelphia	22	0	10	101	130	2	0	0	20	22	0	0	0	3	3	0	0	0	0	0	0	0	0	0	0
Phoenix	77	0	53	275	293	10	0	1	57	71	1	0	0	7	8	0	0	0	1	1	0	0	0	0	0
Provo	51	0	44	160	160	17	0	2	68	70	12	0	0	44	44	7	0	0	43	43	3	0	0	40	40
St. Louis	15	0	5	76	83	1	0	0	20	24	0	0	0	2	5	0	0	0	0	0	0	0	0	0	0
Washington DC	21	0	7	119	143	2	0	0	20	22	0	0	0	4	6	0	0	0	1	2	0	0	0	0	0
Other MSA	10	0	1	79	100	1	0	0	12	18	0	0	0	2	3	0	0	0	0	1	0	0	0	0	0
Other Not MSA	4	0	0	43	65	1	0	0	7	13	0	0	0	2	5	0	0	0	2	2	0	0	0	0	2

**Notes:**

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

ND no available monitoring data.

### 7.3.3 Ambient Air Quality Adjusted to Just Meet the Current and Alternative Standards

As described in section 6.2, each of the current and alternative standards were evaluated using the more recent air quality data set (i.e., 2001-2006) serving as the third air quality scenario. Analysis results are presented for a few selected locations, potential health effect benchmarks, and alternative standard levels, since there were a total of 10 air quality analyses (8 alternative standards, the current standard, and *as is* air quality), for each year-group of data (2001-2003 and 2004-2006), for each of the three monitor-to-road categories ( $\leq 20$  m;  $< 20$  and  $< 100$  m; and  $\geq 100$  m from a major road), and evaluated at five potential health effect benchmark levels (100, 150, 200, 250, 300 ppb 1-hour). All of the results for each location are provided in Appendix A, section 9, much of which is summarized here in a series of key figures.

Figures 7-2 and 7-3 illustrate the estimated mean number of daily maximum exceedances of the three lowest benchmark concentrations (i.e., 100, 150, and 200 ppb) using air quality data adjusted to just meeting the current annual average standard for year-groups 2001-2003 and 2004-2006, respectively. The number of estimated daily maximum exceedances of 100 ppb generally ranges from ten to fifty, with pattern of exceedances based on monitor siting consistent with that noted above for the *as is* data. In general, there were a greater number of daily maximum exceedances at the near road monitors compared with those sited  $\geq 100$  m from a major road, although a few monitors sited at  $\geq 100$  m from a major road contained more estimated exceedances than the monitors sited within 20 m of a major road (e.g., Denver). There were also differences in the estimates for each three-year group from what was expected. For example, a few of the locations had an estimated daily maximum number of exceedances of 100 ppb that were slightly higher for the 2004-2006 year-group when compared with the 2001-2003 year-group (e.g., Detroit in Figures 7-2 and 7-3). The estimated number of daily maximum exceedances of 150 and 200 ppb were much lower than that of 100 ppb, the mean number of exceedances was fewer than 20 for most location years and roadway-monitor groupings. Note that fifty-one of the 81 possible year-group and monitoring site data combinations at the 19 locations did not have any exceedances of the 200 ppb level when using air quality adjusted to just meeting the current standard.

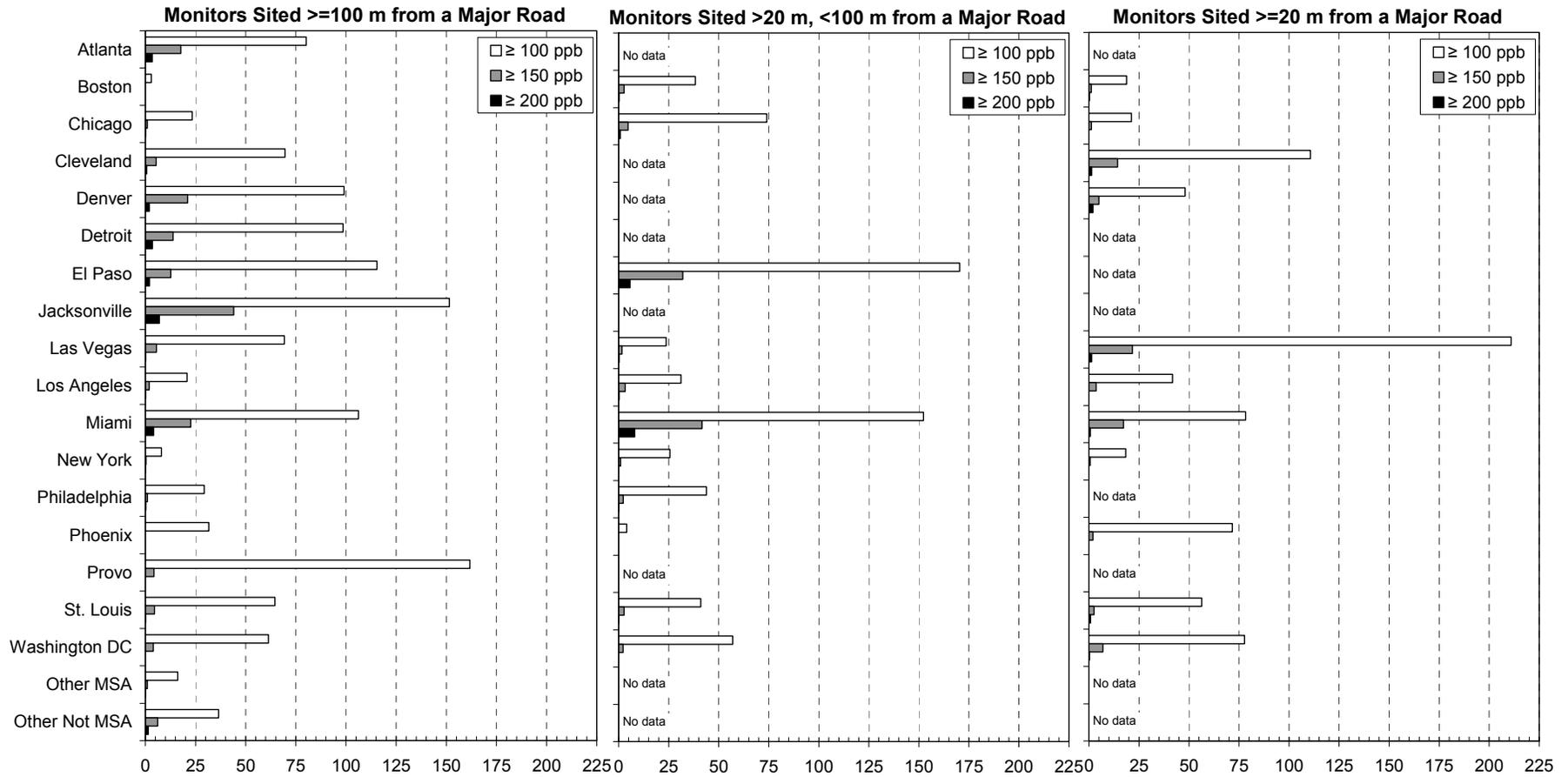
Figure 7-4 presents the mean estimated number of daily maximum exceedances when considering the air quality adjusted to just meeting the potential alternative standard levels, using Chicago as an example to illustrate the patterns in the estimated exceedances for two forms of the standard. These patterns presented for Chicago apply to the other locations, with a few exceptions. As expected, the estimated number of daily maximum exceedances is lower for a 99<sup>th</sup> percentile form compared with each corresponding level using the 98<sup>th</sup> percentile form of alternative standard. In general, the number of estimated daily maximum exceedances of the potential health effect benchmark levels at monitoring sites < 100 m from a major road is greater than the numbers estimated for monitors sited  $\geq$  100 m from a major road. This is what one would expect given the greater potential for vehicle emissions influencing ambient concentrations at near road monitors. There were also a slightly greater number of estimated daily maximum exceedances at the monitors sited  $\leq$  20 m compared with those sited between 20-100 m. As expected, the number of exceedances of the potential health effect benchmark levels decreases with decreasing alternative standard level. Regardless of three-year group or monitoring group, an alternative standard level of 100 ppb tended to reduce the number of estimated exceedances of 100 ppb to either a few to none.

Figure 7-5 presents mean estimated number of daily maximum exceedances of the 200 ppb concentration level for a few additional locations, Phoenix, Los Angeles, Washington DC, and St. Louis. Again, there are trends in these results that are consistent with that reported for the Chicago results, with few exceptions. For example, in St. Louis the estimated number of daily maximum exceedances at monitors located  $\geq$  100 m from a major road were greater than those estimated using the monitoring sites < 100 m from a major road. Also note that there were variable results when comparing year-groups across the different locations within the monitor site-group; sometimes the year 2001-2003 contained greater numbers of exceedances when compared with 2004-2006 (e.g., St. Louis), and other times it did not (e.g. Los Angeles). However, the alternative standard levels of either 100 or 150 ppb at either percentile consistently reduced the mean number of daily maximum exceedances of 200 ppb to about zero.

Tables 7-23, 7-24, and 7-25 summarize the annual mean concentrations and estimated number of exceedances in a year given 2001-2003 air quality adjusted that just meets the 1-hour 100 ppb 98<sup>th</sup> percentile standard at monitors sited  $\geq$  100 m, >20 m and < 100 m, and  $\leq$ 20 m from

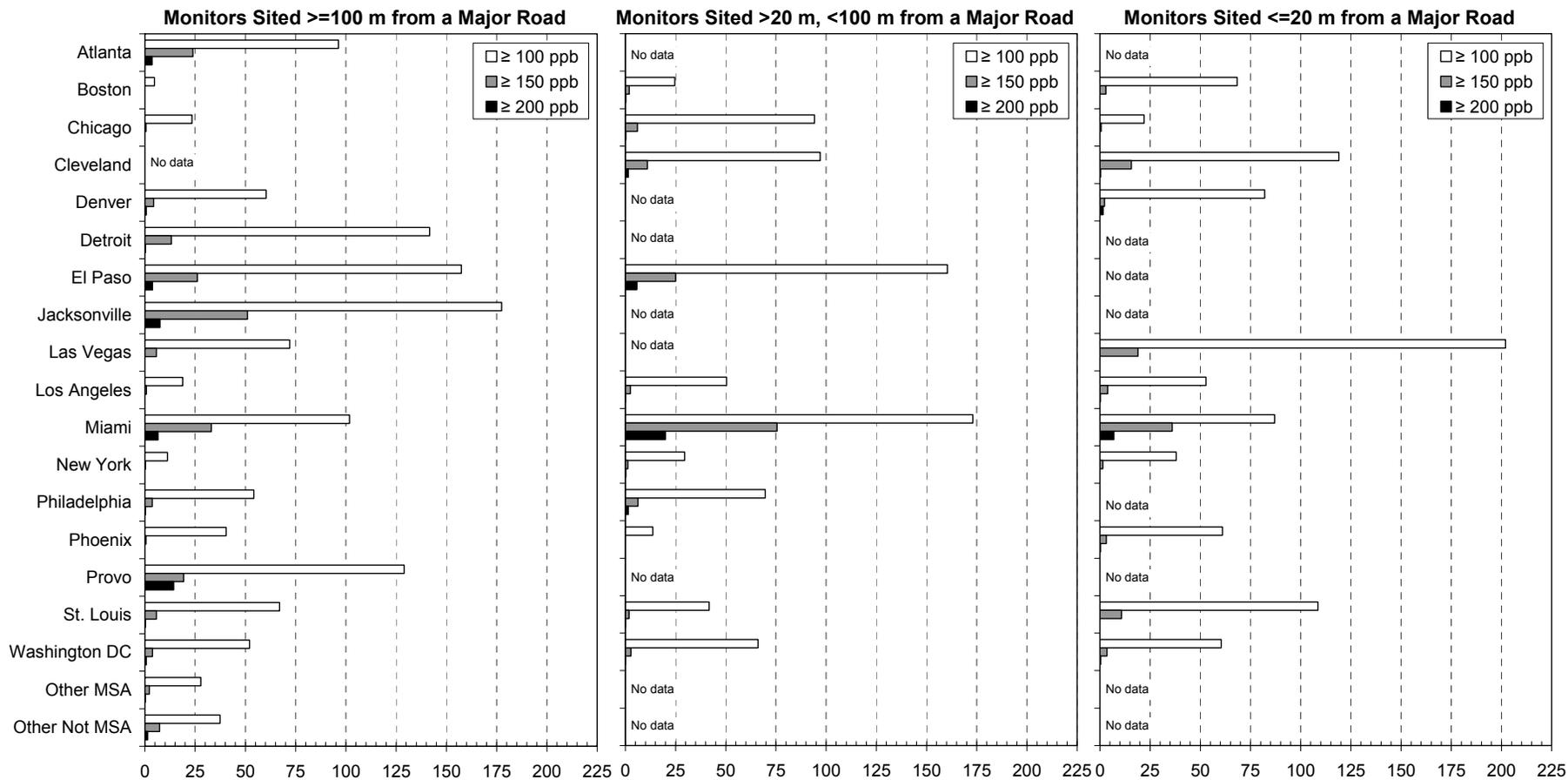
a major road, respectively. The tables provide a more comprehensive comparison of the numbers of daily maximum exceedances for the lowest potential health effect benchmarks (i.e., 100, 150, and 200 ppb), as well as providing upper percentile estimates for each of the parameters. The results for this particular year-group are provided to describe patterns within a given standard level, results were similar for the 2004-2006 air quality data. The complete results for all of the standard levels and year-groups of air quality, including the observed number of daily maximum exceedances (*as is* air quality) are provided in Appendix A, section 9. Most locations contained a mean of fewer than 5 daily maximum exceedances of the 100 ppb concentration level, with upper percentile estimates ranging from the 5 to about 15. These results are comparably less than those estimated using air quality adjusted to just meeting the current standard (Figure 7-2). At potential health effect benchmark levels above 100 ppb, there were few estimated exceedances, particularly at and above the 200 ppb level, considering both the mean and the upper percentiles.

Tables 7-26 summarizes the observed and estimated mean numbers of exceedances of 100 ppb using the 2001-2003 *as is* air quality and air quality adjusted to just meet the current standard and the potential alternative 98<sup>th</sup> percentile standards at each location. The number of daily maximum exceedances for the *as is* air quality generally fell within the number of exceedances estimated using alternative 1-hour 98<sup>th</sup> percentile standards of 50 ppb and 100 ppb at each location. When the air quality was adjusted to just meeting the current annual average standard, the estimated number of daily maximum exceedances was generally near that estimated using the alternative 1-hour 98<sup>th</sup> percentile standard of 150 ppb at each location. In a similar manner, Table 7-27 summarizes the observed and estimated mean numbers of exceedances of 150 ppb 1-hour at each location. The number of daily maximum exceedances using *as is* air quality in each location was most similar to that estimated using the alternative 1-hour 98<sup>th</sup> percentile standard of 50 ppb, while estimates using the air quality adjusted to just meeting the current standard again approached the estimated numbers of exceedance using the alternative 1-hour 98<sup>th</sup> percentile standard of 150 ppb at each location.



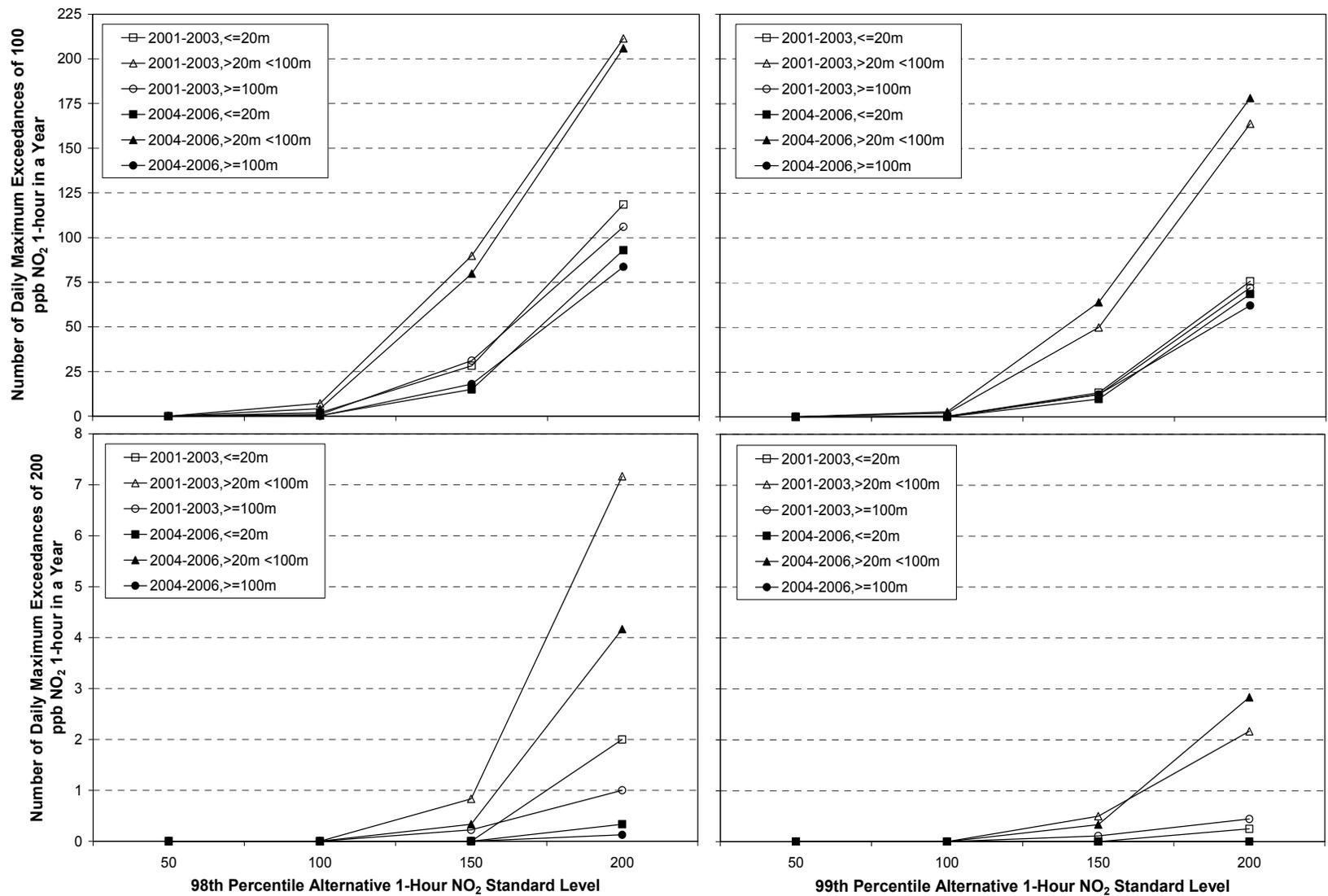
Estimated mean number of daily maximum exceedances of 1-hour NO<sub>2</sub> concentrations of 100, 150, 200 ppb

**Figure 7-2. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using recent NO<sub>2</sub> air quality (2001-2003) adjusted to just meeting the current annual standard (0.053 ppm). Left graph: monitors  $\geq 100$ m from a major road; Middle graph: monitors  $>20$  m and  $<100$  m from a major road; Right graph: monitors  $\leq 20$  m from a major road.**

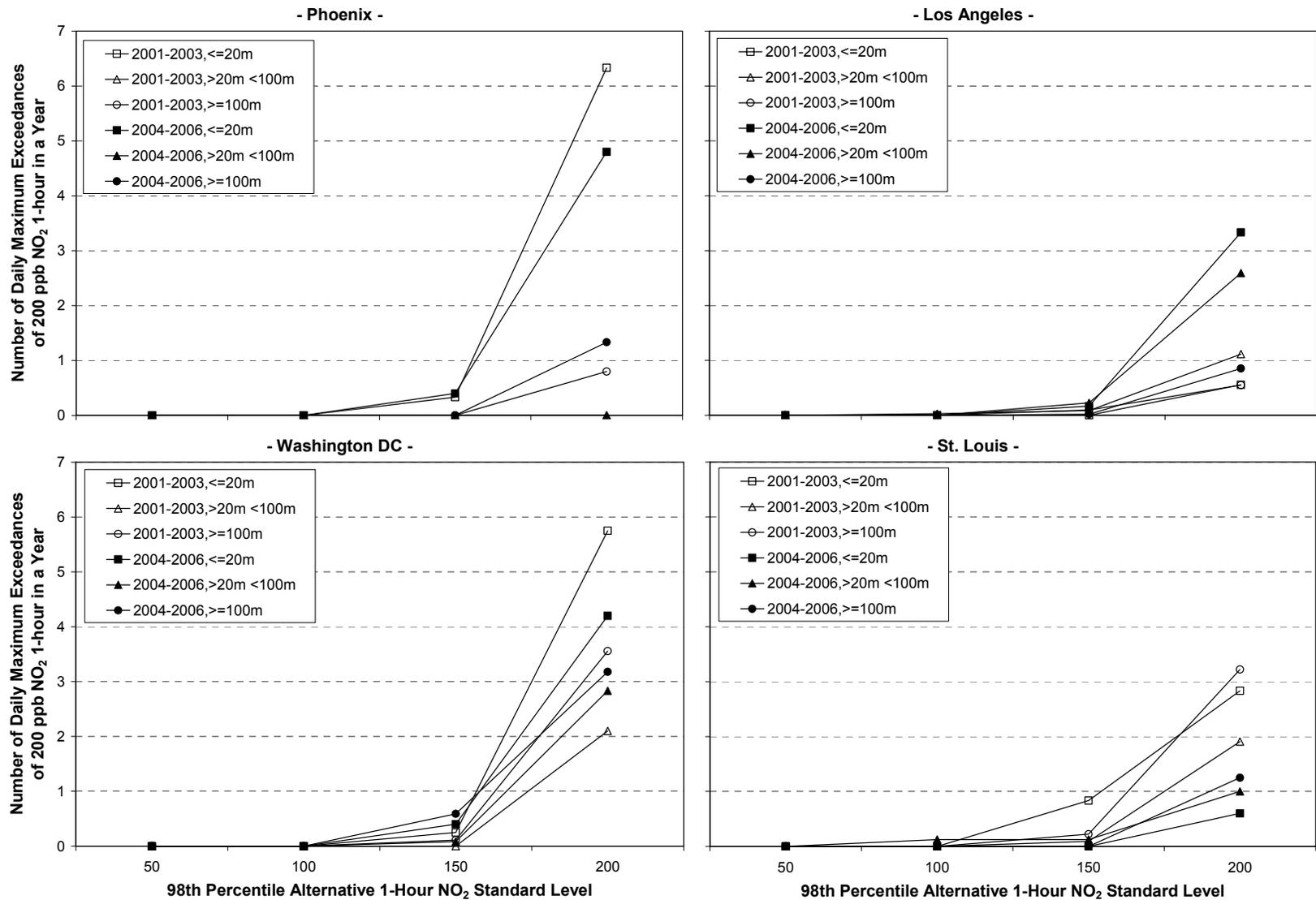


Estimated mean number of daily maximum exceedances of 1-hour NO<sub>2</sub> concentrations of 100, 150, 200 ppb in a Year

**Figure 7-3. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using recent NO<sub>2</sub> air quality (2004-2006) adjusted to just meeting the current annual standard (0.053 ppm). Left graph: monitors  $\geq 100$ m from a major road; Middle graph: monitors  $>20$  m and  $<100$  m from a major road; Right graph: monitors  $\leq 20$  m from a major road).**



**Figure 7-4. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks (100 ppb, top; 200 ppb, bottom) in Chicago in a year, using recent NO<sub>2</sub> air quality data (2001-2006) adjusted to just meeting alternative 1-hour standard levels (98<sup>th</sup> percentile, left; and 99<sup>th</sup> percentile, right) and monitors sited  $\geq 100$  m,  $> 20$  m and  $< 100$  m,  $\leq 20$  m of major roads.**



**Figure 7-5. Estimated mean number of daily maximum exceedances of 200 ppb in four locations (Phoenix, Los Angeles, Washington DC, and St. Louis) in a year, using recent NO<sub>2</sub> air quality data (2001-2006) adjusted to just meeting alternative 1-hour 98<sup>th</sup> percentile standard levels and monitors sited ≥100 m, > 20 m and < 100 m, ≤ 20 m of major roads.**

**Table 7-23. Estimated annual mean NO<sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98th percentile alternative standard and monitors sited ≥ 100 m of a major road.**

Location	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					Number of Daily Maximum Exceedances of 1-Hour Level <sup>3</sup>														
							≥100 ppb					≥150 ppb					≥ 200 ppb				
		Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99
Atlanta	14	15	5	19	29	29	2	0	0	15	15	0	0	0	1	1	0	0	0	1	1
Boston	6	13	7	15	16	16	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0
Chicago	9	25	19	23	32	32	1	0	0	4	4	0	0	0	1	1	0	0	0	0	0
Cleveland	3	25	25	25	26	26	2	1	3	3	3	0	0	0	0	0	0	0	0	0	0
Denver	2	24	22	24	27	27	2	1	2	2	2	0	0	0	0	0	0	0	0	0	0
Detroit	6	23	21	23	26	26	3	1	3	7	7	2	0	1	7	7	1	0	1	4	4
El Paso	12	20	14	21	24	24	3	0	2	8	8	0	0	0	1	1	0	0	0	1	1
Jacksonville	2	26	26	26	26	26	7	3	7	10	10	1	0	1	1	1	1	0	1	1	1
Las Vegas	16	15	3	11	32	32	1	0	0	7	7	0	0	0	0	0	0	0	0	0	0
Los Angeles	51	18	4	19	29	29	1	0	0	5	10	0	0	0	0	5	0	0	0	0	0
Miami	6	16	13	16	19	19	4	0	3	14	14	0	0	0	2	2	0	0	0	0	0
New York	26	22	12	20	34	34	1	0	0	5	5	0	0	0	0	0	0	0	0	0	0
Philadelphia	14	27	20	25	39	39	3	0	2	15	15	0	0	0	1	1	0	0	0	1	1
Phoenix	5	31	26	33	34	34	1	0	1	2	2	0	0	0	0	0	0	0	0	0	0
Provo	3	37	34	38	39	39	6	4	6	9	9	0	0	0	0	0	0	0	0	0	0
St. Louis	9	27	22	26	32	32	3	0	1	15	15	0	0	0	1	1	0	0	0	0	0
Washington DC	18	26	12	29	35	35	4	0	3	11	11	0	0	0	0	0	0	0	0	0	0
Other MSA	612	13	1	13	23	25	0	0	0	1	4	0	0	0	0	0	0	0	0	0	0
Other Not MSA	127	7	1	7	17	18	0	0	0	6	6	0	0	0	1	1	0	0	0	1	1

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group is estimated by dividing the number of site-years by 3.

<sup>2</sup> Annual means for each monitor were first calculated based on all simulated hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual average concentration in any one year within the monitoring period.

<sup>3</sup> The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.

**Table 7-24. Estimated annual mean NO<sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98<sup>th</sup> percentile alternative standard and monitors sited >20 m and <100 m from a major road.**

Location	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					Number of Daily Maximum Exceedances of 1-Hour Level <sup>3</sup>														
							≥ 100 ppb					≥ 150 ppb					≥ 200 ppb				
		Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99
Atlanta	ND																				
Boston	14	23	12	26	35	35	3	0	2	11	11	0	0	0	0	0	0	0	0	0	0
Chicago	6	35	33	35	37	37	7	1	4	21	21	1	0	0	3	3	0	0	0	0	0
Cleveland	ND																				
Denver	ND																				
Detroit	ND																				
El Paso	3	27	26	28	28	28	6	2	7	9	9	0	0	0	0	0	0	0	0	0	0
Jacksonville	ND																				
Las Vegas	3	8	4	8	12	12	1	0	0	4	4	0	0	0	0	0	0	0	0	0	0
Los Angeles	35	19	3	19	32	32	1	0	0	7	7	0	0	0	1	1	0	0	0	1	1
Miami	3	27	24	27	29	29	8	4	6	15	15	0	0	0	1	1	0	0	0	0	0
New York	13	33	23	33	44	44	3	0	1	13	13	0	0	0	2	2	0	0	0	0	0
Philadelphia	7	34	26	33	42	42	5	0	4	11	11	0	0	0	0	0	0	0	0	0	0
Phoenix	2	26	25	26	27	27	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Provo	ND																				
St. Louis	11	22	13	18	38	38	2	0	0	11	11	0	0	0	0	0	0	0	0	0	0
Washington DC	10	29	20	31	36	36	2	0	2	5	5	0	0	0	0	0	0	0	0	0	0
Other MSA	ND																				
Other Not MSA	ND																				

**Notes:**  
<sup>1</sup> The average number of monitors operating per year within the three-year group is estimated by dividing the number of site-years by 3.  
<sup>2</sup> Annual means for each monitor were first calculated based on all simulated hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual average concentration in any one year within the monitoring period.  
<sup>3</sup> The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-25. Estimated annual mean NO<sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks in a year, using 2001-2003 air quality adjusted to just meeting a 1-hour 100 ppb 98<sup>th</sup> percentile alternative standard and monitors sited ≤20 m from a major road.**

Location	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					Number of Daily Maximum Exceedances of 1-Hour Level <sup>3</sup>														
							≥ 100 ppb					≥ 150 ppb					≥ 200 ppb				
		Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99
Atlanta	ND																				
Boston	5	29	10	32	41	41	2	0	0	6	6	0	0	0	1	1	0	0	0	0	0
Chicago	4	26	25	26	27	27	2	0	1	6	6	0	0	0	0	0	0	0	0	0	0
Cleveland	3	32	31	32	34	34	7	3	7	10	10	0	0	0	0	0	0	0	0	0	0
Denver	2	37	37	37	38	38	7	3	7	10	10	2	1	2	3	3	0	0	0	0	0
Detroit	ND																				
El Paso	ND																				
Jacksonville	ND																				
Las Vegas	3	32	31	32	32	32	7	1	10	11	11	0	0	0	0	0	0	0	0	0	0
Los Angeles	9	23	18	23	29	29	1	0	0	3	3	0	0	0	0	0	0	0	0	0	0
Miami	3	12	11	12	12	12	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0
New York	7	30	27	30	33	33	1	0	1	2	2	0	0	0	0	0	0	0	0	0	0
Philadelphia	ND																				
Phoenix	3	41	40	40	43	43	6	4	4	11	11	0	0	0	0	0	0	0	0	0	0
Provo	ND																				
St. Louis	6	28	25	29	30	30	3	1	3	6	6	0	0	0	1	1	0	0	0	0	0
Washington DC	4	33	28	34	36	36	6	3	6	9	9	0	0	0	1	1	0	0	0	0	0
Other MSA	ND																				
Other Not MSA	ND																				

**Notes:**  
<sup>1</sup> The average number of monitors operating per year within the three-year group is estimated by dividing the number of site-years by 3.  
<sup>2</sup> Annual means for each monitor were first calculated based on all simulated hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual average concentration in any one year within the monitoring period.  
<sup>3</sup> The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.  
 ND no available monitoring data.

**Table 7-26. Estimated mean number of daily maximum exceedances of 100 ppb 1-hour NO<sub>2</sub> concentrations in a year, using 2001-2003 air quality *as is* and that adjusted to just meeting the current and alternative standards (98<sup>th</sup> percentile) for monitors sited ≥100 m, >20 m and <100 m, and ≤20 m of a major road.**

Location	Sites ≥100 m of a major road						Sites >20 m and <100 m of a major road						Sites ≤20 m of a major road					
	As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard			
			50	100	150	200			50	100	150	200			50	100	150	200
Atlanta	0	80	0	2	20	65	ND						ND					
Boston	0	3	0	0	3	34	0	38	0	3	35	116	0	19	0	2	29	104
Chicago	0	23	0	1	31	106	2	74	0	7	90	212	0	21	0	2	28	119
Cleveland	0	70	0	2	49	133	ND						0	111	0	7	79	187
Denver	2	99	0	2	37	149	ND						7	48	0	7	68	224
Detroit	3	99	1	3	17	73	ND						ND					
El Paso	0	115	0	3	35	125	2	170	0	6	77	178	ND					
Jacksonville	1	152	1	7	74	152	ND						ND					
Las Vegas	0	69	0	1	41	114	0	24	0	1	11	46	1	211	0	7	141	280
Los Angeles	4	21	0	1	9	37	6	31	0	1	15	53	6	42	0	1	21	72
Miami	0	106	0	4	41	107	0	152	0	8	73	155	0	78	0	1	28	76
New York	0	8	0	1	16	72	1	26	0	3	50	154	1	18	0	1	38	143
Philadelphia	0	29	0	3	53	171	0	44	0	5	84	216	ND					
Phoenix	0	32	0	1	89	213	0	4	0	0	49	210	2	72	0	6	160	293
Provo	0	162	0	6	200	327	ND						ND					
St. Louis	0	65	0	3	60	175	0	41	0	2	37	119	0	56	0	3	46	175
Washington DC	0	61	0	4	58	153	0	57	0	2	57	168	0	78	0	6	73	209
Other MSA	0	16	0	0	3	18	ND						ND					
Other Not MSA	0	37	0	0	1	6	ND						ND					

**Notes:**

The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period.

ND No available monitoring data.

**Table 7-27. Estimated mean number of daily maximum exceedances of 150 ppb 1-hour NO<sub>2</sub> concentrations in a year, using 2001-2003 air quality *as is* and air quality adjusted to just meeting the current and alternative standards (98<sup>th</sup> percentile) for monitors sited ≥100 m, >20 m and <100 m, and ≤20 m of a major road.**

Location	Sites ≥100 m of a major road						Sites >20 m and <100 m of a major road						Sites ≤20 m of a major road					
	As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As Is	Cur Std	Alternative 1-hour 98 <sup>th</sup> percentile standard			
			50	100	150	200			50	100	150	200			50	100	150	200
Atlanta	0	18	0	0	2	10							ND					
Boston	0	0	0	0	0	1	0	3	0	0	3	19	0	1	0	0	2	15
Chicago	0	1	0	0	1	14	0	5	0	1	7	53	0	1	0	0	2	15
Cleveland	0	5	0	0	2	27	ND						0	14	0	0	7	50
Denver	0	21	0	0	2	17	ND						1	5	0	2	7	33
Detroit	1	14	1	2	3	9	ND						ND					
El Paso	0	13	0	0	3	16	0	32	0	0	6	40	ND					
Jacksonville	1	44	1	1	7	41	ND						ND					
Las Vegas	0	6	0	0	1	19	0	2	0	0	1	5	0	22	0	0	7	76
Los Angeles	0	2	0	0	1	5	0	3	0	0	1	8	0	4	0	0	1	7
Miami	0	23	0	0	4	23	0	42	0	0	8	42	0	17	0	0	1	16
New York	0	0	0	0	1	7	0	1	0	0	3	25	0	1	0	0	1	18
Philadelphia	0	1	0	0	3	27	0	2	0	0	5	42	ND					
Phoenix	0	0	0	0	1	41	0	0	0	0	0	11	0	2	0	0	6	88
Provo	0	4	0	0	6	112	ND						ND					
St. Louis	0	5	0	0	3	31	0	3	0	0	2	21	0	3	0	0	3	23
Washington DC	0	4	0	0	4	30	0	2	0	0	2	28	0	7	0	0	6	38
Other MSA	0	1	0	0	0	1	ND						ND					
Other Not MSA	0	6	0	0	0	1	ND						ND					

**Notes:**

The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period.

ND no available monitoring data.

### **7.3.4 On-Road Concentrations Derived From Ambient Air Quality Adjusted to Just Meet the Current and Alternative Standards**

Just as was done with the *as is* air quality data, on-road NO<sub>2</sub> concentrations were estimated using the air quality adjusted to just meeting the current and alternative standard and the approach described in section 7.2.3. The analysis was performed using the more recent air quality separated into two three-year groups (2001-2003 and 2004-2006) based on the form of the potential alternative standards (i.e., a 3-year average) and represents the fourth air quality scenario. Results are presented in a manner consistent with section 7.3.3, whereby the number of daily maximum exceedances of the potential benchmark levels was estimated. However, for the sake of brevity only key figures and tables are provided here. The complete results for the estimated on-road concentrations and numbers of benchmark exceedances are provided in Appendix A, section 9.

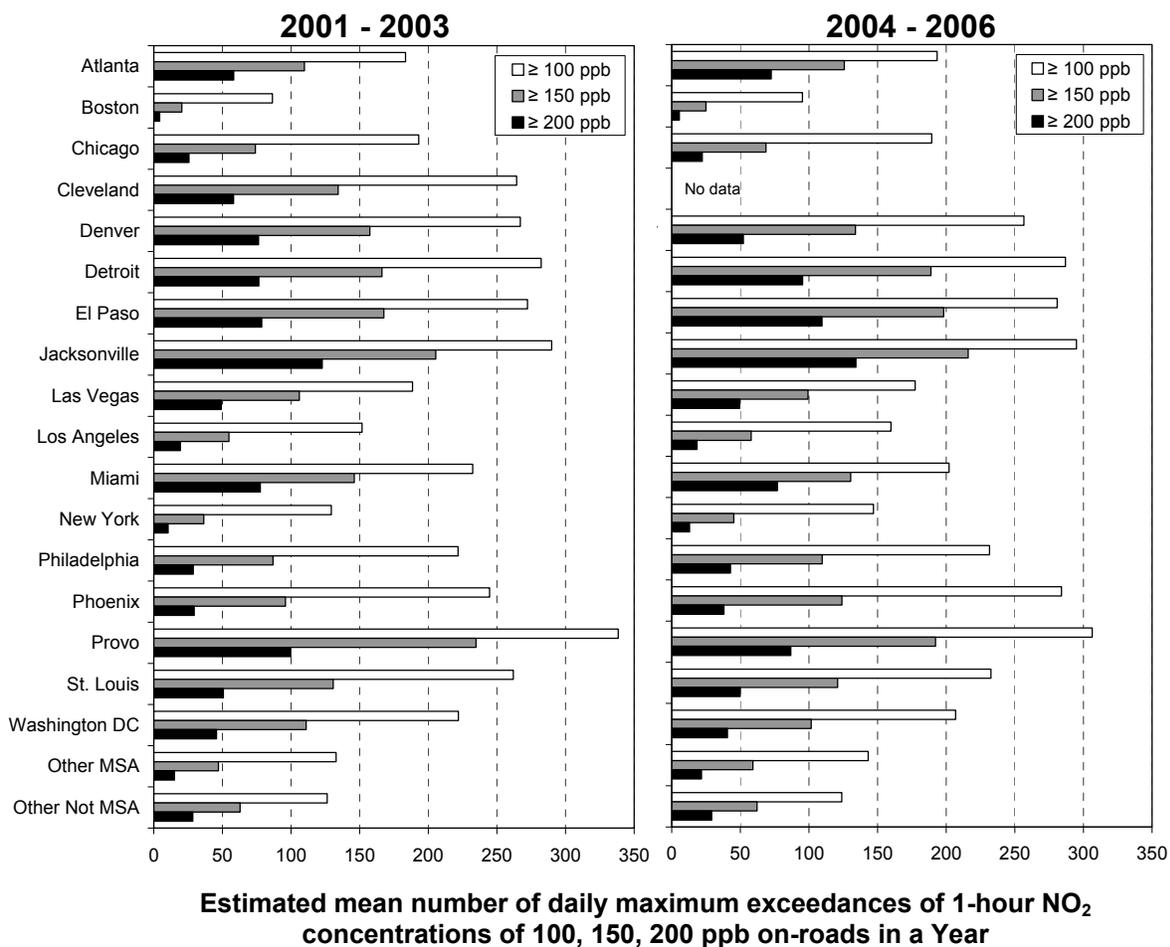
Figures 7-6 illustrates the estimated mean number of daily maximum exceedances of the 100, 150, and 200 ppb levels on-roads, given air quality adjusted to just meeting the current annual average standard for two three-year groups. Most locations contained an average of two-hundred or more estimated daily maximum exceedances of 100 ppb, much greater than those estimated using either the ambient monitors sited  $\geq 100$  m,  $>20$  and  $<100$  m, an  $\leq 20$  m of a major road (Figures 7-2 and 7-3). The estimated numbers of daily maximum exceedances of the 150 and 200 ppb levels were also higher on-roads. Most locations were estimated to contain at least one-hundred exceedances of 150 ppb and between 50 and 100 exceedances of 200 ppb on-roads when using air quality concentrations adjusted to just meeting the current standard.

The effect of the potential alternative standards on the estimated on-road NO<sub>2</sub> concentrations was also analyzed at each of the locations. Figure 7-7 illustrates each of the standard levels (50, 100, 150, and 200 ppb 1-hour) and the two forms (98<sup>th</sup> and 99<sup>th</sup> percentiles) evaluated, again using Chicago as an example to describe patterns in the number of exceedances. The patterns observed in Figure 7-2 and described in section 7.3.3 for the ambient monitors are similar to that observed here, albeit with greater numbers of exceedances estimated on-roads compared with those estimated for monitors near-roads or sited at a distance from major roads. Estimated numbers of daily maximum concentrations above 100 ppb are between 50 and one

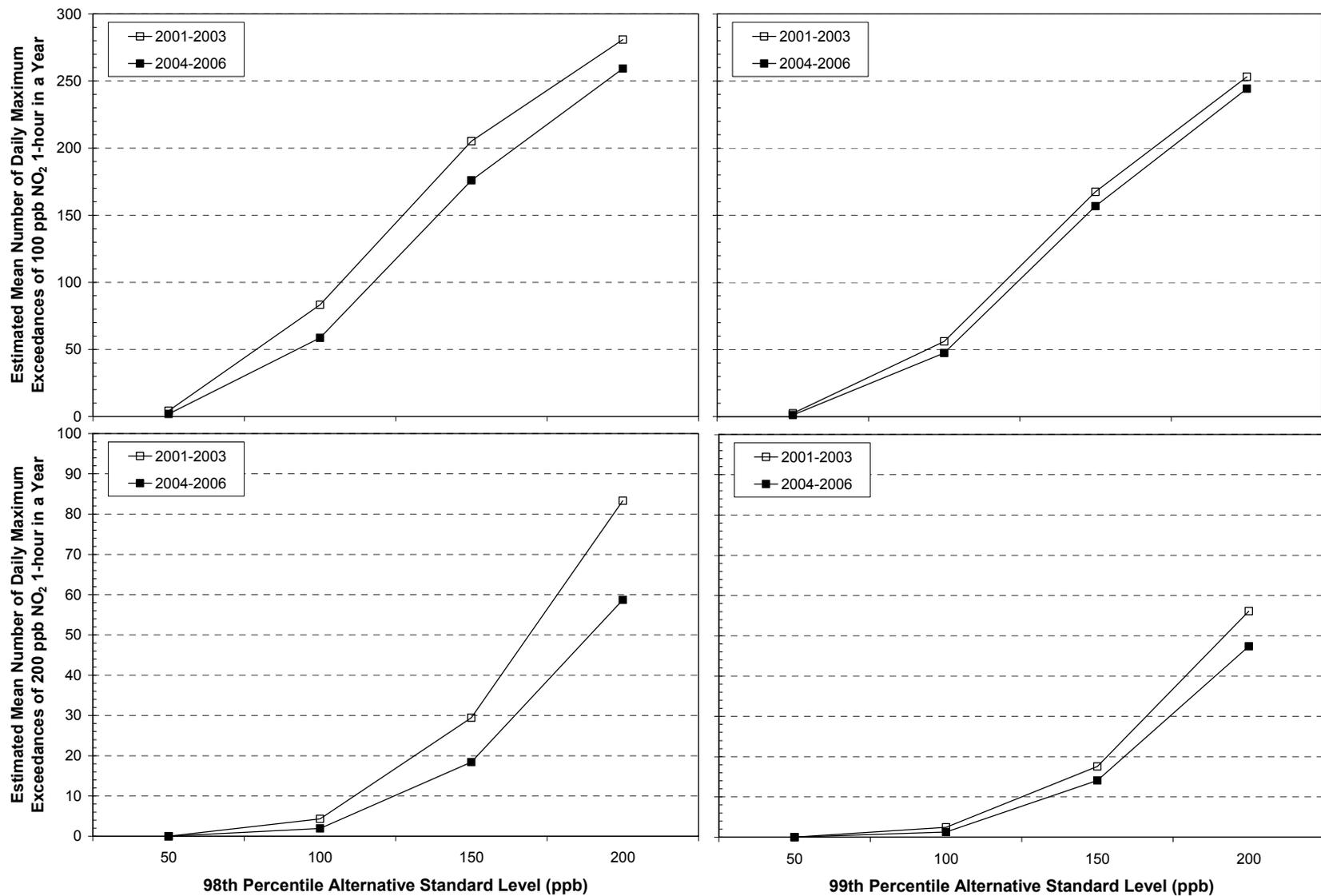
hundred considering a standard level of 100 ppb (either percentile), however daily maximum exceedances of 200 ppb are estimated to be between one and four.

Similar numbers of exceedances on-roads were estimated at other locations using air quality adjusted to just meeting the potential alternative standards. Figure 7-8 illustrates the estimated number of exceedances of 200 ppb at four selected locations as an example, Phoenix, Los Angeles, Washington DC, and St. Louis, using a 98<sup>th</sup> percentile form of a 1-hour standard. The number of concentrations above 200 ppb is similar at each of the locations (including Chicago), particularly when comparing the 100 ppb standard level, ranging from two to seven. Table 7-28 presents a more comprehensive comparison at this particular standard level (98<sup>th</sup> percentile at 100 ppb) using 2001-2003 adjusted air quality at each of the locations. For most locations, the estimated mean number of daily maximum exceedances of 200 ppb on-roads was seven or less, with upper percentiles estimated to number about 30 to 70 of exceedances. The mean number of exceedances of 250 and 300 ppb were less, ranging from a few to tens of occurrences in a year.

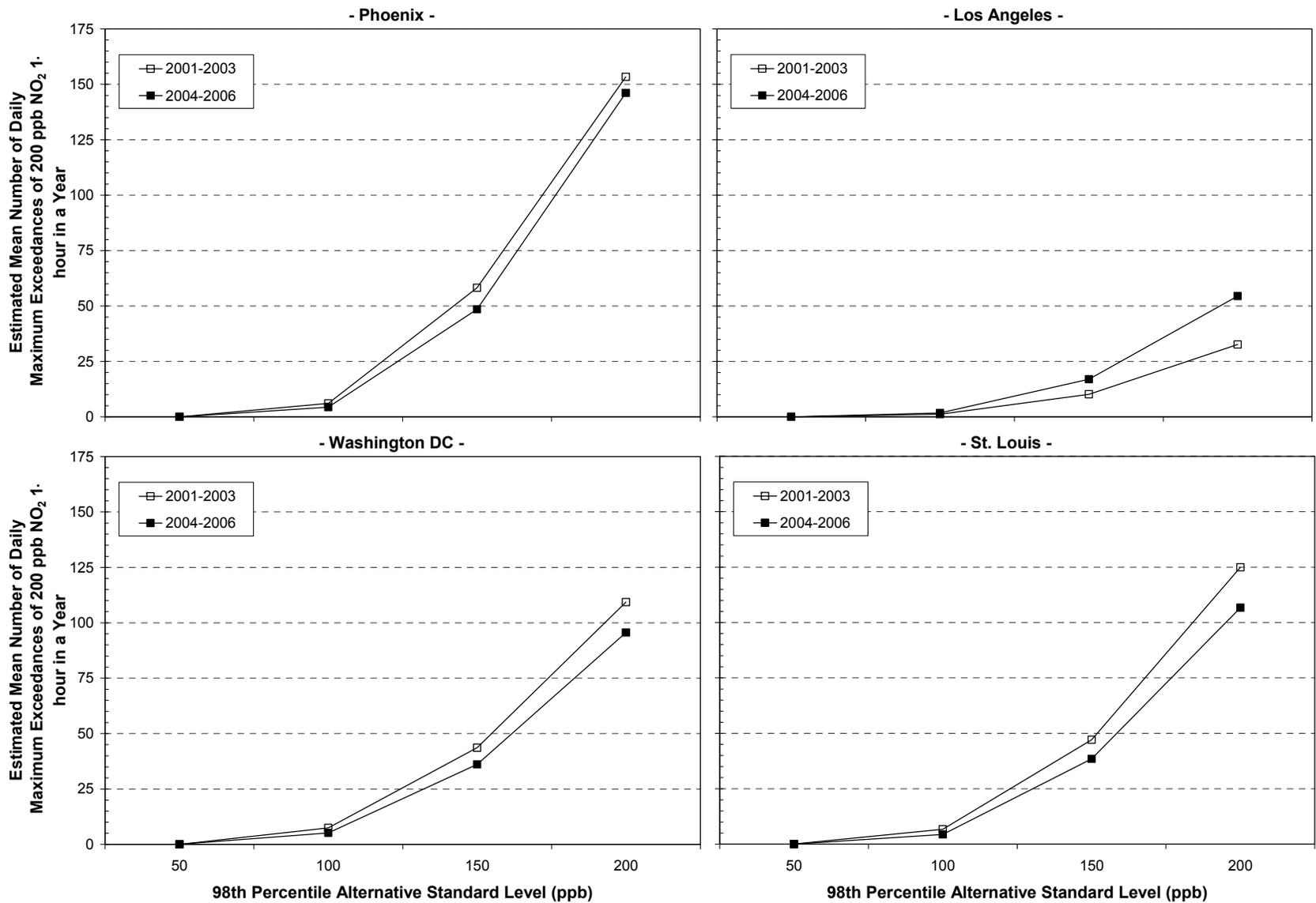
Tables 7-29 and 7-30 summarizes the observed and estimated mean numbers of exceedances of 100 and 150 ppb on-roads, respectively, using all the recent air quality *as is* and that adjusted to just meet the current standard and the potential alternative 98<sup>th</sup> percentile standards at each location. Patterns for the estimated on-road concentrations using *as is* air quality and air quality adjusted to just meet the current annual standard followed similar patterns observed for the monitors sited  $\geq 100$  m,  $> 20$  and  $< 100$  m, and  $\leq 20$  m of a major road (see Tables 7-25 and 7-26, for the daily maximum exceedances of 100 and 150 ppb using 2001-2003 air quality). The estimated number of daily maximum exceedances on-roads using the *as is* air quality was within the range of estimates provided by the alternative 1-hour 98<sup>th</sup> percentile standards of 50 and 100 ppb, while the estimated on-road exceedances of 150 ppb was within the range of estimated exceedances using the 150 and 200 ppb alternative standard levels.



**Figure 7-6. Estimated mean number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using recent NO<sub>2</sub> air quality adjusted to just meeting the current annual standard (0.053 ppm) and an on-road adjustment factor. Left graph: 2001-2003 air quality; Right graph: 2004-2006 air quality.**



**Figure 7-7. Estimated number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks (100 ppb, top; 200 ppb, bottom) on-roads in Chicago in a year, using recent NO<sub>2</sub> air quality (2001-2006) adjusted to just meeting alternative 1-hour standard levels (98<sup>th</sup> percentile, left; and 99<sup>th</sup> percentile) and an on-road adjustment factor.**



**Figure 7-8. Estimated mean number of daily maximum exceedances of 200 ppb on-roads in four locations (Phoenix, Los Angeles, Washington DC, and St. Louis) in a year, using recent NO<sub>2</sub> air quality (2001-2006) adjusted to just meeting alternative 1-hour 98<sup>th</sup> percentile standard levels and an on-road adjustment factor.**

**Table 7-28. Estimated annual mean NO<sub>2</sub> concentration and the number of daily maximum exceedances of short-term (1-hour) potential health effect benchmarks on-roads in a year, using recent air quality (2001-2003) adjusted to just meeting a 1-hour 100 ppb 98<sup>th</sup> percentile alternative standard and an on-road adjustment factor.**

Location	Site-Years <sup>1</sup>	Annual Mean NO <sub>2</sub> (ppb) <sup>2</sup>					Number of Daily Maximum Exceedances of 1-Hour Level <sup>3</sup>														
							≥ 100 ppb					≥ 150 ppb					≥ 200 ppb				
		Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99	Mean	Min	Med	p98	p99
Atlanta	1400	27	6	29	57	65	46	0	22	190	229	10	0	1	70	104	2	0	0	26	38
Boston	600	24	9	24	40	41	26	0	13	125	131	3	0	0	22	25	0	0	0	3	6
Chicago	900	45	25	43	75	78	83	2	68	238	257	18	0	7	92	106	4	0	0	43	49
Cleveland	300	46	32	45	61	64	104	19	98	209	225	24	0	15	73	86	7	0	2	37	38
Denver	200	44	28	42	65	67	99	8	82	252	269	19	0	6	86	103	4	0	1	36	37
Detroit	600	41	26	40	59	63	59	4	46	170	186	13	1	8	56	57	5	0	3	26	28
El Paso	1200	36	17	35	56	58	89	4	79	231	249	17	0	8	80	91	3	0	0	23	24
Jacksonville	200	47	33	47	64	67	119	26	118	217	235	32	2	25	97	114	7	0	4	34	34
Las Vegas	1600	27	4	20	69	73	82	0	43	298	307	19	0	3	149	172	5	0	0	63	71
Los Angeles	5100	32	5	32	61	65	33	0	19	142	160	6	0	1	46	55	1	0	0	15	20
Miami	600	29	16	29	45	46	78	2	74	181	189	19	0	10	92	110	5	0	1	37	48
New York	2600	39	15	37	70	79	57	0	43	212	226	10	0	3	67	73	3	0	0	33	34
Philadelphia	1400	50	26	46	89	92	116	1	102	284	294	27	0	12	118	137	7	0	1	54	68
Phoenix	500	56	33	55	83	88	153	2	152	319	337	35	0	14	182	206	6	0	0	44	48
Provo	300	67	44	65	96	101	257	60	277	353	358	75	1	51	273	301	18	0	4	86	106
St. Louis	900	48	28	47	75	76	125	2	118	274	288	28	0	15	144	153	7	0	1	45	51
Washington DC	1800	47	15	48	82	88	109	0	99	287	310	27	0	10	139	168	7	0	0	56	63
Other MSA	61200	23	1	23	48	51	17	0	4	110	133	2	0	0	23	34	0	0	0	5	8
Other Not MSA	12700	13	1	12	34	37	6	0	0	64	86	1	0	0	12	17	0	0	0	4	8

**Notes:**

<sup>1</sup> The average number of monitors operating per year within the three-year group is estimated by dividing the number of site-years by 300.

<sup>2</sup> Annual means for each monitor were first calculated based on all simulated hourly values in a year. Then the mean of the annual means was estimated as the sum of all the annual means in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the annual average concentration in any one year within the monitoring period.

<sup>3</sup> The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of simulated site-years across the monitoring period. The min, med, p98, and p99 represent the minimum, median, 98<sup>th</sup>, and 99<sup>th</sup> percentiles of the distribution for the number of daily maximum exceedances in any one year within the monitoring period.

**Table 7-29. Estimated mean number of daily maximum exceedances of 100 ppb 1-hour NO<sub>2</sub> concentrations on-roads in a year, using air quality *as is* and air quality adjusted to just meeting the current and alternative standards (98<sup>th</sup> percentile) and an on-road adjustment factor.**

Location	2001-2003 Air Quality						2004-2006 Air Quality					
	As is	Current std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As is	Current std	Alternative 1-hour 98 <sup>th</sup> percentile standard			
			50	100	150	200			50	100	150	200
Atlanta	23	183	2	46	117	170	17	193	3	58	133	181
Boston	5	86	0	26	95	163	2	95	0	19	84	153
Chicago	52	193	4	83	205	281	36	189	2	59	176	259
Cleveland	31	264	7	104	235	305	ND					
Denver	89	267	4	99	232	288	63	257	9	148	263	296
Detroit	41	282	5	59	178	265	20	287	13	165	273	313
El Paso	32	272	3	89	216	278	24	281	5	108	229	281
Jacksonville	13	290	7	119	233	289	11	295	9	127	241	293
Las Vegas	23	189	5	82	167	218	15	177	6	83	161	210
Los Angeles	71	152	1	33	111	191	38	160	2	54	155	227
Miami	7	232	5	78	174	232	6	202	3	56	128	182
New York	42	129	3	57	169	249	35	147	3	75	192	264
Philadelphia	37	222	7	116	254	312	22	232	5	112	237	295
Phoenix	101	245	6	153	293	332	77	284	4	146	299	338
Provo	61	338	18	257	343	351	51	306	13	63	209	298
St. Louis	25	262	7	125	258	316	15	233	4	107	226	287
Washington DC	36	222	7	109	221	279	21	207	5	96	200	260
Other MSA	16	133	0	17	73	138	10	143	0	20	80	143
Other Not MSA	4	126	0	6	28	63	4	124	0	6	28	60

**Notes:**  
The mean number of exceedances represents the sum of daily maximum exceedances occurring at all monitors in a particular location divided by the number of site-years across the monitoring period.  
ND no available monitoring data

**Table 7-30. Estimated mean number of daily maximum exceedances of 150 ppb 1-hour NO<sub>2</sub> concentrations on-roads in a year, using air quality *as is* and air quality adjusted to just meeting the current and alternative standards (98<sup>th</sup> percentile) and an on-road adjustment factor.**

Location	2001-2003 Air Quality						2004-2006 Air Quality					
	As is	Current std	Alternative 1-hour 98 <sup>th</sup> percentile standard				As is	Current std	Alternative 1-hour 98 <sup>th</sup> percentile standard			
			50	100	150	200			50	100	150	200
Atlanta	4	110	0	10	46	95	2	126	0	13	58	111
Boston	0	20	0	3	26	72	0	25	0	1	19	59
Chicago	9	74	0	18	83	168	5	69	0	10	59	138
Cleveland	5	134	1	24	104	196	ND					
Denver	17	157	0	19	99	198	10	134	1	38	148	239
Detroit	9	166	1	13	59	140	2	189	1	50	165	249
El Paso	4	168	0	17	89	180	3	198	0	23	108	197
Jacksonville	1	205	1	32	119	201	2	216	2	35	127	211
Las Vegas	4	106	0	19	82	143	2	99	1	22	83	138
Los Angeles	17	55	0	6	33	81	6	58	0	10	54	122
Miami	1	146	0	19	78	145	0	130	0	13	56	106
New York	7	36	0	10	57	132	5	45	0	14	75	157
Philadelphia	6	87	1	27	116	217	2	110	0	24	112	204
Phoenix	16	96	0	35	153	262	10	124	0	28	146	264
Provo	9	235	2	75	257	331	17	192	4	20	63	160
St. Louis	3	131	1	28	125	224	1	121	0	23	107	194
Washington DC	6	111	0	27	109	190	2	102	0	22	96	171
Other MSA	2	47	0	2	17	51	1	59	0	2	20	57
Other Not MSA	1	63	0	1	6	19	1	62	0	1	6	19

## 7.4 UNCERTAINTY ANALYSIS

*Uncertainty* refers to the lack of knowledge regarding both the actual values of model input variables (parameter uncertainty) and the physical systems or relationships (model uncertainty – e.g., the shape of the concentration-response functions). In any risk assessment, uncertainty is, ideally, reduced to the maximum extent possible, but significant uncertainty often remains. It can be reduced by improved measurement and improved model formulation. In addition, the degree of uncertainty can be characterized, ranging from qualitative to quantitative assessments. Uncertainty can be distinct from *variability*, which commonly refers to the heterogeneity in a population or variable of interest that is inherent and cannot be reduced through further research.

The approach for evaluating uncertainty was adapted from guidelines outlining how to conduct a qualitative uncertainty characterization (WHO, 2008). First, the key sources of the assessment that contribute to uncertainty are identified, and the rationale for why they are included is discussed. Second, a qualitative characterization follows for the types and components of uncertainty, resulting in a summary describing, for each source of uncertainty, the level and direction of influence the uncertainty may have on the air quality characterization results.

The overall characterization of uncertainty is qualitatively evaluated by considering the degree of severity of the uncertainty, implied by the relationship between the source of the uncertainty and the output of the assessment. To the extent possible, an appraisal of the knowledge base (e.g., the accuracy of the data used, acknowledgement of data gaps) and evaluation of the decisions made (e.g., selection of particular model forms) is also included in this uncertainty rating. The characterization is subjectively scaled by the assessors using a designation of low, medium, and high. Briefly, a *low* level of uncertainty suggests large changes within the source of uncertainty would have only a small effect on the results, there is completeness and scientific consistency in the knowledge base, and decisions made regarding the particular source of uncertainty would be widely accepted. A designation of *medium* implies that a change within the source of uncertainty would likely have a proportional effect on the results, there may be limited scientific backing, and limited selection of inputs or models to choose from. A characterization of *high* implies that a small change in the source would have a large effect on

results, there may be inconsistencies present in the scientific support, and assumptions made would be considered unusual and restrictive by others.

The bias direction indicates how the source of uncertainty has been judged to influence estimated concentrations, either the concentrations are likely *over-* or *under-estimated*. In the instance where two or more types or components of uncertainty are present that potentially offset the direction of influence, the bias has been judged as *both*. An *unknown* bias has been assigned where there was no evidence reviewed to judge the direction of uncertainty bias associated with the source. Table 7-31 provides a summary of the sources of uncertainty identified in the air quality characterization, the level of uncertainty, and the overall judged bias of each. A discussion regarding each of these sources of uncertainty and how conclusions were drawn is given in the sections that follow.

**Table 7-31. Summary of qualitative uncertainty analysis for the air quality and health risk characterization.**

Source	Type	Concentration/ Exceedance Bias Direction	Characterization of Uncertainty
Air Quality Data	Database quality	both	Low
Ambient Measurement	Interference	over	Low - Medium
Temporal Representation	Scale	none	Low
	Missing data	both	Low - Medium
	Years evaluated	both	Low
	Emission source changes	over	Low - Medium
Spatial Representation	Scale	unknown	Medium
	Monitor objectives	both	Medium
	Vertical siting of monitor	under	Low - Medium
	Monitor extrapolation < 4m	unknown	Low - Medium
Air Quality Adjustment	Proportional approach used	both	Medium - High
	Spatial scale	over	Medium
On-Road Simulation	Temporal scale	both	Medium - High
	Scenario modeled	over	Low - Medium
	Spatial scale	over	Low - Medium
	Exponential model	both	Medium - High
	Influential factors	unknown	Medium
	Distribution form	both	Low - Medium
	Non US studies used	unknown	Low - Medium
Health Benchmarks	Averaging time	none	Low - Medium
	Susceptibility	under	Medium

### 7.4.1 Air Quality Database

One basic assumption is that the AQS NO<sub>2</sub> air quality data used are quality assured already. Methods exist for ensuring the precision and accuracy of the ambient monitoring data (e.g., EPA, 1983). Reported concentrations contain only valid measures, since values with quality limitations are not entered to the system, removed following determination of being of lower quality or flagged. There is likely no selective bias in retention of data that is not of reasonable quality if the data are in error, it is assumed that selection of high concentration poor quality data would be just as likely as low concentration data of poor quality. Given the numbers of measurements used for this analysis, it is likely that even if a few low quality data are present in the data set, they would not have any significant effect on the results presented here. There are no alternative data sets available that are as comprehensive, and where monitoring data are available that are not included in the AQS, it is expected that given the same methods and quality assurances, would be complimentary to the data existing in the AQS. Therefore, the air quality data and database used likely contributes minimally to the uncertainty level, there is low uncertainty in the knowledge base, and the uncertainty in the subjectivity of choices is also considered low.

Temporally, the data are hourly measurements and appropriately account for variability in concentrations that are commonly observed for NO<sub>2</sub> and by definition are representative of an entire year. In addition, having more than one monitor does account for some of the spatial variability in a particular location. However, the degree of representativeness of the monitoring data used in this analysis can be evaluated from several perspectives, one of which is how well the temporal and spatial variability are represented. In particular, missing hourly measurements at a monitor may introduce bias (if different periods within a year or different years have different numbers of measured values) and increase the uncertainty. Furthermore, the spatial representativeness will be poor if the monitoring network is not dense enough to resolve the spatial variability (causing increased uncertainty) or if the monitors are not appropriately distributed to reflect population exposure (causing a bias). Additional uncertainty regarding temporal and spatial representation by the monitors is expanded below.

### **7.4.2 Measurement Technique for Ambient NO<sub>2</sub>**

One source of uncertainty for NO<sub>2</sub> air quality data is due to interference with other oxidized nitrogen compounds. Nitrogen dioxide is not directly measured. It is estimated by subtracting measured NO from total nitrogen oxides. The ISA identifies several studies conducted that have shown a constant positive interference when oxides of nitrogen other than NO<sub>2</sub> are present (ISA section 2.3). Most commonly the interference is from HNO<sub>3</sub> and has been reported to contribute to up to 50% the calculated NO<sub>2</sub>. This has been shown to occur primarily during the afternoon hours in the summer and would result in an overestimation of ambient NO<sub>2</sub> concentrations. During winter, positive interference in the measurement of NO<sub>2</sub> is estimated to be less, generally at 10% or lower. At any one particular site, however, there is uncertainty in how much the interference will be, and is dependent on the presence of the NO<sub>x</sub> compounds which are largely not measured. In addition, it is not known whether there is a concentration dependence on the amount of interference. This is an important uncertainty when air quality concentrations adjusted upwards to just meet the current standard. Therefore, the bias would be a consistent overestimation of NO<sub>2</sub> concentrations, the level of which may range from affecting the concentrations minimally upwards to a moderate effect. While the science demonstrating the interference is consistent, there remain uncertainties about the application of the level of interference to individual monitoring sites.

### **7.4.3 Temporal Representation**

Data are valid hourly measures and are of similar temporal scale as identified health effect benchmark concentrations. There are frequent missing values within a given valid year which contribute to the uncertainty as well as introducing a possible bias if some seasons, day types (e.g., weekday/weekend), or time of the day (e.g., night or day) are not equally represented. Since a 75 percent daily and hourly completeness rule was applied, some of these uncertainties and biases were reduced in these analyses. Additional validity criteria could have included completeness for monitoring based on quarters, rather than the entire year. This would screen for air quality data potentially missing an entire season of monitoring. The use of validity criteria that included quarterly completeness would likely exclude a few monitor site-years of data considered in the current analysis. This would likely have a greater effect at locations with

fewer site-years of air quality if large numbers of missing data exist in a quarter, although in some locations, actual seasons may be something other than a quarterly classification.

Ambient monitoring data were not interpolated to substitute for any missing values. It is assumed that missing values are not systematic, i.e., high concentrations would be absent as well as low concentrations in equal proportions. There are methods available that can account for time-of-day, day-of-week, and seasonal variation in ambient monitoring concentrations. However, if a method were selected it would have to not simply interpolate the data, but accurately estimate the probability of peak concentrations as well. It was judged that if such a method was available or one was developed to substitute data, it would likely add to a similar level of uncertainty as not choosing to substitute the data. Again, this can be viewed as having a limited effect on uncertainty because using the validity criteria should select for the most representative and complete ambient monitoring data sets possible.

There may be bias and uncertainty in the air quality characterization results if the years monitored vary significantly between locations. Although monitoring locations within a region do change over time, the NO<sub>2</sub> network has been reasonably stable over years 1995-2006, particularly at locations with larger monitoring networks. While it is possible for monitors to move from high concentration areas to low concentration areas or perhaps in the other direction, regulations exist that specify the design and measurement requirements for these networks (e.g., 40 CFR Part 58). Given this, it is expected that the level of uncertainty in the specific monitors operating from year to year is low with a variable bias direction of over-estimation for some years and under-estimation for others.

It should also be noted that use of the older data in some of the analyses here assumes that the sources present at that time are the same as current sources, adding uncertainty to results if this is not the case. Separating the data into two 6-year groups (historical and recent for the *as is* evaluation) and two additional subsets of the recent air quality (2001-2003 and 2004-2006) before analysis reduces the potential impact from changes in national- or location-specific source influences and is judged to have a minimal bias in representing air quality concentrations for those selected years. There is some variability expected from year-to-year, that is, there may be differences in the air quality results if the year-groups included a different 3-year period, such as 2002-2004 or 2003-2005. Deciding to bound the total period rather than characterize all possible

3-year combinations was judged appropriate, given the small differences in the observed results over time and the resources available for the analysis.

#### 7.4.4 Spatial Representation

Relative to the physical area, there are only a small number of monitors in each location. Even considering sparse siting, the monitoring data are assumed to be spatially representative of the locations analyzed here because the monitors are used in determining whether areas meet or do not meet the NAAQS. This could include areas between the ambient monitors that may or may not be influenced by similar local sources of NO<sub>2</sub>. For these reasons the uncertainty and bias due to the spatial network may be moderate, although the monitoring network design should have addressed these issues within the available resources and other monitoring constraints. Bias will depend on ambient monitoring objectives and scale and whether there is large variability in monitoring surface, i.e., areas of differing terrain that are not well represented by the distribution of monitors. The direction of this bias is largely unknown due to the differences in the true representativeness of the network and the particular terrain in each location. In addition, the air quality characterization used all monitors meeting the 75 percent completeness criteria, without taking into account the monitoring objectives or land use for individual monitors. Thus, there will be some lack of spatial representation and uncertainty due to the inclusion/exclusion of some monitors that are very near local sources (including mobile sources) potentially resulting in either over- or under-estimations.

According to one study conducted in the South Bronx, NY in November and December 2001, negative vertical gradients can exist for monitor concentrations (ISA, section 2.5.3.3). On average, measured concentrations can be 2.5 times higher at 4 meters than at a 15 meter vertical monitor siting. Therefore, monitors positioned on building rooftops may underestimate NO<sub>2</sub> concentrations at lower vertical heights and possibly at the standard breathing height of 1.8 m. In this REA, only 7 of the 178<sup>10</sup> monitors in the named locations contained monitoring heights of 15 meters or greater, with 58% at 4 meters or less height, and 79% at 5 meters or less in height. In the aggregate locations (i.e., *Other MSA*, *Other Not MSA*), a total of 4 monitors of 340<sup>11</sup> contained monitoring heights of 15 meters or greater, with 50% at 4 meters or less in height, and

---

<sup>10</sup> 26 monitors in the named locations did not have height reported (therefore,  $178 + 26 = 204$  total number of monitors).

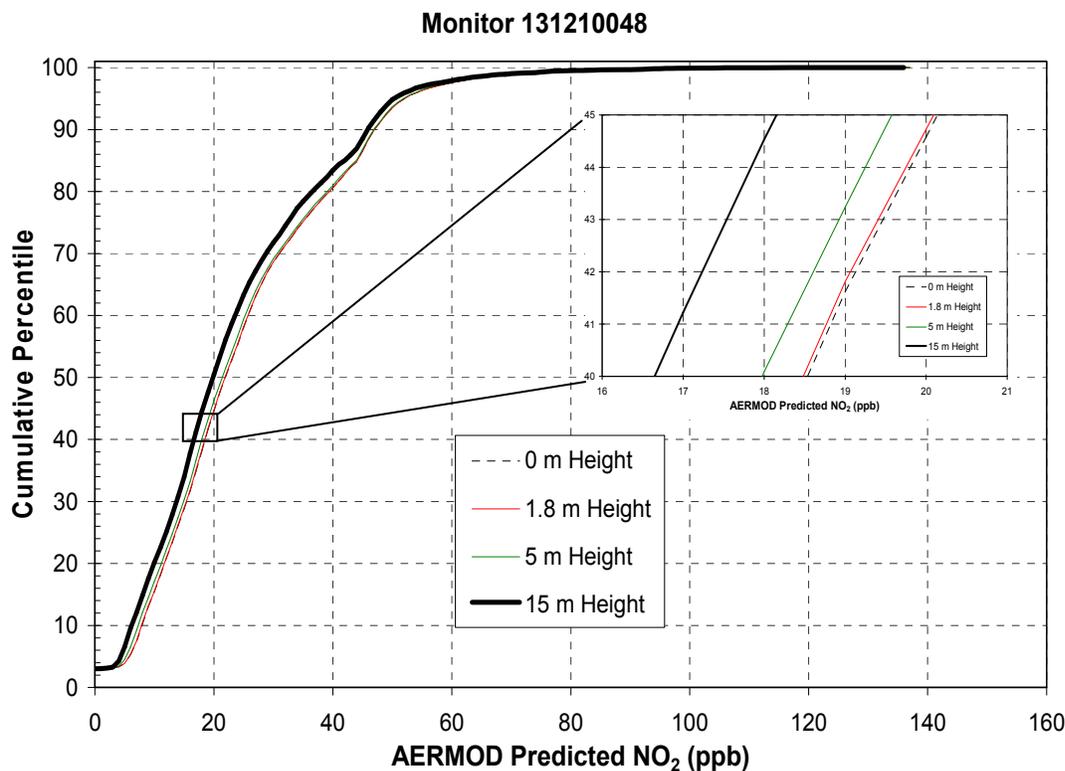
<sup>11</sup> 84 monitors in the aggregate locations did not have height reported (therefore,  $340 + 84 = 424$  total number of monitors).

73% at 5 meters above ground. Not accounting for this potential vertical gradient in NO<sub>2</sub> concentrations may generate underestimates of exceedances for some sites, however the overall impact of inferences made for the locations included in this assessment without considering vertical monitor height is likely minimal since most monitors used for analysis are sited at less than 4-5 meters above ground. In addition, the relationship at heights below 4 meters is largely uncertain (e.g., a breathing height of 1.8 meters is commonly used) and therefore would add an additional unknown bias to the estimated NO<sub>2</sub> concentrations above a benchmark when used as a surrogate for human exposure.

Staff evaluated the potential bias in vertical siting of monitors by using the AERMOD predictions at four receptor heights for each of the three ambient monitors located in the Atlanta exposure modeling domain (monitors 130890002, 130893001, and 131210048), each located at heights 0, 1.8, 5, and 15 meters above ground. An example of the predicted hourly NO<sub>2</sub> concentration distributions at each receptor height for one site (ID 131210048) is presented in Figure 7-9, and is similar to that predicted at each of the other monitors. Consistent with the one study reported in the ISA, the estimated concentrations at the 15 meter monitoring height were the lowest, with progressively greater concentrations with decreasing receptor height. However, the level of differences in concentration at each of the different receptor heights were lower using the modeled concentrations in Atlanta than when compared with those reported for the South Bronx using the measured ambient concentrations. On average, the NO<sub>2</sub> concentrations estimated at a 0 m height were >0.2% than those at a height of 1.8 m, while the largest difference in concentration (7.9%) occurred with comparison of the 0 m to the 15 m height (Table 7-32). When comparing concentrations for each of the receptor heights at the upper tails of the distribution, occasionally a similar pattern was observed, i.e., small increases in the numbers of exceedances of a selected benchmark level (Table 7-33). At one location however (ID 130893001), there were no differences in the number of exceedances of the selected benchmark level.

These modeling results support the ISA cited measurement study in that there is an inverse relationship between monitor vertical siting height and NO<sub>2</sub> concentration, only the magnitude of the relationship differs. The lack of a similar magnitude could be the result of several factors such differing influential features of the study area versus the modeled area (e.g., seasonal/meteorological factors, presence of nearby sources, terrain) or perhaps a limited

sensitivity of the model to variable receptor height. Based on these two limited evaluations, there can be no clear determination as to whether the monitor vertical siting effect is as large as the single study estimated or as small as the dispersion model predicted. Further, since there are limited measurement and model results available to inform a decision and that there were few monitors used above 5 m in vertical height, staff did not adjust concentrations using vertical siting characteristics.



**Figure 7-9. Distribution of 1-hour NO<sub>2</sub> concentrations for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality.**

**Table 7-32. Percent difference in 1-hour NO<sub>2</sub> concentrations for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality.**

Monitor ID	% Difference in 1-hour NO <sub>2</sub> (Lower to higher vertical height)		
	0 m to 1.8 m	1.8 m to 5 m	0 m to 15 m
130890002	0.2	0.9	6.7
130893001	0.1	0.6	4.6
131210048	0.1	1.6	7.9

**Table 7-33. Number of 1-hour NO<sub>2</sub> concentrations above 100 ppb for three modeled receptors in Atlanta at different vertical heights, using AERMOD predicted 2002 air quality.**

Monitor ID	Number of 1-hour NO <sub>2</sub> ≥ 100 ppb (AERMOD Estimated NO <sub>2</sub> , Year 2002)			
	0 m	1.8 m	5 m	15 m
130890002	21	21	21	19
130893001	2	2	2	2
131210048	12	12	11	10

#### 7.4.5 Air Quality Adjustment Procedure

There is uncertainty in the air quality adjustment procedures due to the uncertainty of the true relationship between the adjusted concentrations that are simulating a hypothetical scenario and the *as is* air quality. The adjustment factors used for the current and alternative standards each assumed that all hourly concentrations will change proportionately at each ambient monitoring site. Two principal uncertainties are discussed, namely uncertainty regarding the proportional approach used and the universal application of the approach to all ambient monitors within each location.

Different sources have different temporal emission profiles, so that equally applied changes to the concentrations at the ambient monitors to simulate hypothetical changes in emissions may not correspond well with all portions of the concentration distribution. When adjusting concentrations upward to just meeting the current standard, the proportional adjustment used an equivalent multiplicative factor derived from the annual mean concentration and equally applied to all portions of the concentration distribution, i.e., the upper tails were treated the same as the area of central tendency. This may not necessarily reflect changes in an overall emissions profile that may result from, for example, an increase in the number of sources in a location. It is possible that while the mean concentration measured at an ambient monitor may increase with an increase in the source emissions affecting concentrations measured at the monitor, the tails of the hourly concentration distribution might not have the same proportional increase. The increase could be greater or it could be less than that observed at the mean, dependent largely on the type of sources and inherent operating conditions. Adjusting the ambient concentrations upwards to simulate the alternative standards also carries a similar level of uncertainty although the multiplicative factors were derived from the upper percentiles of the 1-hour NO<sub>2</sub> concentrations, rather than the mean, and then applied to the 1-hour NO<sub>2</sub> concentrations equally.

In each of these instances of adjusting the concentrations upwards, one could argue that there may be an associated over-estimation in the concentrations at the upper tails of the distributions, possibly leading to over-estimation in the numbers of exceedances of benchmark levels. An analysis was performed on monitors within six of the locations used in the air quality characterization to investigate how distributions of hourly nitrogen dioxide concentrations have changed over time (Rizzo, 2008). The analysis indicates that a proportional approach can be appropriate in simulating higher concentrations at most monitoring sites, since historically, NO<sub>2</sub> concentrations have decreased linearly across the entire concentration distribution at each of the monitoring sites and locations evaluated. In addition, when adjusting concentrations downward (e.g., the alternative standard level of 50 ppb 1-hour, 99<sup>th</sup> percentile), the use of the proportional multiplicative adjustment derived from the upper tails and applied to concentration distribution may be less uncertain because NO<sub>2</sub> concentrations have been observed to decrease linearly over time, and only assumes that the downward trend would continue similarly in the future with added source controls.

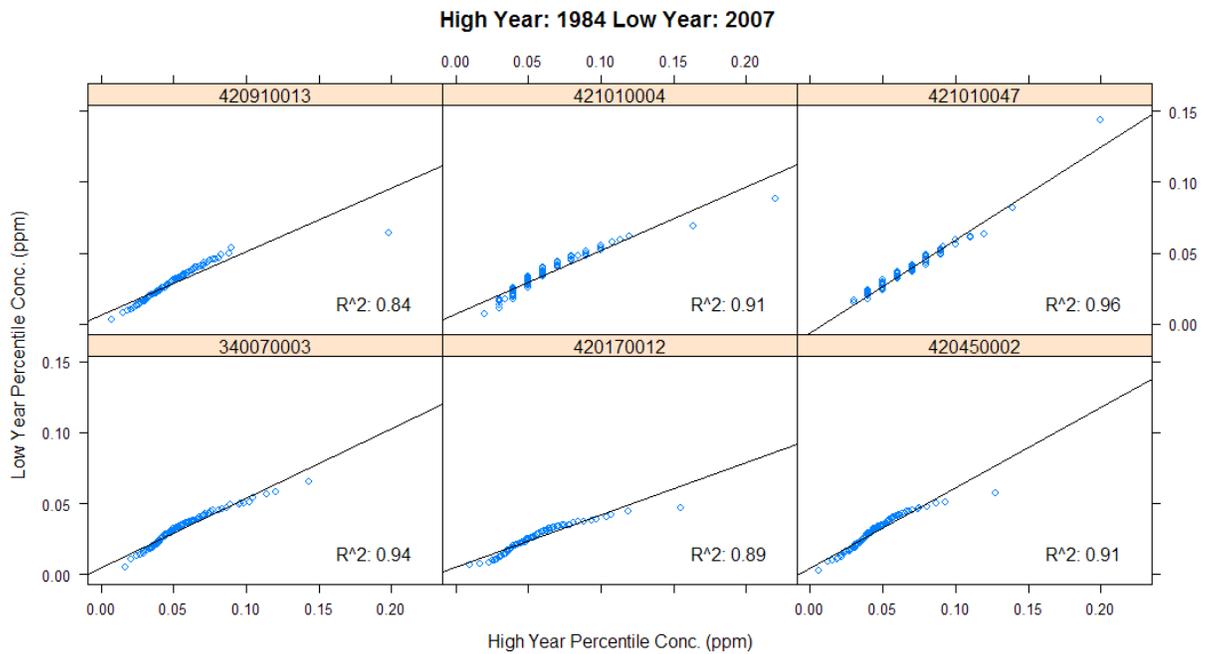
At some of monitoring sites analyzed however, there were features not consistent with a completely proportional relationship, including deviation from linearity primarily at the maximum or minimum percentile concentrations, some indication of curvilinear relationships, and the presence of either a positive or negative regression intercept (Rizzo, 2008). Where multiple monitors were present in a location there tended to be a mixture of each of these condition, including proportionality. Not all of the locations analyzed as part of the air quality characterization were included in the evaluation. It was also assumed that the analysis conducted at the six locations would reflect what would be observed at the other locations if evaluated for trends in concentration over long periods of time. High concentration year to low concentration year comparisons were also limited to 3-years to generate appropriate and a manageable number of comparisons. It was assumed that if additional years of data were compared that similar relationships would be developed. Further, there is uncertainty in adjusting concentrations upwards or downwards considering assumptions regarding future source emission scenarios and how these would relate to observed trends in current and historical air quality. The uncertainty about future source emission scenarios is largely unknown.

Universal application of the proportional simulation approach for each of the locations and within each location was done for consistency and was designed to preserve the inherent

variability in the concentration distribution. There is uncertainty regarding emission changes that would affect the concentrations at the design monitor containing the highest concentration (annual mean, 98<sup>th</sup> or 99<sup>th</sup> percentile 1-hour) that may not necessarily affect lower concentration sites proportionately. This could result in either over- or under-estimations in the number of exceedances at lower concentration sites within a location where the current or alternative standard scenarios were evaluated. When comparing the low concentration years and the high concentration years at multiple ambient monitors within a location however, most monitors contained similar linear relationships (e.g., comparative regression slopes and intercepts). For example, Figure 7-10 shows the daily maximum NO<sub>2</sub> concentration percentiles for four ambient monitors in Philadelphia, where each of 6 ambient monitors were in operation for years 1984 and 2007. The similarity in slope for each of the monitors indicates that an adjustment factor derived from one ambient monitor can be applied to the other monitors in the monitoring network. Furthermore, when calculating the number of exceedances of the potential health effect benchmark levels, the greatest numbers of exceedances typically were noted at the monitoring sites with the highest concentrations within the location (Appendix A, section 7), with little contribution from the low concentration sites within a location. A few locations though were noted that may have an exceptional number of estimated exceedances as a result of the air quality adjustment approach, particularly those locations with few monitoring sites that contained very low concentrations and/or atypical variability in hourly concentrations. These few locations (e.g., Miami, Jacksonville, and Provo) may contain overestimations at the upper tails of the concentration distribution, leading to bias in estimated number of exceedances at both the upper percentiles and the mean when using the air quality simulated to just meet the current and most of the alternative standards. It should also be noted that where deviations from proportionality occur, the magnitude of the uncertainty in the results is likely related to the magnitude of the extrapolation to the adjusted concentration level. This means that there is likely greater uncertainty in the results for evaluating the current annual and the 200 ppb 98<sup>th</sup> percentile alternative standards, than when considering the 50 ppb and 100 ppb 99<sup>th</sup> percentile alternative standard.

Given the limited deviations in linearity and proportionality at each monitor site that may result in both over- or under-estimations in concentrations following either an adjustment upwards or downwards and the limited time and resources available to develop a new universal

approach that addresses each of the observed deviations, staff judged the proportional approach used to simulate just meeting the current and alternative standards as adequate and appropriate for the scenario considered.



**Figure 7-10. Comparison of measured daily maximum NO<sub>2</sub> concentration percentiles in Philadelphia for one high concentration years (1984) versus a low concentration years (2007) at four ambient monitors.**

#### 7.4.6 On-Road Concentration Simulation

On-road and ambient monitoring NO<sub>2</sub> concentrations have been shown to be correlated significantly on a temporal basis (e.g., Cape et al., 2004) and motor vehicles are a significant emission source of NO<sub>x</sub>, providing support for estimating on-road concentrations using ambient monitoring data. The relationship used in this analysis to estimate on-road NO<sub>2</sub> concentrations was derived from data reported in measurement studies containing mostly long-term averaging times, typically 7-14 days or greater in duration (e.g., Roorda-Knape, 1998; Pleijel et al., 2004; Cape et al, 2004). One study was conducted over a one-hour time averaging period however the results were reported for time-averaging of at least 1-day (Rodes and Holland, 1981). Use of such data is considered appropriate in this analysis to estimate on-road hourly concentrations from hourly ambient measures, assuming a direct relationship exists between the short-term

peaks and time-averaged concentrations (e.g., hourly on-road NO<sub>2</sub> concentrations are correlated with 24-hour averages). While this should not impact the overall contribution relationship between vehicles and ambient concentrations on roads, the relationship will likely differ for shorter averaging times. However, the longer-term data used to develop the algorithm were likely collected during variable conditions (e.g., shifting wind direction, variable diurnal rate of transformation of NO to NO<sub>2</sub>) than would be observed across shorter time periods. Therefore, distribution of the adjustment factors based on 7-14 day averaging-times may be biased at the tails, that is, upper percentile values used may be biased low and the lower percentiles may be biased high. This could result in either over- or under-estimations of 1-hour NO<sub>2</sub> concentrations, depending on the time of day. In addition, the ambient concentration level could not be considered in the application of the on-road factor to the hourly concentration because the relationship between the derived adjustment factor and the ambient concentration on that temporal scale is unknown. If there is a concentration dependent relationship, this would bias the estimated on-road concentrations with unknown direction. Application of the on-road concentration estimation also assumes that concentration changes that occur on-roads and at the monitor are simultaneous (i.e., within the hour time period of estimation). While this may not be the case, because time-activity patterns of individuals are not considered in this air quality benchmark characterization, there would be no bias in the number of estimated exceedances.

If assessing personal exposures to individuals near roadways or within vehicles that are traveling on a road, it is likely that their exposure concentrations would also vary due to differing roadway concentrations. There was limited data available in the development of the on-road adjustment factor for differing road-types and most of the data used were reported for urban areas (Appendix A, Table A-108). The factors developed should be appropriate for use in urban areas, as was done in this REA, however representation of all road-types (e.g., freeways, arterials, or local roads) in the urban areas modeled is largely unknown. On-road concentrations were not adjusted in this analysis to account for in-vehicle penetration and decay. Therefore, in-vehicle concentrations would be overestimated if using the estimated on-road concentrations as a surrogate, given that reactive pollutants (e.g., PM<sub>2.5</sub>) tend to have a lower indoor/outdoor (I/O) concentration ratio (Rodes et al., 1998). One study reported mean (I/O) ratios of NO<sub>2</sub> for a few roadways and driving conditions in Hong Kong (Chan and Chung, 2003). On highways and

urban streets, the I/O is centered about 0.6 to 1.0, indicating removal of outdoor NO<sub>2</sub> as it enters inside a vehicle.

At locations where traffic counts are very low (e.g., on the order of hundreds/day) the on-road contribution has been shown to be negligible (Bell and Ashenden, 1997; Cape et al., 2004), therefore any monitors sited in rural areas near roads with minimal traffic volumes could result in small overestimations of NO<sub>2</sub> concentrations when using equation (7-2) at these locations. This is not of great concern because most of the monitors used in the on-road simulation were sited within large CMSA/MSA, likely encompassing urban/suburban features of a location rather than rural areas. Monitors sited within 100 m of the roadway in the named locations were not used in the calculation of on-road concentrations due to the possibility of these monitors already accounting for notable impact from vehicle emissions (e.g., Beckerman et al., 2008), thus controlling for over-estimating the on-road concentrations. However, there is potential for influence by non-road source emissions on the measured concentrations at the monitors used ( $\geq$  100 m from a major road), contrary to an assumption that there is an absence of direct source influence (only that mobile sources were controlled for by selecting these monitors). Therefore, if using ambient monitors directly affected by emissions from non-road sources, the simulated on-road concentrations may be over-estimated. For example, the estimated number of on-road exceedances was greater for Jacksonville and Provo than at the other locations, even though all on-road simulations used monitors sited  $\geq$ 100 m from a major road. The predominant land use however for both of these monitors was commercial, although monitoring objective and measurement scale were unknown. In addition, the ambient monitors in the aggregate locations (i.e., Other MSA, Other Not MSA) did not have distances to major roads calculated and may include a number of site-years of data from monitors <100 m from a major road. The estimated number of daily maximum on-road exceedances may be over-estimated for these aggregate locations.

Another source of uncertainty in the spatial heterogeneity of NO<sub>2</sub> concentrations regards the presence of street canyons on roadways. These localized areas may be subject to highly variable and higher mean concentrations within a short span of a road, often defined by the presence of man-made structures, such as buildings, on both sides of the road. In one study, a comparison of street canyon measured NO<sub>x</sub> concentrations with those measured at a reference site (termed background) indicated that there is about a factor of 2.3 difference in the

concentrations (Ghenu et al., 2007). Vardoulakis et al. (2004) reported mean NO<sub>2</sub> concentrations at a major intersection can be a factor of about 2.1 times greater than on-road concentrations measured at a few hundred meters distance within a street canyon.<sup>12</sup> Because these factors are within the range of adjustment factors used here in estimating the on-road concentration, i.e., ranging from a factor of 1.2 to 3.7 times the ambient concentrations, it is likely that some of the estimated on-road concentrations in the air quality characterization are similar in magnitude to those found in street canyons.

To represent the relationship between the on-road concentrations and the concentrations measured at a distance, a simple exponential model was used. The selection of an exponential model was based on independent peer-reviewed studies that reported this type of relationship using NO<sub>2</sub> measurements. There are uncertainties and possible biases with the selection of the exponential model. For example, NO<sub>x</sub> is primarily emitted as NO (e.g., Heeb et al., 2008; Shorter et al., 2005), with substantial secondary formation due predominantly to NO + O<sub>3</sub> → NO<sub>2</sub> + O<sub>2</sub>. Numerous studies have demonstrated the O<sub>3</sub> reduction that occurs near major roads, reflecting the transfer of odd oxygen to NO to form NO<sub>2</sub>, a process that can impact NO<sub>2</sub> concentrations both on- and downwind of the road. Some studies report NO<sub>2</sub> concentrations increasing just downwind of roadways and that are inversely correlated with O<sub>3</sub> (e.g., Beckerman et al., 2008), suggesting that peak concentration of NO<sub>2</sub> may not always occur on the road, but at a distance downwind. While an exponential model may fit well (or for portions of the data), the peak may be occurring at a distance from the road rather than on the road. Model convergence was one criteria used in selecting for useful parameter estimates. One of the principal reasons for lack of convergence is that the measurement data did not fit the exponential form considered. Therefore, if the study measurement data contained peak concentrations at a distance of the road and other lower concentrations closer to the road and along the transect, it was likely that there were no valid parameters estimated for that study data. It follows that for studies where the nearest roadway distance was not at the edge of the road and the overall concentrations pattern is to increase with decreasing distance from the road, the estimated on-road adjustment factors may be biased high. This would occur when the concentration pattern followed the exponential model well, peaking at the nearest road measurement, but in the absence of additional measurements closer to the road, the model assumes a further increase in concentrations with

---

<sup>12</sup> Ambient concentrations at a site not influenced by mobile sources were not reported by Vardoulakis et al. (2004).

decreased distance to the road. Therefore, the uncertainty regarding where the peak concentration occurs (on-road or at a distance from the road) in combination with the form of the exponential model (the highest concentration occurs at zero distance from road) and the selection of studies that fit the exponential model, may also add a moderate level of uncertainty in the estimated on-road concentrations and the number of exceedances.

The manner in which the on-road adjustment factor distribution was constructed and applied also introduces uncertainty to the results. Based on the few influential variables available from the on-road studies, the number of values derived for the adjustment factors (n=41), a comparison of the distributions that would result in considering the potentially influential variables, and considering how the factors would be applied to the ambient monitoring data, it was decided that a two season categorization was the most appropriate characterization of the derived data (Appendix A, section 8.2). There is some bias in the application of the season categorization due to the presence of on-road adjustment factors derived from annual average data within the *not summer* distribution. Staff judged that on-road adjustment factors that included four seasons (spring, fall, summer, and winter) would be more reflective of expected conditions during non-summer months rather than during summer months. Rather than excluding 27% of the derived ratios, staff decided to retain the ratios and include them in the *not summer* category.

Using an un-weighted mixture of urban and rural on-road adjustment factors within each season category assumes that the distribution of each appropriately reflects the balance of these factors within each location. First, some of the studies used in developing the on-road adjustment factors did not distinctly report whether the data were from urban, suburban, or rural areas. There is a moderate level of uncertainty in the judgment by staff in characterizing the reported study data as urban or rural, an uncertainty that was used to support the decision to not characterize the on-road adjustment factor distribution based on this potentially influential factor. Second, the values for the *m* ratios derived from studies either described as by the original researchers or inferred by staff as rural areas (min 0.36, median 0.89, max 2.44) were comparable to those described as by the original researchers or inferred by staff as urban areas (min 0.21, median 0.74, max 2.70). And finally, given the schedule and resources available to produce the REA, the uncertainty in the categorization by area type, and little difference between

the potential distributions, staff decided not to stratify the on-road adjustment factors by area type.

Each season category was represented by an empirical distribution, with each value from the distribution having an equal probability of selection. While there may be other distribution forms that could be alternatively selected, staff judged that use of a fitted distribution would not improve the representation of the true population of  $m$  values compared with an empirical form, and that there would likely be minimal effect on the estimated number of exceedances. Use of an empirical distribution was done because neither season group of data could be assigned to a particular distribution type (e.g., normal, lognormal, exponential, gamma; see Figures 7-11 and 7-12), although the *summer* data set was significantly different from a normal distribution ( $p < 0.01$ ). While there is uncertainty associated with the use of the empirically-derived data in representing the true population of  $m$  values, assuming a fitted distribution is not without its own uncertainties. For example, using a lognormal distribution may underestimate the observed frequency of certain values of  $m$  (Figures 7-11 and 7-12), and while allowing for values outside of the empirical distribution, would still need to have realistic bounds placed on the minimum and maximum values, further adding to uncertainty regarding the shape and form of the fitted distribution. In addition, allowing for the selection of on-road adjustment factors outside of the range of the empirical data using a fitted distribution would have a low frequency such that the overall impact to the estimated on-road NO<sub>2</sub> concentrations would likely be limited (Table 7-34). Furthermore, the  $m$  ratios were derived from measurement data, they are not actual measurements but are measurement-based. Fitting a distribution from the modeled data may also add to the existing model uncertainty. Each of these factors mentioned (the number of samples, uncertainty in the limits and shape of the distribution, fitting distributions to modeled data) were considered and it was decided by staff that the empirical distribution derived from the measurement data would be most representative.

Staff did however investigate the affect on the number of exceedances when using an alternative fitted distribution. Lognormal distributions were selected, with lower and upper bounds of the on-road adjustment factor defined by the 0.5<sup>th</sup> and 99.5<sup>th</sup> percentiles (Table 7-34). On-road adjustment factors were obtained by sampling from the fitted  $m$  distribution for each season<sup>13</sup>, and then adding 1 (see equation 7-2). The on-road simulation was performed using this

---

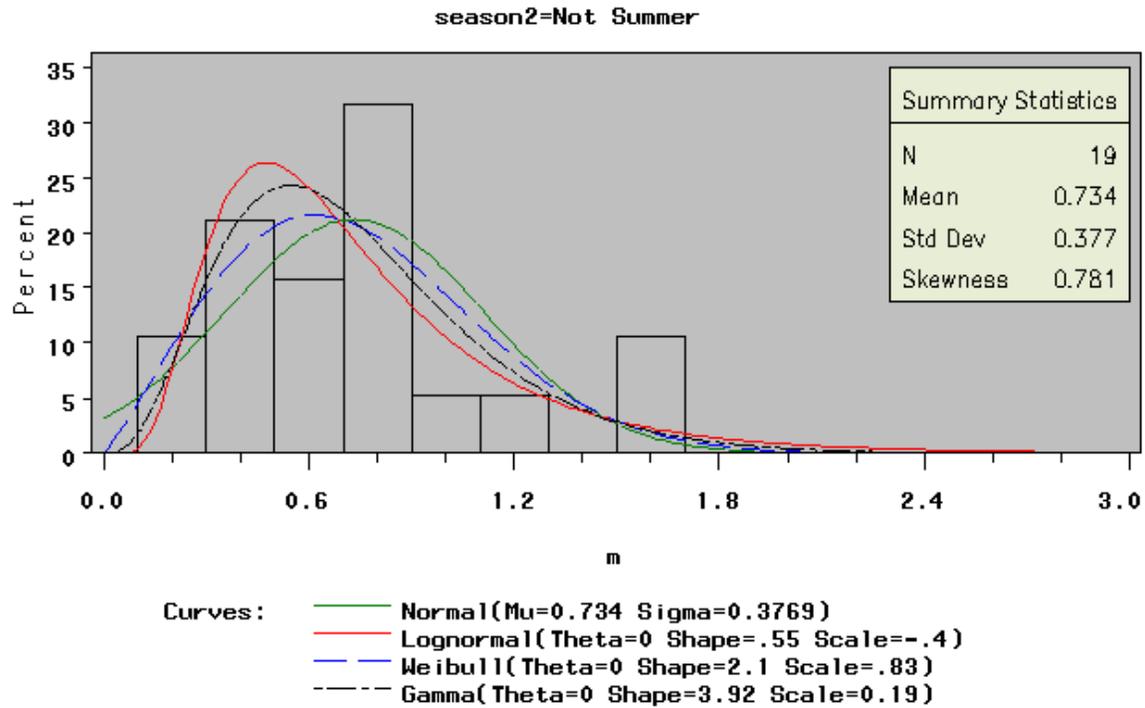
<sup>13</sup> {geometric mean, geometric standard deviation}: Summer {0.989, 1.65}, Not summer {0.643, 1.73}.

fitted distribution in the same manner done using the empirical distribution (section 7.2.4) using 2004-2006 air quality. The average number of estimated exceedances at 100, 200, and 300 ppb were compared with those generated using the empirical distribution by taking the difference of the two exposure estimates. In general, the difference between the estimated number of exceedances obtained using the two different distributions was small when considering unadjusted air quality (Table 7-35). Most locations had between 0 to 3 additional daily maximum on-road exceedances at the 100 ppb benchmark using the fitted lognormal distribution, with no difference in daily maximum exceedances at the 200 ppb or 300 ppb level. Differences between the two on-road simulations were similarly small considering air quality adjusted to just meet the current annual standard, although differences were also present at the higher benchmark levels. In addition, the differences in the two simulations were variable at each location, that is, at some locations, the lognormal distribution generated a greater number of exceedances (e.g., El Paso, New York), while at other locations a fewer number of exceedances (e.g., Atlanta, Detroit) than when using the empirical distribution.

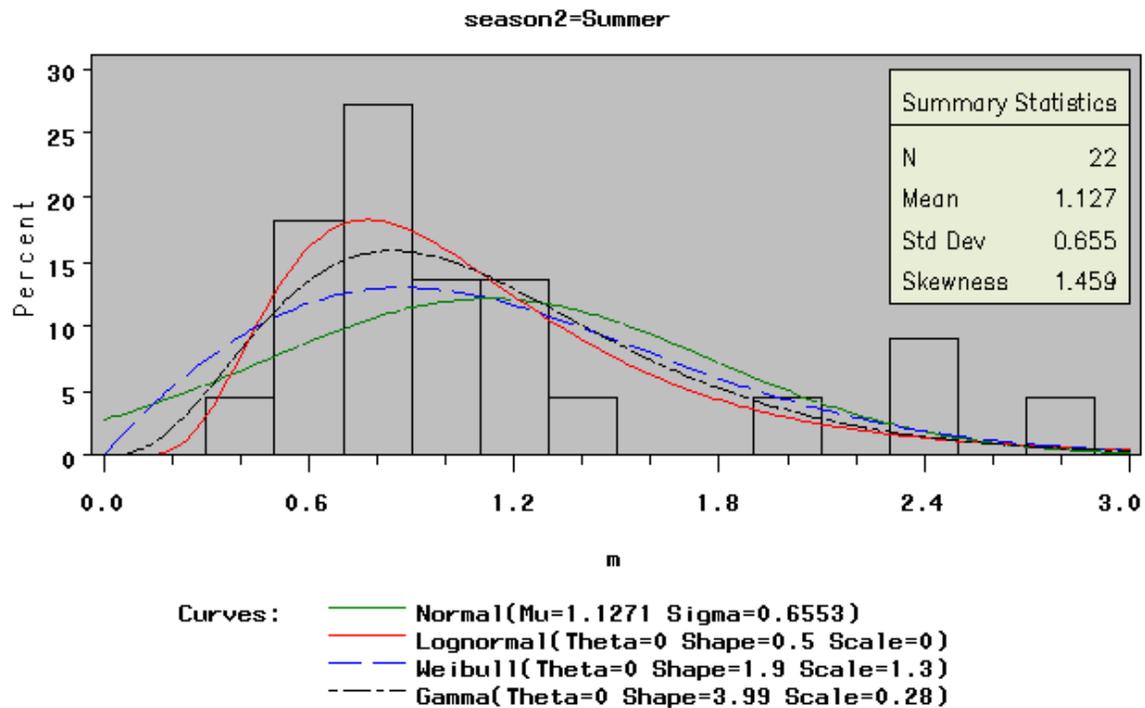
**Table 7-34. Comparison of empirical distribution of on-road adjustment factors used in on-road concentration estimation with a fitted lognormal distribution.**

Season Group	Percentile	Distribution Form	
		Empirical	Lognormal
Not Summer	0.5	1.22	1.16
	1.0	1.22	1.18
	<b>2.3</b>	1.22	<b>1.22</b>
	2.5	1.22	1.22
	5.0	1.22	1.26
	<b>94.4</b>	2.50	<b>2.54</b>
	95.0	2.54	2.59
	97.5	2.54	2.88
	99.0	2.54	3.30
Summer	99.5	2.54	3.64
	0.5	1.49	1.27
	1.0	1.49	1.31
	2.5	1.49	1.37
	5.0	1.51	1.44
	<b>7.8</b>	1.51	<b>1.49</b>
	95.0	3.45	3.25
	97.5	3.70	3.63
	<b>97.8</b>	3.70	<b>3.70</b>
99.0	3.70	4.16	
	99.5	3.70	4.58

**Notes:**  
 Bold font indicates percentile where empirical distribution minimum and maximum intersect with a fitted lognormal distribution.  
 On-road adjustment factors are  $1+m$ , see section 7.2.4 of REA.



**Figure 7-11.** Comparison of the distribution of estimated  $C_v/C_b$  ratios or  $m$  for the *not summer* category with fitted distributions.



**Figure 7-12.** Comparison of the distribution of estimated  $C_v/C_b$  ratios or  $m$  for the *summer* category with fitted distributions.

**Table 7-35. Absolute difference in the estimated number of exceedances of potential health effect benchmarks on-roads using either a fitted lognormal distribution or empirical distribution of the on-road adjustment factors and 2004-2006 air quality *as is* and air quality adjusted to just meet the current annual standard.**

Location	Difference in Mean Number of Daily Maximum Exceedances <sup>2</sup>					
	As Is			Current Annual Standard		
	≥100 ppb	≥ 200 ppb	≥ 300 ppb	≥100 ppb	≥ 200 ppb	≥ 300 ppb
Atlanta	0	0	0	0	2	1
Boston	0	0	0	0	2	0
Chicago	-1	0	0	0	-2	0
Denver	-1	0	0	0	0	-1
Detroit	-1	0	0	1	2	3
El Paso	-2	0	0	-1	-1	-1
Jacksonville	0	0	0	-1	-2	-4
Las Vegas	0	0	0	0	1	1
Los Angeles	0	0	0	0	1	0
Miami	-2	0	0	-1	0	-1
New York	-3	0	0	-1	-3	-1
Philadelphia	-1	0	0	0	-1	-1
Phoenix	0	0	0	0	-3	2
Provo	-9	-1	0	-3	-3	-11
St. Louis	1	0	0	0	3	-1
Washington DC	0	0	0	0	1	-1
Other MSA	0	0	0	0	0	0
Other Not MSA	0	0	0	0	1	1

**Notes:**  
<sup>1</sup> Differences are obtained by subtracting on-road exposure results using fitted lognormal distribution from the results obtained using an empirical distribution.

Another source of uncertainty is the extent to which the near-road study locations used to derive the on-road simulation factors represent the locations in these analyses. The on-road and near-road data were collected in a few locations, most of them outside of the United States. The source mixes (i.e., the vehicle fleet) in study locations may not be representative of the U.S. fleet. Without detailed information characterizing the emissions patterns for the on-road study areas, there was no attempt to match the air quality characterization locations to specific on-road study areas, which might improve the precision of the estimates. When considering the two U.S. studies containing the required measurement data (Rodes et al. 1981; Singer et al.; 2004), three *m* ratios were estimated (i.e., 0.93, 1.54, and 2.43) similar in range with the ratios estimated using data obtained from non-U.S. studies. This evidence implies that the level of uncertainty in

applying the non-US studies for the purposes of this analysis may not be large, although there is limited data available to make this judgment.

#### **7.4.7 Health Benchmark**

The choice of potential health effect benchmarks, and the use of those benchmarks to assess risks, can introduce uncertainty into the risk assessment. For example, the potential health effect benchmarks used were based on studies where volunteers were exposed to NO<sub>2</sub> for varying lengths of time. Typically, the NO<sub>2</sub> exposure durations were between 30 minutes and 2 hours. This introduces some uncertainty into the characterization of risk, which compared the potential health effect benchmarks to estimates of exposure over a 1-hour time period. Therefore, the use of a 1-hour averaging-time could either over- or under-estimate risks.

In addition, the human exposure studies evaluated airways responsiveness in mild asthmatics. For ethical reasons, more severely affected asthmatics and asthmatic children were not included in these studies. Severe asthmatics and/or asthmatic children may be more susceptible than mildly asthmatic adults to the effects of NO<sub>2</sub> exposure. Therefore, the potential health effect benchmarks based on these studies could underestimate risks in populations with greater susceptibility.

## 7.5 KEY OBSERVATIONS

Presented below are key observations resulting from the air quality characterization:

- NO<sub>2</sub> concentrations and estimates of benchmark exceedances are typically higher for monitors that are within 20 m of a major roadway than when monitors are farther (i.e., between 20 m and 100 m or  $\geq 100$  m) from a major roadway.
- Estimated on-road annual average NO<sub>2</sub> concentrations, based on simulated air quality, are, on average, 80% higher than the respective ambient levels at distances  $\geq 100$  m from a road. This falls within the range of on-road to distant monitor concentration ratios reported in the ISA (about 2-fold higher concentrations on-roads) (ISA, section 2.5.4).
- For unadjusted air quality, representing a recent year, many locations are estimated to have, on average, 0 days per year where the 1-hour daily maximum ambient NO<sub>2</sub> concentrations are  $\geq 100$  ppb. Only one location is estimated to experience more than 10 such days, though results were from a monitor sited within a predominantly commercial area. Most locations are estimated to have, on average, 0 days per year with 1-hour daily maximum ambient NO<sub>2</sub> concentrations  $\geq 200$ -300 ppb. No location is estimated to have more than 1 such day per year, on average (Tables 7-14 to 7-19). The corresponding annual average NO<sub>2</sub> concentrations typically ranged from 10 to 30 ppb (Tables 7-11 to 7-13). In contrast, most locations are estimated to have between 10 and 50 days per year where 1-hour daily maximum NO<sub>2</sub> concentrations are  $\geq 100$  ppb based on simulated on-road air quality. On average, most locations are estimated to have fewer than 5 days per year where 1-hour daily maximum on-road NO<sub>2</sub> concentrations are  $\geq 200$  ppb (Table 7-21). The annual average of estimated on-road NO<sub>2</sub> concentrations typically ranged from 15 to 45 ppb (Table 7-20).
- When air quality is adjusted to simulate just meeting the current annual standard, a hypothetical scenario requiring air quality to be adjusted upward, all locations evaluated are estimated to have multiple days per year where 1-hour daily maximum ambient NO<sub>2</sub> concentrations are  $\geq 100$  ppb. Most locations are estimated to have, on average, 50 days or more per year with 1-hour daily

maximum ambient NO<sub>2</sub> concentrations  $\geq$  100 ppb, and six locations are estimated to have 100 days or more per year with 1-hour daily maximum ambient NO<sub>2</sub> concentrations  $\geq$  100 ppb. Fewer benchmark exceedances are estimated to occur with higher benchmark levels. For example, only two locations are estimated to have 10 or more days per year with 1-hour daily maximum ambient NO<sub>2</sub> concentrations that equal or exceed 200 ppb (Figures 7-2 and 7-3). Most locations are estimated to have between 100 and 300 days per year with 1-hour daily maximum on-road NO<sub>2</sub> concentrations  $\geq$  100 ppb and between 25 and 100 days per year with 1-hour daily maximum on-road NO<sub>2</sub> concentrations  $\geq$  200 ppb (Figure 7-6). The corresponding annual average NO<sub>2</sub> concentrations were typically between 30 and 50 ppb (Table 7-28).

In a number of locations, potential alternative standard levels of 0.05 and 0.10 ppm are estimated to result in far fewer days per year than standard levels of 0.15 and 0.20 ppm with NO<sub>2</sub> concentrations  $\geq$  100 ppb (Tables 7-26 and 7-27). When considering the potential alternative standard levels of 0.05 and 0.10 ppm, corresponding annual average NO<sub>2</sub> concentrations were typically between 10 and 30 ppb, similar to a range of concentrations using unadjusted air quality. When considering the potential alternative standard levels of 0.15 and 0.20 ppm, corresponding annual average NO<sub>2</sub> concentrations were typically between 25 and 55 ppb, similar to the range of concentrations observed when using adjusted air quality that just meets the current annual standard.

## 8. EXPOSURE ASSESSMENT AND HEALTH RISK CHARACTERIZATION

### 8.1 OVERVIEW

This section documents the methodology and data used in the inhalation exposure assessment and associated health risk characterization for NO<sub>2</sub> conducted in support of the current review of the NO<sub>2</sub> primary NAAQS. Two important components of the analysis include estimating temporally and spatially variable ambient NO<sub>2</sub> concentrations and simulating human contact with these pollutant concentrations. The approach was designed to better reflect exposures that occur nearby or on a roadway, not necessarily reflected by the existing ambient monitoring data.

Both air quality and exposure modeling approaches have been used to generate estimates of 1-hour NO<sub>2</sub> exposures within Atlanta, Georgia based on a 3-year period (2001-2003). AERMOD, an EPA recommended dispersion model, was used to estimate 1-hour ambient NO<sub>2</sub> concentrations using emissions estimates from stationary and on-road mobile sources. The Air Pollutants Exposure (APEX) model, EPA's human exposure model, was then used to estimate population exposures using the hourly census block level NO<sub>2</sub> concentrations estimated by AERMOD.

Exposure and potential health risk were characterized considering recent air quality conditions (*as is*), for air quality adjusted upward to just meet the current NO<sub>2</sub> standard (0.053 ppm annual average), and for just meeting several potential alternative standards (see chapter 5). The estimated 1-hour exposures for each of these air quality scenarios were compared with the 1-hour potential health effect benchmark levels identified in chapter 6. Specifically, the number of times an individual experienced a daily maximum 1-hour exposure concentration in excess of 100 ppb through 300 ppb was recorded. The exposures for each individual were estimated over an entire year therefore, multiple occurrences of exceedances were recorded, giving the number of days per year with an exceedance of the potential health effect benchmark levels.

The approaches used for assessing exposures in Atlanta are described below. Detailed input data and supporting discussion of the Atlanta case-study is provided in Appendix B-4, in addition to containing the methodology and results for the first exposure modeling case-study

conducted in Philadelphia County as part of the 1<sup>st</sup> draft REA (EPA, 2008b). The Philadelphia County assessment is not included in this REA. There were a few major differences in the approaches used that would not necessarily allow for a direct comparison of the estimated exposures with those estimated for Atlanta, therefore the approach and the exposure results for the Philadelphia County case-study are discussed entirely in Appendix B.

Briefly, the discussion that follows here includes:

- Description of the inhalation exposure model and associated input data used for Atlanta
- Evaluation of estimated NO<sub>2</sub> air quality concentrations and exposures
- Assessment of the quality and limitations of the input data for supporting the goals of the NO<sub>2</sub> NAAQS exposure and risk characterization.

The overall flow of the exposure modeling process performed for this NO<sub>2</sub> NAAQS review is illustrated in Figure 8-1. Several models were used in addition to APEX and AERMOD including emission factors, meteorological processing, and travel demand models, as well as a number of data bases and literature sources to populate the model input parameters. Each of these are described within this chapter, supplemented with additional details in Appendix B.

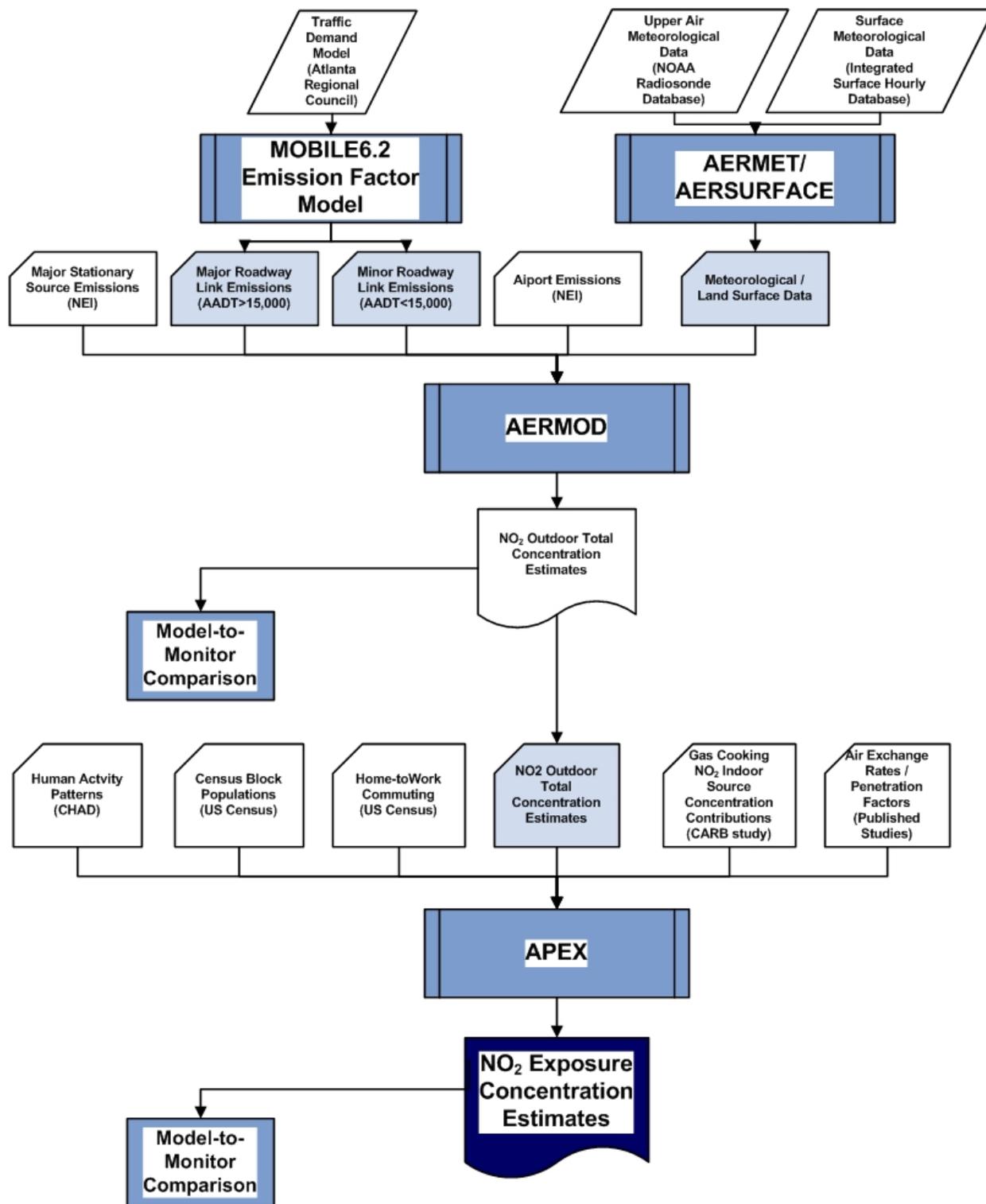


Figure 8-1. General flow used for NO<sub>2</sub> exposure assessment.

## 8.2 OVERVIEW OF HUMAN EXPOSURE MODELING USING APEX

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

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

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

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

The exposure modeling simulations can be summarized by five steps, each of which is detailed in the subsequent sections of this document. Briefly, the five steps are as follows.

1. **Characterize the study area.** APEX selects the census blocks within that study area – and thus identifies the potentially exposed population – based on user-defined criteria and availability of air quality and meteorological data for the area.
2. **Generate simulated individuals.** APEX stochastically generates a sample of hypothetical individuals based on the demographic data for the study area and estimates anthropometric and physiological parameters for the simulated individuals.
3. **Construct a sequence of activity events.** APEX constructs an exposure event sequence spanning the period of the simulation for each of the simulated individuals using the time-location-activity pattern data.
4. **Calculate hourly concentrations in microenvironments.** APEX users define microenvironments that people in the study area visit by assigning location codes in the activity pattern to the user-specified microenvironments. The model then calculates hourly pollutant concentrations in each of these microenvironments for the

period of simulation, based on the user-provided microenvironment descriptions, the hourly air quality data, and for some of the indoor microenvironments, indoor sources of NO<sub>2</sub>. Microenvironmental concentrations are calculated for each of the simulated individuals.

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

## **8.3 CHARACTERIZATION OF STUDY AREA**

### **8.3.1 Study Area Selection**

The selection of the location used for this exposure analysis was based on the location of field and epidemiology studies, the availability of ambient monitoring and other input data, the desire to represent a range of geographic areas, population demographics, general climatology, and results of the ambient air quality characterization.

Atlanta, along with several other locations, was initially selected as a location of interest through statistical analysis of the ambient NO<sub>2</sub> air quality data (see section 7 and Appendix A). Briefly, criteria were established for selecting ambient monitoring sites having high annual mean concentrations and/or exceedances of potential health effect benchmark concentrations. The 90<sup>th</sup> percentile served as the point of reference for the annual mean concentrations and, across all complete site-years for 2001-2006, this value was 23.5 ppb. Seventeen locations had one or more site-years with an annual average concentration at or above the 90<sup>th</sup> percentile, of which Atlanta had one site-year (26.6 ppb annual average). A 1-hour potential health effect benchmark level of 200 ppb was selected as the second criteria for location selection, and Atlanta had one measured concentration above this level. An additional grouping of locations was done base on geographic regions of the U.S., with Atlanta as one of the locations in the southeastern U.S. EPA was also able to obtain measured daily NO<sub>2</sub> exposures of several individuals residing in Atlanta (Suh, 2008), potentially for use in evaluating the APEX estimated exposures. Therefore, in considering each of these selection criteria, 1) the availability of health effects data associated with ambient concentrations (Tolbert et al., 2007), 2) the availability of personal exposure

measurements, 3) the analysis of the air quality data, and 4) geographic representation, Atlanta was selected as the second case-study location.

### **8.3.2 Study Area Description**

The greater Atlanta metropolitan area covers the 13 counties within a radius of approximately 40 km about the Atlanta city center (33.65 °N 84.42 °W) in Fulton County. Due to the complexity of the air quality and exposure modeling to be performed in this exposure assessment, the study location (or modeling domain) was designated as the four counties directly surrounding the city of Atlanta (i.e., Cobb, DeKalb, Fulton, and Gwinnet Counties) (see Figure 8-2). These four counties comprise the urban center of the Atlanta MSA, and contain a large portion of the urbanized road systems in the area. This four county modeling domain contains 27,315 U.S. Census blocks with a combined population of 2,678,078 (2000 Census), comprising approximately 65% of the Atlanta MSA population.

### **8.3.3 Time Period of Analysis**

Calendar years 2001 through 2003 were simulated to envelop the most recent year of emissions data available for the study location (i.e., 2002) and to include a total of 3 years of meteorological data to achieve a degree of representativeness in the dispersion and exposure model estimates. In considering the past 30-years of annual average temperature and precipitation in Atlanta, the three years were variable. On a scale of high to low, in 2001 the temperature was about average (ranked 17<sup>th</sup>) though dry (28<sup>th</sup> lowest precipitation level), 2002 had average precipitation (18<sup>th</sup>) although warmer than most years (12<sup>th</sup>), and 2003 was cooler (25<sup>th</sup>) and wetter (11<sup>th</sup>) than many other years (NCDC, 2007)

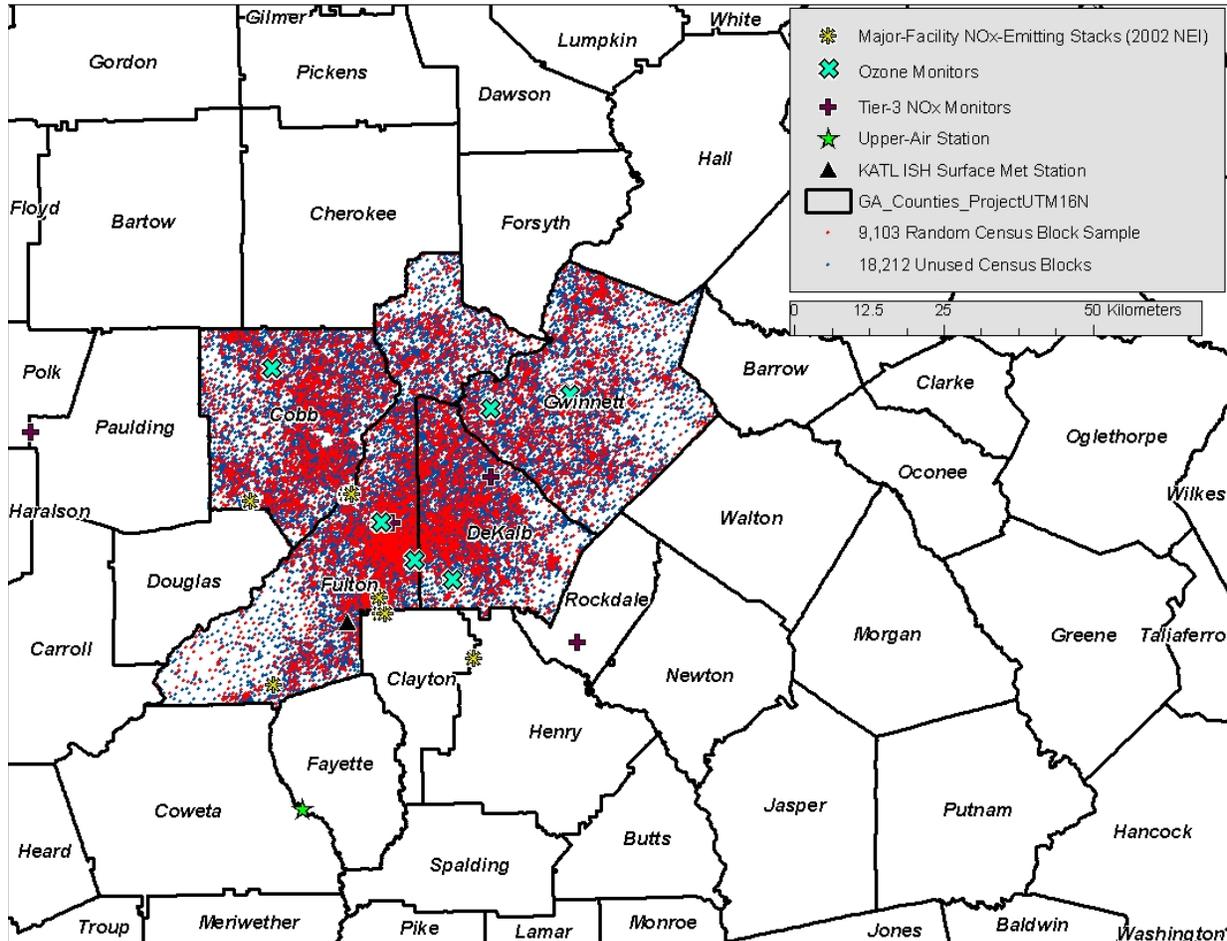
### **8.3.4 Populations Analyzed**

The exposure assessment included the total population residing in each modeled area and considered susceptible and vulnerable populations as identified in the ISA. These include population subgroups defined from either an exposure or health perspective. The population subgroups identified by the ISA (EPA, 2008b) that were included and that can be modeled in the exposure assessment include:

- Children (5-18 years in age)
- Asthmatic children (5-18 years in age)

- All persons (all ages)
- All Asthmatics (all ages)

In addition to these population subgroups, individuals anticipated to be exposed more frequently to NO<sub>2</sub> were assessed, including those commuting on roadways and persons residing near major roadways.



**Figure 8-2. Four county modeling domain used for Atlanta exposure assessment.**

## 8.4 CHARACTERIZATION OF AMBIENT AIR QUALITY USING AERMOD

### 8.4.1 Overview

Air quality data used for input to APEX were generated using AERMOD, a steady-state, Gaussian plume model (EPA, 2004). The following steps were performed to estimate air concentrations using AERMOD.

1. **Collect and analyze general input parameters.** Meteorological data, processing methodologies, and information on surface characteristics and land use were used to determine pollutant dispersion characteristics, atmospheric stability, and mixing heights.
2. **Define sources and estimate emissions.** The emission sources modeled included major stationary emission sources, on-road emissions that occur on major and minor roadways, and emissions from Atlanta Hartsfield International Airport.<sup>1</sup>
3. **Define receptor locations.** Three sets of receptors were identified for the dispersion modeling, and included, ambient monitoring locations, census block centroids, and links along major roadways.
4. **Estimate concentrations at receptors.** Hourly concentrations were estimated for each year simulated (2001-2003) by combining the estimated concentration contributions from each of the emission sources at each of the defined receptors.

A brief description of input data and approaches used for estimating source emissions are described below. Additional details on the inputs and assumptions used in the dispersion modeling are provided in Appendix B-4.

### 8.4.2 General Model Inputs

#### 8.4.2.1 Meteorological Inputs

All meteorological data used for the AERMOD dispersion model simulations were processed with the AERMET meteorological preprocessor, version 06341. Raw meteorological

---

<sup>1</sup> Fugitive emissions from major point sources in the Atlanta area were not included as was done in the Philadelphia County case study, since the NEI shows all emissions to be accounted by stack totals.

data from the Southeast Aerosol Research and Characterization study (SEARCH) site in Atlanta were used as the primary source of meteorology for the AERMOD runs for the years 2001 through 2003. Raw hourly surface meteorological data for the 2001 to 2003 period were obtained from the Integrated Surface Hourly (ISH) Database,<sup>2</sup> primarily for use in modeling the emissions from the Atlanta Hartsfield International Airport (KATL). Upper air data in the Forecast System Laboratory (FSL) format was downloaded from the FSL, (now Global Systems Division) website, <http://www.fsl.noaa.gov/>. Details regarding the data preparation and processing are given in Appendix B, Attachment 1.

#### ***8.4.2.2 Surface Characteristics and Land Use Analysis***

In addition to the standard meteorological observations of wind, temperature, and cloud cover, AERMET analyzes three principal variables to help determine atmospheric stability and mixing heights: the Bowen ratio, surface albedo as a function of the solar angle, and surface roughness. A draft version of AERSURFACE (08256) was used to estimate land-use patterns and calculate these three variables as part of the AERMET processing, using the US Geological Survey (USGS) National Land Cover Data 2001 archives.<sup>3</sup> Details for the seasonal specification definitions, land-use sectors, and data processing are given in Appendix B, Attachment 1.

#### ***8.4.2.4 Other AERMOD Input Specifications***

All emission sources in the Atlanta modeling domain were characterized as *urban*, using the 2000 census population of approximately 4.1 million people in the Atlanta MSA.<sup>4</sup> The AERMOD *toxics* enhancements were also employed to speed calculations from area sources. NO<sub>x</sub> chemistry was applied to all sources to determine NO<sub>2</sub> concentrations. For the roadway and airport emission sources the *Ozone Limiting Method* (OLM) (EPA, 2006c) was used, with plumes considered *grouped*. For all point source simulations, the *Plume Volume Molar Ratio Method* (PVMRM) was used to estimate the conversion of NO<sub>x</sub> to NO<sub>2</sub> (Hanrahan, 1999a, 1999b). The *equilibrium value* for the NO<sub>2</sub>:NO<sub>x</sub> ratio was taken as 75%, the national average ambient background ratio.<sup>5</sup> The initial NO<sub>2</sub> fraction of NO<sub>x</sub> is anticipated to be about 10% or

---

<sup>2</sup> National Climatic Data Center (NCDC), <http://www1.ncdc.noaa.gov/pub/data/techrpts/tr200101/tr2001-01.pdf>

<sup>3</sup> <http://seamless.usgs.gov/>

<sup>4</sup> <http://www.census.gov/Press-Release/www/2001/sumfile1.html>

<sup>5</sup> Appendix W to CFR 51, page 466. [http://www.epa.gov/scram001/guidance/guide/appw\\_03.pdf](http://www.epa.gov/scram001/guidance/guide/appw_03.pdf).

less (Finlayson-Pitts and Pitts, 2000; Yao et al., 2005), therefore a conservative value of 10% was selected from the upper range of this estimate and used for all sources.

Hourly surface O<sub>3</sub> data for years 2001-2003 were obtained from five ambient monitors operating as part of EPA's Air Quality System (AQS)<sup>6</sup> and from one ambient monitor operating as part of the South Eastern Aerosol Research and Characterization (SEARCH) study.<sup>7</sup> Missing data were substituted based on seasonal and time of day characteristics, and hourly values were averaged across each of the O<sub>3</sub> monitors which were available for a particular hour. None of the AQS monitors had data available for November, December, January, and February, for these months only the SEARCH monitor data were used. The locations of these monitors are shown in Figure 8-2.

### **8.4.3 Major Link On-Road Emission Estimates**

Information on traffic data in the Atlanta area was obtained from the Atlanta Regional Commission (ARC) – the regional planning and intergovernmental coordination agency for the 10-county metropolitan area – via their most recent, baseline travel demand modeling (TDM) simulation for year 2005. Although considerable effort was expended to maintain consistency between the ARC approach to analysis of TDM data and that employed in this analysis, complete consistency was not possible due to the differing analysis objectives. The ARC creates county emission inventories. This study created spatially and temporally resolved emission strengths for dispersion modeling. Information about expected differences in traffic between the 2005 data year and 2001-2003 modeled years was not provided by ARC, nor was information about seasonal differences in MOBILE6.2 inputs. The approach used for estimating these major road emissions is discussed further below.

#### ***8.4.3.1 Emission Sources and Locations***

The TDM simulation's data file outputs include a description of the fixed information for the highway network links and traffic descriptors for four time periods: morning, afternoon, evening, and nighttime. Each period's data includes free-flow speed, total vehicle count, total heavy duty truck count, total single occupancy vehicle count, and TDM-calculated congested

---

<sup>6</sup> <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm>

<sup>7</sup> Ambient data were obtained from the Jefferson Street ozone monitor, maintained by Atmospheric Research & Analysis, Inc. Available at <http://www.atmospheric-research.com/studies/SEARCH/index.html>.

speeds for the period. The description of the network consists of a series of nodes joining individual model links (i.e., roadway segments) to which the traffic volumes are assigned, and the characteristics of those links, such as endpoint location, number of lanes, link distance, and TDM-defined link daily capacity.

First, all links with annual average daily traffic (AADT) values greater than 15,000 vehicles per day (one-way) were classified as *major* within the four counties (Cobb, DeKalb, Fulton, and Gwinnett) and a part of a fifth county (Clayton), which contains a small portion of the beltway in the MSA. Then, link locations from the TDM were modified through a GIS analysis to represent the best known locations of the actual roadways, since there was not always a direct correlation between the two (see Appendix B-4.1.1). There were no hourly scaling factors provided for the ARC’s TDM predictions, therefore the total period volume was spread uniformly amongst all hours contributing to that period (morning: 6AM-10AM, midday: 10AM-3PM, afternoon: 3PM-7PM, or nighttime: 7PM-6AM). A 5-hour rolling average, centered on the present hour, was applied to the emission scaling factors to allow for a smoothing of the distribution. The heavy-duty vehicle (HDV) fraction for each hour of each period was obtained by dividing the total period truck count by the total vehicle count, fixing the value as constant for all hours of the period, but allowing it to vary between periods and across links, according to the TDM parameterization. It should be noted that trucks, as defined in the TDM, include heavy and medium duty vehicles as well as commercial vehicles. Because no information on seasonal variation in vehicle activity was available, no seasonal variation was used in the simulations. However, seasonal variations in emission factors from MOBILE6.2 were implemented – see section 8.4.3.2. The AADT and truck fraction from the ARC TDM used in the AERMOD simulations of major links are shown in Tables 8-1 and 8-2, respectively. Note that no rural, local designated links meet the major link AADT criteria, and are thus omitted in Tables 8-1, 8-2, and 8-3.

**Table 8-1. Statistical summary of average annual daily traffic (AADT) volumes (one direction) for Atlanta AERMOD simulations.**

<b>Statistic</b>	<b>Road Type</b>	<b>CBD<sup>1</sup></b>	<b>Fringe</b>	<b>Rural</b>	<b>Suburban</b>	<b>Urban</b>
Count	Arterial	229	180	14	1,299	1,221
	Freeway	109	94	2	616	616
	Local	41	60		168	250
Minimum AADT	Arterial	15,015	15,019	16,603	15,002	15,017
	Freeway	15,049	16,745	23,569	15,111	15,025

Statistic	Road Type	CBD <sup>1</sup>	Fringe	Rural	Suburban	Urban
Maximum AADT	Local	15,442	15,052		15,111	15,017
	Arterial	51,820	49,853	23,433	64,487	46,824
	Freeway	150,047	109,204	24,028	144,434	155,083
Average AADT	Local	110,425	98,420		98,909	127,085
	Arterial	24,814	21,732	19,016	21,383	22,434
	Freeway	73,598	56,741	23,799	59,164	64,744
	Local	25,737	26,536		23,781	25,745
<b>Notes:</b>						
<sup>1</sup> Central business district						

**Table 8-2. Average heavy duty vehicle (HDV) fraction for Atlanta AERMOD simulations.**

Functional Class	Time Period <sup>1</sup>	Region Type				
		CBD <sup>2</sup>	Fringe	Rural	Suburban	Urban
Arterial	Nighttime	12%	18%	15%	12%	13%
	Morning	14%	19%	18%	15%	15%
	Midday	17%	28%	27%	20%	20%
	Afternoon	10%	16%	15%	12%	12%
Freeway	Nighttime	8%	20%	24%	19%	14%
	Morning	9%	20%	26%	19%	15%
	Midday	12%	27%	33%	26%	21%
	Afternoon	7%	16%	21%	15%	12%
Local	Nighttime	10%	24%		18%	15%
	Morning	12%	24%		19%	16%
	Midday	14%	33%		25%	21%
	Afternoon	9%	19%		15%	12%
<b>Notes:</b>						
<sup>1</sup> morning: 6AM-10AM, midday: 10AM-3PM, afternoon: 3PM-7PM, or nighttime: 7PM-6AM						
<sup>2</sup> Central business district						

#### 8.4.3.2 Emission Source Strength

On-road mobile emission factors were derived from the MOBILE6.2 emissions model using ARC input files describing the 2002 vehicle registration distribution and corresponding to the 2008 O<sub>3</sub> season. To maintain consistency with the recent ARC simulations and current modeling parameters and maximize temporal resolution, the ARC's O<sub>3</sub> season input files were used as a basis for all MOBILE6.2 simulations, but were modified as follows. First, the 24-hour

series of temperature and humidity values included in the ARC files were those derived as average values over peak O<sub>3</sub> days. To modify the focus from peak O<sub>3</sub> to average summer days, these values in the input files were modified by converting to average daily minimum and maximum temperature and corresponding specific humidity, determined by the same meteorological record used in the dispersion simulations. Also, winter and summer-specific fuels for the Atlanta region were used for all years, which differ only until the phase-in of Georgia Phase 2 gasoline in 2003, at which point winter and summer sulfur levels are identical. Finally, anti-tampering and inspection/maintenance programs, which were not included in the original ARC input files, were taken from MOBILE input files prepared by the State of Georgia for a previous project.

The simulations were executed to calculate average running NO<sub>x</sub> emission factors in grams per mile for a specific functional class (Freeway, Arterial, Local, or Ramp), speed, and season. Iterative MOBILE6.2 simulations were conducted to create tables of average Atlanta region emission factors resolved by speed (2.5 to 65 mph, in 1 mph increments from 3 to 65 mph), functional class, season, and year (2001, 2002, or 2003) for each of eight combined MOBILE vehicle classes. The resulting tables were then consolidated into speed, functional class, and seasonal values for combined light- and heavy-duty vehicles. To create seasonal-hourly resolved emissions, spring and fall values were taken as the average of corresponding summer and winter values. See Appendix B-4 for an example of the calculated emission factors for Summer, 2001.

To determine the emission strengths for each link for each hour of the year, the Atlanta regional average MOBILE6.2 speed-resolved emissions factor tables were merged with the TDM link data, which had been processed to determine time-resolved speeds. The spatial-mean speed of each link at each time was calculated following the methodology of the Highway Capacity Manual.<sup>8</sup> Table 8-3 shows the resulting average speed for each functional class within each TDM region. The resulting emission factors were then coupled with the TDM-based activity estimates to calculate emissions from each of the major roadway links.

**Table 8-3. Average calculated speed by link type in Atlanta modeling domain.**

	Average Speed (mph)
--	---------------------

<sup>8</sup> 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.

Link Type	CBD <sup>1</sup>	Fringe	Suburban	Urban	Rural
Arterial	22	37	40	30	51
Freeway	54	62	60	57	64
Local	26	40	40	34	N/A
Notes: <sup>1</sup> Central Business District					

#### 8.4.3.3 Other Emission Parameters

Each roadway link is characterized as a rectangular area source with the width given by the number of lanes and an assumed universal lane width of 12 ft (3.66 m). The length and orientation of each link is determined as the distance and angle between end nodes from the adjusted TDM locations. In cases where the distance is such that the aspect ratio is greater than 100:1, the links were disaggregated into sequential links, each with a ratio less than that threshold. There were 737 links that exceeded this ratio and were converted to 1,776 segmented sources. Thus, the total number of area sources included in the dispersion simulations is 5,570. Note that there are some road segments whose length was zero after GIS adjustment of node location. This is assumed to be compensated by adjacent links whose length will have been expanded by a corresponding amount. Table 8-4 shows the distribution of on-road area source sizes.

**Table 8-4. On-road area source sizes.**

Statistic	Number of Lanes	Segment Width (m)	Segment Length (m)
Minimum	1	3.7	0.0
Median	2	7.3	352.8
Mean	2.7	9.9	426.3
1- $\sigma$ Deviation	1.2	4.5	330.0
Maximum	8	29.3	2218.1

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

For light duty vehicles (LDV) it was assumed that the initial vertical extent of the plume is about 1.7 times the average vehicle height, or about 2.6 meters for an average vehicle height of

about 1.53 meters (5 feet), to account for the effects of vehicle-induced turbulence among other factors. The source release height is based on the midpoint of the initial vertical extent, or about 1.3 meters. The initial vertical dispersion coefficient ( $\sigma\text{-}Z_0$ ) was based on the initial vertical extent divided by 2.15, or 1.2 meters.

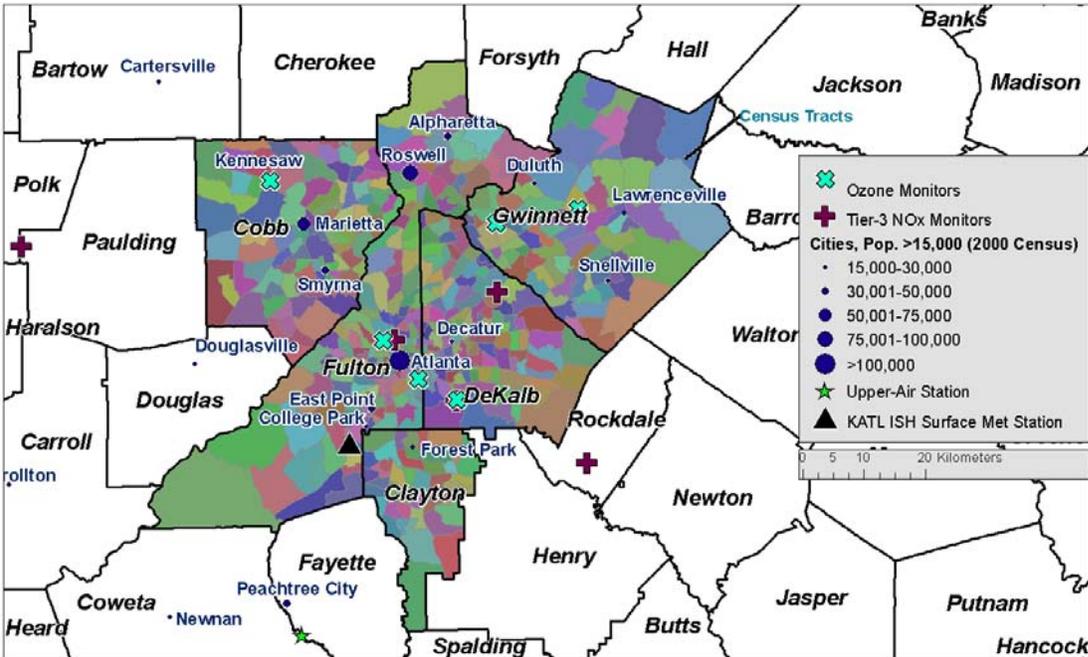
For the heavy duty vehicles (HDV) as with LDVs, the initial vertical extent of the plume was assumed 1.7 times the average vehicle height, or about 6.8 meters for an average vehicle height of about 4.0 meters. Similarly, source release heights were based on the midpoint of the initial vertical extent, or 3.4 meters. The initial  $\sigma\text{-}Z_0$  also based on the initial vertical extent divided by 2.15, was 3.2 meters.

For effective source parameters representing a mix of LDV and HDV for a particular major roadway link, the source release height and initial  $\sigma\text{-}Z_0$  were then assigned using an emissions-weighted average based on the vehicle mix for that roadway link.

The total  $\text{NO}_x$  emissions on the major roadways links were estimated to be 88,438 tons per year (tpy) or approximately 70% of the total on-road emissions.

#### **8.4.4 Minor Link On-road Emission Estimates**

On-road mobile emissions that do not occur on major roadway links were assigned to US Census tracts and simulated as area sources represented by the tract polygons. There are 478 census tract area sources across the 4-county Atlanta modeling domain, and a small part of Clayton County (Figure 8-3). Emission magnitudes and temporal profiles were derived with the same procedure as for the major roadway links, however individual link values were not stored. Instead, each link was assigned to its respective tract and the combined emission total across



**Figure 8-3. The 478 U.S. Census tracts representing area sources for on-road mobile emissions that do not occur on major roadway links.**

links for a specific season and hour were determined for each tract. The resulting total seasonal-hourly emissions profile for each tract area source was then used in AERMOD. Tract-wide emission release parameters for minor links in the dispersion modeling were determined as emissions-weighted averages of light- and heavy-duty vehicle contributions to the tract total values. Estimated NO<sub>x</sub> emissions on the minor roadway links within the five counties was 38,039 tpy (Table 8-5).

**Table 8-5. On-road emissions from major and minor links in Atlanta, 2002.**

County		Total On-Road Emissions (tpy)		% Minor <sup>4</sup>
FIPS <sup>1</sup>	Name	Minor Link <sup>2</sup>	Major Link <sup>3</sup>	
13063	Clayton	1,693	6,185	21%
13067	Cobb	8,329	15,816	34%
13089	DeKalb	7,134	19,871	26%
13121	Fulton	12,047	30,999	28%
13135	Gwinnett	8,835	15,568	36%
<b>Total</b>		<b>38,039</b>	<b>88,438</b>	<b>30%</b>

**Notes:**  
<sup>1</sup> Federal Information Processing Codes for each county.  
<sup>2</sup> Minor links are those roads with ≤ 15,000 AADT.  
<sup>3</sup> Major links are those roads > 15,000 AADT.

4 % Minor is the percent of minor roads in each county.

### 8.4.5 Adjustment of On-road Mobile Source Strengths to 2002 NEI Vehicle Emissions

As noted above, the TDM data received from ARC specified traffic count projections for 2005 instead of the 2001-2003 target years for this analysis. All other model inputs were estimated for the target years, e.g., on-road mobile source emission factors, point source emissions (see section 8.4.6 below), airport emissions (see section 8.4.7 below), and meteorological data. Therefore, to maintain temporal consistency for all emissions used, the on-road emission strengths using the 2005 TDM data were adjusted to match 2002 totals for the 4-county modeling domain from the National Emissions Inventory (NEI).

Table 8-6 compares the on-road mobile source emissions estimated for 2002 as described above (i.e., the 2005 traffic counts combined with 2002 emission factors) with the NEI estimates for 2002. Note that the differences in these estimates may be the result of differences in other factors in addition to the target year traffic counts, such as fleet mix and heavy-duty vehicle fractions. Based on this comparison, an adjustment factor of 0.78 was uniformly applied to all on-road mobile source emission strengths in the Atlanta modeling domain, for both major and minor links.

**Table 8-6. On-road vehicle emission strengths by county for Atlanta modeling domain: modeled vs NEI 2002.**

County	Modeled major link NO <sub>x</sub> emissions (tpy)	Modeled minor link NO <sub>x</sub> emissions (tpy)	Total modeled on-road vehicle NO <sub>x</sub> emissions (tpy)	NEI on-road vehicle NO <sub>x</sub> emissions for 2002 (tpy)	Ratio of NEI-2002-to-modeled NO <sub>x</sub> emissions
Cobb	15,816	8,329	24,145	18,754	0.78
DeKalb	19,871	7,134	27,006	21,715	0.80
Fulton	30,999	12,047	43,046	33,886	0.79
Gwinnett	15,568	8,835	24,403	18,080	0.74
<b>TOTAL</b>	<b>82,254</b>	<b>36,346</b>	<b>118,599</b>	<b>92,434</b>	<b>0.78</b>

#### 8.4.5 Stationary Sources Emissions Preparation

Data for the parameterization of major point sources in Atlanta comes primarily from three sources: the 2002 National Emissions Inventory (NEI; US EPA, 2007e), Clean Air Markets Division (CAMD) Unit Level Emissions Database (US EPA, 2007f), and temporal emission profile information contained in the EMS-HAP (version 3.0) emissions model.<sup>9</sup> The NEI database contains stack locations, emissions release parameters (i.e., height, diameter, exit temperature, exit velocity), and annual emissions for NO<sub>x</sub>-emitting facilities. The CAMD database contains information on hourly NO<sub>x</sub> emission rates for units in the US, where the units are the boilers or equivalent, each of which can have multiple stacks.

First, major stationary sources were selected from the NEI where stacks within facilities contain at least 100 tpy total NO<sub>x</sub> emissions and are located either within the 4-county modeling domain or within 10 km of the modeling domain. Seven NO<sub>x</sub>-emitting facilities met these criteria (Figure 8-4). Stacks within the facilities that were listed separately in the NEI were combined for modeling purposes if they had identical stack physical parameters and were co-located within about 10 m. This resulted in 28 combined stacks (stack parameters are in Appendix B-4) and accounts for 16% of the total number of NO<sub>x</sub> point sources and 51% of the total NO<sub>x</sub> point source emissions in this buffered four county Atlanta area.

The CAMD database was then queried for facilities that matched the facilities identified from the NEI database using the facility name, the Office of Regulatory Information Systems (ORIS) identification code, and facility total emissions. Only one of the 7 major facilities identified was found in the CAMD data base: the Georgia Power Company McDonough Steam-Generating Plant. The CAMD hourly emissions profiles for two units in this facility were summed together and then, after appropriate scaling, used to represent 2 major-facility combined stacks.

For the remaining 26 major-facility combined stacks, hourly NO<sub>x</sub> emissions profiles were created based on the hourly profile typical of that stack's Source Classification Codes (SCC), the season, and the day of week. These SCC-based temporal profiles are year-independent, and were developed for the EPA's EMS-HAP model,<sup>10</sup> described in the EMS-HAP model Version 2

---

<sup>9</sup> <http://www.epa.gov/ttn/chief/emch/projection/emshap30.html>

<sup>10</sup> [http://www.epa.gov/scram001/dispersion\\_related.htm#ems-hap](http://www.epa.gov/scram001/dispersion_related.htm#ems-hap)

User's Guide, Section D-7.<sup>11</sup> As with CAMD hourly emissions, these SCC-based emission profiles are scaled such that the annual total emissions are equal to those of NEI 2002.

#### **8.4.6 Airport Emissions Preparation**

The Atlanta-Hartsfield International Airport emissions were assigned to a polygon that defined an area source for simulation. The perimeter dimensions of the Atlanta-Hartsfield International Airport were determined by GIS analysis of aerial photograph. As with some point source emissions, the annual NO<sub>x</sub> emission totals were extracted from the NEI and the temporal profiles from the EPA's EMS-HAP model. These seasonal, SCC-based emissions were scaled such that the annual total emissions are equal to those of NEI 2002: 5,761 tpy, with about 90% coming from commercial aircraft (see Figure 8-2 for airport location, Appendix B-4 for depiction of area source polygon).

The initial vertical extent of the plume for aircraft emissions was estimated as 10 m to account for typical emission heights and initial dilution parameters. A source release height of 5 m was selected based on the midpoint of the initial vertical extent and the initial vertical dispersion coefficient was estimated using the initial vertical extent divided by 2.15, or 4.6 meters. For cargo-handling equipment a release height of 3.15 m was assumed, which is the average for cargo-handling equipment from a study by the California Air Resources Board (CARB 2006). The initial vertical dispersion coefficient was estimated as the release height divided by 2.15, or 1.47 m. For effective source parameters representing a mix of aircraft and cargo-handling equipment, the source release height and initial sigma-Zo were estimated using an emissions-weighted average with 92% of emissions contributed by aircraft. The aggregate value for release height was 4.85 m with an initial sigma- Zo of 4.22 m.

---

<sup>11</sup> <http://www.epa.gov/scram001/userg/other/emshapv2ug.pdf>

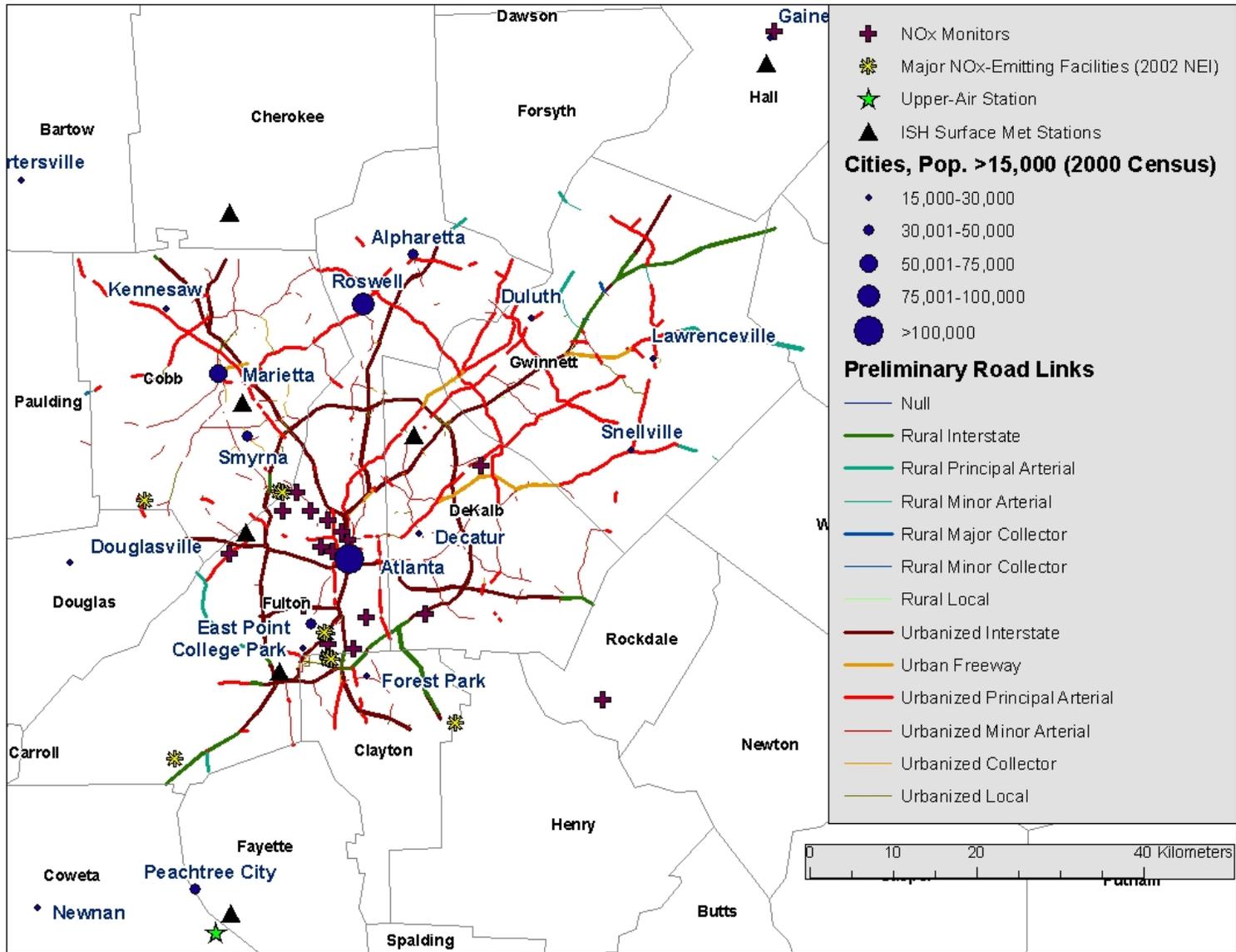


Figure 8-4. Location of major roadway links and major stationary emission sources in Atlanta modeling domain.

### 8.4.7 Receptor Locations

Three sets of receptors were chosen to represent the locations of interest. The first set was selected to represent the locations of the residential population of the modeling domain. These receptors were the 27,315 US Census block centroids<sup>12</sup>. In an effort to make the Atlanta case-study more time efficient, a statistical analysis was performed on the Philadelphia exposure assessment results reported in the 1<sup>st</sup> draft REA (see Appendix B, section 3) to determine the degree of uncertainty introduced by modeling a subset of the block receptors only. The findings of that analysis indicated that the use of a random selection of 1/3 of the block centroids would provide estimates of exposure to exceedances of 200 ppb that were within 4% (90% confidence bounds = 0.3% - 10%) of the estimates obtained based on using all the block receptors. It was judged that this uncertainty was minimal when compared to other uncertainties in the analysis, and therefore, a random selection of 9,103 (1/3) of the block centroids was used for this analysis. These 9,103 Census block receptors are shown along with the other 18,212 block centroids are shown in Figure 8-5. For modeling efficiency, each receptor was assigned a height of 0.0 ft (0.0 m). The effect of this on the exposure estimates in comparison with a standard breathing height of 1.8 m is negligible (see section 7.4.4). Concentrations estimated at these centroid receptors were used by APEX, along with other factors to estimate an individual's microenvironmental concentrations (see section 8.7).

The second set of receptors was chosen to represent the on-road microenvironment (Figure 8-5). For this set, one receptor was placed at the center of each of the 5,570 sources. Receptor concentration estimates were used by APEX, along with other factors, to estimate an individual's on-road and in-vehicle microenvironmental exposures (see section 8.7). The distribution of distances of the on-road and the block centroid receptors was estimated to determine the distance relationship between the on-road emissions and population-based receptors. Approximately 1% of the block centroids are within 50 m of the center of a major roadway link and 26% within 400 m, with a geometric mean of the distribution between 750 m and 800 m (a detailed distribution is provided in Appendix B-4).<sup>13</sup> The population distribution

---

<sup>12</sup> The block centroids used for this analysis are actually population-weighted locations reported in the ESRI data base. They were derived from geocoded addresses within the block taken from the Acxiom Corporation InfoBase household database (Skuta and Wombold 2008; ESRI 2008). These centroids differ from the "internal points" reported by the US Census, which are often referred to as centroids because they are designed to represent the approximate geographic center of the block.

<sup>13</sup> The distance to the roadway edge is shorter by half the roadway width. As noted in Table 8-4 modeled roadway widths ranged from 3.7 m to 29.3 m with an average of 9.9 m and a median of 7.3 m.

within close proximity of the road is actually a different value than the block centroids. For the population simulated in Atlanta, 17% of the population resides within 75 m, 25% were between 75 and 200 m, and 58% were > 200 m of a major road.

The third set of receptors included the locations of the available ambient NO<sub>x</sub>/NO<sub>2</sub> monitors. These receptors were used in evaluating the dispersion model performance. When considering the four Atlanta counties and period of analysis, there were three monitors within the modeling domain with valid ambient NO<sub>2</sub> monitoring concentrations (Figure 8-5).

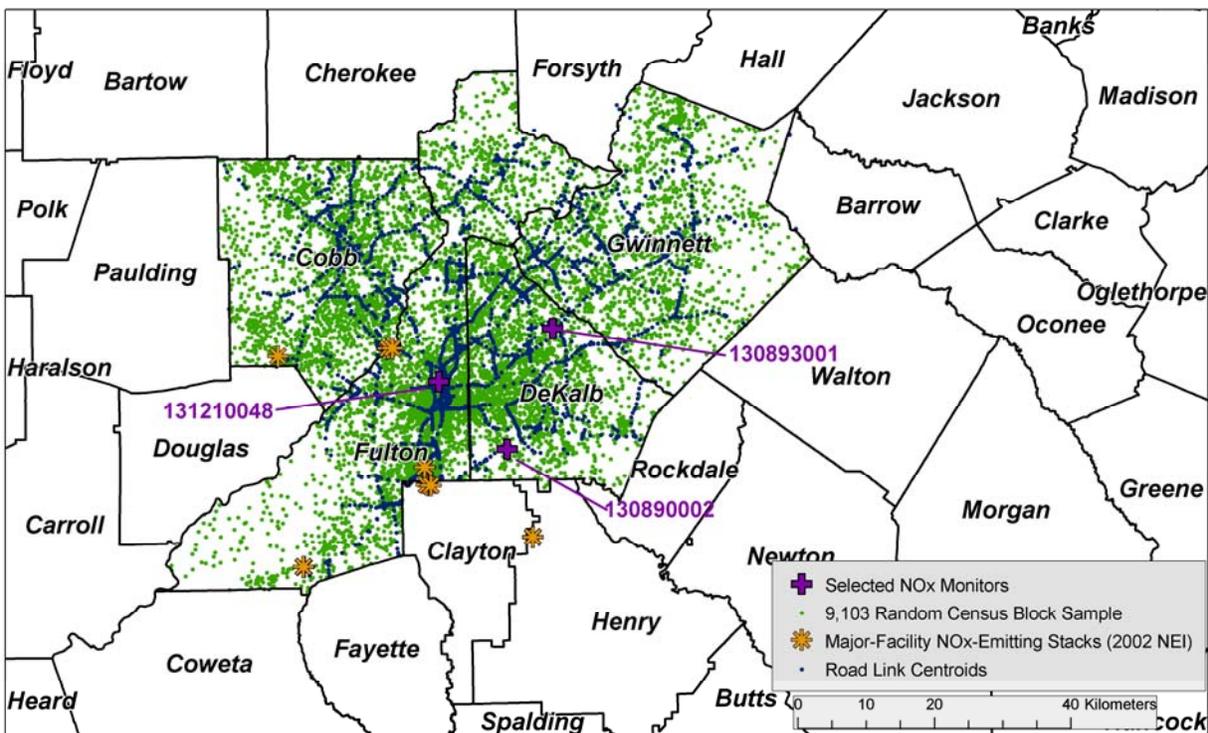


Figure 8-5. Location of modeled receptors in Atlanta modeling domain.

## 8.4.8 Modeled Air Quality Evaluation

### 8.4.8.1 Comparison of Hourly Cumulative Density Functions

The hourly NO<sub>2</sub> concentrations estimated from each of the four source categories were combined at each receptor. These concentration predictions were then compared with measured concentrations at ambient NO<sub>2</sub> monitors. Rather than compare concentrations just at the single modeled receptor point to the ambient monitor concentrations, a distribution of concentrations was developed for the predicted concentrations for all receptors within a 4 km distance of the

monitors, not including receptors within 100 m of a major road. Further, instead of a comparison of central tendency values alone for the number of receptors modeled (mean or median), the complete modeled and measurement concentration distributions were used for comparison.

As an initial comparison of modeled versus measured air quality, all modeled receptors within 4 km of each ambient monitor location, excluding those receptors on roadways or within 100 m of a major roadway, were used to generate a prediction envelope.<sup>14</sup> This envelope was constructed based on selected percentiles from the modeled concentration distribution at each receptor for comparison to the ambient monitor concentration distribution. The 2.5<sup>th</sup> and 97.5<sup>th</sup> percentiles from all monitor distribution percentiles<sup>15</sup> were selected to create the lower and upper bounds of the envelope, while the 50<sup>th</sup> percentile concentrations were combined to create a distribution representing the central tendency (Figure 8-6). The distribution of the modeled values estimated for the monitor receptor is also presented, along with the complete hourly concentration distribution measured at each ambient monitor. A table providing the values used to generate the figure is provided in Appendix B-4.

The hourly concentration distributions modeled at receptors within 4 km of each of the ambient monitor locations provide a reasonable representation of the measured ambient NO<sub>2</sub> concentrations. The lower and upper bounds of the predicted concentration distributions surround the measured ambient concentration distribution at many of the percentiles. The actual modeled monitor receptor concentration distributions were generally above that of the corresponding measured concentrations, resulting in overestimation at some of the upper percentiles by about 20-50%. At monitor 131210048 however, the overestimate in concentrations was generally less than 10 ppb, or within 10-20% of that measured. In fact the maximum estimated concentration at this monitor was 137 ppb, just 1 ppb above that measured (136 ppb).

When considering the lowest potential health effect benchmark levels, the modeled monitor receptor had 19, 2, and 9 daily maximum predicted values above 100 ppb 1-hour, compared with 0, 0, and 3 of the measured values at monitors 130890002, 130893001, and

---

<sup>14</sup> 500 m to 4 km is the area of representation of a neighborhood-scale monitor, according to EPA guidance.

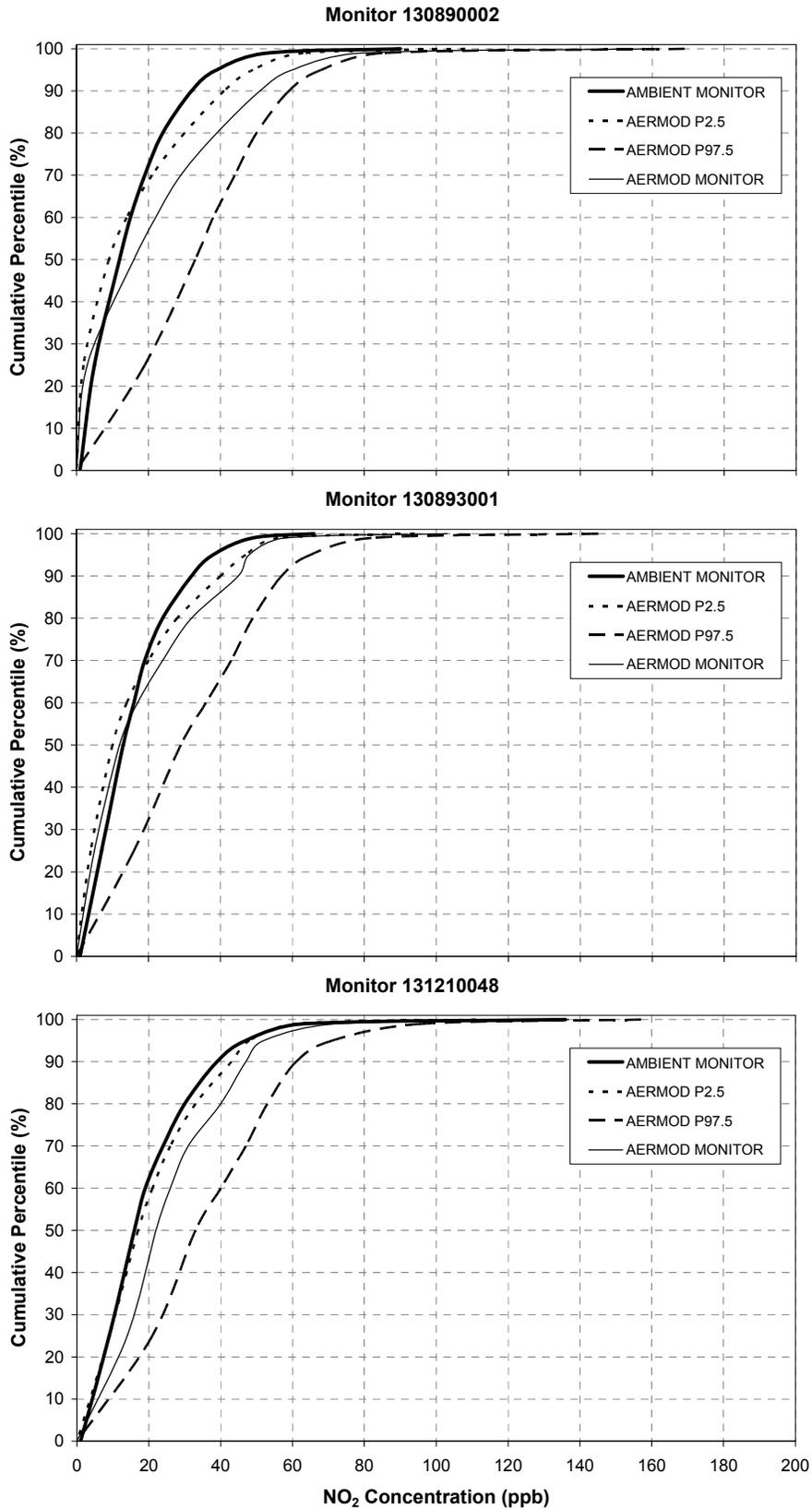
<sup>15</sup> As an example, suppose there are 1000 receptors surrounding a monitor, each receptor containing 8,760 hourly values used to create a concentration distribution. Then say the 73<sup>rd</sup> percentile concentration prediction is to be estimated for each receptor. The lower bound of the 73<sup>rd</sup> percentile of the modeled receptors would be represented by the 2.5<sup>th</sup> percentile of all the calculated 73<sup>rd</sup> percentile concentration predictions, i.e., the 25<sup>th</sup> highest 73<sup>rd</sup> percentile concentration prediction across the 1000 73<sup>rd</sup> percentile values generated from all of the receptors. Note that, at any given percentile along either of the envelope bounds as well as at the central tendency distribution (the receptor 50<sup>th</sup> percentile), the concentration from a different receptor may be used.

131210048, respectively. There were only two predicted daily maximum exceedances of 150 ppb 1-hour at one monitor (ID 130890002), although there were no measured exceedances at this monitor. None of the modeled monitors had estimated daily maximum NO<sub>2</sub> concentrations above 200 ppb 1-hour as observed with the measurement data for all three monitors. This indicates that while overestimating the number of exceedances of the lowest potential health effect benchmark level of 100 ppb at these three locations, there is not an overestimate of higher benchmark levels, such as those  $\geq 200$  ppb considering *as is* air quality.

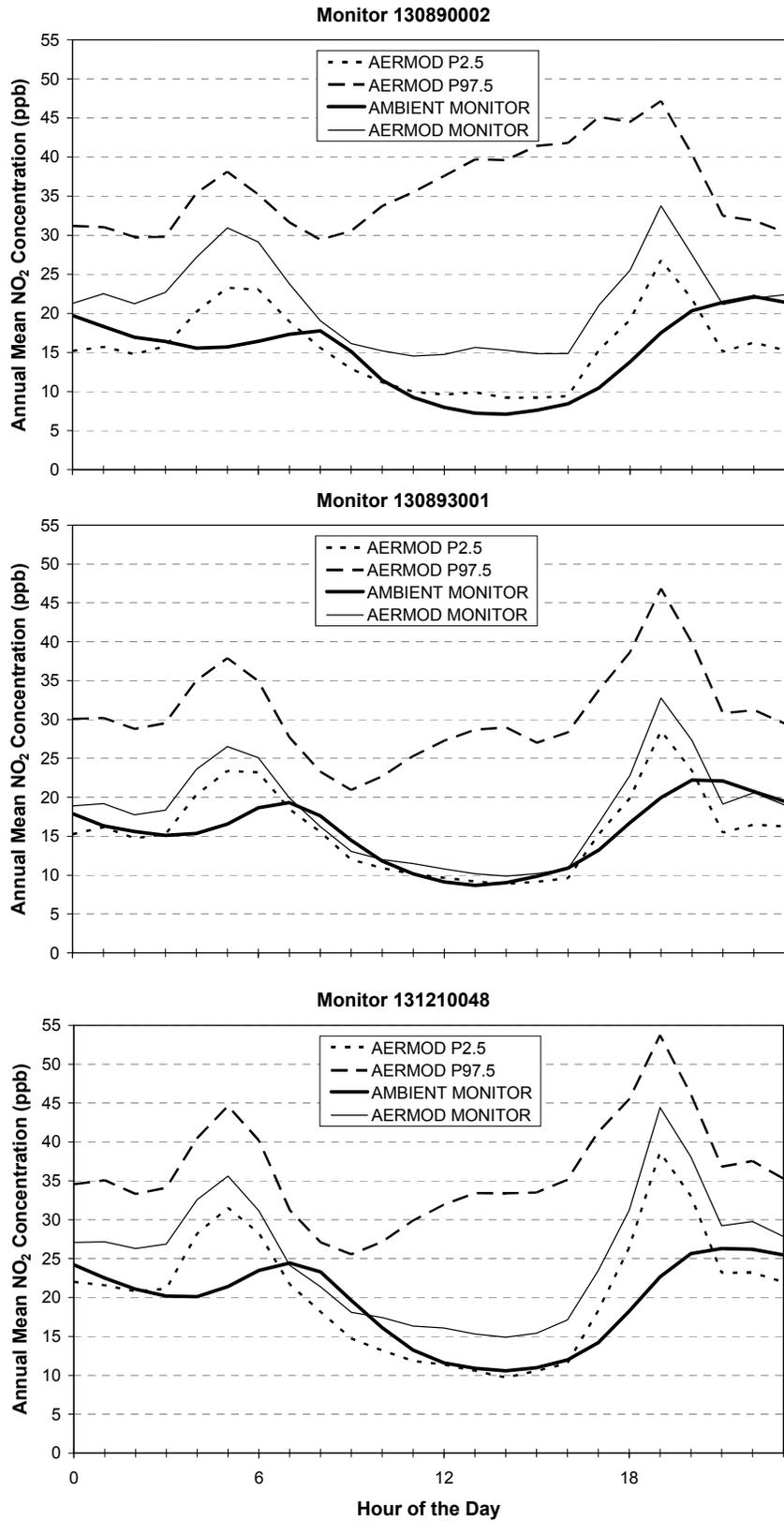
#### ***8.4.8.2 Comparison of annual average diurnal concentration profiles***

A second comparison between the modeled and monitored data was performed to evaluate the diurnal variation in NO<sub>2</sub> concentrations. For AERMOD, receptor concentrations during each hour-of-the-day were averaged (i.e., 365 values for hour 1, 365 values for hour 2, and so on), to generate an annual average NO<sub>2</sub> concentration for each hour at each receptor. Then a prediction envelope was constructed similar to that described above from modeled receptors located within 4 km of each ambient monitor. These modeled diurnal distributions, along with that of each ambient monitor hour-of-the-day annual average concentration are illustrated in Figure 8-7. A table providing the values used to generate the figure is provided in Appendix B-4.

When comparing the modeled predicted and ambient measured diurnal profiles, there was agreement between the patterns and several hours of the day where the observed values fell within the model prediction envelope. This occurred primarily during the late night (9PM-12AM), early morning (1AM-3AM), and late morning through many of the midday hours (8AM-4PM). However, NO<sub>2</sub> concentrations were overestimated at certain times of the day, generally between the hours of 4-6AM and 5-8PM. The overestimation in concentrations is not entirely unexpected given the results of the distribution of hourly concentrations illustrated in Figure 8-6.



**Figure 8-6. Comparison of measured ambient monitor NO<sub>2</sub> concentration distribution with the modeled monitor receptor and receptors within 4 km of the monitors at three locations in Atlanta for Year 2002.**



**Figure 8-7. Comparison of measured ambient monitor NO<sub>2</sub> concentration diurnal profile with the modeled monitor receptor and receptors within 4 km of the monitors at three locations in Atlanta for Year 2002.**

#### ***8.4.8.3 Comparison of estimated on-road NO<sub>2</sub> concentrations***

The two independent approaches used to estimate on-road NO<sub>2</sub> concentrations, one using ambient monitor data combined with an on-road simulation factor (section 7) and the other using the AERMOD dispersion model, were compared to one another. There are no on-road NO<sub>2</sub> concentration measurements in Atlanta for the modeled data to be compared with, although it should be noted that the data used to estimate the simulation factors and applied to the monitor data were measurement based.

First a comparison can be made between the adjustment factors used for estimating on-road concentrations in the air quality analysis and similar factors that can be generated using AERMOD estimated concentrations for year 2002. As described above in section 7, an empirical distribution of on-road simulation factors was derived from on-road and near-road NO<sub>2</sub> concentration measurements published in the extant literature. The derived empirical distribution was separated into two components, one for application to summertime ambient concentrations, and the second for all other seasons. The two empirical distributions are presented in Figure 8-8, and represent the factors that are multiplied by the ambient monitor concentration (i.e., at monitors  $\geq 100$  m from a major road) and used to estimate the on-road concentration in the air quality characterization. The measurement data from which these were derived were mainly time-averaged over 7 to 14 days. The one-hour NO<sub>2</sub> concentrations estimated at AERMOD receptors  $\geq 100$  m from a major road were compared with the concentrations estimated at their closest on-road receptor to generate a similar ratio (i.e., on-road/non-road NO<sub>2</sub> concentrations). In this case, 7-day averages were calculated using the hourly AERMOD concentration predictions. These 7-day average AERMOD generated ratios were also stratified into two seasonal categories, one containing the summer ratios (June, July, and August) and the other for all other times of the year. The AERMOD on-road factor distributions in semi-empirical form are also presented in Figure 8-8. The values for each of the methods and season distributions are provided in Appendix B-4.

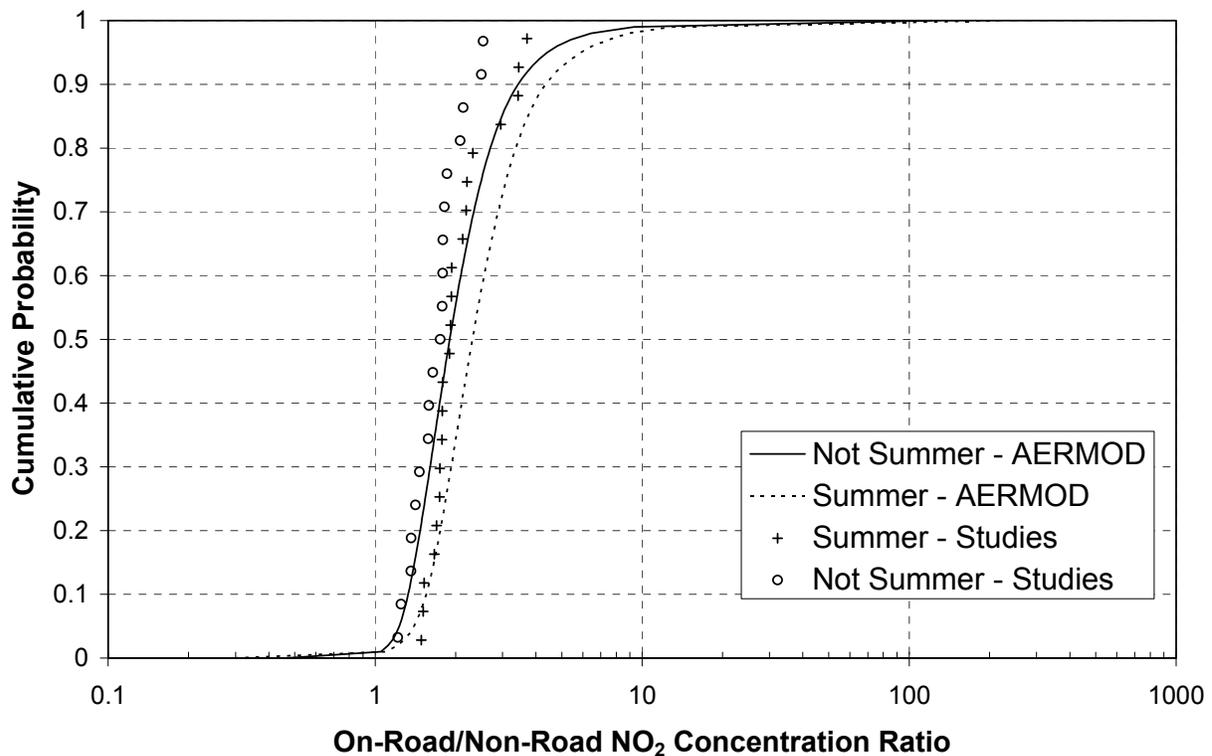
Both the modeled and measurement derived distributions have similar seasonal relationships, that is the summer ratios are consistently greater than the non-summer ratios throughout the entire distribution. There are small differences when comparing the two approaches at the lowest distribution percentiles, with the AERMOD ratios consistently below

that of the empirically derived factors. This is likely due to the differences in the population of samples used to generate each type of distribution. The measurement study-derived distribution used data from on-road concentration measurements and from monitoring sites located at a distance from the road, sites that by design of the algorithm and the factor selection criteria are likely not under the influence of non-road NO<sub>2</sub> emission sources. The measurement study derived ratios never fall below one, there are no on-road concentrations less than any corresponding non-road influenced concentrations. This is, by design, a function of the algorithm used to derive the ratio (i.e., concentrations away from the road are always less). This was reasonable and conservative assumption used in estimating the on-road concentrations for the air quality characterization performed in section 7. The population of AERMOD receptors used to generate the depicted distribution however includes concentrations estimated at non-road receptors that are greater than corresponding on-road receptors. This is likely a more realistic depiction of the actual relationship between on-road and non-road receptors. It is possible that an NO<sub>2</sub> emission source at a distance from a road could contribute to local concentrations more than a mobile source contributes to corresponding on-road concentrations.

There are some similarities that follow when comparing each of the AERMOD with the measurement study derived distributions the lower to mid percentiles. Overlap of the two different approaches occurs through about the 40<sup>th</sup> percentile and tracks closely through the 90<sup>th</sup> percentile. The AERMOD predicted ratio distributions then extend beyond the range of values offered by the measurement study derived ratios at about the 90<sup>th</sup> percentile. Given the greater number of receptors modeled by AERMOD, the AERMOD approach may be better representing the variability in NO<sub>2</sub> concentrations than when using the on-road adjustment factor approach.

While not directly comparable to Atlanta, a few U.S studies report similar concentrations for inside vehicles and near roads. For example, Riediker et al. (2003) measured NO<sub>2</sub> concentrations inside North Carolina State patrol cars while on duty in Raleigh as well as at a fixed site monitor located near local major roadways. Mean concentrations inside the vehicles averaged over about 9 hours were 41.7 ppb (minimum 1.6, maximum 548.5 ppb), similar to the mean and range of concentrations estimated by AERMOD and using the on-road adjustment factor (Table 8-7). Reported roadside NO<sub>2</sub> concentrations were also comparable in mean concentration, (49.9 ppb), although they had a smaller range (minimum 13.0, maximum 212.1 ppb). This is likely a result of a much greater averaging time (i.e., approximately 4-days) used

for these roadside measurements. CARB (2003) measured NO<sub>2</sub> concentrations inside school buses during several commutes on urban and suburban/rural bus routes in Los Angeles, CA. Measurements were collected over an average commute time of approximately 85 minutes and corresponded to average concentrations of about NO<sub>2</sub> 70 ppb along an urban route (minimum 34, maximum 120 ppb). Rural/suburban areas averaged less, NO<sub>2</sub> concentrations were about 45 ppb (minimum 23, maximum 68 ppb).



**Figure 8-8. Comparison of on-road/non-road ratios developed from AERMOD concentration estimates for year 2002 and those derived from data reported in published NO<sub>2</sub> measurement studies.**

A second comparison was conducted using the hourly on-road NO<sub>2</sub> concentrations estimated by AERMOD for 3,259 on-road receptors in Atlanta for the years 2001-2003. The 24 hourly values modeled for each day at each receptor were rounded to the nearest 1 ppb. The second set of estimated on-road NO<sub>2</sub> concentrations was generated as part of the Air Quality Characterization by applying randomly selected on-road adjustment factors to the ambient monitor concentrations in the Atlanta MSA, using the same three ambient monitors which were all located > 100 m from a major road. Table 8-7 compares the summary statistics of the hourly

concentrations and the number of estimated exceedances of three potential health effect benchmark levels (i.e., 100, 150, and 200 ppb) using the two different approaches to estimate on-road concentrations. The AERMOD predicted and ambient monitor simulated concentration distributions have very similar variances, although the AERMOD estimated concentrations are about 40% greater at the mean and about 15 ppb higher at each of the percentiles (save the max). The AERMOD on-road receptors also consistently had a greater number of exceedances of potential health effect benchmark levels than that estimated using the on-road monitor simulation. For example, the AERMOD receptors had an average of 241 exceedances of 100 ppb per site-year while the simulated on-road monitors had an average of 169 exceedances per year, a difference of about 40%. This difference between the two approaches was prevalent throughout each of the percentiles and when considering each of the 1-hour concentration levels. The differences could be due to the greater number of receptors modeled by AERMOD (n=3,259) compared with the on-road monitor simulation (n=800) and that the AERMOD generated on-road receptors could include locations with greater influence by roadway emissions that are not captured by the simplified approach conducted in the Air Quality Characterization.

**Table 8-7. Summary statistics of estimated on-road hourly NO<sub>2</sub> concentrations (ppb) and the numbers of hourly concentrations above 100, 150, and 200 ppb in a year using both the AERMOD and the on-road ambient monitor simulation approaches in Atlanta.**

Statistic	On-Road Hourly NO <sub>2</sub> (ppb)		Number of hours >100 ppb		Number of hours >150 ppb		Number of hours >200 ppb	
	AERMOD	AQ Monitors	AERMOD	AQ Monitors	AERMOD	AQ Monitors	AERMOD	AQ Monitors
Mean	43	31	241	169	28	20	5	3
Std	25	25	307	227	51	43	14	7
Var	631	646	94,102	51,427	2,577	1,856	190	54
N <sup>1</sup>	28,548,840	6,622,300	3,259	800	3,259	800	3,259	800
p0	0	0	0	0	0	0	0	0
p5	9	3	2	2	0	0	0	0
p10	15	6	4	8	0	0	0	0
p15	19	9	7	11	0	0	0	0
p20	23	11	11	16	0	0	0	0
p25	26	13	16	23	0	0	0	0
p30	29	15	23	33	0	0	0	0
p35	32	17	30	42	0	1	0	0
p40	34	20	41	55	0	1	0	0
p45	37	22	56	67	1	3	0	0
p50	40	25	79	87	1	3	0	0
p55	44	28	119	111	3	5	0	0
p60	46	31	185	132	6	8	0	1

Statistic	On-Road Hourly NO <sub>2</sub> (ppb)		Number of hours >100 ppb		Number of hours >150 ppb		Number of hours >200 ppb	
	AERMOD	AQ Monitors	AERMOD	AQ Monitors	AERMOD	AQ Monitors	AERMOD	AQ Monitors
p65	49	35	253	160	12	9	1	1
p70	52	38	317	184	22	13	2	1
p75	56	43	399	220	37	21	4	2
p80	60	49	485	280	50	26	7	3
p85	66	56	584	353	71	36	11	3
p90	74	65	706	426	96	53	18	9
p95	88	81	879	649	132	110	31	17
p100	556	437	1,929	1,595	542	373	181	55

**Notes:**  
<sup>1</sup> For the on-road hourly NO<sub>2</sub> concentration, N is the number of 1-hour concentrations generated for each simulation. In the exceedance columns, N represents the number of AERMOD receptors or monitor site-years simulated.

#### 8.4.8. Using unadjusted AERMOD predicted NO<sub>2</sub> concentrations

The NO<sub>2</sub> concentrations estimated using AERMOD may be biased upwards based on a comparison with measurement data from the three available ambient monitors. Given an apparent systematic bias, one could argue for adjusting concentrations to improve the comparison of the model predictions with the measurement data. However, data were not adjusted based on these model-to-monitor comparisons for a few reasons, primarily regarding the confidence in the dispersion modeling system, the spatial representation of the monitors compared with receptors modeled, and the number of comparisons available. Details on the reasoning are provided in section 8.12.1.

### 8.5 SIMULATED POPULATION

One of the important population subgroups for the exposure assessment is asthmatics. Evaluating exposures for this population requires an estimation of both adult and children asthma prevalence rates. The proportion of the population of children characterized as being asthmatic was estimated by statistics on asthma prevalence rates recently used in the NAAQS review for O<sub>3</sub> (US EPA, 2007g). See Appendix B, Attachment 4 for details in the derivation. Specifically, the analysis generated age and gender specific asthma prevalence rates for children ages 0-17 using data provided in the National Health Interview Survey (NHIS) for 2003 (CDC, 2007). Adult asthma prevalence rates for Atlanta were derived from the Behavioral Risk Factor

Surveillance System (BRFSS) survey information for years 2004 – 2005 (Blackwell and Kanny, 2007). Table 8-8 provides a summary of the prevalence rates used in the exposure analysis by age and gender. Additional information on the variability in these prevalence rates is given in Appendix B-4.

**Table 8-8. Asthma prevalence rates by age and gender used for Atlanta.**

Region (Study Area)	Age <sup>1</sup>	Asthma Prevalence <sup>2</sup>	
		Female	Male
Atlanta (South)	0	0.034	0.041
	1	0.052	0.070
	2	0.071	0.102
	3	0.088	0.129
	4	0.099	0.144
	5	0.119	0.165
	6	0.122	0.164
	7	0.112	0.133
	8	0.093	0.138
	9	0.091	0.168
	10	0.108	0.178
	11	0.132	0.162
	12	0.123	0.145
	13	0.097	0.143
	14	0.095	0.153
	15	0.100	0.151
	16	0.115	0.140
	17	0.145	0.122
	>17	0.083	0.050

**Notes:**  
<sup>1</sup> Ages 0-17 from the National Health Interview Survey (NHIS) for 2003 (CDC, 2007), ages >17 from the Behavioral Risk Factor Surveillance System (BRFSS) survey information (Blackwell and Kanny, 2007)  
<sup>2</sup> Asthma prevalence is given as fraction of the population. Multiply by 100 to obtain the percent.

The total population simulated within the Atlanta model domain was approximately 2.68 million persons, of which there was a total simulated population of about 212,000 asthmatics. The model simulated approximately 500,000 children, of which there were about 64,000 asthmatics. For comparison, the Georgia Department of Human Resources reports the 2001 asthma prevalence for children in middle and high school as ranging from 9.6 to 13.8%, though for Fulton County middle school estimates were higher (15.8%) (Blackwell et al, 2003).

And when considering this population simulated in Atlanta and their proximity to roadways, 17% of the population resides within 75 m, 25% were between 75 and 200 m, and 58% were > 200 m of a major road.

## **8.6 CONSTRUCTION OF LONGITUDINAL ACTIVITY SEQUENCES**

Exposure models use human activity pattern data to predict and estimate exposure to pollutants. Different human activities, such as spending time outdoors, indoors, or driving, will result in varying pollutant exposure concentrations. To accurately model individuals and their exposure to pollutants, it is critical to understand their daily activities. EPA's Consolidated Human Activity Database (CHAD) provides data for where people spend time and the activities performed. Typical time-activity pattern data available for inhalation exposure modeling consist of a sequence of location/activity combinations spanning 24-hours, with 1 to 3 diary-days for any single study individual.

The exposure assessment performed here requires information on activity patterns over a full year. Long-term multi-day activity patterns were estimated from single days by combining the daily records using an algorithm that represents the day-to-day correlation of activities for individuals. The algorithm first uses cluster analysis to divide the daily activity pattern records into groups that are similar, and then select a single daily record from each group. This limited number of daily patterns is then used to construct a long-term sequence for a simulated individual, based on empirically-derived transition probabilities. This approach is intermediate between an assumption of no day-to-day correlation (i.e., re-selection of diaries for each time period) and perfect correlation (i.e., selection of a single daily record to represent all days). Details regarding the algorithm and supporting evaluations are provided in Appendix B-4, Attachments 2 and 3.

## **8.7 CALCULATING MICROENVIRONMENTAL CONCENTRATIONS**

Probabilistic algorithms are used to estimate the pollutant concentration associated with each exposure event. The estimated pollutant concentrations account for temporal and spatial variability in ambient (outdoor) pollutant concentration and factors affecting indoor microenvironment, such as a penetration, air exchange rate, and pollutant decay or deposition rate. APEX calculates air concentrations in the various microenvironments visited by the

simulated person by using the ambient air data estimated for the relevant blocks/receptors, the user-specified algorithm, and input parameters specific to each microenvironment. The method used by APEX to estimate the microenvironmental concentration depends on the microenvironment, the data available for input to the algorithm, and the estimation method selected by the user. The current version of APEX calculates hourly concentrations in all the microenvironments at each hour of the simulation for each of the simulated individuals using one of two methods: by mass balance or a transfer factors method. Details regarding the algorithms used for estimating specific microenvironments and associated input data derivations are provided in Appendix B.

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

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

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

### **8.7.1 Microenvironments Modeled**

In APEX, microenvironments represent the exposure locations for simulated individuals. For exposures to be estimated accurately, it is important to have realistic microenvironments that

match closely to the locations where actual people spend time on a daily basis. As discussed above, the two methods available in APEX for calculating pollutant levels within microenvironments were mass balance or a transfer factors approach. Table 8-9 lists the microenvironments used in this study, the calculation method used, and the type of parameters needed to calculate the microenvironment concentrations.

**Table 8-9. List of microenvironments modeled and calculation methods used.**

<b>Microenvironment</b>	<b>Calculation Method</b>	<b>Parameter Types used<sup>1</sup></b>
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
<sup>1</sup> AER=air exchange rate, DE=decay-deposition rate, PR=proximity factor, PE=penetration factor		

## **8.7.2 Microenvironment Descriptions**

### **8.7.2.1 Microenvironment 1: Indoor-Residence**

The Indoors-Residence microenvironment uses several variables that affect NO<sub>2</sub> exposure: whether or not air conditioning is present, the average outdoor temperature, the NO<sub>2</sub> removal rate, and an indoor concentration source.

Air conditioning (A/C) status of an individual’s residential microenvironment was simulated randomly using the probability that a residence has an air conditioner. For the Atlanta modeling domain an air-conditioning prevalence of 97.0 % was used (American Housing Survey or AHS, 2004).

Air exchange rate (AER) data for the indoor residential microenvironment were the same used in APEX for the most recent O<sub>3</sub> NAAQS review (EPA, 2007g; see Appendix B,

Attachment 5). Briefly, AER data were reviewed, compiled, and evaluated from the extant literature to generate location-specific AER distributions categorized by influential factors, namely, location, temperature, and presence of A/C. The AER data obtained was limited in the number of samples, particularly when considering these influential factors. When categorizing by temperature, a range of temperatures was used to maintain a reasonable number of samples within each category to allow for some variability within the category, while still allowing for differences across categories. Several distribution forms were investigated (i.e., exponential, log-normal, normal, and Weibull) and in general, lognormal distributions provided the best fit. Fitted lognormal distributions were defined by a geometric mean (GM) and standard deviation (GSD). Because no fitted distribution was available specifically for Atlanta, distributions were selected from other locations thought to have similar characteristics, qualitatively considering factors that might influence AERs including 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. To avoid unusually extreme simulated AER values, bounds of 0.1 and 10 were selected for minimum and maximum AER, respectively. Table 8-10 summarizes the distributions used by A/C prevalence and temperature categories. See Appendix B, Attachment 2 for additional details.

**Table 8-10. Geometric means (GM) and standard deviations (GSD) for air exchange rates by A/C type and temperature range used for Atlanta exposure assessment.**

<b>A/C Type</b>	<b>Temp (°C)</b>	<b>N</b>	<b>GM</b>	<b>GSD</b>
No A/C <sup>1</sup>	<=10	61	0.9258	2.0836
	10-20	87	0.7333	2.3299
	>20	44	1.3782	2.2757
Central or Room A/C <sup>2</sup>	<=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
<b>Notes:</b>				
<sup>1</sup> Distribution derived from Research Triangle Park study. See Appendix B, Attachment 5.				
<sup>2</sup> Distribution derived from non-California cities. See Appendix B, Attachment 5.				

The same NO<sub>2</sub> removal rate distribution was used for all indoor microenvironments that use the mass balance method. This removal rate is based on data provided by Spicer et al. (1993) and was approximated with a uniform distribution, U{1.02, 1.45 h<sup>-1</sup>} based on the six reported values from a single house (Table 8-12).

**Table 8-11. Data used to estimate removal rate constant for indoor microenvironments.**

Source Introduction	House Temperature °C	Relative Humidity (%)	Air Exchange Rate (h <sup>-1</sup> )	Removal Constant k (h <sup>-1</sup> )
injection	23	52	0.15	1.28
injection	21	54	0.16	1.02
radiant heater	26	54	0.15	1.15
convective heater	24	55	0.12	1.04
range	22	54	-	1.45
injection	22	55	0.15	1.13
<b>Notes:</b> Data from Table 1 of Spicer et al. (1993).				

An indoor emission source term was included in the APEX simulations to estimate NO<sub>2</sub> exposure to gas cooking (hereafter referred to as “indoor sources”). This was the only indoor source considered in this assessment. Three types of data were used to generate the emission factor for this indoor source: (1) the fraction of households in the Atlanta MSA that use gas for cooking fuel, (2) the range of contributions to indoor NO<sub>2</sub> concentrations that occur from cooking with gas, and (3) the diurnal pattern of cooking in households.

The fraction of households in Atlanta that use gas cooking fuel (i.e., 39%) was obtained from AHS (2004). Data used for estimating the contribution to indoor NO<sub>2</sub> concentrations that occur during cooking with gas fuel were derived from a study sponsored by the California Air Resources Board (CARB, 2001). A uniform distribution of concentration contributions for input to APEX was estimated as U{4, 188 ppb}. An analysis by Johnson et al (1999) of survey data on gas stove usage collected by Koontz et al (1992) showed an average number of meals prepared each day with a gas stove of 1.4. The diurnal allocation of these cooking events was estimated using food preparation time obtained from CHAD diaries, stratified by hour of the day, and normalized to the expected value of daily food preparation events of 1.4 (Table 8-12).

**Table 8-12. Probability of gas stove cooking by hour of the day.**

Hour of the Day	Probability of Cooking (%) <sup>1</sup>
0	0
1	0
2	0
3	0
4	0

Hour of the Day	Probability of Cooking (%) <sup>1</sup>
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
<b>Notes:</b> <sup>1</sup> Values rounded to the nearest 5%. Data sum to 145% due to rounding convention and the scaling to represent 1.4 cooking events/day.	

**8.7.2.2 Microenvironments 2-7: All other indoor microenvironments**

The remaining five indoor microenvironments, which represent Bars and Restaurants, Schools, Day Care Centers, Office, Shopping, and Other environments, were all modeled using the same data and functions. An air exchange rate distribution (GM = 1.109, GSD = 3.015, Min = 0.07, Max = 13.8) was based on an indoor air quality study (Persily et al, 2005). This is the same distribution in APEX used for the most recent O<sub>3</sub> NAAQS review (EPA, 2007g). See Appendix B, Attachment 5 for details in the data used and derivation. The removal rate is the same uniform distribution used in the Indoor-Residence microenvironment (section 8.7.2.1). The Bars and Restaurants microenvironment included an estimated contribution from indoor sources as was described for the Indoor-Residence, only there was an assumed 100% prevalence rate for cooking with a gas appliance and it occurred at any hour of the day.

**8.7.2.3 Microenvironments 8 and 9: Outdoor Microenvironments**

Two outdoor microenvironments, the Near Road and Public Garage/Parking Lot, used the transfer factors method to calculate pollutant exposure. Penetration factors are not applicable to

outdoor environments (effectively, PEN=1). The distribution for proximity factors were developed from the dispersion model estimated concentrations, using the relationship between on-road to receptor estimated concentrations.

#### ***8.7.2.4 Microenvironment 10: Outdoors-General***

The general outdoor environment concentrations are represented by the AERMOD predicted concentrations. Therefore, both the penetration factor and proximity factor for this microenvironment were set to 1.

#### ***8.7.2.5 Microenvironments 11 and 12: In Vehicle- Cars and Trucks, and Mass Transit***

Penetration factors were developed from data provided in Chan and Chung (2003). Since major roads were the focus of this assessment, reported indoor/outdoor ratios for highway and urban streets were used here. Mean values range from about 0.6 to just over 1.0, with higher values associated with increased ventilation (i.e., window open). A uniform distribution U{0.6, 1.0} was selected for the penetration factor for Inside-Cars/Trucks due to the limited data available to describe a more formal distribution and the lack of data available to reasonably assign potentially influential characteristics such as use of vehicle ventilation systems for each location. Mass transit systems, due to the frequent opening and closing of doors, was assigned a uniform distribution U{0.8, 1.0} based on the reported mean values for fresh-air intake (0.796) and open windows (1.032) on urban streets. Proximity factors were developed from the dispersion model estimated concentrations, using the relationship between the on-road to receptor estimated concentrations. The proximity distributions were stratified based using time-of day and season bins and are provided in Appendix B-4.

## **8.8 EXPOSURE MEASURES AND HEALTH RISK CHARACTERIZATION**

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

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

where,

$C_i$  = Hourly exposure concentration at clock hour  $i$  of the simulation period (ppm)

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

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

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

$T$  = 60 minutes

From the hourly exposures, APEX calculates time series of 1-hour average exposure concentrations that a simulated individual would experience during the simulation period. APEX then statistically summarizes and tabulates the hourly (or daily, annual average) exposures. In this analysis, the exposure indicator is 1-hr exposures above selected health effect benchmark levels. From this, APEX can calculate two general types of exposure estimates: counts of the estimated number of people exposed at or above a specified NO<sub>2</sub> concentration level and the number of times per year that they are so exposed; the latter metric is in terms of person-occurrences or person-days. The former highlights the number of individuals exposed at least *one or more* times per modeling period to the potential health effect benchmark level of interest. APEX can also report counts of individuals with multiple exposures. This person-occurrences measure estimates the number of times per season that individuals are exposed to the exposure indicator of interest and then accumulates these estimates for the entire population residing in an area.

APEX tabulates and displays the two measures for exposures above levels ranging from 100 to 300 ppb by 50 ppb increments for 1-hour average exposures. These results are tabulated for the population and subpopulations of interest.

### 8.8.1 Adjustment for Just Meeting the Current and Alternative Standards

We used a different approach to simulate just meeting the current and alternative standards than was used in the Air Quality Characterization (Appendix A). In this case, instead of adjusting upward<sup>16</sup> the air quality concentrations, to reduce computer processing time, we adjusted the health effect benchmark levels by the same factors described for each specific location and simulated year (Table 8-13). Since it is a proportional adjustment, the end effect of adjusting concentrations upwards versus adjusting benchmark levels downward within the model is the same. The same follows for where as is concentrations were in excess of an alternative standard level (e.g., 50 ppb for the 98<sup>th</sup> percentile averaged over three years), only the associated benchmarks are adjusted upwards (i.e., a higher threshold simulating lower exposures).

**Table 8-13. Adjusted potential health effect benchmark levels used by APEX to simulate just meeting the current standard and various alternative standards considered.**

Model Scenario	Averaging Time	Conc (ppb)	Conditions	Potential health effect benchmark level (ppb)				
				100	150	200	250	300
As-is				100	150	200	250	300
Current Standard	Annual	53	Year 2001	44	66	88	110	132
			Year 2002	37	55	73	91	110
			Year 2003	31	46	62	77	93
Alternative Standards	1 hour	50	98 <sup>th</sup> %ile	163	nd	327	nd	490
			99 <sup>th</sup> %ile	177	nd	355	nd	532
		100	98 <sup>th</sup> %ile	82	nd	163	nd	245
			99 <sup>th</sup> %ile	89	nd	177	nd	266
		150	98 <sup>th</sup> %ile	54	nd	109	nd	163
			99 <sup>th</sup> %ile	59	nd	118	nd	177
		200	98 <sup>th</sup> %ile	41	nd	82	nd	123
			99 <sup>th</sup> %ile	44	nd	89	nd	133

**Notes:**  
<sup>nd</sup> not done due to model constraints on number of levels possible in one model simulation.

When modeling indoor sources, the indoor concentration contributions needed to be scaled by the similar proportions. This additional scaling was necessary so as not to affect the impact of the estimated indoor concentrations while adjusting the benchmark levels. The following presents the justification of why it can appropriate to use the same proportional factor to adjust the indoor source concentration contribution.

<sup>16</sup> To evaluate the current and most of the alternative standards proposed, ambient concentrations were lower than air quality that would just meet the standards.

Exposure concentrations an individual experiences are first defined as the sum of the contribution from ambient concentrations and from indoor sources (if present) and this concentration can be either above or below a selected concentration level of interest. This is represented by the following equation:

$$C_{\text{exposure}} = aC_{\text{ambient}} + bC_{\text{indoor}} > C_{\text{threshold}}$$

where,

- $C_{\text{exposure}}$  = individual exposure concentration (ppm)
- $a$  = proportion of exposure concentration from ambient (unitless fraction)
- $C_{\text{ambient}}$  = ambient concentration in the absence of indoor sources
- $b$  = proportion of exposure concentration from indoor (unitless fraction, equivalent to 1-a)
- $C_{\text{indoor}}$  = direct indoor source concentration contribution in the absence of ambient influence (ppm)
- $C_{\text{threshold}}$  = an exposure concentration of interest (ppm)

It follows that if we are interested in adjusting the ambient concentrations upwards by some proportional factor  $f$  (a unitless number), this can be described with the following:

$$fa C_{\text{ambient}} + bC_{\text{indoor}} > C_{\text{threshold}}$$

This is equivalent to

$$aC_{\text{ambient}} + b(C_{\text{indoor}} / f) > (C_{\text{threshold}} / f)$$

Therefore, if the potential health effect benchmark level and the indoor concentrations are both proportionally scaled downward by the same adjustment factor, the contribution of both sources of exposure (i.e., ambient and indoor) are maintained and the same number of estimated exceedances would be obtained as if the ambient concentration were proportionally adjusted upwards by factor  $f$ .

## **8.9 EXPOSURE MODELING AND HEALTH RISK CHARACTERIZATION RESULTS**

### **8.9.1 Overview**

The results of the exposure and risk characterization are presented here for the four county modeling domain in Atlanta. Several exposure scenarios were considered for the exposure assessment including an analysis of three averaging times for NO<sub>2</sub> concentrations (annual, 24-hour, and 1-hour), an analysis of the contribution to NO<sub>2</sub> exposures of both indoor and outdoor sources, and an analysis of NO<sub>2</sub> exposures assuming air quality that just meets the current annual and several alternative 1-hour daily maximum standards. The year 2002 served as the base year for all scenarios, while 2001 and 2003 were only evaluated for a limited number of scenarios. Exposures were simulated for four population groups; all persons, all children (ages 5-17), all asthmatics, and asthmatic children (ages 5-17).

The exposure results that are summarized below focus on asthmatics. Key results are presented in the next three subsections, with complete results for each of these two population subgroups provided in Appendix B-4. In addition, due to limitations in the data summaries output from the current version of APEX, certain exposure data could only be output for the entire population modeled (i.e., all persons - includes asthmatics and healthy persons of all ages) rather than the particular subpopulation. The summary exposure results for the entire population (e.g., annual average exposure concentrations, time spent in microenvironments at or above a potential health effect benchmark level) is assumed representative of the asthmatic population in the modeling results because the asthmatic population does not have its microenvironmental concentrations and activities estimated any differently from those of the total population. The assumption of modeling asthmatics similarly to healthy individuals (i.e., using the same time-location-activity profiles) is supported by the findings of van Gent et al. (2007), at least when considering children 7-10 years in age. These researchers used three different activity-level measurement techniques; an accelerometer recording 1-minute time intervals, a written diary considering 15-minute time blocks, and a categorical scale of activity level. Based on analysis of 5-days of monitoring, van Gent et al. (2007) showed no difference in the activity data collection methods used as well as no difference between asthmatic children and healthy children when comparing their activity levels.

### **8.9.2 Annual Average Exposure Concentrations (as is)**

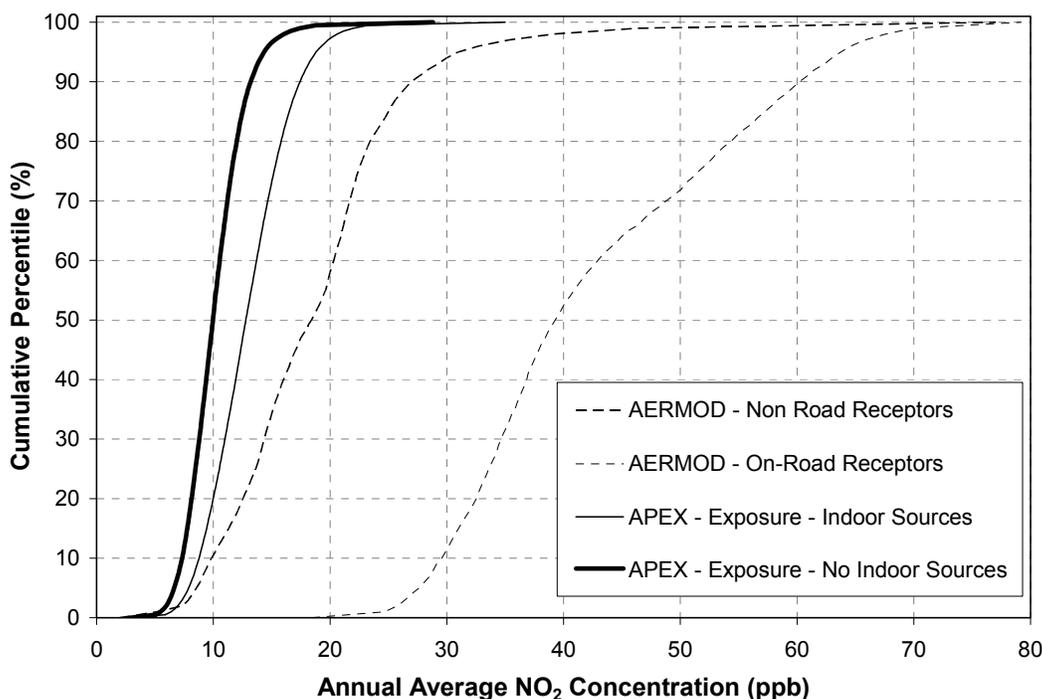
Figure 8-9 illustrates the annual average exposure concentrations for the total simulated population (i.e., both asthmatics and healthy individuals of all ages), considering the modeled year 2002 air quality (as is) and both with and without indoor sources. Also plotted on this figure is the distribution of the annual average NO<sub>2</sub> concentrations predicted by AERMOD separated into two broad receptor categories. As a point of reference, the measured annual average concentration for the three ambient monitors in the Atlanta modeling domain ranged from 15 ppb to 19 ppb in year 2002. About one-half of the AERMOD predicted annual average NO<sub>2</sub> concentrations for the non-road receptors were below the range of the ambient monitoring annual average concentrations, with most receptors predicted to be less than 30 ppb. About 5% of these receptors had concentrations above this level. It should be noted that the non-road receptors included here could have a number of block centroids located near a major road. Consistent with what was observed in the air quality characterization data for on-road concentration estimates, the AERMOD long-term average concentrations predicted at the roadway links are about twice that of the estimated concentrations at non-road receptors.

The hourly NO<sub>2</sub> concentrations output from AERMOD were input into the exposure model, providing a wide range of estimated exposures calculated by APEX (Figure 8-9). All persons were estimated to experience exposures below an annual average exposure of 53 ppb, even when considering indoor source concentration contributions. The estimated annual average exposures were below that of both the modeled receptors and the measured air quality. For example, the median annual average exposure was about 6 ppb less than the modeled median non-road receptor concentration when the exposure estimation included indoor sources, and about 9 ppb less when annual average exposures were estimated without the indoor sources. In the absence of indoor source contributions, personal exposure concentrations for most of the simulated individuals are estimated to be about 40 to 70 percent that of the local ambient or outdoor concentration. This estimate is consistent with studies reporting such a relationship based on measurements of personal exposure and ambient concentrations that ranges from around 0.3 to 0.6 (Table AX3.5-1b, ISA ANNEX).

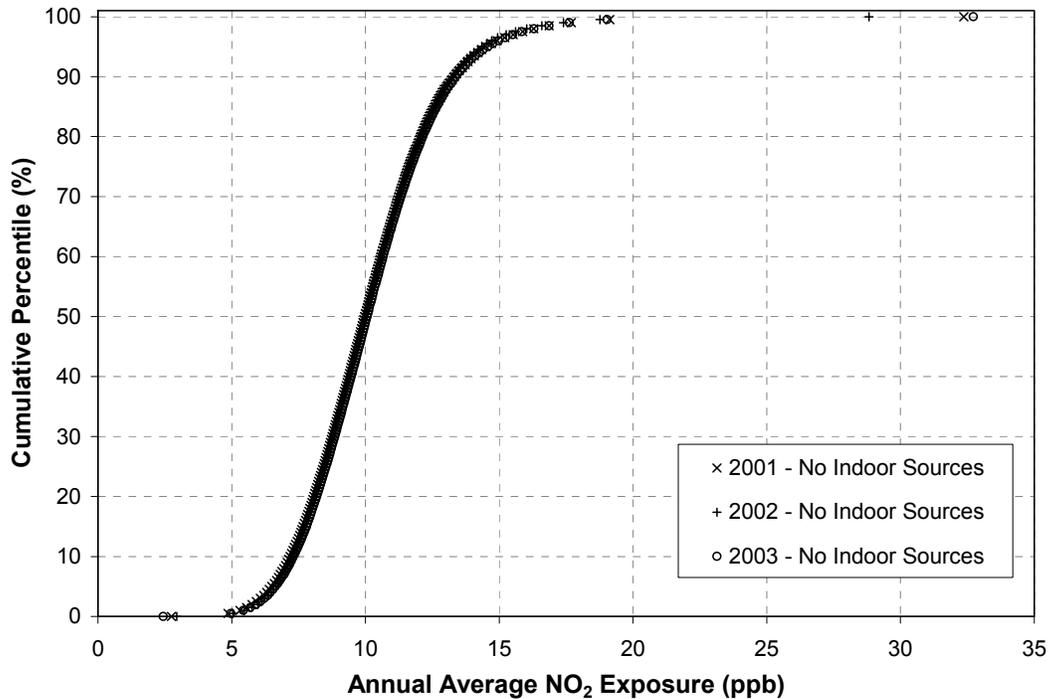
In comparing the estimated exposures with and without indoor sources, indoor sources were estimated to contribute between 1 and 4 ppb to the total annual average exposures. This would correspond to indoor sources contributing approximately 1/3 of the annual average

exposures for persons using gas cooking appliances. Again, while Figure 8-9 summarizes the entire population, the data are representative of what would be observed for the population of asthmatics or asthmatic children.

Year-to-year-variation was evaluated by comparing the estimated annual average exposure distributions for each year simulated. Each simulated year of data was very similar, with estimated median annual average exposures at about 10 ppb and 95% of the simulated individuals' annual average exposures within the interval from 5.9 to 15.8 ppb (Figure 8-10).



**Figure 8-9. Comparison of annual average AERMOD predicted NO<sub>2</sub> concentrations (on-road and non-road receptors) and APEX modeled NO<sub>2</sub> exposures (with and without modeled indoor sources) in Atlanta modeling domain for year 2002.**



**Figure 8-10. Comparison of estimated annual average NO<sub>2</sub> exposures for Years 2001-2003 in Atlanta modeling domain without modeled indoor sources.**

### 8.9.3 Daily Average Exposures (as is)

As mentioned earlier, APEX is capable of providing exposure results across a variety of averaging times, including 24-hour average exposures. This averaging time serves as a good point of comparison with the personal exposures reported in the published literature. As mentioned above regarding APEX default results, the daily mean exposures were estimated for the total simulated population. In this simulation, each person has 365 daily mean personal exposures, thus each individual experiences a daily average concentration distribution (i.e., each person has a median daily average exposure, a 99<sup>th</sup> percentile daily average exposure, etc.). These modeled exposures were compared with personal NO<sub>2</sub> measurement data obtained from Suh (2008) for the participants of an Atlanta epidemiological study conducted by Wheeler et al. (2006). The personal exposure measurements were collected across two seasons (fall and spring)<sup>17</sup> and considered cooking fuel (gas or electric cooking) as an influential variable for personal exposures. A total of 30 individuals participated in the study, of whom 13 subjects had personal exposure measurements for both seasons, with no persons using both cooking fuels.

<sup>17</sup> Fall was designated here for sample collection dates reported in the months of September, October, and November 1999; Spring was designated where sample collection dates were reported in the months of April and May 2000.

An average of 6 daily average personal exposure measurements was available for each individual when stratified by season and cooking fuel (minimum number of days = 3, max = 7). Because there were few personal exposure measurements, an exposure distribution was constructed for each individual, simply using their minimum, median, and maximum daily mean exposures and are summarized in Figure 8-11. In comparing the median personal daily mean exposures using the two stratification variables, two patterns can be noted. First, the use of gas as a cooking fuel increased daily median personal exposures by about 3 to 10 ppb in both seasons. Second, seasonal differences were also present, with personal daily average exposures higher during the spring by about 1 to 3 ppb when comparing the individual median values for the persons employing gas or electric cooking. While these general patterns are noted, it should be added that the maximum daily average exposures were highest in the spring and similar for both of the cooking fuel categories.

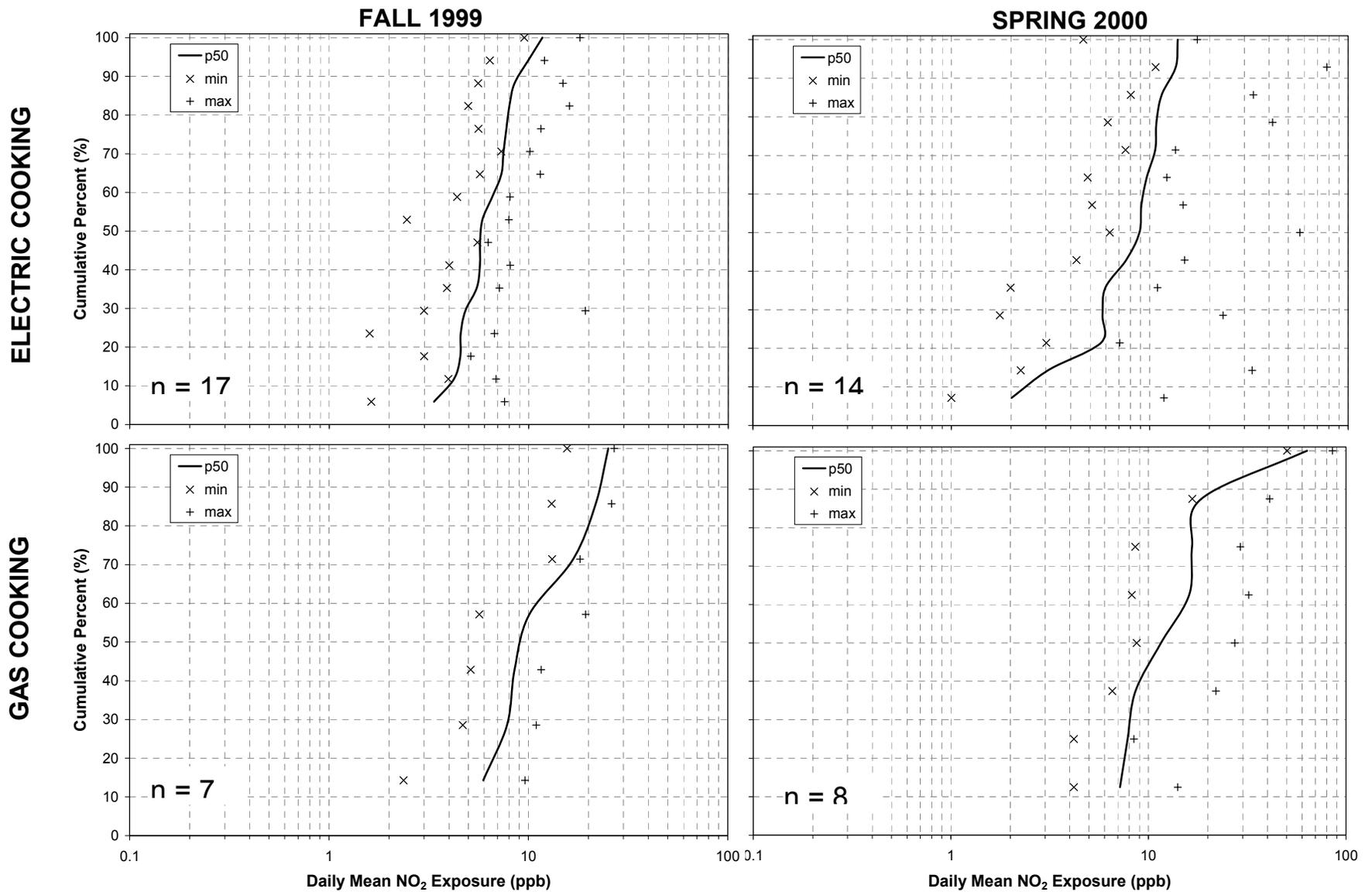
Daily mean exposures estimated using APEX were also evaluated in a similar manner, by stratifying the results based on the same seasons and whether or not indoor sources were included in the model simulation. The specific period from 1999-2000 was not modeled by APEX although this period was included in the personal exposure measurement study. The APEX simulation results for year 2002 were selected for comparison with the exposure measurements obtained from Suh (2008). A distribution of each person's estimated daily exposure was constructed, using the median daily mean exposure to represent the central tendency and a 95 % prediction interval to represent the lower and upper bounds of exposure (i.e., the 2.5<sup>th</sup> and the 97.5<sup>th</sup> percentiles). This prediction interval was chosen rather than using the minimum and maximum as done with the personal measurements because APEX estimated 61 and 91 days of exposure for each individual in the spring and fall months, respectively. The APEX results would likely capture more variability in exposures given the greater number of days in comparison with the personal exposure measures that contained at most 7 days of data per season.

The daily mean exposures estimated using APEX, stratified by season and by inclusion of indoor sources, are presented in Figure 8-12. The distributions of median daily mean exposures are comparable to one another, although the fall season was about 1 ppb higher than the spring exposures. The range of estimated daily mean exposures, given by the 95% prediction interval, was also similar across the season categories. In comparing the simulations where indoor

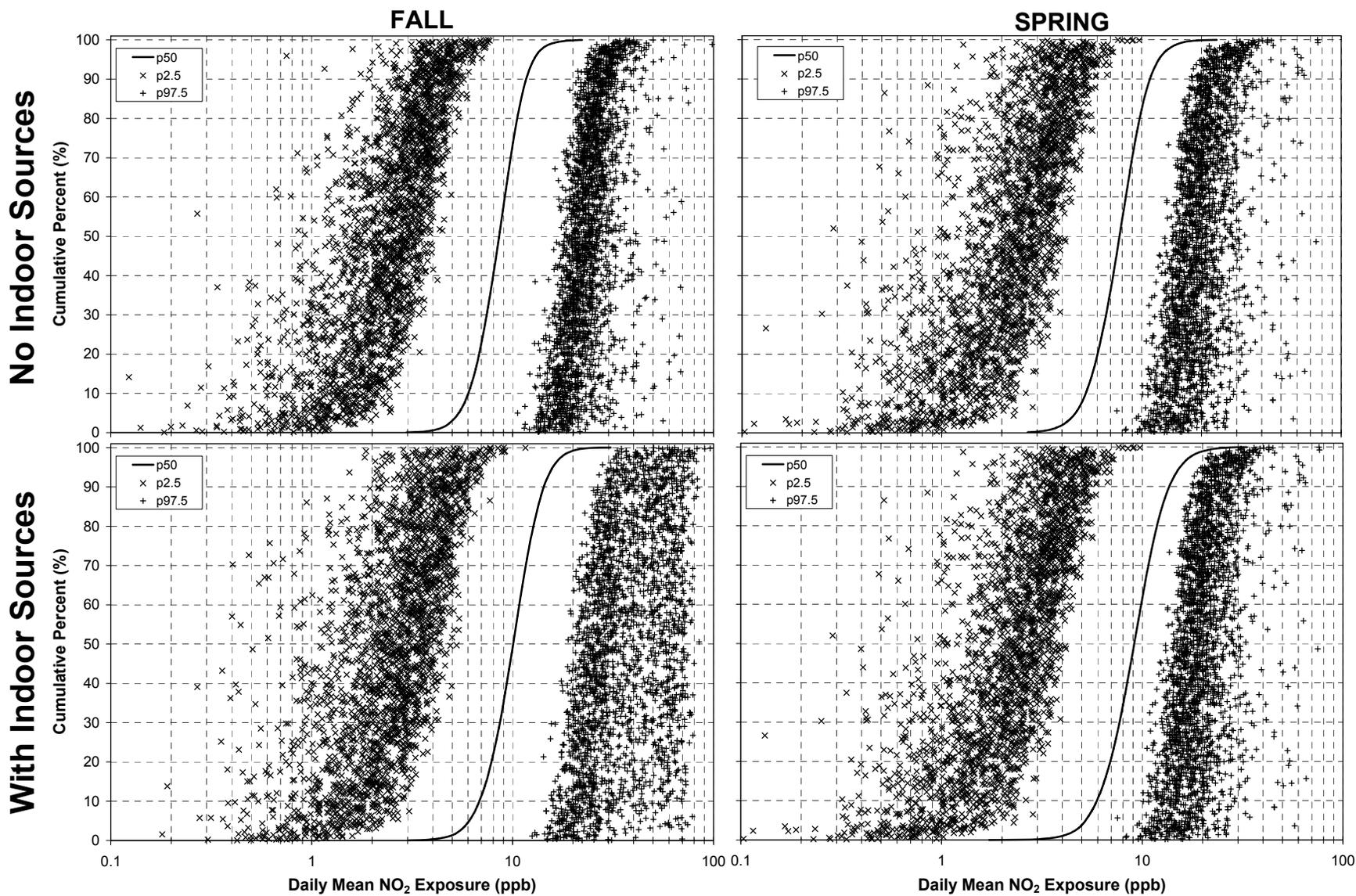
sources were modeled to the simulations conducted without indoor source contributions, the estimated exposures were between 1 to 4 ppb greater for the indoor source simulations. It should be noted that the indoor source exposure distributions include exposures for all of the simulated individuals, some of which do not have gas cooking occurring at home. The APEX simulated daily mean exposures are similar to the measured personal exposures (Figure 8-11) when considering the values and range of the median concentrations as well as the values and range of the bounding percentiles given by the 95% prediction intervals.<sup>18</sup>

---

<sup>18</sup> While a direct comparison of APEX estimated maximum daily exposure concentrations with the maximum observed daily personal exposure concentrations is considered questionable given the large discrepancy in sample sizes, it should be noted that approximately 99.1% of APEX simulated persons had their estimated maximum daily exposure concentrations within the maximum observed daily personal exposure measurement of 78.2 ppb without gas cooking. Approximately 97.5% of APEX simulated persons had their estimated maximum daily exposure concentrations within the maximum observed daily personal exposure measurement of 85.4 ppb with gas cooking.



**Figure 8-11. Distribution of measured daily average personal NO<sub>2</sub> exposures for individuals in Atlanta, stratified by two seasons (fall or spring) and cooking fuel (gas or electric). Minimum (min), median (p50), and maximum (max) were obtained from each individual’s multi-day exposure measurements. The figure generated here was based on personal exposure measurements obtained from Suh (2008).**



**Figure 8-12. Distribution of estimated daily average NO<sub>2</sub> exposures for individuals in Atlanta, stratified by two seasons (fall or spring) and with and without indoor sources, for Year 2002 APEX simulation. Lower bound (2.5th percentile, p2.5), median (p50), and upper bound (97.5th percentile, p97.5) were calculated from each simulated persons 365 days of exposure. A random sample of 5% of persons (about 2,500 individuals) is presented in each figure to limit the density of the graphs.**

## **8.9.4 One-Hour Exposures**

### ***8.9.4.1 Overview***

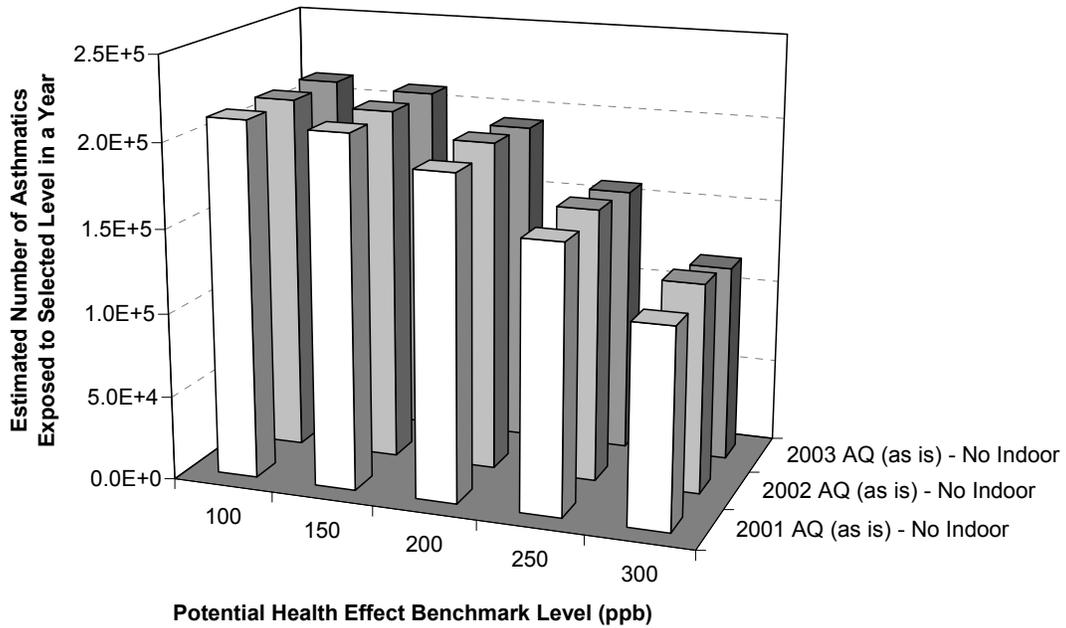
Because the focus of the exposure and risk characterization is on short-term 1-hour daily maximum exposures, analyses were performed using the APEX estimated 1-hour exposure concentrations. The number of exposures above the selected potential health effect benchmark levels (i.e., 100, 150, 200, 250, and 300 ppb, 1-hour average) were estimated. An exceedance was recorded when the maximum exposure concentration estimated for the individual was above the selected benchmark level in a day. Estimates of repeated exposures are also recorded, that is where 1-hour exposure concentrations were above a selected benchmark level in a day added together across multiple days (therefore, the maximum number of multiple exceedances per individual is 365). Persons of interest in this exposure analysis are those with particular susceptibility to NO<sub>2</sub> exposure, namely individuals with asthma. The potential health effect benchmark levels used are appropriate for characterizing the potential risk of adverse health effects for asthmatics. The majority of the results presented in this section are for the entire (i.e., all ages) simulated asthmatic population because the pattern of exposure results for asthmatic children were very similar. However, the exposure analysis was also performed for the total population to assess numbers of persons exposed to these levels and to provide additional information relevant to the asthmatic population (such as time spent in particular microenvironments). The 1-hour exposure results are presented separately for three scenarios, (1) considering the exposures associated with as is air quality, (2) simulating exposures with air quality adjusted upwards to represent just meeting the current annual average standard, and (3) simulating exposures associated with air quality adjusted to represent just meeting alternative 1-hour daily maximum standards. In addition, the presence (or not) of indoor sources was also considered within each of these three scenarios.

### ***8.9.4.2 Estimated Number of 1-hour Exposures Above Selected Levels (as is)***

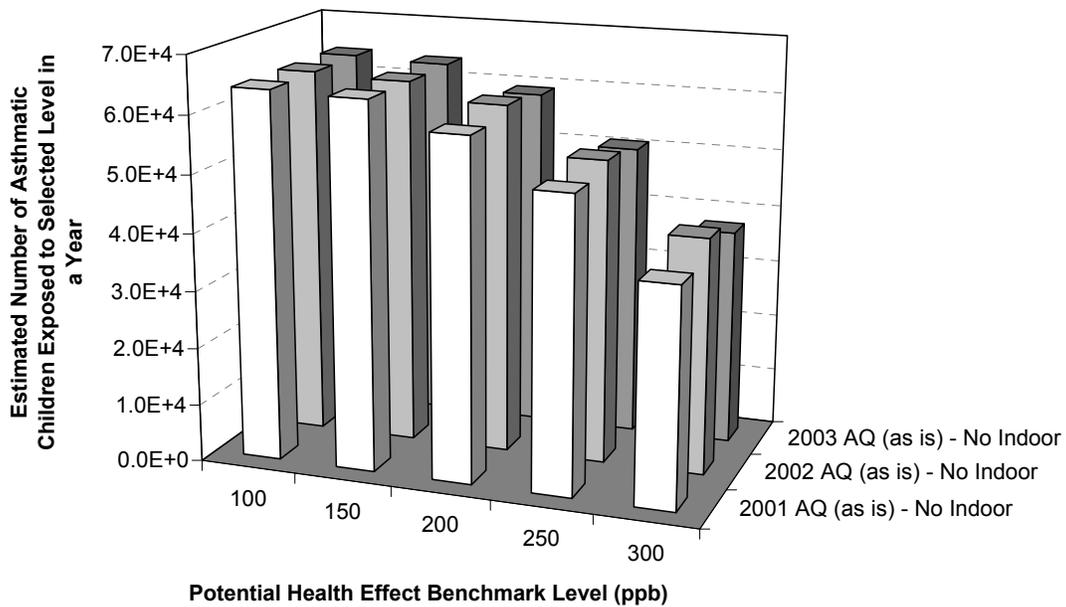
The results presented in this section were generated from the modeled air quality as input to APEX without any adjustment to the air concentrations or the potential health effect benchmark levels. Figure 8-13 summarizes the estimated number of asthmatics exposed at each of the potential health effect benchmark levels using the modeled air quality for each year,

without any contribution from indoor sources. As observed with the annual average exposure concentrations, there is great similarity in the estimated numbers of exceedances for each of the three years modeled. Year-to-year variability in the number of asthmatics exposed as indicated by a coefficient of variation (COV=mean/standard deviation) was at most 3.3%, calculated for the 300 ppb benchmark level. All persons (i.e., just over 212,000) were estimated to be exposed at least one time to a 1-hour daily maximum concentration of 100 ppb in a year. The number of asthmatics exposed to greater concentrations (e.g., 200 or 300 ppb) drops only slightly and is estimated to be somewhere between 117,000 – 196,000 depending on the 1-hour concentration level and year of air quality simulated. Similar patterns across the benchmark levels were observed for simulated asthmatic children, albeit with lower total numbers of asthmatic children with exposures at or above the potential health effect benchmark levels.

The results for all asthmatics and asthmatic children were similar in terms of the proportion of the population exposed and the year-to-year variability in numbers of exceedances. For example, nearly 61,000 asthmatic children were estimated to be exposed one time to a 1-hour daily maximum NO<sub>2</sub> concentration of at least 200 ppb for year 2002, comprising about 95% of that subpopulation (Figure 8-14). The number of children with at least one exceedance of 300 ppb was less, estimated to be about 41,000 using the 2002 air quality, or about 64% of all asthmatic children. As a comparison, the percent of all asthmatics experiencing exposures at or above 200 and 300 ppb was 92% and 59%, respectively. The year-to-year variability in the number of asthmatic children exposed at or above the selected benchmark levels was also small, although slightly higher than that estimated for all asthmatics. The highest COV for asthmatic children using the 3-year exposure estimates was also observed for exceedances of the 300 ppb benchmark (COV = 4.9%).



**Figure 8-13. Estimated number of all simulated asthmatics in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark levels, using modeled 2001-2003 air quality (as is), without indoor sources.**



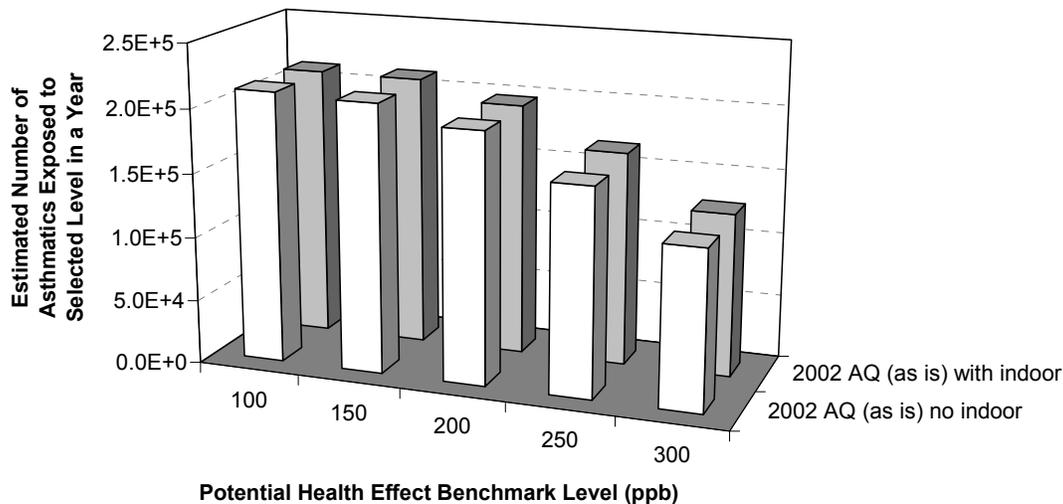
**Figure 8-14. Estimated number of simulated asthmatic children in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark levels, using modeled 2001-2003 air quality (as is), without modeled indoor sources.**

Additional exposure estimates were generated using the modeled 2002 air quality (as is). Those estimates include an evaluation of the contribution of indoor sources. APEX allows for the same persons to be simulated (i.e., demographics of the population were conserved), as well as using the same individual time-location-activity profiles generated for each person. Figure 8-15 illustrates the estimated number of asthmatics experiencing exposures above the potential health effect benchmarks, both with indoor sources and without indoor sources included in the model runs. The number of asthmatics at or above the selected benchmark levels at least one time in a year is very similar when including indoor source concentration contributions (i.e., gas cooking) compared to the number of persons whose exposure estimates did not include indoor sources. The reduction in numbers of asthmatics exposed at least once at or above any potential health effect benchmark level ranged from 0 to around 5,000 when indoor source contributions were excluded.

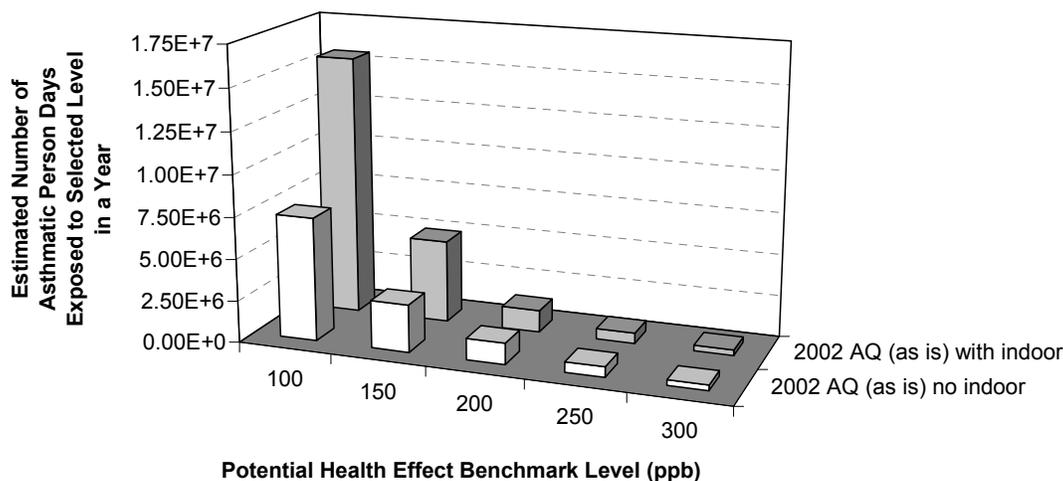
The number of person-days of exposure at or above a given potential benchmark level gives a different perspective on the contribution of indoor sources. Figure 8-16 illustrates the total number of days where the particular concentration level was exceeded, representing the sum of all multiple exposures (in contrast to focusing on persons as was done for example in Figure 8-13) for the simulated population in a given year. Since most individuals were exposed at least one time at many of the 1-hour levels, it was difficult to discern the effect that indoor sources had on the estimated exposures. Now it can be seen that the indoor source contribution increases not just the number of persons exposed, but more importantly how many times they would be exposed per year above the selected benchmark level. It appears that on average, there is an increase in the number of person-days by about a factor of 2.1 and 1.8 for the 100 and 150 ppb 1-hour concentration levels, respectively, while the higher benchmark levels are largely unaffected by the presence of indoor sources.

An evaluation of the time spent in the 12 microenvironments was performed to estimate where simulated individuals are exposed to concentrations above the potential health effect benchmark levels. Currently, the output generated by APEX is limited to compiling the microenvironmental time for the total population (includes both asthmatic individuals and healthy persons) and the summaries provide the total time spent above the selected potential health effect benchmark levels. As mentioned above, the data still provide a reasonable approximation for each of the population subgroups (e.g., asthmatics or asthmatic children)

because their microenvironmental concentrations and activities are not estimated any differently from those of the total population simulated by APEX.



**Figure 8-15. Estimated number of all simulated asthmatics in the Atlanta model domain with at least one NO<sub>2</sub> exposure at or above potential health effect benchmark levels, using modeled 2002 air quality (as is), both with and without modeled indoor sources.**



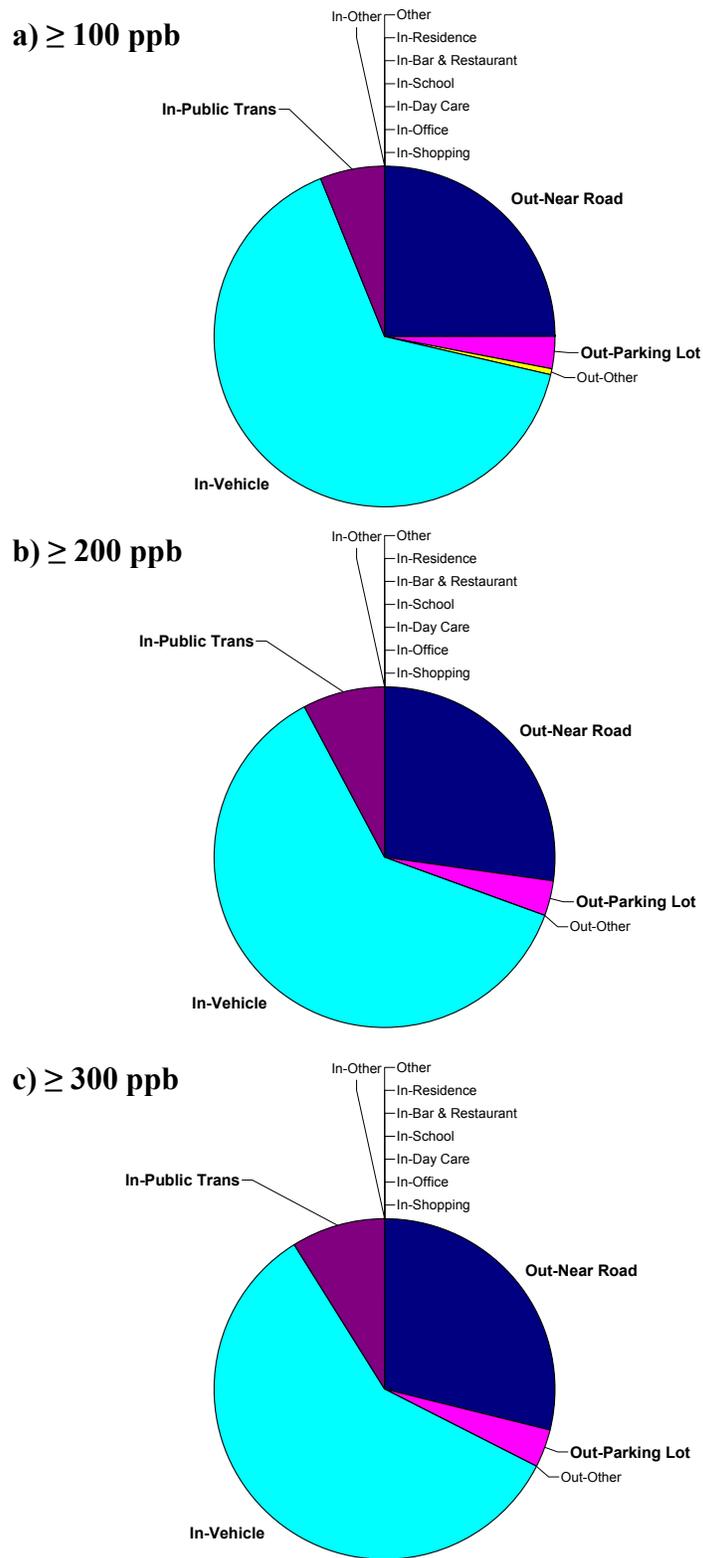
**Figure 8-16. Estimated number asthmatic person-days in the Atlanta model domain with an NO<sub>2</sub> exposure at or above potential health effect benchmark levels, using modeled 2002 air quality (as is), both with and without modeled indoor sources.**

As an example, Figure 8-17 (a, b, c) summarizes the percent of total time spent in each microenvironment for simulation year 2002 that was associated with estimated exposure concentrations at or above 100, 200, and 300 ppb (results for years 2001 and 2003 were similar). These estimated exposures summarized in this figure did not include the contribution from indoor sources. Time spent in the indoor microenvironments contributed little to the occurrence of estimated exposures at or above the selected benchmark levels. Most indoor microenvironments contributed < 1% of exposures to 1-h concentrations above 100 ppb and none of them contributed at all to exceedances of the 200 and 300 ppb benchmark levels. Most of the time associated with the high short-term exposures was associated with the transportation microenvironments (In-Vehicle or In-Public Transport) or outdoors (Outdoors-Near Road, Outdoors-Parking Lot, Outdoors-Other). The time spent outdoors near roadways exhibited an increase in contribution of exceedances of potential health benchmark levels, increasing from around 25 to 29% of time associated with concentrations of 100 and 300 ppb, respectively. The in-vehicle microenvironment showed a corresponding decrease, estimated as contributing to 65% of the time associated with 100 ppb exceedances, while contributing to 58% of 1-hour daily maximum exposures at or above 300 ppb. While more persons are likely to spend more time inside a vehicle than outdoors near roads, there is attenuation of the estimated on-road concentration that penetrates the in-vehicle microenvironment, leading to lowered concentrations. The result of this is that exposures above 300 ppb occur less frequently in-vehicles when compared with the outdoor near-road microenvironment that involves no attenuation of concentrations.

The microenvironments where the exposure exceedances occur were also identified for the estimated exposures that included indoor source contributions (Figure 8-18). While the transportation-associated microenvironments remained important for exposures above each of the selected levels, the time spent in the indoor microenvironments was also important for exceedances of hourly levels of 100 ppb, contributing to approximately 26% (inside a home) and 33% (inside bar/restaurant) of the time persons were exposed (Figure 8-18a). This is a result of the indoor source contribution to each individual's exposure concentrations and is consistent with what was observed regarding the effect of indoor sources on the total person-days of exposure. However, the importance of the indoor microenvironments decreases with the increasing benchmark levels. Exposures at or above 200-300 ppb occur rarely in the indoor

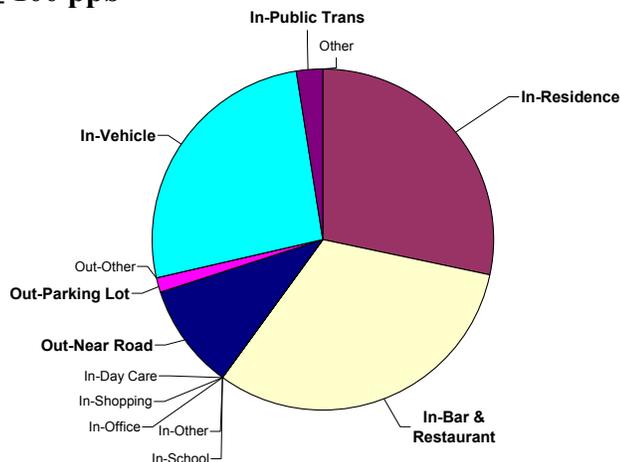
microenvironments, even when considering the indoor source contributions. The exposures at the higher benchmarks are associated mainly with the transportation microenvironments, increasing from about 39% of the time exposures occurred at the lowest potential health effect benchmark level (100 ppb) to comprising 100% of the time exposures occurred at the highest benchmark level (300 ppb, Figure 8-18c).

In the above analysis of persons exposed, the results show the number or percent of those with at least one day on which the 1-hour exposure was at or above the selected potential health effect benchmark level. Given that the benchmark is for a relatively short averaging time (i.e., 1-hour) it may be possible that individuals are exposed to concentrations at or above the potential health effect benchmark levels on several days in a given year. Since APEX simulates the longitudinal diary profile for each individual, the number of days with a 1-hour daily maximum exposure above a selected level is retained for each person. Figure 8-19 presents such an analysis for the year 2002, where the estimated exposures did not include indoor source NO<sub>2</sub> contributions. Nearly all simulated asthmatics (98.7%) experienced up to six exposures at or above 100 ppb, with nearly 78% experiencing at least six exposures at or above the 150 ppb level. Multiple exposures at or above the higher potential health effect benchmark levels were less frequent, with around 58, 28, and 12 percent of asthmatics exposed annually to four or more 1-hour NO<sub>2</sub> concentrations greater than or equal to 200, 250, and 300 ppb, respectively.

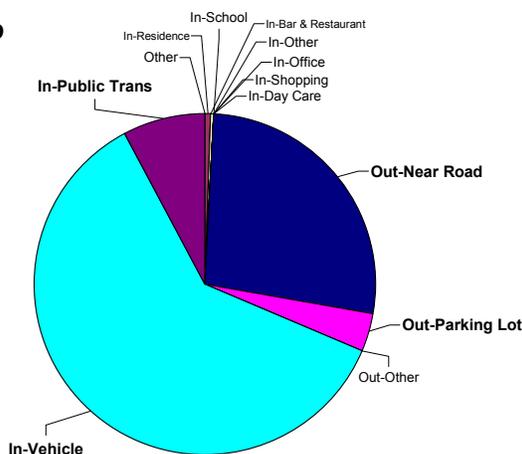


**Figure 8-17. Fraction of time all simulated persons in the Atlanta model domain spend in the twelve microenvironments that corresponds with exceedances of the potential NO<sub>2</sub> health effect benchmark levels, a)  $\geq 100$  ppb, b)  $\geq 200$  ppb, and c)  $\geq 300$  ppb, year 2002 air quality (as is) without indoor sources.**

a)  $\geq 100$  ppb



b)  $\geq 200$  ppb



c)  $\geq 300$  ppb

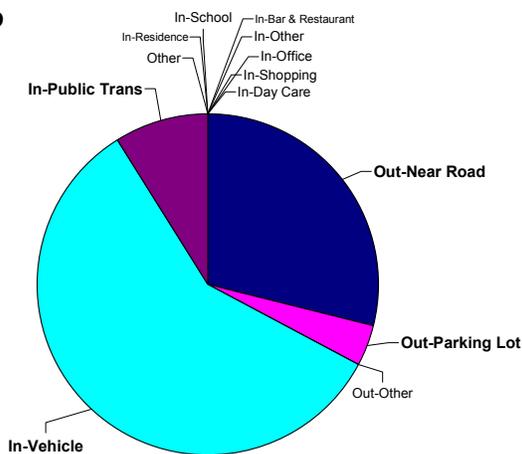
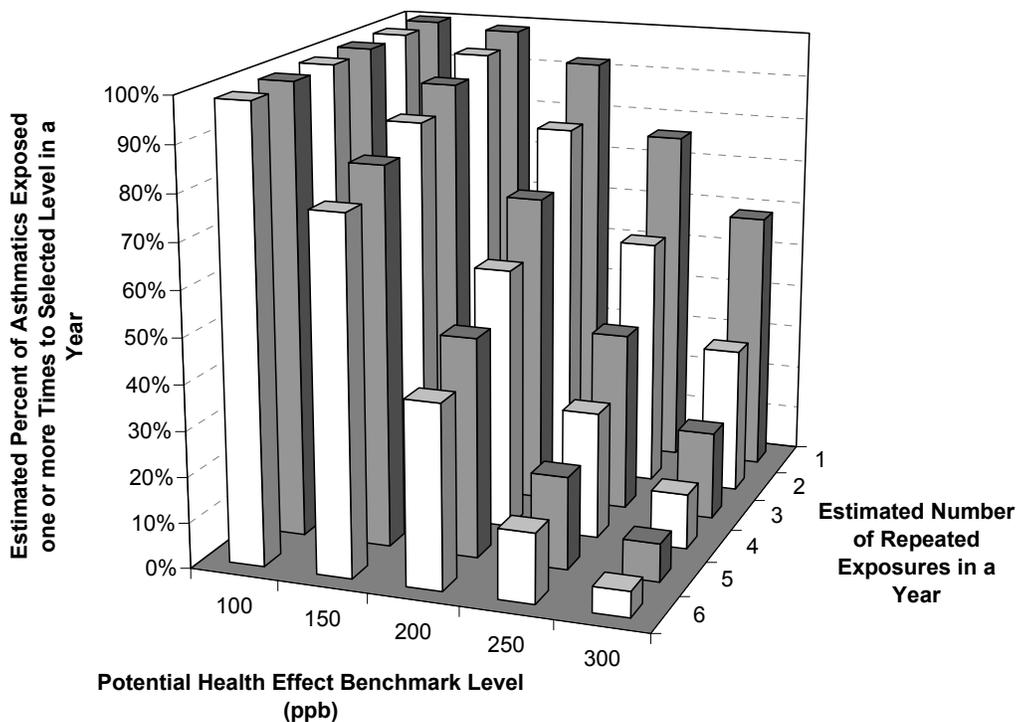
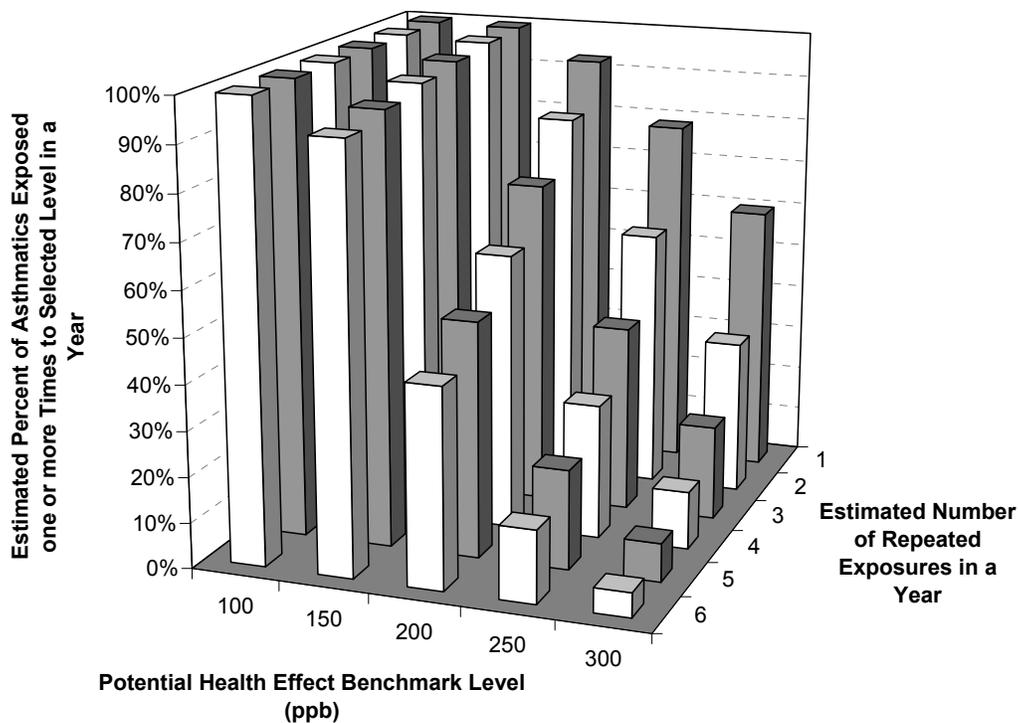


Figure 8-18. Fraction of time all simulated persons in the Atlanta model domain spend in the twelve microenvironments that corresponds with exceedances of the potential NO<sub>2</sub> health effect benchmark levels, a)  $\geq 100$  ppb, b)  $\geq 200$  ppb, and c)  $\geq 300$  ppb, year 2002 air quality (as is) with indoor sources.

The contribution of indoor sources to the occurrence of repeated exposure exceedances was also evaluated. Figure 8-20 illustrates that nearly all asthmatics (about 93%) would be exposed at least six times to either the 1-hour daily maximum 100 ppb or 150 ppb concentration level in a year when considering exposure to ambient NO<sub>2</sub> combined with indoor source emissions. This is approximately 15% more persons than was estimated for the simulations without indoor source contributions. The percent of asthmatics experiencing multiple exposures above the 200, 250 and 300 ppb was only about 1-4% greater than that observed for asthmatics without indoor sources. This is consistent with the person-day results that indicate the indoor source emissions contribute primarily to numbers of exposures experienced at or above the 100 or 150 ppb benchmark levels.



**Figure 8-19. Estimated percent of all asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above potential health effect benchmark levels, using modeled 2002 air quality (as is), without indoor sources.**



**Figure 8-20. Estimated percent of all asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above potential health effect benchmark levels, using modeled 2002 air quality (as is), with indoor sources.**

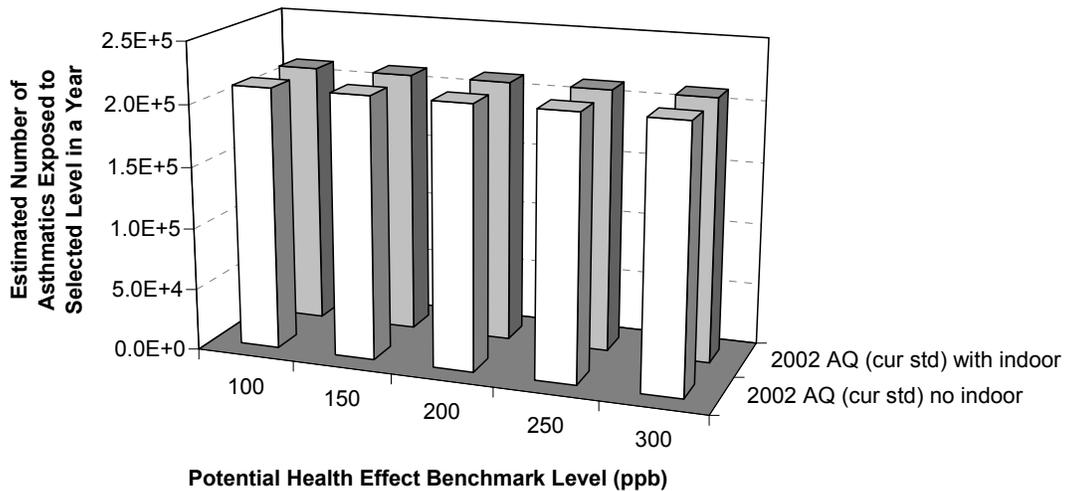
**8.9.4.3 Estimated Number of 1-hour Exposures Above Selected Levels (current standard)**

To simulate just meeting the current annual average NO<sub>2</sub> standard, the potential health effect benchmark levels were adjusted in the exposure model, rather than adjusting all of the hourly concentrations for each receptor and year simulated (see section 8.8). Similar to what was performed for the as is air quality, estimates of short-term exposures (i.e., 1-hour daily maximum) were generated for the total population and population subgroups of interest (i.e., asthmatics and asthmatic children).

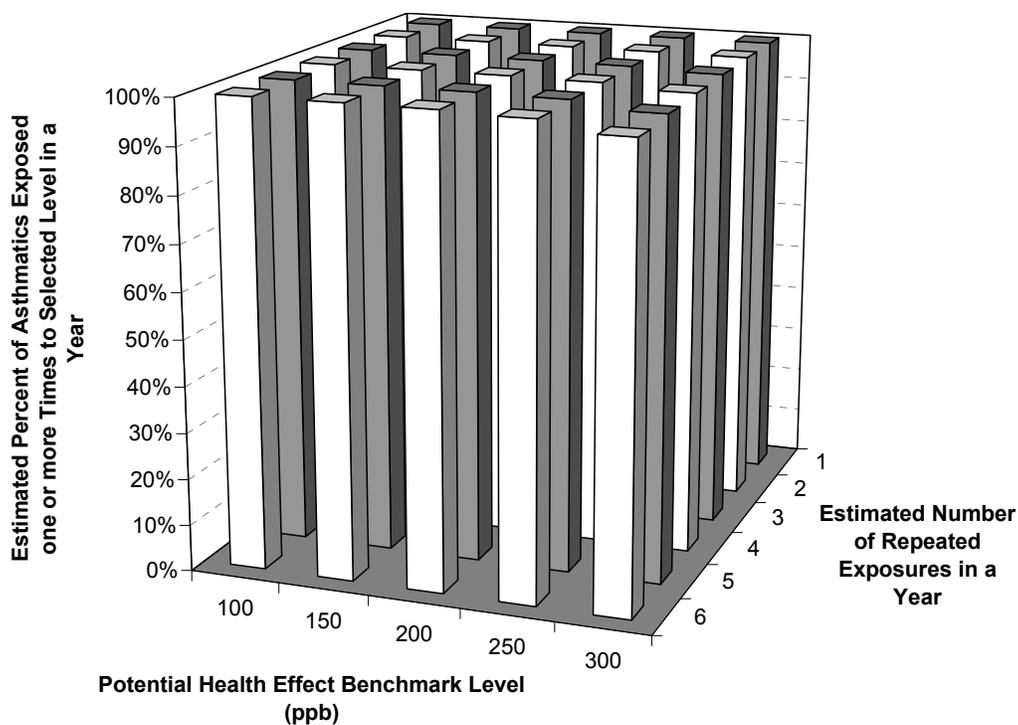
When considering the estimated exposures associated with air quality simulated to just meet the current annual average NO<sub>2</sub> standard, the number of persons experiencing concentrations at or above the potential health effect benchmarks is increased in comparison with as is air quality. Figure 8-21 illustrates the percent of asthmatics estimated to experience at least one exposure at or above the selected potential health effect benchmark concentrations, with air quality adjusted to just meet the current standard. The exposure results for both including and excluding indoor source contributions are presented. While it was estimated that about 92, 76,

and 59% of asthmatics would be exposed to 200, 250, and 300 ppb (1-hour average) at least once in a year for the as is air quality, it was estimated that nearly all asthmatics would experience at least one exposure above any of the potential health effect benchmark levels in a year when air quality is adjusted to just meet the current standard. Exposure estimates where indoor sources were included were not greatly different than the results without indoor source contributions, with nearly all asthmatics estimated to have at least one exposure at or above even the highest potential health effect benchmark level.

For air quality simulated to just meet the current standard, repeat exposures at the selected potential health effect benchmarks are more frequent than that estimated for the modeled as is air quality. Figure 8-22 illustrates this using the simulated asthmatic population for year 2002 data as an example. Nearly all asthmatics (>97%) were estimated to be exposed at or above any one of the selected levels for at least six times in a year. Results for asthmatics when exposures were estimated considering the contribution from indoor sources were similar, only slightly higher (data not shown).



**Figure 8-21. Estimated number of all asthmatics in the Atlanta modeling domain with at least one NO<sub>2</sub> exposure at or above the potential health effect benchmark level, using modeled 2002 air quality just meeting the current standard (cur std), with and without modeled indoor sources.**



**Figure 8-22. Estimated percent of asthmatics in the Atlanta modeling domain with repeated NO<sub>2</sub> exposures above health effect benchmark levels, using modeled 2002 air quality just meeting the current standard, without modeled indoor sources.**

**8.9.4.4 Estimated Number of 1-hour Exposures Above Selected Levels (alternative standards)**

To simulate just meeting the alternative NO<sub>2</sub> standards, the potential health effect benchmark level was adjusted in the exposure model, rather than adjusting all of the hourly concentrations for each receptor and year simulated (see section 8-8). Similar to exposure analyses performed with the as is air quality, estimates of short-term exposures (i.e., 1-hour daily maximum) were generated for the total population and population subgroups of interest (i.e., asthmatics and asthmatic children). Due to limitations on the number of concentration levels allowed in an APEX simulation, only the potential health effect benchmark levels of 100, 200, 300 ppb were evaluated for the alternative 1-hour daily maximum standards.

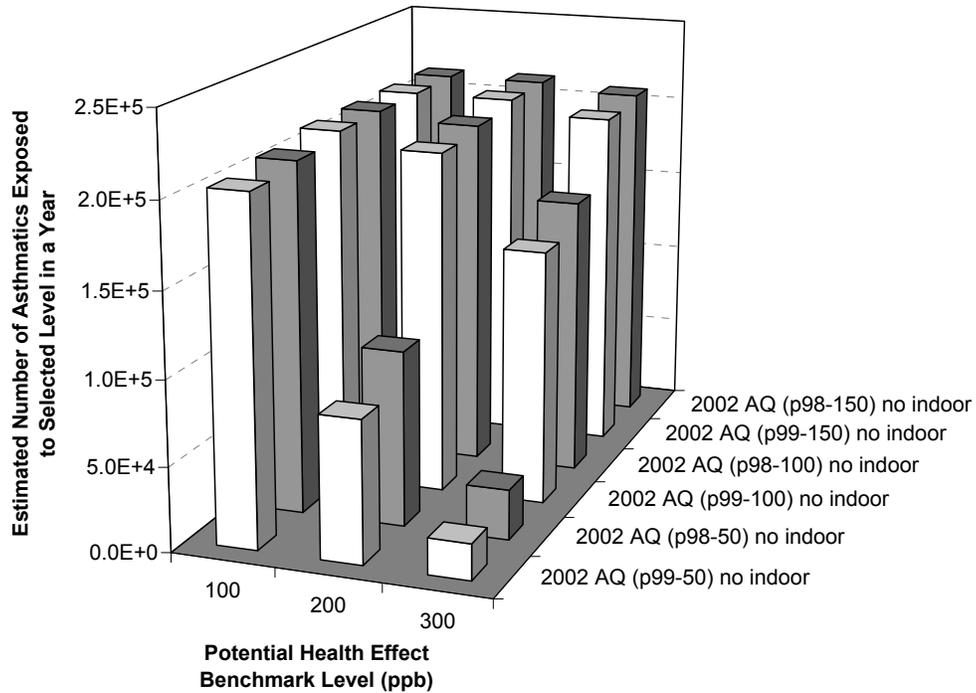
In considering exposures estimated to occur associated with air quality simulated to just meet the alternative NO<sub>2</sub> 1-hour daily maximum standards, the number of persons experiencing concentrations at or above the potential health effect benchmarks varied, depending on the form and level of the standard. Figure 8-23 illustrates the different forms (a 98<sup>th</sup> percentile or p98; 99<sup>th</sup> percentile or p99) at various 1-hour concentration levels of the standard. The number of

persons exposed at least once at each of the 98<sup>th</sup> and 99<sup>th</sup> percentiles alternative standards and considering a potential benchmark level of 100 ppb is similar to that observed for the as is air quality and for air quality adjusted to just meet the current standard. That is, most persons are exposed at least once to 100 ppb in a year, regardless of the standard form and level chosen. It is not until the level of the 1-hour daily maximum standard approaches 50 ppb for either of the percentile forms that the number of persons exposed to the higher benchmark levels is substantially reduced. For example, while nearly all asthmatics are exposed to 100 ppb at least once in a year as was observed in the above analyses, the percent of asthmatics exposed to at least one 1-hour concentration at or above the 200 or 300 ppb is reduced to about 49% and 14% of the subpopulation, respectively, when considering the 50 ppb, 98<sup>th</sup> percentile standard.

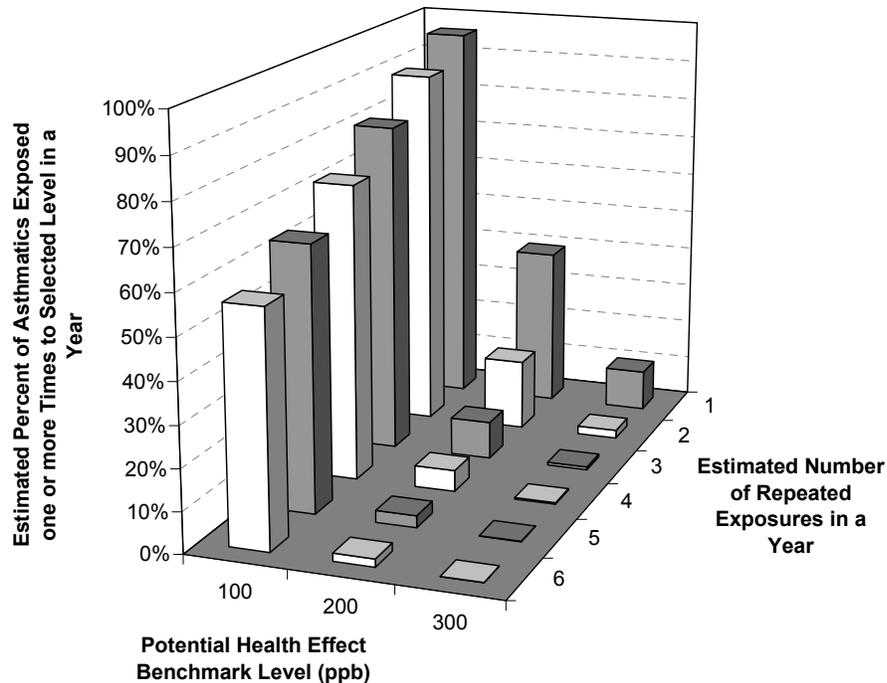
The estimated number of repeated NO<sub>2</sub> exposures above selected levels can be sharply reduced for potential alternative standards at the lower end of the range of alternative standards considered. As an example, Figure 8-24 illustrates the number of multiple exposures above the potential health effect benchmark levels using a 50 ppb, 99<sup>th</sup> percentile alternative standard. This is the first instance where multiple exposures of the 100 ppb benchmark are estimated to be reduced, with about 57% of asthmatics estimated to have greater than six in a year. A greater reduction in the number of multiple exposures is observed when considering the 200 ppb benchmark level. For example, only 5% of asthmatics are estimated to be exposed four or more times, compared with 58% using the 2002 air quality as is.

The effect of indoor source contributions to the exposures was also evaluated for the same level and form of alternative standard (50 ppb, 99<sup>th</sup> percentile). Figure 8-25 illustrates what has been consistently shown in the above analyses, the indoor sources primarily affect the numbers of persons and the number of times a person is exposed at or above 100 or 150 ppb, with limited contribution to the higher potential health effect benchmark levels.

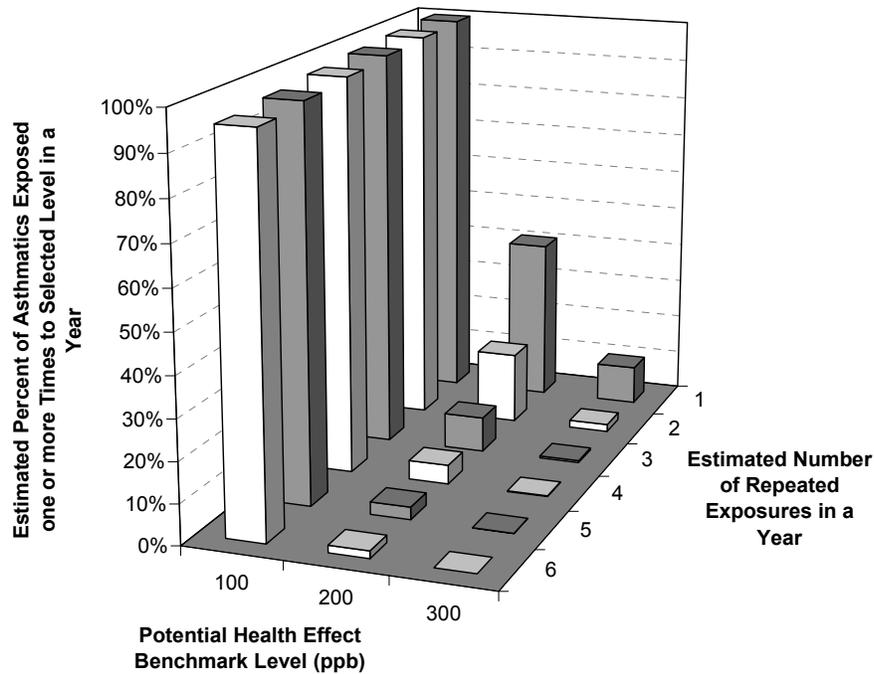
In addition, for comparison with the results presented in Figure 8-24, the percent of asthmatics exposed to the selected health effect benchmark levels considering the 100 ppb, 99<sup>th</sup> percentile alternative standard is presented in Figure 8-26. A greater proportion of asthmatics have multiple exposures at all of the 1-hour benchmarks, nearly all of which were estimated to have at least six exposures at or above a 1-hour concentration of 100 ppb.



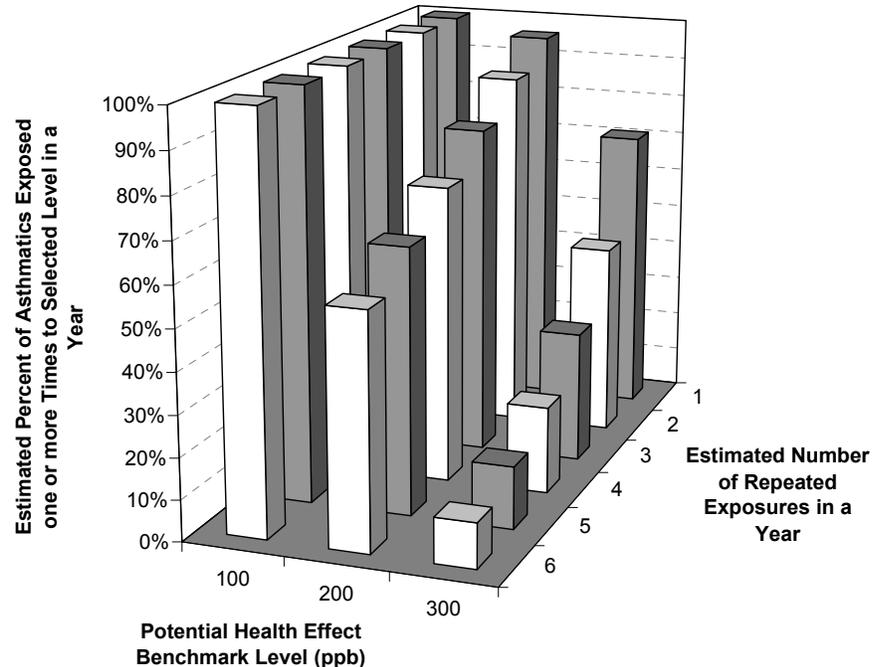
**Figure 8-23. Estimated percent of asthmatics in the Atlanta modeling domain with NO<sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting potential alternative standards, without indoor sources.**



**Figure 8-24. Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO<sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 50 ppb level 99<sup>th</sup> percentile form alternative standard, without indoor sources.**



**Figure 8-25.** Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO<sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 50 ppb level 99<sup>th</sup> percentile form alternative standard, with indoor sources.



**Figure 8-26.** Estimated percent of asthmatics in the Atlanta modeling domain with multiple NO<sub>2</sub> exposures at or above potential health effect benchmark levels, using modeled 2002 air quality adjusted to just meeting a 100 ppb level 99<sup>th</sup> percentile form alternative standard, without indoor sources.

## 8.10 KEY OBSERVATIONS

Presented below are key observations resulting from the exposure assessment:

- Modeled concentrations are reasonable given comparisons to available data
  - AERMOD predicted upper percentile NO<sub>2</sub> concentrations are about 10 to 50% (or 10 to 30 ppb) higher than ambient measurements at three fixed-site monitors
  - APEX modeled daily mean NO<sub>2</sub> exposures in Atlanta (medians 3-24 ppb) are comparable to personal exposure measurements in Atlanta (median 3-14 ppb)
  - APEX modeled annual average NO<sub>2</sub> exposures, expressed as a percent of the ambient NO<sub>2</sub> concentration (40 to 70%), are consistent with findings reported in the ISA (30 to 60%)
- Estimated exposures above 1-hour potential health effect benchmark levels using APEX were due largely to roadway-related exposures (>99%). Of this, approximately 70% were from in-vehicle exposures, with the remainder associated with outdoor near-road exposures
- When included, indoor sources contribute to the occurrence of NO<sub>2</sub> exposures at or above 100 ppb (61%), but little to the occurrence of higher exposures (i.e., above 200, 300 ppb)
- The estimated effect of air quality on benchmark exceedances differs by benchmark level considered:
  - 100 ppb
    - For all air quality scenarios considered, more than 90% of asthmatics in Atlanta are estimated to be exposed at least one time per year
    - Of the standard levels evaluated, 50 ppb was the only level estimated to reduce repeat exposures to NO<sub>2</sub> concentrations above 100 ppb compared to recent air quality levels
  - 200 ppb
    - Of all the air quality scenarios considered, only alternative standards set at 50 ppb are estimated to reduce the percent of

asthmatics exposed at least one time (by approximately 40 to 50%) relative to recent air quality levels

- 300 ppb
  - Of all the air quality scenarios considered, only alternative standards set at 50 ppb or 100 ppb are estimated to reduce the percent of asthmatics exposed at least one time (by approximately 80% and 15% respectively) relative to recent air quality levels
- When simulating air quality that just meets the current annual standard, virtually all asthmatics in Atlanta are estimated to experience six or more daily maximum 1-hour exposures per year to NO<sub>2</sub> concentrations above the highest benchmark level evaluated (300 ppb)
- Using a 98<sup>th</sup> versus a 99<sup>th</sup> percentile form made a difference of approximately 5 to 10% in the number of daily maximum benchmark exceedances, with the 99<sup>th</sup> percentile form generating fewer exceedances.

## **8.11 REPRESENTATIVENESS OF EXPOSURE RESULTS**

### **8.11.1 Introduction**

Due to time and resource constraints the exposure assessment evaluating the current and alternative standards was only applied to the Atlanta urban area. A natural question is how representative are the estimates from this assessment of exposures in Atlanta to other urban areas in the United States. To address this question, additional data were compiled and analyzed to provide perspective on how representative the Atlanta exposure modeling results might be for other urban areas. Because most estimated exceedances were associated with the near-road or in-vehicle microenvironments, the analysis and discussion is centered on a variety of population and road statistics to allow for comparison of Atlanta with several other urban locations.

### **8.11.2 Description of Data Compiled and Summarized**

Three sources were used for comparing Atlanta with several urban locations: 1) the Human Air Pollutants Exposure Model near-road population data base, 2) American Housing Survey (AHS) data, and 3) statistics from the Federal Highway Administration (FHWA).

### **8.11.2.1 HAPEM6 Near-Road Population Data Base**

The first type of data considered employs a data base developed for the Human Air Pollutants Exposure Model (HAPEM6) and is available as part of the HAPEM6 installation package.<sup>19</sup> The data base provides estimates of the fraction of people in each of 6 age groups in each US Census tract living near major roadways.<sup>20</sup> The distance-from-roadway bins are: 0 m to 75 m, 75 m to 200 m, and > 200 m. Details regarding the data base development are provided in Appendix B, Attachment 6 and 7.

Briefly, the development of the HAPEM6 near-road population data base was for use in estimating the enhancement near major roadways of air toxic pollutant concentrations from on-road motor vehicle emissions relative to concentrations at other outdoor locations. First, several measurement studies of near roadway concentration gradients were reviewed (Kwon, 2005; Meng et al., 2004; Riediker et al., 2003; Rodes et al., 1998; Weisel et al., 2004; Zhu et al., 2002). None of these studies provided sufficient data to estimate the required concentration ratios. Measurements in the Riediker et al. (2003) and Rodes et al. (1998) studies were made at distances shorter than those of interest for this study. Available ratios for Zhu et al. (2002) were only for downwind distances, and did not represent ratios under more general meteorological conditions. The ability of regression models applied to the Relationships of Indoor, Outdoor, and Personal Air (RIOPA) study data (Kwon et al., 2005; Meng et al., 2004; and Weisel et al., 2004) to predict the near roadway concentrations was generally poor, likely due to the problem that the near roadway concentrations are also impacted by other emissions sources that cannot be easily adjusted for.

CALPUFF air dispersion model predictions from the Portland (OR) Air Toxics Assessment were analyzed (PATA; Cohen et al., 2005) using summary statistics and regression modeling in order to obtain distributions of concentration ratios. A distribution was developed based on the ratio of concentrations within a distance  $D_1$  meters of a major roadway, to concentrations at locations greater than a second distance,  $D_2$  meters from a major roadway. A second distribution was developed using the ratio of concentrations at  $D_1 - D_2$  meters of a major roadway to concentrations at locations greater than  $D_2$  meters from a major roadway.

---

<sup>19</sup> [http://www.epa.gov/ttn/fera/human\\_hapem.html](http://www.epa.gov/ttn/fera/human_hapem.html).

<sup>20</sup> Ages 0-1, 2-4, 5-15, 16-17, 18-64, and  $\geq 65$ .

To determine the best choices for the distance-to-roadway bins, regression models were developed to estimate two distance terms  $D_1$  and  $D_2$  for each season and for the annual mean. Generally, the two-distance term regression models applied to the PATA CALPUFF data showed that the optimal road distances, based on  $R^2$  and Akaike Information Criterion (AIC) statistics, were  $D_1 = 75$  m and  $D_2 = 300$  m. The regression models favored the higher distances for  $D_2$  because the CALPUFF estimates for PATA continue to decrease significantly with distance from the road. However, the  $R^2$  and AIC values were not much different between the models with  $D_2 = 200$  or 300 m. Because a few other studies, including Zhu et al. (2002), have shown typical zones of influence for roadways no further than 200 m, distances of  $D_1 = 75$  m and  $D_2 = 200$  m were selected for development of the population data base.

Then an analysis was conducted using the US Census block level populations stratified by age. The block level data were then aggregated up to the tract level for populations stratified by age for use in HAPEM6. The data bases used were:

- The Environmental Sciences Research Center (ESRI) StreetMap US roadway geographic database (which includes NavTech, GDT and TeleAtlas rectified street data)
- A geographic database of US Census block boundaries, extracted using the PCensus 2000 Census data extraction tool for Census file SF1
- A geographic database for US Census block boundaries in Puerto Rico and the US Virgin Islands obtained from Proximity

Because populations are not generally evenly distributed within blocks, it was assumed that the block populations all reside within 150 m of at least one road within the block of designation “local” or greater as defined by the Census Feature Class Codes (CFCC). Thus, the first step was to create a 150 m buffer around all roadways within the block. This buffer served as a “clipped” block boundary defining the portion of the block containing residential populations. The block population was assumed to be uniformly distributed within the “clipped” block boundary. Next, a 75 m buffer and a 200 m buffer were created around all major roadways within the block. These buffers were overlaid on the “clipped” block boundary, and the fraction of the “clipped” block area that fell within each buffer was calculated. This area fraction was assumed to equal the population fraction that fell within each buffer, and the fractions were applied to each population subgroup. The 75 m buffer and the 200 m buffer were also overlaid

on the unclipped block boundary to determine the fraction of the total block area that fell within each of the buffers. The block level fractions for area and populations were then aggregated up to the tract level.

For each tract in the CMSA or MSA, the tract-level, age-stratified distance-to-roadway population fractions were combined with 2000 US Census tract-level, age-stratified residential populations to calculate the CMSA- or MSA-level distance-to-roadway population fractions, as follows.

$$\text{CMSA fraction} = \frac{\sum_{\text{tracts}} (\text{tract-fraction} * \text{tract-population})}{\sum_{\text{tracts}} (\text{tract-population})}$$

Results of the estimated population within the selected roadway distances are provided in Table 8-14 for each of the 18 named locations identified in the air quality characterization. Based on the HAPEM6 data base, Atlanta has the lowest percent of its population living within each of the near-roadway distance bins. Absolute differences for the closest road distance bin ranged from 4 to 16 percentage points lower for Atlanta compared with all other locations. Atlanta also was estimated to have the greatest percent of the population living at distances  $\geq 200$  m from a major road. Consideration of just this attribute would suggest that on a population-weighted basis, the number of daily maximum exposures of NO<sub>2</sub> concentrations above benchmark levels may be lower for Atlanta than many of the other urban areas listed in Table 8-14. However, note that most of the exceedances were associated with in-vehicle exposures.

**Table 8-14. Percent of population within selected distances of a major road in several locations.**

Location	Percent of Population at Distance from a Major Road <sup>1</sup>		
	<75 m	>75 m, <200 m	$\geq 200$ m
Atlanta	16%	23%	62%
Boston	27%	38%	35%
Chicago	27%	38%	35%
Cleveland	20%	36%	44%
Colorado Springs	21%	32%	47%
Denver	23%	34%	43%
Detroit	22%	33%	45%
El Paso	24%	35%	41%
Jacksonville	21%	29%	49%
Las Vegas	22%	33%	45%
Los Angeles	26%	38%	37%
Miami	26%	37%	37%
New York	32%	37%	30%

Location	Percent of Population at Distance from a Major Road <sup>1</sup>		
	<75 m	>75 m, <200 m	≥200 m
Philadelphia	25%	34%	42%
Phoenix	22%	34%	44%
Provo	19%	31%	51%
St. Louis	20%	31%	49%
Washington DC	25%	27%	48%
<b>Notes:</b> <sup>1</sup> Major roadways were all roadways except those classified as local by the US Census Bureau.			

### 8.11.2.2 American Housing Survey (AHS) Data

The American Housing Survey (AHS), conducted by the Bureau of the Census for the Department of Housing and Urban Development (HUD), collects data on the nation's housing (AHS, 2008). Relevant housing characteristic data, including housing units within 300 m of a major highway, railroad, or airport and residential prevalence of air conditioning are summarized for 14 of the named locations<sup>21</sup> evaluated in the air quality characterization, using the available metropolitan areas surveyed by the AHS (Table 8-15). Because survey years differ for each location and some locations contained more than one survey, the most recent data or data closest to 2002 were selected (the base year for the exposure modeling). Consistent with the pattern noted for the population living near roadways, Atlanta also has the lowest percent of housing units (9.7%) within 300 m of a 4 or more lane highway, railroad, or airport (AHS, 2008). Denver, Phoenix, and St. Louis were only slightly higher though, estimated to have 10.1, 11.2, and 11.4% of housing units, respectively < 300 m from these same locations. Again, consideration of this attribute alone, suggests daily maximum exposures to NO<sub>2</sub> concentrations above selected benchmarks would tend to be lower for Atlanta than many of the other urban areas listed in Table 8-15. As noted previously, the estimated exceedances were dominated by exposure occurring in-vehicles.

The AHS also provides data on A/C prevalence rate for several urban areas (Table 8-15). The A/C prevalence can vary greatly across urban areas, based largely on climate differences. The air conditioning prevalence can influence the air exchange rate in a residence, potentially affecting the infiltration rate of outdoor air concentrations into the indoors residential microenvironment. Atlanta was estimated to have one of the highest air conditioning prevalence

<sup>21</sup> There are no AHS data for Colorado Springs, El Paso, Jacksonville, and Las Vegas.

rates (97.2%), though similar rates could be found in Miami, Phoenix, St. Louis, and Washington D.C. A few of the urban areas listed have much lower A/C prevalence rates, including Los Angeles with 57.4% and Boston with 63.1%. For locations having a low A/C prevalence, it is expected that the number of indoor residential exposures to daily maximum NO<sub>2</sub> concentrations above selected benchmarks would be greater compared to those estimated in Atlanta. However, results of this exposure assessment indicate the indoor residential microenvironment does not contribute to exceedances of the selected benchmarks (see Figure 8-17) even when considering alternative A/C prevalence (section 8.12.2.6).

**Table 8-15. Residential A/C prevalence and roadway distance statistics for housing units in several locations (AHS, 2008).**

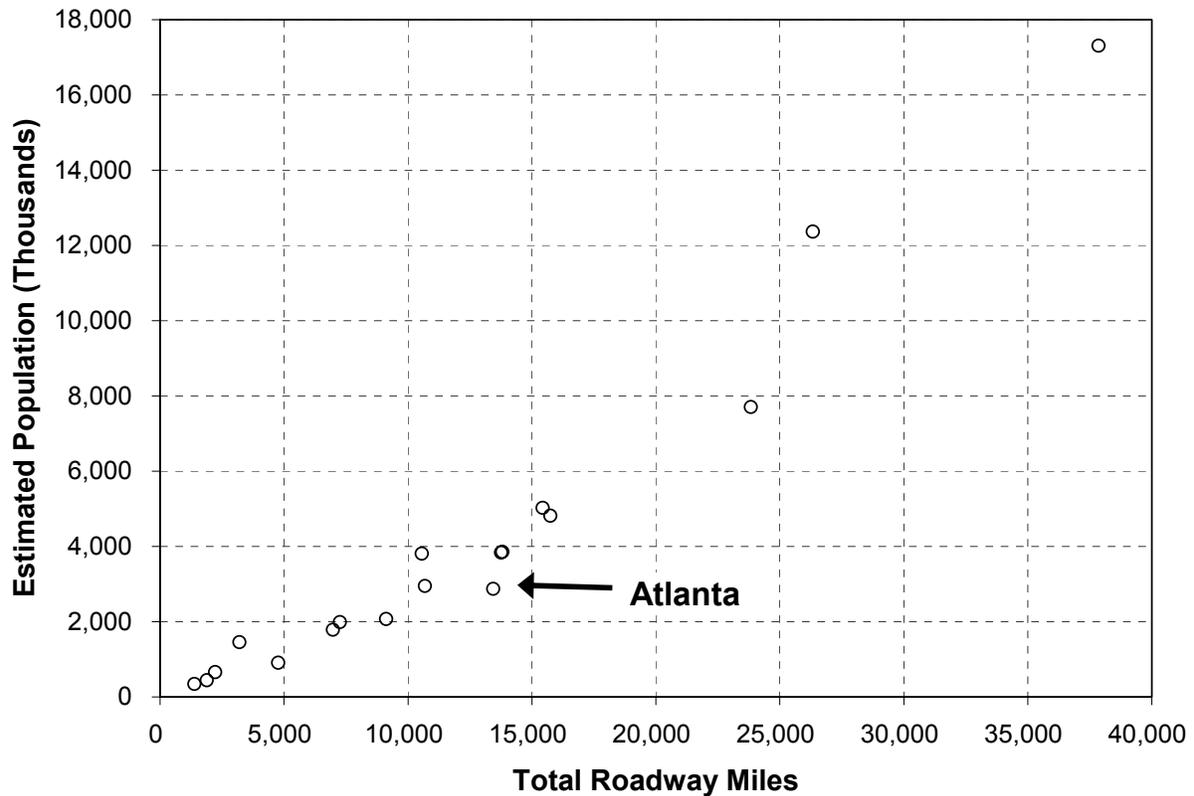
Location	AHS Survey Year	Housing units < 300 m from a mobile source <sup>1</sup> (%)	A/C Use <sup>2</sup> (%)
Atlanta	2004	9.7	97.2
Boston	1998	14.3	63.1
Chicago	2003	18.8	89.6
Cleveland	2004	19.0	75.8
Denver	2004	10.1	66.9
Detroit	2003	17.7	82.4
El Paso	ND	ND	ND
Jacksonville	ND	ND	ND
Las Vegas	ND	ND	ND
Los Angeles	2003	14.2	57.4
Miami	2002	15.8	98.1
New York	2003	14.8	83.3
Philadelphia	2003	14.3	91.4
Phoenix	2002	11.2	94.4
Provo	ND	ND	ND
St. Louis	2004	11.4	96.7
Washington DC	1998	17.6	96.0
<b>Notes:</b>			
ND No data available			
<sup>1</sup> Represents the percent of total year-round housing units located within 300 meters of a 4 or more lane highway, railroad, or airport (AHS, 2008).			
<sup>2</sup> Represents the percent of total year-round housing units having central or room unit air conditioners (AHS, 2008).			

### 8.11.2.3 Federal Highway Administration (FHWA) Data

The Federal Highway Administration (FHWA) produces annual highway statistics reports based on highway data submitted by individual States. Because 2002 served as the base year for the exposure assessment, these data were compiled for each of the named locations evaluated in the air quality characterization and are presented in Table 8-16. We note that Atlanta contains the highest per capita daily vehicle miles traveled and also the highest miles of roadway per 1,000 persons, both attributes which would tend to result in greater population exposure to peak NO<sub>2</sub> concentrations associated with roadway exposures. However, as shown in Figure 8-27, Atlanta is roughly in the middle of the distribution with respect to estimated population and total roadway miles when compared with the other locations examined.

**Table 8-16. Population and roadway statistics for several locations (FHWA, 2002).**

<b>Location</b>	<b>Total roadway miles</b>	<b>Estimated Population (thousands)</b>	<b>Miles of roadway per 1,000 persons</b>	<b>Total DVMT<sup>1</sup> per capita</b>
Atlanta	13,438	2,873	4.7	35.3
Boston	13,809	3,854	3.6	20.9
Chicago	23,832	7,702	3.1	21.5
Cleveland	6,975	1,785	3.9	20.6
Colorado Springs	1,887	439	4.3	20.4
Denver	7,261	1,989	3.7	22.9
Detroit	13,755	3,835	3.6	25.1
El Paso	2,225	657	3.4	17.3
Jacksonville	4,769	906	5.3	31.0
Las Vegas	3,206	1,456	2.2	18.1
Los Angeles	26,329	12,365	2.1	23.7
Miami	15,436	5,021	3.1	23.9
New York	37,854	17,307	2.2	15.9
Philadelphia	15,743	4,813	3.3	19.4
Phoenix	10,684	2,949	3.6	21.2
Provo	1,384	345	4.0	21.2
St. Louis	9,123	2,067	4.4	29.2
Washington DC	10,561	3,807	2.8	22.7



**Figure 8-27. Comparison of estimated population and total roadway miles in 18 locations (data from FHWA (2002) provided in Table 8-16).**

### 8.11.4 Discussion

There are a number of important attributes to take into account in considering the representativeness of the exposure modeling results from Atlanta with regard to other urban locations not modeled in the exposure assessment. As noted above, Atlanta falls on the low end relative to the other urban areas examined (i.e, 18 locations in all listed in Table 8-14) with respect to percent of population living within selected distances of a major road (see Table 8-14) and percent of the population residing in housing units < 300 m from a mobile source (see Table 8-15). Being on the low end with respect to these two attributes would likely result in Atlanta having a lower proportion of the population likely being exposed to daily maximum 1-hour NO<sub>2</sub> concentrations above selected benchmark levels than other locations included in Tables 8-14 and 8-15, in the absence of other influential attributes. However, as noted above, Atlanta contains

the highest per capita daily vehicle miles traveled and also the highest miles of roadway per 1,000 persons, both attributes which would tend to result in greater population exposure to NO<sub>2</sub> concentrations above selected benchmark levels associated with increased roadway exposures. While the miles of roads per person is generally accounted for by the current exposure modeling, the DVMT specific to Atlanta is not well represented and is likely more similar to a national DVMT.

Furthermore, Atlanta is roughly in the middle of the distribution with respect to estimated population and total roadway miles when compared with the other locations examined. Given that there are attributes that go in both directions with respect to the influence of on-road and other mobile sources on the representativeness of Atlanta relative to the other 17 urban areas examined in the air quality analysis, staff judges that the Atlanta exposure estimates are likely representative of other moderate to large urban areas included in this comparison. Staff does recognize that the Atlanta exposure results are likely lower on a population-weighted basis compared to the largest urban areas such as Los Angeles, New York, and Chicago given the greater proximity of the population to mobile sources in these large urban areas. For example, 64, 69, and 65%, respectively, of Los Angeles, New York, and Chicago's populations live within 200 m of a major road compared to only 39% for Atlanta. Similarly, there is a much higher percentage of housing units within 300 m of a mobile source (i.e., 14.2, 14.8, and 18.8%, respectively, for Los Angeles, New York, and Chicago), compared to only 9.7% for Atlanta.

As discussed above, with respect to residential A/C prevalence, we expect that urban areas with lower A/C prevalence would tend to result in higher exposures to NO<sub>2</sub> of ambient origin, all other factors being equal. We note that in comparing A/C prevalence rates for each location, Boston, Cleveland, Denver, and Los Angeles had between 20 to 40 percentage points less prevalence of A/C in residences than that observed for Atlanta. Thus, based on this consideration, the estimated percent of population exposed and person days with exposures above the selected health effect benchmarks for Atlanta may be somewhat lower than would be expected for other urban areas within the parts of the U.S. (e.g., urban areas in the midwest, northeast, southern California, and northwest) with lower prevalence of residential A/C.

## 8.12 UNCERTAINTY ANALYSIS

The methods and the model used in this exposure assessment conform to the most contemporary modeling methodologies available. APEX is a powerful and flexible model that allows for the realistic estimation of air pollutant exposure to individuals. Since it is based on human time-location-activity diaries and accounts for the most important variables known to affect exposure (where people are located and what they are doing), it has the ability to effectively approximate actual exposure conditions. In addition, the input data selected were the best available data to generate the exposure results. However, there are constraints and uncertainties with the modeling approaches and the input data that limit the realism and accuracy of the modeling results.

Uncertainties and assumptions associated with NO<sub>2</sub> specific model inputs, their utilization, and application are discussed in the following sections. Analyses for certain components of APEX performed previously in other NAAQS reviews (see EPA, 2007g; Langstaff, 2007) that are relevant to the NO<sub>2</sub> NAAQS review are only summarized below. This includes a sensitivity analyses performed on the CHAD data base using O<sub>3</sub> exposures and an analysis of the air exchange rate data.

Following the same general approach described in section 7.8 and adapted from WHO (2008), a qualitative analysis of the components contributing to uncertainty in the exposure results was performed. This includes an identification of the important uncertainties, an indication of the potential bias direction, and a scaling of the uncertainty using *low*, *medium*, and *high* categories. Even though uncertainties in AERMOD concentrations predictions are an APEX input uncertainty, they are addressed separately here for clarity.

**Table 8-17. Summary of qualitative uncertainty analysis for the exposure assessment.**

Source	Type	Concentration/ Exceedance Bias Direction	Characterization of Uncertainty
AERMOD Inputs and Algorithms	AERMOD formulations for mobile sources	unknown	Low
	On-road emissions	over	Low – Medium
	O <sub>3</sub> monitoring data	over	Low
	Use of unadjusted NO <sub>2</sub> concentrations	unknown	Low – Medium
	Meteorological data	unknown	Low – Medium
APEX Inputs and	Population data base	both	Low
	Commuting data base	both	Low - Medium

Algorithms	CHAD data base	under	Low - High
	Meteorological data	both	Low
	Air exchange rates	unknown	Medium
	A/C prevalence	none	Low
	Indoor sources not modeled	under	Medium
	Indoor decay distribution	under	Low - Medium
	Indoor concentration distribution	under	Medium - High
	Longitudinal profile	both	Low

### 8.12.1 Dispersion Modeling Uncertainties

Air quality data used in the exposure modeling was determined through use of EPA’s recommended regulatory air dispersion model, AERMOD (version 07026 (EPA, 2004)), with meteorological data discussed above and emissions data based on the EPA’s National Emissions Inventory for 2002 (EPA, 2007e) and the CAMD Emissions Database (EPA, 2007f) for stationary sources and mobile sources determined from local travel demand modeling and EPA’s MOBILE6.2 emission factor model. All of these are high quality data sources. Parameterization of meteorology and emissions in the model were made in as accurate a manner as possible to ensure best representation of air quality for exposure modeling. Thus, the resulting air quality values are likely free of systematic errors to the best approximation available through application of modeled data.

An analysis of uncertainty associated with application of a model is generally broken down into two main categories of uncertainty: 1) model algorithms, and 2) model inputs. While it is convenient to discuss uncertainties in this context, it is also important to recognize that there is some interdependence between the two in the sense that an increase in the complexity of model algorithms may entail an increase in the potential uncertainty associated with model inputs.

#### 8.12.1.1 AERMOD Algorithms

The AERMOD model was promulgated by in 2006 as a “refined” dispersion model for near-field applications (with plume transport distances nominally up to 50 kilometers), based on a demonstration that the model produces largely unbiased estimates of ambient concentrations across a range of source characteristics, as well as a wide range of meteorological conditions and topographic settings (Perry, *et al.*, 2005; EPA, 2003). While a majority of the 17 field study

databases used in evaluating the performance of AERMOD are associated with elevated plumes from stationary sources (typically power plants), a number of evaluations included low-level releases more typical of the dominant emissions category in this assessment. Moreover, the range of dispersion conditions represented by these evaluation studies provides some confidence that the fundamental dispersion formulations within the model will provide robust performance in other settings.

AERMOD is a steady-state, straight-line plume model, which implies limitations on the model's ability to simulate certain aspects of plume dispersion. For example, AERMOD treats each hour of simulation as independent, with no memory of plume impacts from one hour to the next. As a result, AERMOD may not adequately treat dispersion under conditions of atmospheric stagnation or recirculation when emissions may build up within a region over several hours. This could lead to a bias toward under-prediction by AERMOD during such periods. On the other hand, AERMOD assumes that each plume may impact the entire domain for each hour, regardless of whether the actual transport time for a particular source-receptor combination exceeds an hour. While these assumptions imply some degree of physically unrealistic behavior when considering the impacts of an individual plume simulation, their importance in terms of overall uncertainty will vary depending upon the application. The degree of uncertainty attributable to these basic model assumptions is likely to be more significant for individual plume simulations than for a cumulative analysis based on a large inventory. This question deserves further investigation to better define the limits and capabilities of a modeling system such as AERMOD for large scale exposure assessments such as this. The evidence provided by the model-to-monitor comparisons presented in Chapter 8.4.8 is encouraging as to the viability of the approach in this application when adequate meteorological and other inputs are available. However, each modeling domain and inventory will present its own challenges and will require a separate assessment based on the specifics of the application.

Beyond the basic dispersion algorithms in AERMOD, another component of model formulation uncertainty for this application is the use of the Ozone Limiting Method (OLM) and Plume Volume Molar Ratio Method (PVMRM) algorithms for simulating the conversion of NO to NO<sub>2</sub>. Model performance evaluations for these NO<sub>x</sub> chemistry algorithms are more limited than for non-reactive plume dispersion. However, an assessment of the potential for bias

associated with the PVMRM algorithm showed generally good performance based on the available evaluation data (MACTEC, 2005).

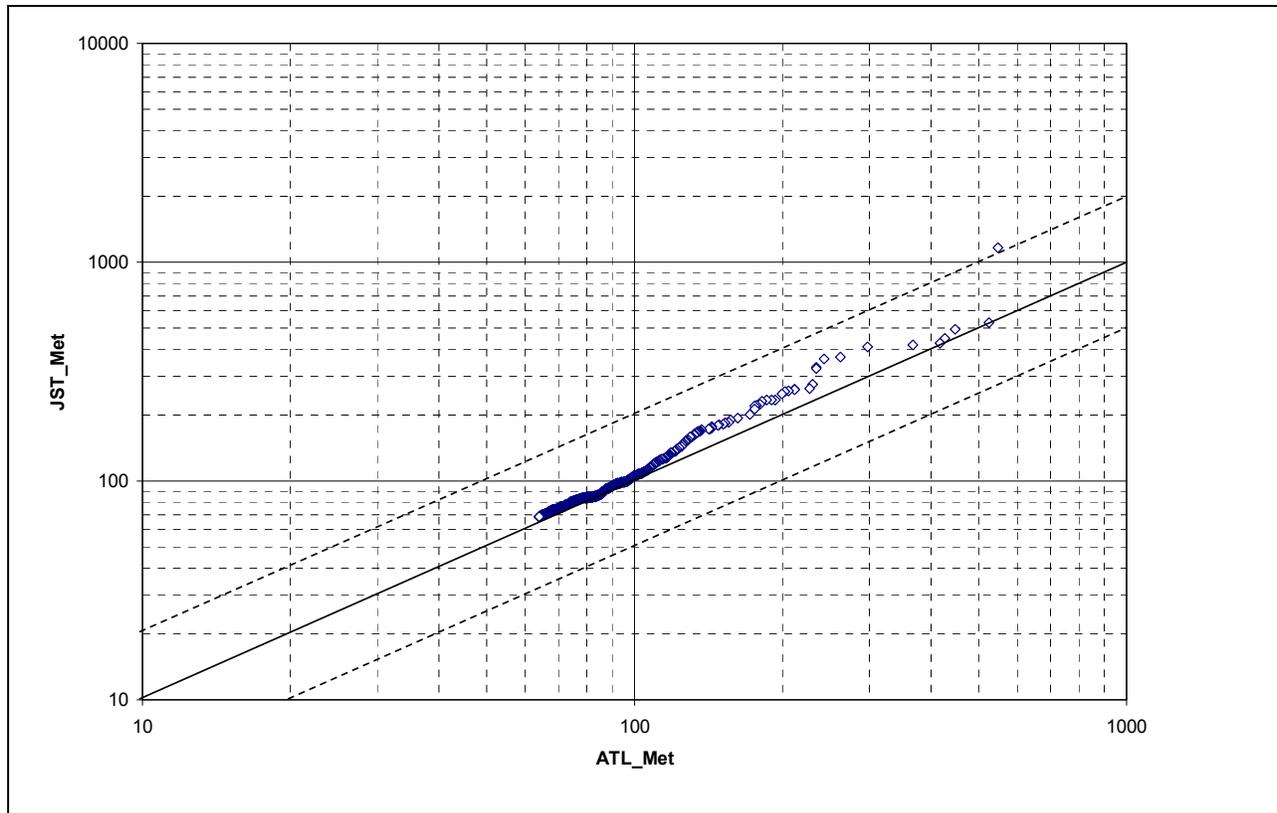
While the AERMOD model algorithms are not considered to be a significant source of uncertainty for this assessment, the representativeness of modeled concentrations for any application are strongly dependent on the quality and representativeness of the model inputs. The main categories of model inputs that may contribute to bias and uncertainty are emission estimates and meteorological data. Use of the OLM and PVMRM chemistry algorithms also introduces the potential uncertainty associated with the dependence on ambient O<sub>3</sub> concentration inputs since both algorithms treat the reaction of NO with O<sub>3</sub> to produce NO<sub>2</sub> as the primary mechanism for conversion of NO<sub>x</sub> emissions. These issues are addressed in the following sections.

#### ***8.12.1.2 Meteorological Inputs***

Details regarding the representativeness of the meteorological data inputs for AERMOD are addressed separately in Attachment 1 to Appendix B-4 of the REA. One of the main issues associated with the representativeness for this application is the sensitivity of the AERMOD model to the surface roughness of the meteorological tower site used to process the meteorological data for use in AERMOD relative to the surface roughness across the full domain of sources. This issue has been shown to be more significant for low-level sources, including mobile sources, due to the importance of mechanical shear-stress induced turbulence on dispersion for such sources. In particular, concerns were raised in preliminary modeling due the typically low surface roughness associated with the meteorological tower located at ATL airport compared with the much higher roughness environment of most low-level emission sources. Based on this concern, alternative meteorological data from the Jefferson Street (JST) Southeast Aerosol Research and Characterization study (SEARCH) site were determined to be more representative of the majority of NO<sub>x</sub> emissions across the modeling domain, as discussed in Attachment 1 to Appendix B-4. The meteorological data obtained for the JST site were used to model all emissions with the exception of the Atlanta airport. Meteorological data for ATL were used to model the airport emissions.

To assess the uncertainty associated with the sensitivity of AERMOD to surface roughness effects, a comparison was made between modeled NO<sub>2</sub> concentrations from mobile

source emissions based on the JST meteorological data versus the ATL meteorological data. Surface roughness lengths were generally about an order of magnitude higher at the JST site relative to the ATL site. Results of this comparison are presented in Figure 8-28, which shows a plot of high ranked 1-hour concentrations, unpaired in time and space, calculated at four Atlanta NO<sub>2</sub> monitoring locations for 2002. This figure shows relatively good agreement in modeled concentrations based on the two sets of meteorological inputs, at least for the peak of the concentration distribution at these four receptor locations. This suggests that the sensitivity of AERMOD model results to variations in surface roughness may be less significant than commonly believed, provided that meteorological data inputs are processed with surface characteristics appropriate for the meteorological site. The overall peak concentration should about a factor of 2 bias between the two sets of meteorological inputs, with the concentrations based on the higher roughness JST data showing higher impacts. While these results are encouraging in relation to the assessment of uncertainty for the REA, they may not be indicative of the degree of sensitivity to surface roughness for individual sources in other modeling contexts.



**Figure 8-28. Comparison of high ranked AERMOD 1-hour NO<sub>2</sub> concentrations (µg/m<sup>3</sup>) from mobile sources across four NO<sub>2</sub> monitoring locations based on JST vs. ATL meteorological inputs for 2002.**

### ***8.12.1.3 Mobile Source Characterization***

Given the predominance of mobile source emissions in this assessment, source characterization of non-stationary mobile sources is another important category to consider for the uncertainty analysis. Determining the most appropriate source characteristics for modeling emissions from mobile sources presents several technical challenges and involves a number of uncertainties. Unlike typical stationary emission sources simulated by AERMOD, for which source characteristics such as release height and effluent parameters can be clearly defined and measured, emissions from mobile sources represent an aggregate of emissions from non-stationary sources of various sizes, shapes, and speeds. Since mobile source emissions (other than aircraft) are emitted near the ground, the plumes can be significantly influenced by the turbulent wake associated with the emitting vehicle, as well as turbulence generated by nearby vehicles and other roughness elements such as sound barriers, median barriers, trees, buildings, etc. Some of these effects may vary depending upon the orientation of the roadway to the wind direction since the vehicle wake effect is most pronounced for wind directions parallel to the

road, where the wakes of multiple vehicles can combine to enhance dispersion of the roadway emissions.

Representative source characteristics for mobile emissions may also depend on the pollutant of interest, and whether the pollutant is primarily associated with direct vehicle exhaust, as in the case of NO<sub>2</sub>, or includes mechanical sources, such as re-entrained road dust, tire wear and brake wear for PM. Emissions associated with vehicle exhaust may experience some buoyancy due to the exhaust temperature exceeding the ambient temperature. Such buoyancy effects might be negligible for vehicles moving at highway speeds, where mechanically-induced turbulence would likely dominate the initial plume characteristics, but could be a significant factor for slow moving vehicles on a cold day during rush hour. In addition to the influence of roughness elements, characteristics of the roadway itself may be a factor. For example, thermally-induced turbulence generated by direct sunlight on dark asphalt could enhance the initial plume spread compared to the same conditions for a more reflective concrete road surface. The best approach for determining source characteristics for mobile emissions may also depend on the scope and nature of the application. Characterizing mobile sources for a large-scale urban study, such as the Atlanta NO<sub>2</sub> modeling, may necessitate a different approach than characterizing mobile sources for a particular highway project within a more localized modeling domain. A detailed treatment accounting for influences of specific local features may be possible for the latter. However, for larger scale applications such as this, a simplified approach with the goal of characterizing emissions based on a reasonable estimate of the aggregate effect of these various factors is necessitated by practical limitations.

The factor of 1.7 times the vehicle height used to account for vehicle-induced turbulence is cited in Gilles et al. (2005) based on some field measurements for an unpaved road. The factor of 1.7 is somewhat less than the typical formula for the turbulent wake downwind of a building, which is 2.5 times the building height. This difference seems reasonable based on the more aerodynamic shape of vehicles as compared to buildings. While some differences may be expected between paved and unpaved roads, these differences are probably minor compared to other uncertainties.

However, the value used to estimate vehicle-induced turbulence could be conservative in terms of modeled concentrations since it may not account for the other influences mentioned above, especially the influence of roadway orientation relative to the wind direction. These

influences may include considerable variability both temporally and spatially, and this variability is difficult to quantify within the current model formulations. However, given the size of the domain, the number of sources included in the modeled inventory, and the length of period modeled, it is likely that some of the uncertainties associated with source characterization will tend to cancel out in the cumulative impact assessment. This expectation is supported for off-road exposures by the generally good agreement in model-to-monitor comparisons presented in Chapter 8.4.8, but is more difficult to assess in relation to on-road exposures.

The matching of TDM links, which are straight segments by their nature, to roads, which may be curvilinear, was done as objectively as possible. It is possible that once links were segmented to avoid aspect ratio issues in the modeling, these segments could deviate from the actual road layout more than they did prior to segmenting. However, the GIS analysis and matching was done before the segmenting and no revision was done after. Thus staff cannot quantify any additional spatial mismatch that came from segmenting. Spatial mismatch from segmenting could affect offroad concentrations relative to a different methodology where predicted concentrations from segments were mated to road locations rather than full TDM links due to both orientation of the areas and location relative to receptors. However, these segments were not considered individual links, but rather partial values to avoid numerical modeling issues and were maintained as consistent with the original data source from which they were drawn (i.e., the TDM), which did not have data at the resolution of the individual segments. On-road concentrations using an alternative methodology, where segments rather than links were mated to actual road locations, would differ due to orientation of the area relative to wind direction. However, as the onroad receptors are centered within the source, any differences are likely to be small.

#### ***8.12.1.4 On-Road Emissions Estimates***

It should be noted that free flow speed represents all traffic, that is, cars and trucks on each link are assigned the same speed. This adds to uncertainty due to the prominence of trucks in the NO<sub>x</sub> inventories, and may contribute positive bias to diurnal patterns in AERMOD predictions.

The truck fractions used in the traffic modeling may also contribute to bias and uncertainty in the exposure results, although adjustments made based on the NEI on-road

emissions may correct for the potential bias. A comparison with National estimates of vehicle miles traveled for year 2002 (FHWA, 2002) and a study conducted in Georgia (FHWA, 2007) indicate that the average truck fractions produced by the Atlanta Regional TDM are potentially higher, possibly contributing to overestimations in NO<sub>x</sub> emissions. Table 8-18 summarizes National estimates of the vehicle mile traveled (VMT) by two highway categories and vehicle types. Depending on roadway category, rural VMT consisted of about 7 to 20% trucks and urban road miles ranged from 4 to 8% trucks. Comparable results can be found in data reported by FHWA (2007) for a roadway in Atlanta, where urban traffic counts were estimated to be comprised of 3 to 8% trucks and rural traffic counts ranged from 10 to 20% trucks (Table 8-19).

The rural roads were estimated to have from 15 to 33% of VMT as trucks based on the Atlanta TDM modeling, while urban roads ranged from 12 to 20% (Table 8-2). The discrepancy is likely due to an inconsistency between the definitions of trucks between the present and FHWA documents. The truck fraction used in the exposure assessment is based directly on the total truck volume included in the TDM, defined as:

$$\text{Total Trucks} = \text{Heavy (class 8-13)} + \text{Medium (class 4-7)} + \text{Commercial (any vehicle used for commercial purposes)}$$

By definition this truck fraction contains a larger fraction of vehicles than those from the DEIS. To maintain internal consistency in our approach, we used the Atlanta-provided MOBILE6 input files and fleet characterizations and computed composite emissions for light and heavy duty vehicles, where the heavy duty fraction is a VMT-weighted composite of all medium and heavy duty vehicles from MOBILE classes 2B to class 8B. It is possible that bias could result from the addition of commercial vehicles in the TDM, but this was not resolvable with the data provided by ARC. However, even with a mismatched HDV fraction and emission factor, for example by coupling MDV+HDV VMT to an HDV8B emission factor, this would have been corrected by the scaling of the emissions down to the 2002 NEI on-road emissions to account for differences between 2005 and 2001-03 activity (see Table 8-6). In addition, the reported truck fractions in Table 8-18 are likely associated with peak counts for light duty vehicles during work commutes. Therefore these may be biased low, even when comparing with morning and afternoon HDV fractions in Table 8-2 which are based on broader time periods.

**Table 8-18. National vehicle miles traveled by roadway category and vehicle type.**

Area	Roadway	Millions of Vehicle-Miles Traveled				% Trucks + Buses
		Buses	Single Unit Trucks	Combination Trucks	All Vehicles	
Rural	Interstate	941	8,745	45,633	279,962	20
	Arterial	1,104	14,606	27,818	433,805	10
	Other	1,901	14,963	14,090	414,393	7
Urban <sup>1</sup>	Interstate	802	9,106	23,887	408,618	8
	Other	2,101	28,467	27,215	1,318,978	4

**Notes:**

<sup>1</sup>Urban consists of travel on all roads and streets in urban places with 5,000 or greater population.

Data from table vm1.xls of FHWA (2002).

**Table 8-19. Observed peak hour truck percentages on Interstate 75 (I-75) using 2002 traffic count data.**

Roadway <sup>1</sup>	Peak Hour <sup>2</sup>	Direction	FHWA Vehicle Classes <sup>3</sup>		Total Trucks <sup>4</sup>
			6-7 (MDT)	8-13 (HDT)	
I-75 N of I-285	AM	Northbound	1	7	8
		Southbound	0	3	3
	PM	Northbound	1	5	6
		Southbound	1	6	6
I-75 N of Wade Green Road	AM	Northbound	1	19	20
		Southbound	1	11	12
	PM	Northbound	1	10	10
		Southbound	1	14	14

**Notes:**

<sup>1</sup> I-75 N of I-285 is just east of Fulton county, likely represents an urban area. The roadway I-75 N of Wade Green Road in northern Cobb county likely represents a rural area.

<sup>2</sup> Peak hours for AM were between 7:30-9:15, PM peak hours were 4:45-5:14.

<sup>3</sup> MDT and HDT are medium and heavy duty trucks, respectively.

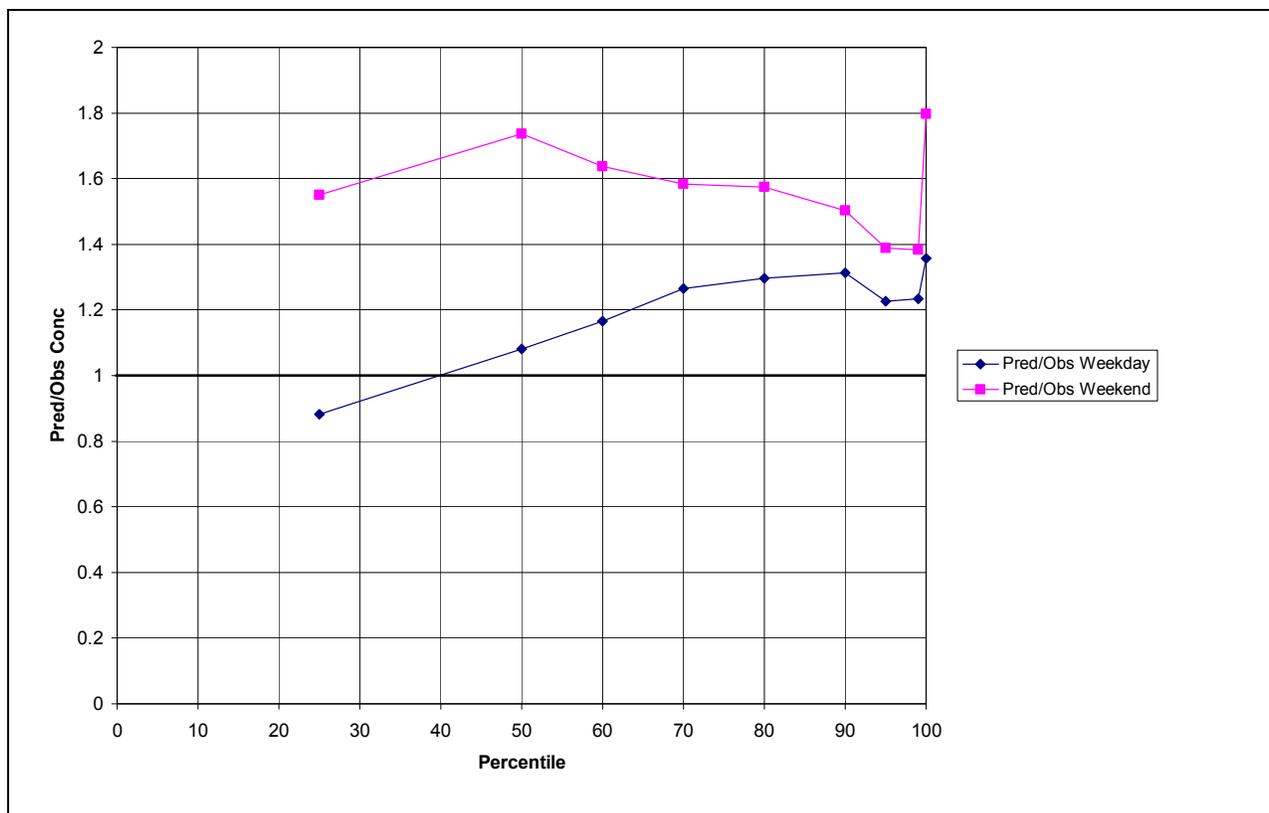
<sup>4</sup> Totals were summed before rounding, therefore MDT +HDT may not always sum to total trucks

Data are from Table 2-4 of FHWA (2007).

Another important source of uncertainty is the diurnal pattern of on-road vehicle activity and emissions. Vehicle activity data was provided by the Atlanta Regional Council (ARC) showing average daily traffic for 4 daily time periods on each roadway link (see section 8.4.3.1). However, the air dispersion model requires hourly allocations of the data. Assigning equal vehicle activity levels to each hour in an ARC-specified time period would have created an unrealistic “step function” time series. In order to create a more realistic diurnal pattern while minimizing alteration of the provided data, a 5-hour running averages of vehicle activity were used to smooth the “step function”. There is uncertainty about how well the smoothed diurnal

pattern represents the unknown actual hourly diurnal pattern. This uncertainty is important, because typical meteorological conditions at various times of day have differential effects on the relationship between emissions and concentrations. For example, at dawn and dusk typical low mixing heights and low wind speeds enhance the concentration impacts of emissions, so that any emission overestimates at those times are likely to result in disproportionate concentration overestimates. The model-to-monitor comparisons in Figure 8-7 show disproportionate overestimates of concentrations during those hours of the day, suggesting that corresponding vehicle activity levels may be overestimated.

Another temporal component of the emission profile for mobile sources worth noting is the variability by day-of-week. The temporal profile of mobile source emissions used for the REA accounts for variability by season and hour-of-day, but does not distinguish between weekday and weekend emission profiles. An analysis of model-to-monitor comparisons for weekday vs. weekend periods was conducted to assess this potential source of uncertainty. Figure 8-29 compares the average ratios of predicted to observed NO<sub>2</sub> concentrations at selected percentiles across the concentration distribution based on the four NO<sub>2</sub> monitors (including the SEARCH JST monitor). While this analysis showed that model performance was significantly better for weekdays than weekends, the affect of this simplification on the overall concentration distributions was not significant. The absence of the morning rush hour peak in NO<sub>2</sub> concentrations evident in the ambient concentrations for the weekend comparison suggests that this factor may have contributed somewhat to the conservative bias in modeled concentrations for the morning rush hour period, but was not the major factor.



**Figure 8-29. Comparison of the average ratios of predicted/observed concentrations of NO<sub>2</sub> across four ambient monitors based on weekday vs. weekend only.**

Another potential source of uncertainty in relation to on-road emission estimates is the assumption of 0.1 as the “in-stack” ratio of NO<sub>2</sub> to NO<sub>x</sub> emissions. While this ratio is considered to be generally representative of many sources of NO<sub>x</sub> emissions and is considered a default value for applications of the OLM option, modeled concentrations of NO<sub>2</sub> will be sensitive to this user-specified value, especially for modeled impacts close to low-level sources. As a result, the uncertainty associated with this issue is likely to be greater for on-road concentration estimates than for off-road locations. However, the overall uncertainty associated with this modeling input is expected to be low.

#### ***8.12.1.5 O<sub>3</sub> Monitoring Data for OLM and PVMRM Options***

Monitored hourly O<sub>3</sub> concentrations were used as input to the OLM and PVMRM atmospheric chemistry modules in AERMOD for conversion of NO to NO<sub>2</sub>. Since AERMOD is currently limited to the use of a single hourly O<sub>3</sub> concentration for all sources within the modeled inventory, this may introduce some uncertainty in the modeled concentrations if there is significant spatial variability of O<sub>3</sub> concentrations across the domain. One suggested source of

this uncertainty and potential bias is the influence of an O<sub>3</sub> deficit over major roadways due to the high concentration of NO<sub>x</sub> emissions depleting O<sub>3</sub>, especially during rush hour. Lacking more complete spatial coverage of O<sub>3</sub> concentrations, it is difficult to quantify this potential cause of bias and uncertainty. If such spatial variability of O<sub>3</sub> concentrations were present, it would likely contribute to more conservative modeled concentrations during periods of higher than average NO<sub>x</sub> emissions and lower than average O<sub>3</sub> concentrations, which would limit conversion of NO to NO<sub>2</sub>. This may be one factor contributing to the apparent conservative bias that appears at all NO<sub>2</sub> monitors during the morning rush hour period, in addition to the uncertainties regarding temporal profiles of mobile source emissions. The simple linear treatment of NO<sub>x</sub> chemistry in the AERMOD model options does not explicitly account for the O<sub>3</sub> depleting effect of the NO-to-NO<sub>2</sub> conversion.

Another issue that has been raised as a potential source of uncertainty in relation to the NO<sub>x</sub> chemistry for this application is that the AERMOD modeling was conducted separately for mobile sources, point sources, and the ATL airport source. The OLM option was used for the mobile source and airport source emissions, while the PVMRM option was used for the point sources. As a result, the ozone-limiting effects simulated by these chemistry options may have been underestimated and may lead to a conservative bias in modeled concentrations. While this may have been an issue with preliminary modeling that separated the on-road and off-road components of mobile source emissions into separate AERMOD runs, the final application of AERMOD combined the on-road and off-road emissions into a single run. Given the relative contributions of these three categories of emissions, the potential impact of O<sub>3</sub> depletion due to point source and airport emissions on total modeled concentrations, which are dominated by the mobile source category, is likely to be insignificant.

#### ***8.12.1.6 Use of Unadjusted AERMOD NO<sub>2</sub> Concentrations***

While the number and range of field study data bases used in AERMOD's evaluation is large relative to evaluation of other models, these field studies are predominantly associated with elevated buoyant releases from power plant stacks. In most of these cases emission rates are known with a high degree of accuracy, often based on continuous emission monitors (CEMs) for operational facilities or based on controlled emissions in the case of tracer releases. Model performance for mobile source emissions across an urban area is not well documented, and

evaluating model performance for such applications is complicated by a number of challenges and uncertainties. These include uncertainties regarding the temporal and spatial distribution of emissions, physical characteristics of the emission sources (such as release height and initial dilution parameters), and the influence of spatial variability of surface characteristics on dispersion of low-level plumes, among other factors. In addition, application of the AERMOD model to simulate NO<sub>x</sub> emissions across an urban domain in support of an exposure assessment imposes different requirements than a more typical application of AERMOD to support a specific regulatory permit application. Regulatory applications of AERMOD are generally motivated by a need to estimate the peak concentrations across a domain of interest without regard to any specific spatial or temporal pattern of impacts. In contrast, the use of AERMOD to support an exposure assessment implies that modeled concentrations will be coupled with population and other information in a way that implies some spatial and temporal pairing of impacts, and also places more emphasis on the significance of the full concentration distribution predicted by the model.

Despite these additional concerns and caveats regarding the use of AERMOD in this REA, the evaluation of modeled air quality presented in Chapter 8.4.8 shows overall good agreement between AERMOD modeled NO<sub>2</sub> concentrations and available ambient monitored NO<sub>2</sub> concentrations. The model evaluation results are consistent across the available ambient monitors and across all seasons. Bias in the predicted concentration distribution at each of the monitor locations is generally within the range expected of refined dispersion models across the cumulative concentration distribution. While some systematic positive bias is evident in the diurnal profiles associated with morning rush hour peak in mobile source emissions, the degree of bias in those cases is within the factor 2 commonly used to indicate relatively unbiased model performance. In considering that this upward bias occurs mainly in the early morning hours, it is possible that there may not be a large proportion of the simulated population exposed at these times of the day. Therefore the upward bias may not have a large effect on the exposure results presented. The actual affect on the exposure results from this bias remains uncertain, because the time-of-day simulated individuals were exposed was not generated by APEX.

Given the uncertainties associated with determining emission profiles and source characteristics for an urban-wide exposure assessment, previous assessments have often included adjustments to modeled concentrations based on available ambient monitored concentrations as a

means of reducing overall uncertainty in the analysis. Two main factors have argued against such an approach for this REA. First, advances in the science of dispersion modeling, including the promulgation of AERMOD model and more refined techniques for treating the temporal and spatial variation of emissions, provide a basis for more confidence in the representativeness of modeled concentrations for the exposure assessment. Second, the limited number of available ambient NO<sub>2</sub> monitors, the predominance of mobile source emissions of NO<sub>x</sub> within the inventory, and the significant horizontal gradients of concentration associated with low-level emissions from major roadways raise significant concerns regarding the spatial representativeness of the ambient NO<sub>2</sub> concentrations for purposes of the exposure assessment. Given that the air quality concentration estimates are estimated to be conservative, and that the values at the upper tails of the hourly distribution are not unusual in comparison with the other portions of the concentration distribution, it was determined that adjustment of the modeled air quality based on the three monitors was not necessary. Any effort to adjust concentration estimates based on monitored values would present a range of options and issues regarding how the modeled concentrations would be adjusted both temporally and spatially in relation to the observations. Based on these considerations, such an approach could actually increase the uncertainty of the REA in ways that are difficult to characterize or justify.

### **8.12.2 Exposure Modeling Uncertainties**

#### ***8.12.2.1 Population Data Base***

The population and commuting data are drawn from U.S. Census data from the year 2000. This is a high quality data source for nationwide population data in the U.S. however, the data do have limitations. The Census used random sampling techniques instead of attempting to reach all households in the U.S., as it has in the past. While the sampling techniques are well established and trusted, they may introduce uncertainty to exposure results. The Census has a quality section (<http://www.census.gov/quality/>) that discusses these and other issues with Census data. It is likely the bias within this data would not affect the results in any particular direction, and given the use of the sampled demographics to represent the simulated population, it is expected that the uncertainty in the exposure results from this source is low.

#### ***8.12.2.2 Commuting Data Base***

Commuting pattern data were derived from the 2000 U.S. Census. The commuting data address only home-to-work travel. A few simplifying assumptions needed to be made to allow for practical use of this data base to reflect a simulated individual's commute. First, there were a few commuter identifications that necessitated a restriction of their movement from a home block to a work block. This is not to suggest that they never travelled on roads, only that their home and work blocks were the same. This includes the population not employed outside the home, individuals indicated as commuting within their home block, and individuals that commute over 120 km a day. This could lead to either over- or under-estimations in exposure if they were in fact to visit a block with either higher or lower NO<sub>2</sub> concentrations. Given that the number of individuals who meet these conditions is likely a small fraction of the total population and that the bias is likely in either direction, the overall uncertainty is considered low.

Second, although several of the APEX microenvironments account for time spent in travel, the travel is assumed to always occur in basically a composite of the home and work block. No other provision is made for the possibility of passing through other census blocks during travel. This could also contribute to bias in either direction, dependent on the number of blocks the simulated individual would actually traverse and the spatial variability of the concentration across different blocks. This could potentially affect a large portion of the population, since we expect that at the block level, many persons would have a commute transect that included more than two blocks, although the actual number of persons and the number of blocks per commute and the spatial variability across blocks has not been quantified. In addition, the commuting route (i.e., which roads individuals are traveling on during the commute) is not accounted for. This may bias the exposure results in either direction, with some individual under-estimated and others over-estimated.

Furthermore, the estimation of block-to-block commuter flows relied on the assumption that the frequency of commuting to a workplace block within a tract is proportional to the amount of commercial and industrial land in the block. This assumption may introduce a bias in overestimating exposures if 1) the blocks with greater commercial/industrial land density also have greater concentrations when compared with lower density commercial/industrial density blocks, and 2) most persons commute to lower commercial/industrial density blocks. It should also be noted that recent surveys, notably the National Household Transportation Survey

(NHTS), have found that most trips taken and most VMT accrued by households are non-work trips, particularly social/recreational and shopping-related travel (Hu and Reuscher, 2004). This constitutes an unquantified source of uncertainty that is not be addressed by the Census commuter dataset.

#### ***8.12.2.2 Human Time-Location-Activity Pattern Data***

The CHAD time-location activity diaries used are the most comprehensive source of such data and realistically represent where individuals are located and what they are doing. The diaries are sequential records of each persons activities performed and microenvironments visited. There are however, uncertainties the exposure results as a result of the CHAD diaries used for simulating individuals may introduce uncertainty to the exposure results. First, much of the data used to generate the daily diaries were collected in surveys conducted over 20 years ago. While the trends in people's daily activities may not have changed much over the years, it is certainly possible that some differences do exist. For example, it is estimated that between 1983 and 2001, the average miles traveled by people in the U.S. increased by 55%, corresponding to a 2.4% annual increase in miles traveled per person (Hu and Reuscher, 2004). Therefore, it is possible that the overall commute times in the CHAD diaries used to estimate in-vehicle exposures are biased low, resulting in under-estimation of exposures.

Second, the CHAD data are taken from numerous surveys that were performed for different purposes. Some of these surveys collected only a single diary-day while others went on for several days. Some of the studies were designed to not be representative of the U.S. population, although a large portion of the data is from National surveys. In addition, study collection periods occur at different times of the year, possibly resulting in seasonal differences. This could add uncertainty to the results if there are characteristics of the survey population that are distinct from the simulated population.

The CHAD diaries that are selected from APEX to represent the Atlanta population are not all from Atlanta, the state of Georgia, or from the Southeast, albeit some of the diaries may be. As stated above, most of the diaries are from National surveys, therefore there are diaries from locations other than Atlanta that are used to simulate the Atlanta population. A few of the limitations associated with the use of diaries from different locations or seasons are corrected by the approaches used in the exposure modeling. For example, diaries used are weighted by

population demographics (i.e., age and gender) for a particular location and temperature is used as a classification variable to account for its affect on human activities.

A sensitivity analysis was performed to evaluate the affect of using different CHAD studies has on APEX results for the recent O<sub>3</sub> NAAQS review (see Langstaff (2007) and EPA (2007g)). Briefly, O<sub>3</sub> exposure results were generated using APEX with all of the CHAD diaries and compared with results generated from running APEX using only the CHAD diaries from the National Human Activity Pattern Study (NHAPS), a nationally representative study in CHAD. There was agreement between the APEX exposure results for the 12 metropolitan areas evaluated (one of which was Atlanta), whether all of CHAD or only the NHAPS component of CHAD is used. The absolute difference in percent of persons above a particular concentration level ranged from -1% to about 4%, indicating that the exposure model results are not being overly influenced by any single study in CHAD. It is likely that similar results would be obtained here for NO<sub>2</sub> exposures, although it remains uncertain due to different averaging times (1-hour vs. 8-hour average).

This is not to suggest that the uncertainty is low in using the CHAD data to represent the Atlanta area, but that similar results would be obtained in using the diaries available, so long as the population was appropriately stratified and certain characteristics influencing exposure were considered. One particular influential factor that is not modeled by APEX is the commute time/distances for the Atlanta population. The Atlanta population is spread over a larger area than most other locations and as a result, individual spend more time driving (Table 8-16). Not taking this added drive time into account when using the CHAD diaries could lead to underestimation of exposures for the Atlanta population. Given the difference in Atlanta DVMT in comparison with other locations, it is possible that this underestimation is large. However, in considering this lack of accounting for the greater Atlanta commute times that exist and that an important driver for exposures above selected levels was from the in-vehicle microenvironment, the Atlanta exposure results may to some degree be representative of other locations in the U.S. with more nationally representative commute times.

### ***8.12.2.3 Longitudinal Profile***

APEX creates seasonal or annual sequences of daily activities for a simulated individual by sampling human activity data from more than one subject. Each simulated person essentially

becomes a composite of several actual people in the underlying activity data. Certain aspects of the personal profiles are held constant, though in reality they change as an individual ages. This is only important for simulations with long timeframes, particularly when simulating young children (e.g., over a year or more).

The cluster algorithm used in constructing longitudinal profiles was evaluated against a sequence of available multiday diaries sets collected as part of the Harvard Southern California Chronic Ozone Exposure Study (Xue et al. 2005, Geyh et al. 2000). Briefly, the activity pattern records were characterized according to time spent in each of 5 aggregate microenvironments: indoors-home, indoors-school, indoors-other, outdoors, and in-transit. The predicted value for each stratum was compared to the value for the corresponding stratum in the actual diary data using a mean normalized bias statistic. See Appendix B, Attachment 2 and 3 for details. The evaluation indicated the cluster algorithm can replicate the observed sequential diary data, with some exceptions. The predicted time-in-microenvironment averages matched well with the observed values. For combinations of microenvironment/age/gender/season, the normalized bias ranges from -35% to +41%. Sixty percent of the predicted averages have bias between -9% and +9%, and the mean bias across any microenvironment ranges from -9% to +4%. Although, on occasion there were large differences in replicating variance across persons and within-person variance subsets, about two-thirds of the predictions for each case were within 30% of the observed time spent in each microenvironment.

The longitudinal approach used in the exposure assessment was an intermediate between random selection of diaries (a new diary used for every day for each person in the year) and perfect correlation (same diary used for every day for each person in the year). The cluster algorithm used here was previously compared with two other algorithms, one that used random sampling and the other employing both diversity (*D*) and autocorrelation (*A*) statistics (see EPA, 2007g for details on this algorithm). The number of persons with at least one or more exposures to a given O<sub>3</sub> concentration was about 30% less when using the cluster algorithm than when using random sampling, while the number of multiple exposures for those persons exposed was greater using the cluster algorithm (by about 50%). The algorithm employing the *D* and *A* statistics exhibited similar patterns, although were lower in magnitude when compared with random sampling (about 5% fewer persons with one or more exposures, about 15% greater multiple exposures). These exposure results using the cluster algorithm in APEX appeared to be

the result of a greater correlation of diaries selected in comparison with the other two algorithms. This outcome conforms to an expectation of correlation between the daily activities of individuals. While the evaluation was performed using 8-hour O<sub>3</sub> as the exposure output it is expected that similar results would be obtained for 1-hour NO<sub>2</sub> exposures. That is, the characteristics of the diaries that contribute greatly to any pollutant exposure above a given threshold (e.g., time spent outdoors, vehicle driving time) are likely a strong component in developing each longitudinal profile. Given these results and that the REA is not necessarily focused on health effects resulting from multi-day exposures, the particular longitudinal approach used likely contributes minimally to uncertainty. See Appendix B, Attachments 2 and 3 for further details in the cluster algorithm and the evaluations performed.

#### ***8.12.2.4 Meteorological Data***

Meteorological data are taken directly from monitoring stations in the assessment areas. It is assumed that most of the data used are error free and have undergone required quality assurance review. One strength of these data is that it is relatively easy to see significant errors if they appear in the data. Because general climactic conditions are known for the simulated area, it would have been apparent upon review if there were outliers in the dataset, and at this time none were identified. If there were a bias in the data, it would be expected to be limited in extent and randomly occurring, therefore contributing to both under and over-estimations equally to a marginal degree. To reduce the number of calms and missing winds in the 1-hour MET data, archived one-minute winds for the ASOS station at ATL were used to calculate hourly average wind speed and directions. This approach reduces the number of estimated zero concentrations that would be output by AERMOD if not supplemented by the additional wind data, thus preventing a downward bias in the predicted 1-hour concentrations.

However, there are limitations in the use of these data. APEX only uses one temperature value per day in selecting an appropriate CHAD diary and indoor microenvironment air exchange rate. Because the model does not represent hour-to-hour variations in meteorological conditions throughout the day, there may be uncertainty in some of the exposure estimates for indoor microenvironments (see the next section).

#### ***8.12.2.5 Air Exchange Rates (AER)***

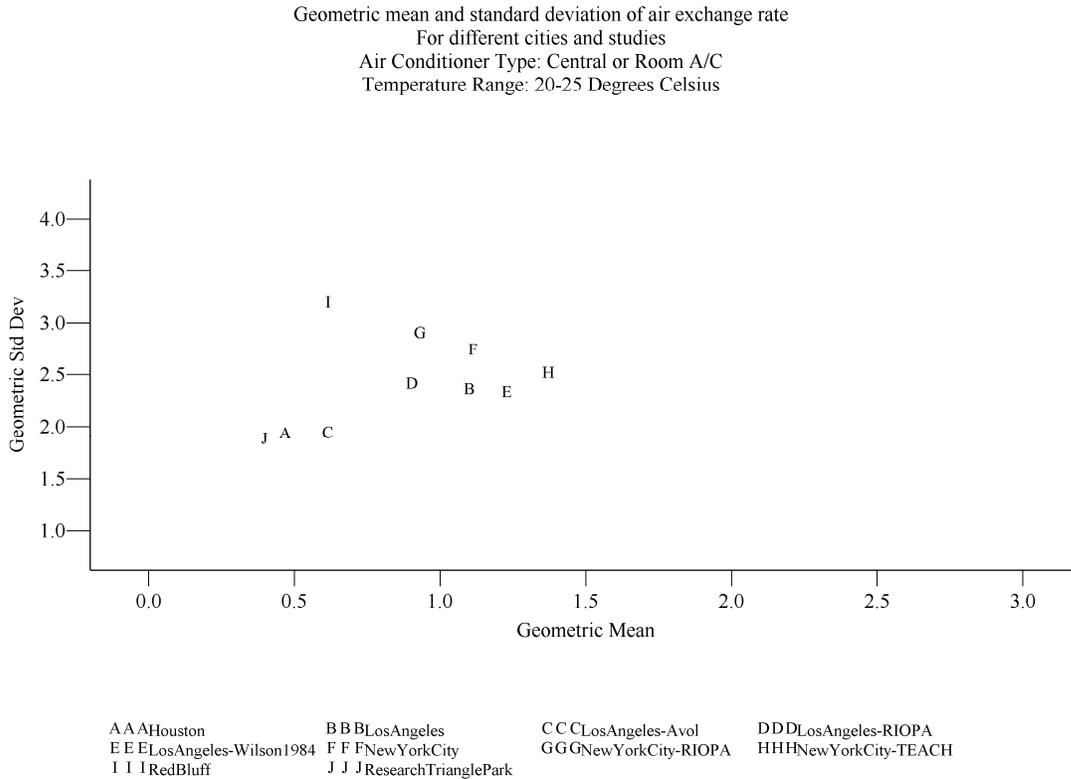
The residential air exchange rate (AER) distributions used to estimate indoor exposures contribute to uncertainty in the exposure results. Three components of the AER analyzed previously by EPA (2007g) include 1) the extrapolation of air exchange rate distributions between-CMSAs, 2) analysis of within-CMSA uncertainty due to sampling variation, and 3) the uncertainty associated with estimating daily AER distributions from AER measurements with different averaging times. The results of those previous investigations are briefly summarized here. See Appendix B, Attachments 8 and 9 for details.

#### ***Extrapolation of AER among locations***

Air exchange rate (AER) distributions were assigned in the APEX model, as detailed in the indoors-residential microenvironment. Since specific AER data for Atlanta were not available, data from another location were used to represent AERs in Atlanta based on having potentially similar influential characteristics. Such factors include age composition of housing stock, construction methods used, and other meteorological variables not explicitly treated in the analysis, such as humidity and wind speed patterns. AER data from measurements in Research Triangle Park, NC (RTP) were selected to represent the distribution of AERs in Atlanta (see Appendix B, Attachment 5).

In order to assess the uncertainty associated with this extrapolation, between-location uncertainty was evaluated by examining the variation of the geometric means and standard deviations across several cities and studies. The evaluation showed a relatively wide variation across different cities in their AER geometric means and standard deviations, stratified by air-conditioning status, and temperature range. For example, Figure 8-30 illustrates the GM and GSD of AERs estimated for several cities in the U.S. where A/C was present and within the temperature range of 20-25 °C. The wide range in GM and GSD pairs implies that the modeling results may be very different if the matching of modeled location to study location was changed. For example, the NO<sub>2</sub> exposure estimates may be sensitive to use of an alternative distribution, say those in New York City, compared with results generated using the RTP AER distributions. It is likely though that the true distribution is more similar to the selected distribution from RTP than New York City or some other location given the population of available AER data. It is unclear as to the direction of bias given the limited number of data available for comparison. It

should be noted that Houston, the only other “southern” location, generally coincided with the RTP AERs distribution in the 20-25 °C and other temperature ranges for homes with A/C (see Appendix B, Attachment 8).



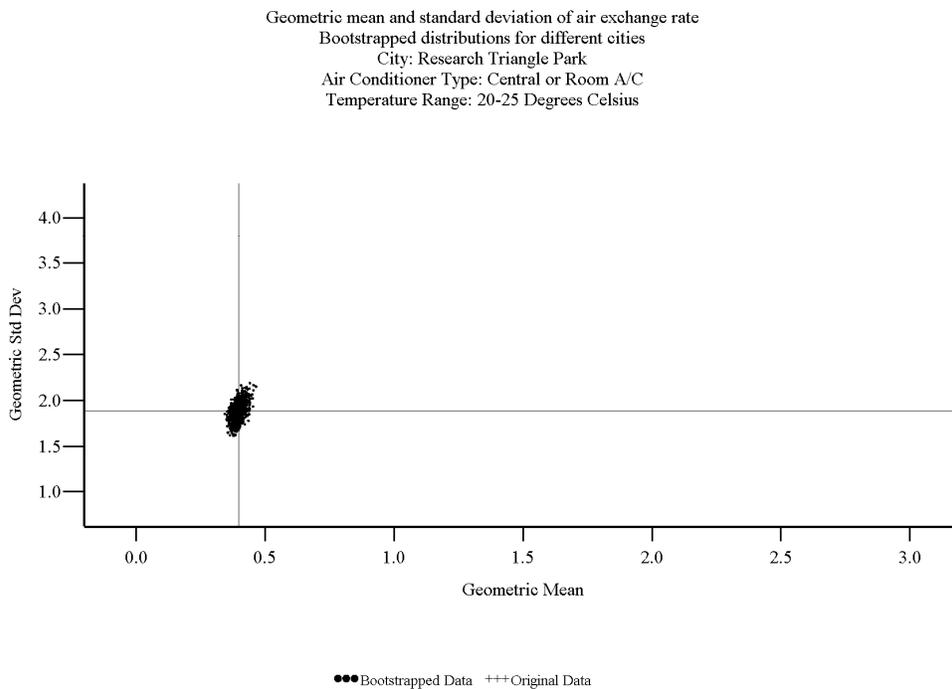
**Figure 8-30. Example comparison of estimated geometric mean and geometric standard deviations of AER (h-1) for homes with air conditioning in several cities.**

***Within location uncertainty***

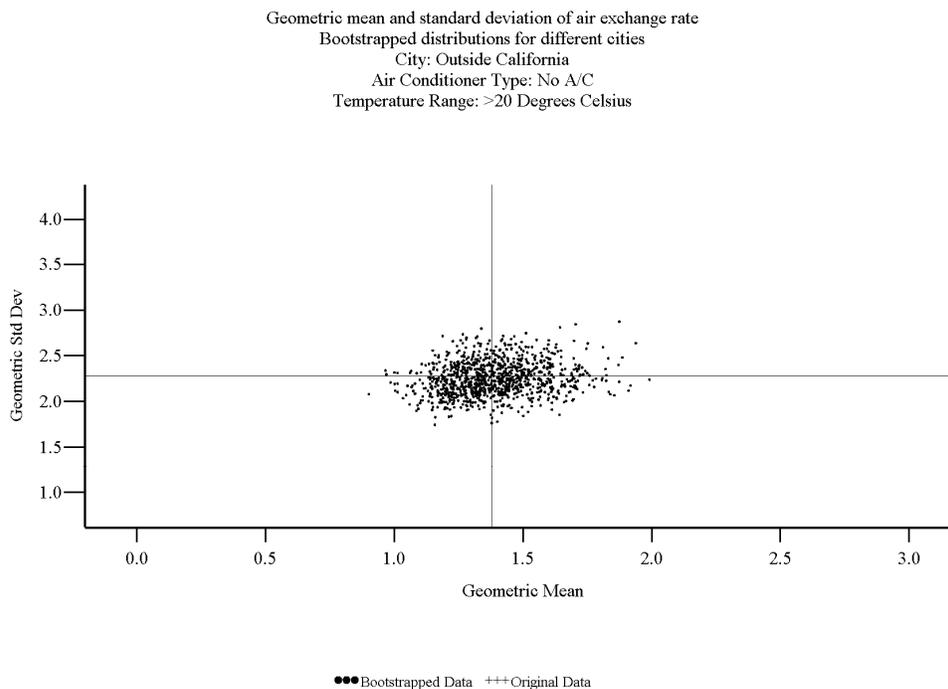
There is also variation in AERs within studies for the same location (e.g., Research Triangle Park, NC), but this is much smaller than the observed variation across different CMSAs. This finding tends to support the approach of combining different studies for a CMSA, where data were available. The within-city uncertainty was assessed by using a bootstrap distribution to estimate the effects of sampling variation on the fitted geometric means and standard deviations for the RTP data used to represent the Atlanta AERs. These bootstrap distributions assess the uncertainty due to random sampling variation. They do not address other

uncertainties such as the lack of representativeness of the available study data or the variation in the lengths of the AER monitoring periods. Because only the GM and GSD were used, the bootstrap analyses does not account for uncertainties about the true distributional shape, which may not necessarily be lognormal.

One-thousand bootstrap samples were randomly generated for each AER subset (of size N), producing a set of 1,000 geometric mean (GM) and geometric standard deviation (GSD) pairs. The analysis of the RTP data used to represent Atlanta indicated that the GSD uncertainty for a given AER temperature group tended to have a range within  $\pm 0.25$  fitted GSD ( $\text{hr}^{-1}$ ), with smaller intervals surrounding the GM (i.e, about  $\pm 0.10$  fitted GM ( $\text{hr}^{-1}$ )) (Figure 8-31). Broader ranges were generated from the bootstrap simulation for AER distributions used for Atlanta homes without A/C (Figure 8-32), although both still within  $\pm 0.5$  of the fitted GM and GSD values. See Appendix B, Attachment 8 for further details.



**Figure 8-31. Example of boot strap simulation results used in evaluating random sampling variation of AER ( $\text{h}^{-1}$ ) distributions (RTP data).**



**Figure 8-32. Example of boot strap simulation results used in evaluating random sampling variation of AER (h-1) distributions (outside CA).**

***Variation in AER measurement averaging times***

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

***8.12.2.6 Air Conditioning Prevalence***

Because the selection of an air exchange rate distribution is conditioned on the presence or absence of an air-conditioner, the air conditioning status of the residential microenvironment was simulated randomly using the probability that a residence has an air conditioner, i.e., the residential air conditioner prevalence rate. For this study we used location-specific data for

Atlanta AHS, 2004). EPA (2007g) details the specification of uncertainty estimates in the form of confidence intervals for the air conditioner prevalence rate, and compares these with prevalence rates and confidence intervals developed from the Residential Energy Consumption Survey (RECS) of 2001 for several aggregate geographic subdivisions (e.g., states, multi-state Census divisions and regions) (EIA, 2001).

Briefly, Air conditioning prevalence rates were 97% for Atlanta, with reported standard errors of 1.2% (AHS, 2004). Estimated 95% confidence intervals were also small and span approximately 4.6% (AHS, 2003). The RECS prevalence estimates for Atlanta and confidence intervals compared well with a value of 95.0% and a 95% confidence interval spanning 5.8%. This suggests that there is limited bias in the A/C prevalence estimates used and that the uncertainty in the estimated value is likely low.

A sensitivity analysis was performed to evaluate changes in the estimated exposures when using a lower A/C prevalence. Changing the A/C prevalence from the actual Atlanta value used allows for a greater percentage of homes to use estimated AER distributions for homes without A/C. An A/C prevalence of 0.55 (or 55%) was input to a new simulation using 2002 air quality without indoor sources, based on the lower bound of observed A/C prevalence rates in Table 8-15). Table 8-21 indicates that there is no change in the percent of asthmatics exposed at or above each of the potential health effect benchmark levels, whether considering a single exposure or up to six exposures in a year. There are however, several thousand additional person days, or additional days for persons that are already exposed to daily maximum concentrations at or above the benchmarks when considering the simulation conducted using the lower prevalence rate. Only a few additional persons were exposed to benchmarks  $\geq 200$  ppb that did not have such exposures in the simulation using the higher A/C prevalence rate. These results suggest that the indoor-residential microenvironment contributes much less to exposures above any of the benchmarks when compared with the estimated contribution from on-road and near-road microenvironments. Most individuals (>99%) were already estimated to experience at least one exposure at concentrations at or above 100 to 150 ppb through the roadway related exposures, one of the main reasons why there are no additional persons exposed at these benchmark levels when considering the lower A/C prevalence. Even though there is a large fraction of the population not exposed to benchmark levels  $\geq 200$  ppb (18-41%) using the higher prevalence rate, the outdoor ambient concentrations rarely would exceed these concentrations. Thus only a

few additional persons experience these higher benchmark level concentrations with the lower A/C prevalence.

**Table 8-20. Comparison of exposure results using a 0.55 versus 0.97 A/C prevalence for 2002 air quality without indoor sources.**

Simulation	Benchmark Level (ppb)	Percent of All Asthmatics with Indicated Number of Multiple Daily Maximum 1-hour Exposures At or Above Benchmark Level						Person Days Above Benchmark	Number of Persons with at Least One Exposure
		1	2	3	4	5	6		
no-indoor - 0.97 AC prevalence	100	100%	100%	100%	99%	99%	99%	7393854	212426
	150	99%	96%	93%	88%	83%	78%	2839603	209855
	200	92%	80%	69%	58%	48%	40%	1271622	195766
	250	76%	56%	40%	28%	20%	15%	618725	161863
	300	59%	32%	19%	12%	8%	6%	323273	124531
no indoor- 0.55 AC prevalence	100	100%	100%	100%	99%	99%	99%	7442239	212426
	150	99%	96%	93%	88%	83%	78%	2849885	209855
	200	92%	81%	69%	58%	48%	40%	1274646	195875
	250	76%	56%	40%	28%	20%	15%	620237	161918
	300	59%	33%	20%	12%	8%	6%	323878	124637
Absolute Difference (0.97 ACprev- 0.55 ACprev)	100	0	0	0	0	0	0	-48385	0
	150	0	0	0	0	0	0	-10282	0
	200	0	0	0	0	0	0	-3024	-109
	250	0	0	0	0	0	0	-1512	-54
	300	0	0	0	0	0	0	-605	-106

### 8.12.2.7 Indoor Source Estimation

Other indoor NO<sub>2</sub> emission sources, such as emissions from gas pilot lights, gas heating, unvented gas fire places, gas water heaters, or gas clothes drying were not included in this analysis due to lack of adequate data readily usable for characterization, modeling complexities regarding the assignment of particular sources to the simulated population (e.g, correlations of sources), a limited time to conduct analyses of potential data distributions including the analysis of their uncertainties, and limited resources allocated for inclusion in the review. Exclusion of these sources would bias all indoor concentrations low when these sources are present, however, it is largely uncertain how much it would affect any estimates of the benchmark level exceedances, and the number of persons affected in Atlanta in the absence of source emission and prevalence data.

There may be uncertainty added to the exposure results when considering the form (i.e., uniform) and limits (limited by the bounds of the measurement data) of the distribution used to

represent indoor decay. The measurement data used to develop the distribution were obtained from a single study, conducted in a single home, under limited temperature and humidity conditions (see Table 8-11). The experimental design included four different methods for introducing NO<sub>2</sub> into the home, including direct injection and home appliances. Different homes, with varying construction materials and furnishings may have different NO<sub>2</sub> decay rates, and as a result of the uniform distribution selected, the number of exposures may be either over- or under-estimated depending on the extent of how far the true population of NO<sub>2</sub> values extend outside the range of the uniform distribution used (i.e., 1.02-1.45).

A sensitivity run was performed using an alternative fitted distribution, assuming a lognormal form with GM and GSD of 1.17 and 1.14, respectively, and lower and upper bounds defined by 50% of observed minimum (0.51) and 100% of observed maximum (2.9). The exposure results generated using the lognormal decay distribution were compared with the simulations performed using the uniform distribution for 2002 air quality and without indoor sources (Table 8-21). There was no difference in the percent of asthmatics estimated to experience one through six daily maximum exposures in a year above any of the benchmarks. There were however a few additional person days above each of the benchmark levels except for <300 ppb and 54 additional persons exposed at or above 200 and 250 ppb when using the lognormal distribution. This suggests that the simulated exposures above the selected benchmarks are not sensitive changes in the indoor decay rate NO<sub>2</sub>. Whether the same outcome would occur with additional alternative distributions of different forms and bounds or that the indoor microenvironment is not sensitive to indoor decay based on the algorithm used by APEX remains largely unknown.

**Table 8-21. Comparison of exposure results using a uniform versus lognormal NO<sub>2</sub> indoor decay distribution for 2002 air quality without indoor sources.**

Simulation	Benchmark Level (ppb)	Percent of All Asthmatics with Indicated Number of Multiple Daily Maximum 1-hour Exposures at or Above Benchmark Level						Person Days Above Benchmark	Number of Persons with at Least One Exposure
		1	2	3	4	5	6		
no-indoor uniform decay	100	100%	100%	100%	99%	99%	99%	7393854	212426
	150	99%	96%	93%	88%	83%	78%	2839603	209855
	200	92%	80%	69%	58%	48%	40%	1271622	195766
	250	76%	56%	40%	28%	20%	15%	618725	161863
	300	59%	32%	19%	12%	8%	6%	323273	124531
no indoor-	100	100%	100%	100%	99%	99%	99%	7402926	212426

lognormal decay	150	99%	96%	93%	88%	83%	78%	2842022	209855
	200	92%	80%	69%	58%	48%	40%	1272227	195821
	250	76%	56%	40%	28%	20%	15%	619027	161918
	300	59%	32%	19%	12%	8%	6%	323273	124531
absolute difference (uniform-lognormal)	100	0	0	0	0	0	0	-9072	0
	150	0	0	0	0	0	0	-2419	0
	200	0	0	0	0	0	0	-605	-54
	250	0	0	0	0	0	0	-302	-54
	300	0	0	0	0	0	0	0	0

The data used to estimate the average number of daily food preparation events is older than the time period assessed (1992 versus 2001-2003) and may therefore not reflect current conditions to some degree, possibly leading to either under- or over-estimates of exposure to concentration exceedances. For example, if the population of Atlanta in 2003 that uses gas stoves to prepare food at home does so less frequently than reported the 1992 survey population, then the number of such exposures may be over-estimated. The variability associated with the mean usage of 1.4 that was used in the model is also under-represented in that there are likely some individuals that cook more or less than this value. Furthermore, the estimate is not specific for the Atlanta population. The uncertainty regarding each of these issues and how they may affect the exposure results is largely unknown.

As noted in the microenvironmental description, it was assumed that the probability that a food preparation event included stove use was the same no matter what hour of the day the food preparation event occurred. If such probabilities differ, then the diurnal allocation of cooking events may differ from the actual pattern. To the extent that the gas stove usage patterns may correlate with ambient concentration patterns, the number of exposures to exceedances of threshold concentrations of concern may be under- or over-estimated. For example, if gas stove usage and ambient concentrations are positively correlated (e.g., if cooking tends to occur during evening rush hour) and the diurnal allocation assumed here results in a lower correlation (e.g., if the diurnal allocation understates the probability of gas stove usage at times of high ambient concentrations) then the number of such exposures may be under-estimated. As another example, if the diurnal pattern allocation assumed here understates the probability of gas stove usage at times when simulated subjects are assumed to be at home, then the number of such exposures may be under-estimated.

There is also uncertainty regarding the distribution used to estimate the indoor source concentration contribution. Concentrations were obtained from CARB (2001) from a variety of described cooking conditions (e.g., with or without ventilation, different pans) and foods cooked (e.g., bacon, french fries, broiled fish, lasagna) in a single test home. While an alternative distribution form may be fitted to such data, there already exist large uncertainties regarding the representation of these measured concentrations obtained under the limited experimental conditions to the population of all possible cooking conditions, the foods cooked, and the proper weighting of such cooking events for the simulated population in Atlanta. In the absence of such knowledge, it is likely that a fitted distribution would be biased high. In considering these uncertainties, staff elected to use a uniform distribution, bounded by the upper and lower range of the experimental data. Use of this uniform distribution does exclude concentrations outside the upper value, suggesting that concentrations in excess of the upper bound are an impossibility. This is unlikely and may add to the uncertainty in the estimated exposures when cooking with gas, and implies a bias in underestimating indoor source contribution to indoor concentrations. Although it appears that the study was primarily designed to estimate upper percentile PM concentrations, it is possible that the uniform distribution selected for NO<sub>2</sub> does capture the upper range of concentrations very well due to the presence of study-designed “worst-case” cooking scenarios.

The durations of the CARB (2001) cooking tests ranged from 21 minutes to 3 hours with an average of about 70 minutes. For implementation in APEX it was assumed that each cooking event lasts exactly an hour. That is, the randomly selected net concentration contribution was added to hourly average indoor concentration for the hour it was selected to occur. Because the mass balance algorithm leads to carryover from one hour to the next, some of the indoor cooking impact will influence subsequent hours. However, the affect of the cooking event may be overstated or understated for cooking events longer or shorter than 1 hour.

## 9. CHARACTERIZATION OF HEALTH RISKS USING DATA FROM EPIDEMIOLOGICAL STUDIES

### 9.1 INTRODUCTION

As mentioned above in chapter 6, in response to advice received from the CASAC NO<sub>2</sub> Panel on the 1<sup>st</sup> draft REA, we have conducted a focused quantitative risk assessment in which estimates of respiratory ED visits as a function of ambient levels of NO<sub>2</sub> have been developed for a single urban area (i.e., the Atlanta MSA). In this approach, concentration-response functions derived from NO<sub>2</sub> epidemiological studies are used in conjunction with 1) ambient air quality data representing as is and alternative air quality scenarios and 2) baseline incidence data for respiratory ED visits to estimate the impact of ambient levels of NO<sub>2</sub> on ED visits associated with these air quality scenarios.

The purpose of this chapter is to present the results for the current risk assessment which is an illustrative case study that provides information on the magnitude and potential changes in NO<sub>2</sub>-related public health impacts associated with recent air quality and alternative air quality scenarios simulating attainment of the current and alternative NO<sub>2</sub> standards. We note that chapters 4 and 5 of this document provide additional qualitative assessment of the epidemiological evidence most relevant to characterizing NO<sub>2</sub>-related health effects in the United States; this includes both a discussion of respiratory-related ED visits as well as other health endpoints. We also note that integration of the scientific evidence presented in the ISA (EPA, 2008a) with the air quality, exposure, and risk characterization results presented in chapters 7 through 9 of this document is presented in chapter 10. Chapter 10 also discusses staff's assessment of how this information might be considered in evaluating the adequacy of the current NO<sub>2</sub> NAAQS and the need for potential alternative primary NO<sub>2</sub> standards.

Previous reviews of the NO<sub>2</sub> primary NAAQS, completed in 1985 and 1996, did not include quantitative health risk assessments. Thus, the risk assessment described in this document builds upon the methodology and lessons learned from the risk assessment work conducted for the recently concluded PM and O<sub>3</sub> NAAQS reviews (Abt Associates, 2005; Abt Associates, 2007). Many of the same methodological issues are present in conducting a risk

assessment for each of these criteria air pollutants where epidemiological studies provided the basis for the concentration-response relationships used in the quantitative risk assessment.

The NO<sub>2</sub> health risk assessment described in this chapter estimates the incidence of respiratory-related ED visits associated with short-term exposures to NO<sub>2</sub> under recent (“as is”) air quality levels, upon just meeting the current NO<sub>2</sub> standard of 0.053 ppm annual average, and upon just meeting several potential alternative NO<sub>2</sub> primary NAAQS in the Atlanta MSA.<sup>1</sup> As discussed in more detail in chapter 6 above, staff has elected to evaluate daily maximum 1-h standard levels of 0.05, 0.10, 0.15, and 0.20 ppm using both 98<sup>th</sup> and 99<sup>th</sup> percentile forms and averaged over a three-year period.<sup>2</sup> The risk assessment is intended as a tool that, together with other information on this health endpoint and other health effects evaluated in the final ISA and discussed elsewhere in this document, can aid the Administrator in judging whether the current primary standard protects public health with an adequate margin of safety, or whether revisions to the standard are appropriate.

Section 9.2 describes the general approach used to conduct the risk assessment for ED visits. Sections 9.3, 9.4, and 9.5 discuss in more detail the three types of inputs required to conduct the assessment. Section 9.6 presents a discussion of uncertainties and variability and section 9.7 presents a summary of results from the assessment and key observations.

## 9.2 GENERAL APPROACH

The general approach used for the NO<sub>2</sub>-related ED risk assessment is dictated by the fact that it is based on concentration-response functions which have been estimated in epidemiological studies evaluated in the final ISA. Since these studies estimate concentration-response functions using ambient air quality data from fixed-site, population-oriented monitors, the appropriate application of these functions in a risk assessment similarly requires the use of ambient air quality data at fixed-site, population-oriented monitors. In order to estimate the incidence of respiratory-related ED visits associated with recent air quality conditions in a set of counties attributable to ambient NO<sub>2</sub> exposures, as well as the change in incidence of this health effect in that set of counties corresponding to a given simulated change in NO<sub>2</sub> levels

---

<sup>1</sup> The current NO<sub>2</sub> standard refers to a two-year period and requires that the annual average NO<sub>2</sub> level be less than or equal to 0.053 ppm in each of the two years.

<sup>2</sup> As an example, for the alternative standards using the 98<sup>th</sup> percentile form, the standard is met when the average of the annual 98<sup>th</sup> percentile daily maximum 1-hour concentrations for a 3-year period is at or below the specified standard level.

representing just meeting the current or alternative 1-h daily maximum NO<sub>2</sub> standards, the following three elements are required:

- **Air quality information** including: (1) “as is” air quality data for NO<sub>2</sub> from ambient monitors in the assessment location, and (2) “as is” concentrations adjusted to reflect patterns of air quality estimated to occur under a simulation where the area’s air quality is adjusted to just meet the specified standard. (These air quality inputs are discussed in more detail in section 6.2 of this document).
- **Concentration-response functions** which provide an estimate of the relationship between the health endpoint of interest and ambient NO<sub>2</sub> concentrations.
- **Baseline health effects incidence.** The baseline incidence of the health effect in the assessment location in the target year is the incidence corresponding to “as is” NO<sub>2</sub> levels in that location in that year.

Figure 9-1 provides a broad schematic depicting the role of these components in the NO<sub>2</sub> risk assessment. Each of the key components (i.e., air quality information, estimated concentration-response functions, and baseline incidence) is discussed below, highlighting those points at which judgments have been made.

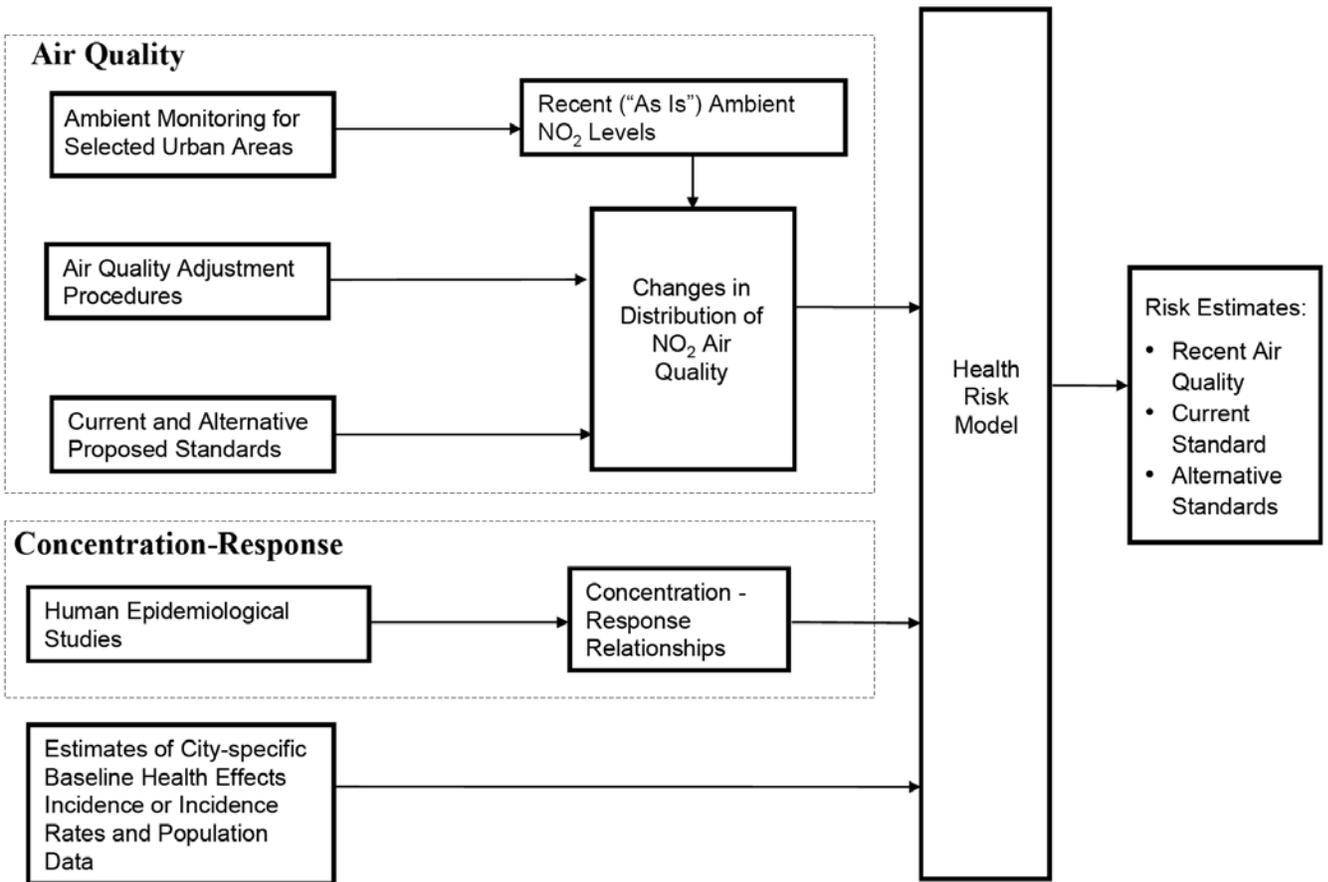
These inputs are combined to estimate health effect incidence changes associated with specified changes in NO<sub>2</sub> levels. Although some epidemiological studies have estimated linear or logistic concentration-response functions, by far the most common form, and the form relevant for the epidemiological study used in the current risk assessment is the exponential (or log-linear) form:

$$y = Be^{\beta x}, \quad (\text{Equation 9-1})$$

where  $x$  is the ambient NO<sub>2</sub> level,  $y$  is the incidence of the health endpoint of interest at NO<sub>2</sub> level  $x$ ,  $\beta$  is the coefficient of ambient NO<sub>2</sub> concentration (describing the extent of change in  $y$  with a unit change in  $x$ ), and  $B$  is the incidence at  $x=0$ , i.e., when there is no ambient NO<sub>2</sub>. The relationship between a specified ambient NO<sub>2</sub> level,  $x_0$ , for example, and the incidence of a given health endpoint associated with that level (denoted as  $y_0$ ) is then

$$y_0 = Be^{\beta x_0}. \quad (\text{Equation 9-2})$$

If we let  $x_0$  denote the baseline (upper) NO<sub>2</sub> level, and  $x_1$  denote the lower NO<sub>2</sub> level, and  $y_0$  and  $y_1$  denote the corresponding incidences of the health effect, we can derive the following



**Figure 9-1. Major components of nitrogen dioxide health risk assessment for emergency department visits.**

relationship between the change in  $x$ ,  $\Delta x = (x_0 - x_1)$ , and the corresponding change in  $y$ ,  $\Delta y$ , from equation (9-1)<sup>3</sup>:

$$\Delta y = (y_0 - y_1) = y_0[1 - e^{-\beta\Delta x}]. \quad (\text{Equation 9-3})$$

Alternatively, the difference in health effects incidence can be calculated indirectly using relative risk. Relative risk (RR) is a measure commonly used by epidemiologists to characterize the comparative health effects associated with a particular air quality comparison. The risk of ED visits for respiratory illness at ambient NO<sub>2</sub> level  $x_0$  relative to the risk of ED visits for respiratory illness at ambient NO<sub>2</sub> level  $x_1$ , for example, may be characterized by the ratio of the two rates: the rate of ED visits for respiratory illness among individuals when the ambient NO<sub>2</sub> level is  $x_0$  and the rate of ED visits for respiratory illness among (otherwise identical) individuals when the ambient NO<sub>2</sub> level is  $x_1$ . This is the RR for ED visits for respiratory illness associated with the difference between the two ambient NO<sub>2</sub> levels,  $x_0$  and  $x_1$ . Given a concentration-response function of the form shown in equation (9-1) and a particular difference in ambient NO<sub>2</sub> levels,  $\Delta x$ , the RR associated with that difference in ambient NO<sub>2</sub>, denoted as  $RR_{\Delta x}$ , is equal to  $e^{\beta\Delta x}$ . The difference in health effects incidence,  $\Delta y$ , corresponding to a given difference in ambient NO<sub>2</sub> levels,  $\Delta x$ , can then be calculated based on this  $RR_{\Delta x}$  as

$$\Delta y = (y_0 - y_1) = y_0[1 - (1/RR_{\Delta x})]. \quad (\text{Equation 9-4})$$

Equations (9-3) and (9-4) are simply alternative ways of expressing the relationship between a given difference in ambient NO<sub>2</sub> levels,  $\Delta x > 0$ , and the corresponding difference in health effects incidence,  $\Delta y$ . These health impact equations are the key equations that combine air quality information, concentration-response function information, and baseline health effects incidence information to estimate health risks related to changes in ambient NO<sub>2</sub> concentrations.

---

<sup>3</sup> If  $\Delta x < 0$  – i.e., if  $\Delta x = (x_1 - x_0)$  – then the relationship between  $\Delta x$  and  $\Delta y$  can be shown to be  $\Delta y = (y_1 - y_0) = y_0[e^{\beta\Delta x} - 1]$ . If  $\Delta x < 0$ ,  $\Delta y$  will similarly be negative. However, the *magnitude* of  $\Delta y$  will be the same whether  $\Delta x > 0$  or  $\Delta x < 0$  – i.e., the absolute value of  $\Delta y$  does not depend on which equation is used.

### 9.3 AIR QUALITY INFORMATION

As illustrated in Figure 9-1, and noted earlier, air quality information required to conduct the NO<sub>2</sub> risk assessment includes (1) recent air quality data for NO<sub>2</sub> from a suitable monitor for the assessment location and (2) air quality adjustment procedures to modify the recent data to simulate air quality data just meeting the current annual and potential alternative 1-h daily maximum standards. The approach used to adjust air quality data to simulate meeting specified standards is discussed above in section 6.2.

In the first part of the risk assessment, we estimate the incidence of the health effect associated with “as is” levels of NO<sub>2</sub> (or equivalently, the change in health effect incidence,  $\Delta y$ , associated with a change in NO<sub>2</sub> concentrations from “as is” levels of NO<sub>2</sub> to 0 ppb). In the second part, we estimate the incidence of the health effect associated with NO<sub>2</sub> concentrations simulated to just meet a specified standard (i.e., the current NO<sub>2</sub> standard of 0.053 ppm annual average as well as each of potential alternative 1-h daily maximum standards).

To estimate the incidence of a health effect associated with “as is” NO<sub>2</sub> levels in a location, we need a time series of hourly “as is” NO<sub>2</sub> concentrations for that location. We have used monitor data from the Georgia Tech monitor (monitor id =131210048), the monitor that was used in Tolbert et al. (2007), the epidemiological study from which we obtained the concentration-response functions (see section 9.4 below). Complete hourly data were available on over 93 percent of the days – 348 days in 2005, 345 days in 2006, and 340 days in 2007. Missing NO<sub>2</sub> concentrations were filled in, as described in section 3.5 of Appendix C.

Because Tolbert et al. (2007) estimated a relationship between daily respiratory-related ED visits and the 3-day moving average (i.e., NO<sub>2</sub> levels on the same day, the previous day, and the day before that) of the daily 1-h maximum NO<sub>2</sub> concentrations, we calculated the 3-day moving average of the daily 1-h maximum NO<sub>2</sub> concentrations at the monitor to provide the air quality input to the risk assessment.

The calculations for the second part of the risk assessment, in which we estimated risks associated with NO<sub>2</sub> levels simulated to just meet the current annual standard and potential alternative 1-h daily maximum standards were done analogously, using the monitor-specific series of adjusted daily maximum hourly concentrations rather than the monitor-specific series of “as is” daily maximum hourly concentrations.

## 9.4 CONCENTRATION-RESPONSE FUNCTIONS

As indicated in Figure 9-1, another key component in the risk assessment model is the set of concentration-response functions which provide estimates of the relationship between the health endpoint of interest and ambient NO<sub>2</sub> concentrations. As discussed above, the health endpoint of interest for this focused quantitative risk assessment is respiratory-related ED visits. As discussed in sections 4.2.2 and 4.5.2 several community epidemiological studies have been conducted in the U.S. that examined the relationship between NO<sub>2</sub> and other air pollutants and increased ED visits either for all respiratory causes or for asthma-related visits. Figure 5-1 in this document summarizes the single pollutant model effect estimates from these studies. As discussed in section 4.5.2, staff has considered several factors in selecting the urban area and epidemiological studies upon which the current risk assessment is based. First, we have judged that studies conducted in the United States are preferable to those conducted outside the United States given the potential for effect estimates to be impacted by factors such as the ambient pollutant mix, the placement of monitors, activity patterns of the population, and characteristics of the healthcare system. Second, we judged that studies of ambient NO<sub>2</sub> are preferable to those of indoor NO<sub>2</sub> given that studies of indoor NO<sub>2</sub> focus on exposures in locations with indoor sources of NO<sub>2</sub>. These indoor sources can result in exposure patterns, NO<sub>2</sub> levels, and co-pollutants that are different from those typically associated with ambient NO<sub>2</sub>. Third, we judged it appropriate to focus on studies of ED visits. When compared to studies of respiratory symptoms, the public health significance of ED visits is less ambiguous for the individuals affected. In addition, baseline incidence data are more readily available for these endpoints. Finally, we judged it appropriate to focus on studies that evaluated NO<sub>2</sub> health effect associations using both single- and multi-pollutant models. Taking these factors into consideration, we have chosen to focus on the studies by Tolbert and colleagues (2007) in Atlanta, Georgia that address ED visits for respiratory causes as a case study to illustrate the magnitude and changes in estimated NO<sub>2</sub>-related risks for this endpoint for various air quality scenarios.

Tolbert et al. (2007) estimated concentration-response functions using both single pollutant models (i.e., where NO<sub>2</sub> was the only pollutant entered into the health effects model) and multi-pollutant models (i.e., where one or two co-pollutants (PM<sub>10</sub>, O<sub>3</sub>, CO) were entered into the health effects model). To the extent that any of the co-pollutants present in the ambient air may have contributed to the health effects attributed to NO<sub>2</sub> in single pollutant models, risks

attributed to NO<sub>2</sub> might be overestimated where concentration-response functions are based on single pollutant models. However, if co-pollutants are highly correlated with NO<sub>2</sub>, their inclusion in an NO<sub>2</sub> health effects model can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, inclusion of multiple pollutants in models often produces unstable and statistically insignificant effect estimates for both NO<sub>2</sub> and the co-pollutants. Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases, we report risk estimates based on both single- and multi-pollutant models in the NO<sub>2</sub> risk assessment.

All of the models in Tolbert et al. (2007) used a 3-day moving average of pollution levels (i.e., the average of 0-, 1-, and 2-day lags), so the issue of which of several different lag structures to select does not arise. The issue of how well a given lag structure captures the actual relationship between the pollutant and the health effect, however, is still relevant. Models in which the pollutant-related incidence on a given day depends only on same-day or previous-day pollutant concentration (or some variant of those, such as a two- or three-day average concentration) necessarily assume that the longer pattern of pollutant levels preceding the pollutant concentration on a given day does not affect incidence of the health effect on that day. To the extent that a pollutant-related health effect on a given day is affected by pollutant concentrations over a longer period of time, then these models would be mis-specified, and this mis-specification would affect the predictions of daily incidence based on the model. The extent to which short-term NO<sub>2</sub> exposure studies may not capture the possible impact of long-term exposures to NO<sub>2</sub> is unknown. A number of epidemiologic studies have examined the effects of long-term exposure to NO<sub>2</sub> and observed associations with decrements in lung function and partially irreversible decrements in lung function growth. The final ISA (EPA, 2008a) concludes, however, that “overall, the epidemiological evidence was suggestive but not sufficient to infer a causal relationship between long-term NO<sub>2</sub> exposure and respiratory morbidity” (ISA, section 3.4). Currently, there is insufficient information to adequately adjust for the potential impact of longer-term exposure on respiratory ED visits associated with NO<sub>2</sub> exposures, if any, and this uncertainty should be kept in mind as one considers the results from the short-term exposure NO<sub>2</sub> risk assessment.

## 9.5 BASELINE HEALTH EFFECTS INCIDENCE DATA

As illustrated in Equation 9-1, the most common health risk model based on air pollution epidemiological studies expresses the reduction in health risk ( $\Delta y$ ) associated with a given reduction in  $\text{NO}_2$  concentrations ( $\Delta x$ ) as a percentage of the baseline incidence ( $y$ ). To accurately assess the impact of changes in  $\text{NO}_2$  air quality on health risk in a given urban area, information on the baseline incidence of health effects in that location is therefore needed. For this assessment, baseline incidence is the incidence under recent (“as is”) air quality conditions.

We obtained annual estimates of the baseline incidence of respiratory ED visits in Atlanta, GA via personal communication with the authors of the study conducted in the Atlanta area (Tolbert, 2007). Tolbert et al. (2007) notes that there are 42 hospitals with emergency departments in the 20-county Atlanta MSA. Of these, 41 were able to provide incidence data for at least part of the study period (1993 – 2004). For purposes of the  $\text{NO}_2$  risk assessment, we need incidences for the years of the risk assessment (2005 – 2007). Assuming that baseline incidence of respiratory ED visits does not change appreciably in the span of a few years, we have used the incidence of respiratory ED visits for the most recent year (i.e., 2004) in the Tolbert et al. study, which was 121,818 respiratory ED visits.<sup>4</sup> Because this baseline incidence estimate is based on 36 hospitals, rather than the total 42 hospitals in Atlanta, this will be an underestimate of baseline incidence. This is a source of downward bias in our estimates of  $\text{NO}_2$ -related risk. While there is some year-to-year variability in respiratory-related baseline incidence (e.g., there were roughly 130,000 and 140,000 respiratory-related ED visits in 2002 and 2003, respectively, in the Atlanta area), the estimate used for the risk assessment based on 2004 was within 10% of the average for the most recent three year period available.

Average daily baseline incidences, necessary for short-term daily concentration-response functions, were calculated by dividing the annual incidence by the number of days in the year for which the baseline incidences were obtained. To the extent that  $\text{NO}_2$  affects health, however, actual incidence rates would be expected to be somewhat higher than average on days with high  $\text{NO}_2$  concentrations; using an average daily incidence would therefore result in underestimating the changes in incidence on such days. Similarly, actual incidence rates would be expected to be

---

<sup>4</sup> The specific definition of “respiratory-related” emergency department visits used in Tolbert et al. (2007) included visits with the following respiratory illnesses as the primary diagnosis (specified by ICD-9 diagnostic codes): asthma (493, 786.07, and 786.09), COPD (491, 492, and 496), upper respiratory illness (460 – 465, 460.0, and 477), pneumonia (480 – 486), and bronchiolitis (466.1, 466.11, and 466.19).

somewhat lower than average on days with low NO<sub>2</sub> concentrations; using an average daily incidence would, therefore, result in overestimating the changes in incidence on low NO<sub>2</sub> days. Both effects would be expected to be small, however, and should largely cancel one another out.

## 9.6 ADDRESSING UNCERTAINTY AND VARIABILITY

An important issue associated with any population health risk assessment is the characterization of uncertainties and variability. *Uncertainty* refers to the lack of knowledge regarding both the actual values of model input variables (parameter uncertainty) and the physical systems or relationships (model uncertainty – e.g., the shape of the concentration-response functions). In any risk assessment, uncertainty is, ideally, reduced to the maximum extent possible, but significant uncertainty often remains. It can be reduced by improved measurement and improved model formulation. In addition, the degree of uncertainty can be characterized, sometimes quantitatively. For example, for the NO<sub>2</sub> risk assessment the statistical uncertainty surrounding the estimated NO<sub>2</sub> coefficients in the concentration-response functions is reflected in the confidence intervals provided for the risk estimates presented in this chapter and in Appendix C. Additional uncertainties are discussed briefly below and in more detail in Appendix C.

*Variability* refers to the heterogeneity in a population or variable of interest that is inherent and cannot be reduced through further research. The current risk assessment for Atlanta is based on locations-specific inputs (i.e., air quality data, baseline incidence data, and concentration-response functions are for the Atlanta MSA). Variability in air quality data is considered to some extent by the inclusion of three years of data. Temporal variability is more difficult to address, because the risk assessment focuses on some unspecified time in the future when a given standard is just being met. To minimize the degree to which values of inputs to the analysis may be different from the values of those inputs at that unspecified time: we have used recent input data – for example, air quality data for the period 2005-2007 and baseline incidence data for 2004. However, future changes in these inputs have not been predicted (e.g., future population levels or changes in baseline incidence).

A number of important sources of uncertainty have been addressed qualitatively. Using a similar approach to that described in section 7.8 for the air quality characterization and in section 8.12 for the exposure assessment in this document, staff have evaluated uncertainty in the

respiratory-related ED visits risk assessment using an approach adapted from the recent guidelines for qualitative uncertainty characterization (WHO, 2008). Uncertainties have been qualitatively evaluated with respect to the level of uncertainty and the direction of bias. The level of uncertainty was evaluated by considering the degree of severity of the uncertainty, implied by the relationship between the source of the uncertainty and the output of the assessment. We have used a designation of low, medium, and high as described in WHO (2008).

The bias direction indicates how the source of uncertainty was judged to influence estimated ED visits associated with NO<sub>2</sub> concentrations, either the estimated number or percent of ED visits are likely “over-“ or “under-estimated”. In the instance where two or more types or components of uncertainty result in offsetting direction of influence, the bias was judged as “both”. An “unknown” bias was assigned where there was no evidence reviewed to judge the uncertainty associated with the source. Table 9-1 provides a summary of the sources of uncertainty identified in the health risk assessment, the level of uncertainty, and the overall judged bias of each. A brief summary discussion regarding those sources of uncertainty not already examined in chapter 7 is included in the comments section of Table 9-1 and is elaborated on in the bulleted points below.

- Causality. There is uncertainty about whether the association between NO<sub>2</sub> and ED visits actually reflects a causal relationship. Our judgment, drawing on the conclusions in the ISA and as discussed in more detail in chapter 4, is that there is, at a minimum, a likely causal relationship with either short-term NO<sub>2</sub> itself or with NO<sub>2</sub> serving as an indicator for itself and other components of ambient air associated with combustion processes.
- Empirically estimated concentration-response relationships. In estimating the concentration-response relationships, there are uncertainties: (1) surrounding estimates of NO<sub>2</sub> coefficients in concentration-response functions used in the assessment, (2) concerning the specification of the concentration-response model (including the shape of the relationships) and whether or not a population threshold or non-linear relationship exists within the range of concentrations examined in the studies, and (3) concerning the possible role of co-pollutants. The uncertainty resulting from the statistical uncertainty associated with the estimated NO<sub>2</sub> coefficient in the concentration-response function has been characterized by confidence intervals reflecting sample size. These confidence intervals do not reflect the uncertainties related to the concentration-response functions, such as whether or not the model

**Table 9-1. Characterization of Key Uncertainties in the Emergency Department Visits Health Risk Assessment for the Atlanta Region.**

Uncertainty	Level of Uncertainty	Direction of Bias	Comments
Causality	low	Upward, if causality assumption isn't true.	Statistical association does not prove causation. However, the risk assessment considers only a health endpoint for which the overall weight of the evidence supports the assumption that NO <sub>2</sub> is likely causally related based on the totality of the health effects evidence. If the assumption of a causal relationship is incorrect, then a positive estimated coefficient in the concentration-response function would be upward biased, since it is greater than zero.
Empirically estimated concentration-response relations	medium	No obvious bias, if concentration-response model is correctly specified. Otherwise, unclear.	Because concentration-response functions are empirically estimated, there is uncertainty surrounding these estimates. If the model is correctly specified, there is no bias in the coefficient estimates. If the model is mis-specified, there can be bias. Omitted confounding variables, for example, could cause upward bias in the estimated NO <sub>2</sub> coefficients if the omitted variables are positively correlated with both NO <sub>2</sub> and the health effect. However, including potential confounding variables that are highly correlated with one another can lead to unstable estimators. Because both single- and multi-pollutant models were available, both were used.
Functional form of concentration-response relation	medium	Unclear	Statistical significance of coefficients in an estimated concentration-response function does not necessarily mean that the mathematical form of the function is the best model of the true concentration-response relation. If the "true" functional relationship between NO <sub>2</sub> and a health effect is different from the one specified, there can be bias in the resulting estimates of effect. The direction of the bias will depend on how the specified model differs from reality. For example, if the specified concentration-response function is log-linear down to 0 ug/m <sup>3</sup> , but there is actually a threshold in the true relationship, then the effect will be overstated by the model corresponding to levels of NO <sub>2</sub> below the threshold.

Uncertainty	Level of Uncertainty	Direction of Bias	Comments
Lag structure of concentration-response relation	low	<p>Downward, if important lags are omitted (e.g., if concentration-response function includes a single lag, while “truth” is a distributed lag).</p> <p>Unclear, if concentration-response function includes a single lag, but it’s the wrong lag.</p>	<p>The actual lag structure for short-term NO<sub>2</sub> exposures is uncertain. Omitted lags could cause an underestimation in the predicted incidence associated with a given reduction in NO<sub>2</sub> concentrations. The level of uncertainty (in the sense of the <i>impact</i> of the uncertainty) may depend on the situation. For example, suppose the health effect is actually affected largely by same-day NO<sub>2</sub> concentrations but the model (incorrectly) includes only a 1-day lag. In this case, the impact on the outcome of the analysis may be minimal if, as is likely, there is a high degree of autocorrelation in NO<sub>2</sub> concentrations from day to day (so that yesterday’s NO<sub>2</sub> level would act as a good proxy for today’s NO<sub>2</sub> level). If, on the other hand, there is a distributed lag – e.g., if risk of the health effect on day <i>t</i> depends on NO<sub>2</sub> concentrations for the entire week leading up to day <i>t</i> – and the model includes only a single lag, then the understatement of effect could be substantial.</p>
Transferability of concentration-response relations	low	No obvious bias.	<p>Concentration-response functions may not provide an adequate representation of the concentration-response relationship in times and places other than those in which they were estimated. For example, populations in the assessment location/time period may have more or fewer members of sensitive subgroups than the location/time period in which functions were derived, which would introduce additional uncertainty related to the use of a given concentration-response function in the analysis. This problem was minimized in the NO<sub>2</sub> risk assessment, however, because it relies on concentration-response functions estimated in a recent study conducted in the assessment location.</p>

<b>Uncertainty</b>	<b>Level of Uncertainty</b>	<b>Direction of Bias</b>	<b>Comments</b>
Extrapolation of concentration-response relations beyond the range of observed NO <sub>2</sub> data	low	Unclear.	A concentration-response relationship estimated by an epidemiological study may not be valid at concentrations outside the range of concentrations observed during the study. This problem should be minimal in the NO <sub>2</sub> risk assessment, however, because the NO <sub>2</sub> concentrations observed in the study from which C-R functions were obtained covered a wide range – from 1 ppb to 181 ppb.
Adequacy of ambient NO <sub>2</sub> monitors as surrogate for population exposure	low	No obvious bias.	Possible differences in how the spatial variation in ambient NO <sub>2</sub> levels across an urban area are characterized in the original epidemiological study compared to the more recent ambient NO <sub>2</sub> data used to characterize current air quality would contribute to uncertainty in the health risk estimates. The NO <sub>2</sub> risk assessment uses the same monitor used in the epidemiological study from which the C-R functions were obtained, which should minimize this source of uncertainty.
Adjustment of air quality distributions to simulate just meeting current and alternative NO <sub>2</sub> standards.	medium to high	No obvious bias.	The pattern and extent of daily reductions in NO <sub>2</sub> concentrations that would result if the current NO <sub>2</sub> standard or alternative NO <sub>2</sub> standards were just met is not known. There remains significant uncertainty about the shape of the air quality distribution of hourly levels upon just meeting an NO <sub>2</sub> standard, especially for alternative standards that are at levels higher than recent NO <sub>2</sub> air quality levels.
Baseline health effects data	Low-medium	Downward bias.	Data on baseline incidence may be uncertain for a variety of reasons. For example, location- and age-group-specific baseline rates may not be available in all cases. Baseline incidence may change over time for reasons unrelated to NO <sub>2</sub> . This source of uncertainty is relatively minor in the NO <sub>2</sub> risk assessment, however, because a baseline incidence estimate has been obtained from the study authors for the assessment area. There is a known downward bias to this estimate, however, because it is based on an incomplete set of hospitals providing ED data (36 out of 42) in the Atlanta MSA.

used in the epidemiological study is the correct model form. With respect to uncertainties about model form and whether or not a population threshold exists, the available epidemiological studies neither support nor refute the existence of thresholds at the population level. Concerning the possible role of co-pollutants in the Tolbert et al. (2007) study, NO<sub>2</sub> was only moderately correlated with the other pollutants considered (i.e., PM<sub>10</sub>, O<sub>3</sub>) that produced the concentration-response functions that have been used in the risk assessment, although it was fairly highly correlated ( $r = 0.7$ ) with CO. When a study, such as Tolbert et al. (2007) is conducted in a single location, the problem of possible confounding is particularly difficult. Single-pollutant models, which omit co-pollutants, may produce overestimates of the NO<sub>2</sub> effect, if some of the effects are really due to one or more of the other pollutants. On the other hand, effect estimates based on a multi-pollutant model can be uncertain and even result in statistically insignificant estimates where there is a true relationship, if the co-pollutants included in the model are highly correlated with NO<sub>2</sub>. As a result of these considerations, we report risk estimates based on both the single- and multi-pollutant models from Tolbert et al. (2007). It should be noted that use of a concentration-response relationship based on an epidemiological study conducted in the same location for this risk assessment reduces some potential uncertainties since it does not involve extrapolation of the relationship across different geographic areas with different population characteristics, land uses, source mixtures and other factors.

- Adequacy of ambient NO<sub>2</sub> monitors as surrogate for population exposure. The Tolbert et al. (2007) study used ambient concentrations at fixed-site monitors to represent ambient exposure and for several reasons this may or may not provide a good representation of ambient NO<sub>2</sub> exposure for the population. The final ISA identifies the following three components to exposure measurement error: (1) the use of average population rather than individual exposure data; (2) the difference between average personal ambient exposure and ambient concentrations at central monitoring sites; and (3) the difference between true and measured ambient concentrations (final ISA, section 1.3.2, p.1-5). While a concentration-response function may understate the effect of personal exposure to NO<sub>2</sub> on the incidence of a health effect, it will give an unbiased estimate of the effect of ambient concentrations on the incidence of the health effect, if the ambient concentrations at monitoring stations provide an unbiased estimate of the ambient concentrations to which the population is exposed. If NO<sub>2</sub> is the causal agent, the understatement of the impact of personal exposures is not a concern, since NO<sub>2</sub> NAAQS are expressed in terms of ambient, not personal exposure, levels. However, if NO<sub>2</sub> is not the causal agent, and the effects are due to confounding copollutants or other factors, then reducing ambient NO<sub>2</sub> levels might not result in the estimated reductions in the health effects.
- Adjustment of air quality distributions to simulate just meeting the current annual standard and alternative 98<sup>th</sup> and 99<sup>th</sup> percentile daily maximum 1-h standards. The current annual standard and many of the alternative 1-h standards analyzed in the current risk assessment requires an upward adjustment of recent ambient NO<sub>2</sub> levels. In adjusting air quality to simulate just meeting these standards, we have assumed that the overall shape of the distribution of 1-h and 24-h concentrations would not change. While we believe this is a reasonable assumption in the absence of evidence

supporting a change in the distribution, we recognize this as an important additional uncertainty, especially for those scenarios where considerable upward adjustment is required to simulate just meeting some of the standards.

- Baseline incidence. There are uncertainties related to the baseline incidence including: (1) the extent to which baseline incidence varies between the year used in the assessment (i.e., 2004) and some unspecified future year when air quality is adjusted to simulate just meeting the current and alternative standards; (2) the extent to which baseline incidence is underestimated because only 36 of the 42 emergency departments provided baseline incidence for the study in 2004; (3) the use of annual incidence data to develop daily baseline incidence; and (4) the extent to which Atlanta area residents visited emergency departments outside of the Atlanta MSA. As noted previously, the use of the available baseline incidence for 2004 results in some underestimation of the risk for the Atlanta MSA since data were only available from 36 of the 42 emergency departments for that year (i.e., about 14% of emergency departments were not included). Concerning the use of annual baseline incidence to estimate daily incidence, to the extent that NO<sub>2</sub> affects health, actual incidence would be expected to be somewhat higher than average on days with high NO<sub>2</sub> concentrations and using an average daily incidence would result in underestimating the changes in incidence on such days. Similarly, actual incidence would be expected to be somewhat lower on days with low NO<sub>2</sub> concentrations and using an average daily incidence would result in overestimating the changes in incidence on such days. Both of these effects would be expected to be small and should largely cancel each other out. With respect to the last uncertainty, we consider this to be a relatively minor uncertainty since most ED visits are likely to be made to the closest emergency department available, which, for residents of the Atlanta MSA are likely to be within that MSA. The baseline incidence data has not been adjusted for any future changes

such as aging of the population over time or possible changes in ED visits due to increased in-migration of younger individuals.

## **9.7 RISK ESTIMATES FOR EMERGENCY DEPARTMENT VISITS**

In this section, we present risk estimates associated with several air quality scenarios, including three recent years of air quality as represented by 2005, 2006, and 2007 monitoring data. In addition, risk estimates are presented for a hypothetical scenario, where air quality from 2006 and 2007 is adjusted upward to simulate just meeting the current annual NO<sub>2</sub> standard, and for scenarios where the three year period (2005-2007) is adjusted (either up or down) to simulate just meeting potential alternative 98<sup>th</sup> and 99<sup>th</sup> percentile daily maximum 1-h standards. As discussed previously in chapter 5, potential alternative 1-h standards with levels set at 0.05, 0.10, 0.15, and 0.20 have been included in the risk assessment.

Throughout this section and Appendix C the uncertainty surrounding risk estimates resulting from the statistical uncertainty of the NO<sub>2</sub> coefficients in the concentration-response functions used is characterized by ninety-five percent confidence intervals around estimates of incidence, incidence per 100,000 population, and percent of total incidence that is NO<sub>2</sub>-related. In some cases, the lower bound of a confidence interval falls below zero. This does not imply that additional exposure to NO<sub>2</sub> has a beneficial effect but only that the estimated coefficient in the concentration-response function was not statistically significantly different from zero. Lack of statistical significance could reflect insufficient statistical power to detect a relationship that exists or could reflect that no relationship exists.

Tables 9-2, 9-3, and 9-4 present the risk estimates for NO<sub>2</sub>-related ED visits associated with recent air quality (2005, 2006, and 2007, respectively). Table 9-2 for 2005 also includes risk estimates for just meeting several alternative 1-h daily maximum standards based on adjusting 2005-2007 air quality data to simulate just meeting these alternative standards. Similarly, Tables 9-3 (based on 2006) and 9-4 (based on 2007) include risk estimates associated with just meeting these same alternative 1-h standards, as well as risk estimates associated with a simulation where air quality is adjusted upward to represent just meeting the current 0.053 ppm annual NO<sub>2</sub> standard. Since attainment of the current annual standard is based on the most recent two year period, risk estimates for the annual standard are only included in the tables based on 2006 and 2007 air quality.

**Table 9-2. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO<sub>2</sub> Concentrations and NO<sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2005 NO<sub>2</sub> Concentrations.\***

Other Pollutants in Model	Percent of Total Incidence of Respiratory Emergency Department Visits Associated with "As is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet Alternative Standards**								
	"as is"	Alternative 98th percentile 1-hr daily maximum standards (ppm)				Alternative 99th percentile 1-hr daily maximum standards (ppm)			
		0.05***	0.1	0.15	0.2	0.05	0.1	0.15	0.2
none	3% (1.6% - 4.3%)	2.1% (1.1% - 3.1%)	4.2% (2.2% - 6.1%)	6.2% (3.3% - 8.9%)	8.1% (4.4% - 11.7%)	2% (1% - 2.9%)	3.9% (2.1% - 5.7%)	5.8% (3.1% - 8.3%)	7.6% (4.1% - 10.9%)
CO	2.5% (0.8% - 4.2%)	1.8% (0.6% - 3%)	3.6% (1.2% - 5.9%)	5.3% (1.8% - 8.7%)	7% (2.4% - 11.3%)	1.7% (0.6% - 2.8%)	3.3% (1.1% - 5.5%)	4.9% (1.7% - 8.1%)	6.5% (2.2% - 10.6%)
O <sub>3</sub>	1.5% (-0.1% - 3.1%)	1.1% (0% - 2.2%)	2.1% (-0.1% - 4.3%)	3.2% (-0.1% - 6.3%)	4.2% (-0.2% - 8.4%)	1% (0% - 2%)	2% (-0.1% - 4%)	2.9% (-0.1% - 5.9%)	3.9% (-0.2% - 7.8%)
PM <sub>10</sub>	1.1% (-0.6% - 2.7%)	0.8% (-0.4% - 1.9%)	1.5% (-0.9% - 3.8%)	2.2% (-1.3% - 5.6%)	3% (-1.7% - 7.4%)	0.7% (-0.4% - 1.8%)	1.4% (-0.8% - 3.5%)	2.1% (-1.2% - 5.2%)	2.8% (-1.6% - 6.9%)
PM <sub>10</sub> , O <sub>3</sub>	0.6% (-1.1% - 2.3%)	0.4% (-0.8% - 1.7%)	0.9% (-1.6% - 3.3%)	1.3% (-2.5% - 4.9%)	1.7% (-3.3% - 6.4%)	0.4% (-0.8% - 1.5%)	0.8% (-1.5% - 3%)	1.2% (-2.3% - 4.5%)	1.6% (-3.1% - 6%)

\*Estimated incidences of respiratory emergency department visits are based on the concentration-response functions estimated in Tolbert et al. (2007) [results corresponding to Figure 2 in Tolbert et al. (2007) were obtained via personal communication with P. Tolbert]. All models use a 3-day moving average of the daily 1-hr. maximum NO<sub>2</sub> concentration and apply to all ages.

\*\*Incidence was quantified down to 0 ppb. Percents are rounded to the nearest tenth.

\*\*\*Alternative 1-hr daily maximum standards are characterized by a concentration of m ppm and an nth percentile, requiring that the average of the 3 annual nth percentile 1-hr daily maxima over a 3-year period be at or below m ppm.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the NO<sub>2</sub> coefficient.

**Table 9-3. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO<sub>2</sub> Concentrations and NO<sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2006 NO<sub>2</sub> Concentrations.\***

Other Pollutants in Model	Percent of Total Incidence of Respiratory Emergency Department Visits Associated with "As is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet the Current and Alternative Standards**									
	"as is"	current annual standard	Alternative 98th percentile 1-hr daily maximum standards (ppm)				Alternative 99th percentile 1-hr daily maximum standards (ppm)			
			0.05***	0.1	0.15	0.2	0.05	0.1	0.15	0.2
none	3.1% (1.6% - 4.5%)	9% (4.9% - 12.9%)	2.2% (1.2% - 3.2%)	4.3% (2.3% - 6.3%)	6.4% (3.5% - 9.3%)	8.5% (4.6% - 12.2%)	2% (1.1% - 3%)	4% (2.2% - 5.9%)	6% (3.2% - 8.7%)	7.9% (4.3% - 11.4%)
CO	2.6% (0.9% - 4.4%)	7.7% (2.6% - 12.5%)	1.9% (0.6% - 3.1%)	3.7% (1.2% - 6.1%)	5.5% (1.8% - 9%)	7.3% (2.5% - 11.8%)	1.7% (0.6% - 2.9%)	3.4% (1.2% - 5.7%)	5.1% (1.7% - 8.4%)	6.8% (2.3% - 11%)
O <sub>3</sub>	1.6% (-0.1% - 3.2%)	4.6% (-0.2% - 9.2%)	1.1% (-0.1% - 2.3%)	2.2% (-0.1% - 4.5%)	3.3% (-0.2% - 6.6%)	4.4% (-0.2% - 8.7%)	1% (0% - 2.1%)	2.1% (-0.1% - 4.1%)	3.1% (-0.1% - 6.2%)	4.1% (-0.2% - 8.1%)
PM <sub>10</sub>	1.1% (-0.6% - 2.8%)	3.3% (-1.9% - 8.2%)	0.8% (-0.4% - 2%)	1.6% (-0.9% - 3.9%)	2.3% (-1.3% - 5.8%)	3.1% (-1.8% - 7.7%)	0.7% (-0.4% - 1.8%)	1.4% (-0.8% - 3.7%)	2.2% (-1.2% - 5.4%)	2.9% (-1.7% - 7.2%)
PM <sub>10</sub> , O <sub>3</sub>	0.6% (-1.2% - 2.4%)	1.9% (-3.6% - 7.1%)	0.4% (-0.8% - 1.7%)	0.9% (-1.7% - 3.4%)	1.3% (-2.5% - 5.1%)	1.8% (-3.4% - 6.7%)	0.4% (-0.8% - 1.6%)	0.8% (-1.6% - 3.2%)	1.2% (-2.4% - 4.7%)	1.6% (-3.2% - 6.2%)

\*Estimated incidences of respiratory emergency department visits are based on the concentration-response functions estimated in Tolbert et al. (2007) [results corresponding to Figure 2 in Tolbert et al. (2007) were obtained via personal communication with P. Tolbert]. All models use a 3-day moving average of the daily 1-hr. maximum NO<sub>2</sub> concentration and apply to all ages.

\*\*Incidence was quantified down to 0 ppb. Percents are rounded to the nearest tenth.

\*\*\*Alternative 1-hr daily maximum standards are characterized by a concentration of m ppm and an nth percentile, requiring that the average of the 3 annual nth percentile 1-hr daily maxima over a 3-year period be at or below m ppm.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the NO<sub>2</sub> coefficient.

**Table 9-4. Estimated Percent of Total Annual Incidence of Respiratory ED Visits Associated with "As Is" NO<sub>2</sub> Concentrations and NO<sub>2</sub> Concentrations that Just Meet Alternative Standards in Atlanta, GA, Based on Adjusting 2007 NO<sub>2</sub> Concentrations.\***

Other Pollutants in Model	Percent of Total Incidence of Respiratory Emergency Department Visits Associated with "As is" NO <sub>2</sub> Concentrations and NO <sub>2</sub> Concentrations that Just Meet the Current and Alternative Standards**									
	"as is"	current annual standard	Alternative 98th percentile 1-hr daily maximum standards (ppm)				Alternative 99th percentile 1-hr daily maximum standards (ppm)			
			0.05***	0.1	0.15	0.2	0.05	0.1	0.15	0.2
none	2.8% (1.5% - 4%)	8.1% (4.4% - 11.6%)	2% (1% - 2.9%)	3.9% (2.1% - 5.7%)	5.8% (3.1% - 8.4%)	7.6% (4.1% - 11%)	1.8% (1% - 2.7%)	3.6% (1.9% - 5.3%)	5.4% (2.9% - 7.8%)	7.1% (3.8% - 10.2%)
CO	2.4% (0.8% - 3.9%)	6.9% (2.3% - 11.3%)	1.7% (0.6% - 2.8%)	3.3% (1.1% - 5.5%)	4.9% (1.7% - 8.1%)	6.5% (2.2% - 10.6%)	1.6% (0.5% - 2.6%)	3.1% (1% - 5.1%)	4.6% (1.5% - 7.5%)	6.1% (2% - 9.9%)
O <sub>3</sub>	1.4% (-0.1% - 2.8%)	4.1% (-0.2% - 8.3%)	1% (0% - 2%)	2% (-0.1% - 4%)	2.9% (-0.1% - 5.9%)	3.9% (-0.2% - 7.8%)	0.9% (0% - 1.9%)	1.8% (-0.1% - 3.7%)	2.7% (-0.1% - 5.5%)	3.6% (-0.2% - 7.3%)
PM <sub>10</sub>	1% (-0.6% - 2.5%)	2.9% (-1.7% - 7.3%)	0.7% (-0.4% - 1.8%)	1.4% (-0.8% - 3.5%)	2.1% (-1.2% - 5.2%)	2.8% (-1.6% - 6.9%)	0.6% (-0.4% - 1.7%)	1.3% (-0.7% - 3.3%)	1.9% (-1.1% - 4.9%)	2.6% (-1.5% - 6.4%)
PM <sub>10</sub> , O <sub>3</sub>	0.6% (-1.1% - 2.2%)	1.7% (-3.2% - 6.4%)	0.4% (-0.8% - 1.5%)	0.8% (-1.5% - 3%)	1.2% (-2.3% - 4.5%)	1.6% (-3% - 6%)	0.4% (-0.7% - 1.4%)	0.7% (-1.4% - 2.8%)	1.1% (-2.1% - 4.2%)	1.5% (-2.8% - 5.6%)

\*Estimated incidences of respiratory emergency department visits are based on the concentration-response functions estimated in Tolbert et al. (2007) [results corresponding to Figure 2 in Tolbert et al. (2007) were obtained via personal communication with P. Tolbert]. All models use a 3-day moving average of the daily 1-hr. maximum NO<sub>2</sub> concentration and apply to all ages.

\*\*Incidence was quantified down to 0 ppb. Percents are rounded to the nearest tenth.

\*\*\*Alternative 1-hr daily maximum standards are characterized by a concentration of m ppm and an nth percentile, requiring that the average of the 3 annual nth percentile 1-hr daily maxima over a 3-year period be at or below m ppm.

Note: Numbers in parentheses are 95% confidence intervals based on statistical uncertainty surrounding the NO<sub>2</sub> coefficient.

In Table 9-2, and similarly in Tables 9-3 and 9-4, the first row of percent of total annual incidence estimates is based on a single pollutant model (i.e., NO<sub>2</sub> only) and results in the largest estimates for NO<sub>2</sub>-related respiratory ED visits. The next three rows present risk estimates based on two pollutant models (i.e., NO<sub>2</sub> + CO, NO<sub>2</sub> + O<sub>3</sub>, NO<sub>2</sub> + PM<sub>10</sub>). The last row presents risk estimates based on a three pollutant model (i.e., NO<sub>2</sub> + PM<sub>10</sub> + O<sub>3</sub>). As noted above in this chapter, effect estimates based on a multi-pollutant model can be uncertain and even result in statistically insignificant estimates where there is a true relationship, if the co-pollutants included in the model are highly correlated with NO<sub>2</sub>. In the case of this study in Atlanta, NO<sub>2</sub> was moderately correlated with PM<sub>10</sub> and O<sub>3</sub> concentrations. The negative lower bounds of the confidence intervals for many of the risk estimates based on multi-pollutant models may in part be due to correlation in these pollutant concentrations and staff do not view these estimates as suggesting any health beneficial health effect of increasing NO<sub>2</sub> exposure levels.

Tables 4-1, 4-2, and 4-3 in Appendix C present these same results expressed in terms of incidence of respiratory-related ED visits in the Atlanta MSA based on recent air quality and just meeting alternative standards based on 2005, 2006, and 2007 air quality data. Finally, Tables 4-4, 4-5, and 4-6 in Appendix C present these same risk estimates expressed in terms of incidence per 100,000 general population in the Atlanta MSA based on recent air quality and simulating just meeting alternative standards based on the same three years of air quality data.

### **Key Observations**

Presented below are key observations resulting from the respiratory-related ED visits risk assessment:

- Respiratory-related ED visits estimated to result from exposures to NO<sub>2</sub> were estimated for a single urban area (i.e., Atlanta) for several recent years of air quality (2005-2007) and for air quality adjusted to simulate just meeting the current annual NO<sub>2</sub> standard and several alternative 1-hour daily maximum NO<sub>2</sub> standards. While we would expect some differences in estimated NO<sub>2</sub>-related ED respiratory visits across different locations due to differences in populations, land use patterns, access to medical facilities, co-pollutants and other factors affecting exposure and the concentration-response relationships, we believe that the risk estimates do provide a useful perspective on the likely overall magnitude and pattern of ED visits associated with various NO<sub>2</sub> air quality scenarios in urban areas within the U.S.
- The largest risk estimates were associated with single-pollutant NO<sub>2</sub> concentration-response functions based on the effect estimates reported in Tolbert et al. (2007). Risk estimates based on various co-pollutant models with O<sub>3</sub>, CO,

and PM<sub>10</sub> resulted in significant reduction in the risk estimates, often by a factor of two or greater and resulted in much wider confidence intervals.

- The only standards that resulted in a reduction in risk estimates from the baseline of recent air quality for the three year period examined were the 98<sup>th</sup> and 99<sup>th</sup> percentile 1-hour daily maximum standards set at the level of 0.05 ppm.
- The impact of changing the level of the alternative 1-hour daily maximum standards is substantially greater than the impact of changing from a 98<sup>th</sup> to a 99<sup>th</sup> percentile standard. For example, changing from a 98<sup>th</sup> percentile 1-hour daily maximum standard set at 0.10 ppm to one set at 0.05 ppm reduces the estimated incidence of respiratory-related ED visits in Atlanta by about 49 percent in 2007 (from 4700 to 2400); however, changing from a 98<sup>th</sup> percentile 1-hour daily maximum standard based on 0.05 ppm to a 99<sup>th</sup> percentile 1-hour daily maximum standard based on 0.05 ppm reduces the incidence in 2007 by only about 8 percent (from 2400 to 2200).
- The overall pattern of risk estimates is similar across the three years examined. For the three years examined, there was not significant year-to-year variability in the risk estimates.
- Important uncertainties and limitations associated with the risk assessment which were discussed above in section 9.6 and which should be kept in mind as one considers the quantitative risk estimates include:
  - uncertainty about the extent to which the associations between NO<sub>2</sub> and ED visits for respiratory causes actually reflect causal relationships;
  - statistical uncertainty due to sampling error which is characterized in the assessment;
  - uncertainties associated with the air quality adjustment procedure that was used to simulate just meeting the current annual and several alternative 1-h daily maximum standards;
  - uncertainties associated with the estimated baseline incidence for ED respiratory visits;
  - uncertainties related to how changes in population, activity patterns, air quality, and other factors over time might impact the risk estimates;
  - there is uncertainty about the extent to which the risk estimates presented for the Atlanta urban area are representative of other urban locations in the U.S.

# 10. EVIDENCE- AND EXPOSURE/RISK-BASED CONSIDERATIONS RELATED TO THE PRIMARY NO<sub>2</sub> NAAQS

## 10.1 INTRODUCTION

This chapter considers the scientific evidence in the ISA (EPA, 2008a) and the exposure and risk characterization results presented in this document as they relate to the adequacy of the current NO<sub>2</sub> primary NAAQS and potential alternative primary NO<sub>2</sub> standards. The available scientific evidence includes epidemiologic, controlled human exposure, and animal toxicological studies. The NO<sub>2</sub> exposure and risk characterizations described in chapters 6-9 of this document include estimates of exposures and health risks associated with recent NO<sub>2</sub> concentrations and with NO<sub>2</sub> concentrations adjusted to simulate scenarios just meeting the current and potential alternative standards. In considering the scientific evidence and the exposure- and risk-based information, we have also considered relevant uncertainties. Section 10.2 of this chapter presents our general approach to considering the adequacy of the current standard and potential alternative standards. Section 10.3 focuses on evidence- and exposure-/risk-based considerations related to the adequacy of the current standard, and section 10.4 focuses on such considerations related to potential alternative standards (in terms of the indicator, averaging time, form, and level).

These considerations are intended to inform the Agency's policy assessment of a range of options with regard to the NO<sub>2</sub> NAAQS. We note that the final decision on retaining or revising the current NO<sub>2</sub> primary standard, taking into account the Agency's policy assessment, is largely a public health policy judgment. A final decision will draw upon scientific information and analyses about health effects, population exposure and risks, and policy judgments about the appropriate response to the range of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on a recognition that the available health effects evidence reflects a continuum consisting of ambient levels at which scientists generally agree that health effects are likely to occur through lower levels at which the likelihood and magnitude of the response become increasingly uncertain. This approach

is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that primary standards be set at a zero-risk level but rather at a level that avoids unacceptable risks to public health, including the health of sensitive groups.

## **10.2 GENERAL APPROACH**

This section describes the general approach that staff is taking to inform decisions regarding the need to retain or revise the current NO<sub>2</sub> NAAQS. The current standard, which is an annual average of 0.053 ppm, was retained by the Administrator in the most recent review in 1996 (61 FR 52854 (October 8, 1996)). The decision in that review to retain the annual standard was based on consideration of available scientific evidence for health effects associated with NO<sub>2</sub> and on air quality information. With regard to these considerations, the Administrator noted that “a 0.053 ppm annual standard would keep annual NO<sub>2</sub> concentrations considerably below the long-term levels for which serious chronic effects have been observed in animals” and that “Retaining the existing standard would also provide protection against short-term peak NO<sub>2</sub> concentrations at the levels associated with mild changes in pulmonary function and airway responsiveness observed in controlled human studies” (60 FR 52874, 52880 (Oct. 11, 1995)). As a result, the Administrator concluded that “the existing annual primary standard appears to be both adequate and necessary to protect human health against both long- and short-term NO<sub>2</sub> exposures” and that “retaining the existing annual standard is consistent with the scientific data assessed in the Criteria Document (U.S. EPA, 1993) and the Staff Paper (U.S. EPA, 1995a) and with the advice and recommendations of CASAC” (61 FR 52852 at 52854).

To inform the range of options that the Agency will consider in this review of the current primary NO<sub>2</sub> standard, the general approach we have adopted builds upon the approaches used in reviews of other criteria pollutants, including the most recent reviews

of the Pb, O<sub>3</sub>, and PM NAAQS (EPA, 2008c; EPA, 2007h; EPA, 2005). As in these other reviews, we consider the implications of placing more or less weight or emphasis on different aspects of the scientific evidence and the exposure/risk-based information, recognizing that the weight to be given to various elements of the evidence and exposure/risk information is part of the public health policy judgments that the Administrator will make in reaching decisions on the standard.

A series of general questions frames our approach to considering the scientific evidence and exposure/risk-based information. First, our consideration of the scientific evidence and exposure/risk-based information with regard to the adequacy of the current standard is framed by the following questions:

- To what extent does evidence and exposure/risk-based information that has become available since the last review reinforce or call into question evidence for NO<sub>2</sub>-associated effects that were identified in the last review?
- To what extent has evidence for different health effects and/or sensitive populations become available since the last review?
- To what extent have uncertainties identified in the last review been reduced and/or have new uncertainties emerged?
- To what extent does evidence and exposure/risk-based information that has become available since the last review reinforce or call into question any of the basic elements of the current standard?

To the extent that the available evidence and exposure/risk-based information suggests it may be appropriate to consider revision of the current standard, we consider that evidence and information with regard to its support for consideration of a standard that is either more or less protective than the current standard. This evaluation is framed by the following questions:

- Is there evidence that associations, especially causal or likely causal associations, extend to ambient NO<sub>2</sub> concentrations as low as, or lower than, the concentrations that have previously been associated with health effects? If so, what are the important uncertainties associated with that evidence?
- Are exposures above benchmark levels and/or health risks estimated to occur in areas that meet the current standard? If so, are the estimated exposures and health

risks important from a public health perspective? What are the important uncertainties associated with the estimated risks?

To the extent that there is support for consideration of a revised standard, we then consider the specific elements of the standard (indicator for gaseous NO<sub>x</sub>, averaging time, form, and level) within the context of the currently available information. In so doing, we address the following questions:

- Does the evidence provide support for considering a different indicator for gaseous NO<sub>x</sub>?
- Does the evidence provide support for considering different averaging times?
- What ranges of levels and forms of alternative standards are supported by the evidence, and what are the associated uncertainties and limitations?
- To what extent do specific averaging times, levels, and forms of alternative standards reduce the estimated exposures above benchmark levels and risks attributable to NO<sub>2</sub>, and what are the uncertainties associated with the estimated exposure and risk reductions?

The following discussion addresses the questions outlined above and presents staff's conclusions regarding the scientific evidence and the exposure-/risk-based information specifically as they relate to the current and potential alternative standards. This discussion is intended to inform the Agency's consideration of policy options that will be presented in an Advanced Notice of Proposed Rulemaking (ANPR), together with the scientific support for such options, and which will be further considered in the Agency's proposed and final rule-making notices. Section 10.3 considers the adequacy of the current standard while section 10.4 considers potential alternative standards in terms of indicator, averaging time, form, and level. Each of these sections considers key conclusions as well as the uncertainties associated with the evidence and/or exposure/risk analyses.

### **10.3 ADEQUACY OF THE CURRENT ANNUAL STANDARD**

In the last review of the NO<sub>2</sub> NAAQS, the AQCD for NO<sub>x</sub> concluded that there were two key health effects of greatest concern at ambient or near-ambient concentrations of NO<sub>2</sub> (ISA, section 5.3.1). The first was increased airway hyperresponsiveness in

asthmatic individuals after short-term exposures. The second was increased respiratory illness among children associated with longer-term exposures to NO<sub>2</sub>. Evidence also was found for increased risk of emphysema, but this appeared to be of major concern only with exposures to levels of NO<sub>2</sub> that were much higher than current ambient levels (ISA, section 5.3.1). Controlled human exposure and animal toxicological studies provided qualitative evidence for airway hyperresponsiveness and lung function changes while epidemiologic studies provided evidence for increased respiratory symptoms with increased indoor NO<sub>2</sub> exposures. Animal toxicological findings of lung host defense system changes with NO<sub>2</sub> exposure provided a biologically-plausible basis for the epidemiologic results. Subpopulations considered potentially more susceptible to the effects of NO<sub>2</sub> exposure included persons with preexisting respiratory disease, children, and the elderly. The epidemiologic evidence for respiratory health effects was limited, and no studies had considered effects such as hospital admissions, ED visits, or mortality (ISA, section 5.3.1).

### **10.3.1 Evidence-based considerations**

Evidence published since the last review generally has confirmed and extended the conclusions articulated in the 1993 AQCD (ISA, section 5.3.2). The epidemiologic evidence has grown substantially with the addition of field and panel studies, intervention studies, time-series studies of effects such as hospital admissions, and a substantial number of studies evaluating mortality risk associated with short-term NO<sub>2</sub> exposures. As noted above, no epidemiologic studies were available in 1993 that assessed relationships between NO<sub>2</sub> and outcomes such as hospital admissions, ED visits, or mortality. In contrast, dozens of epidemiologic studies on such outcomes are now included in this evaluation (ISA, chapter 3). While not as marked as the growth in the epidemiologic literature, a number of recent toxicological and human clinical studies also provide insights into relationships between NO<sub>2</sub> exposure and health effects.

In considering this evidence, we note that different scientific methodologies provide different types of information. For example, controlled human exposure studies provide information on health effects that are specifically associated with exposure to NO<sub>2</sub> in the absence of the co-pollutants that are commonly found in ambient air.

However, these studies do not provide information directly related to the public health implications of real-world NO<sub>2</sub> air quality. Epidemiologic studies provide information on the public health implications of real-world NO<sub>2</sub> concentrations; however, interpretation of specific NO<sub>2</sub>-related effects in these studies is complicated by a number of factors, including the presence of co-pollutants in the ambient air.

As an initial consideration with regard to the adequacy of the current standard, staff notes that the evidence relating long-term (weeks to years) NO<sub>2</sub> exposures to adverse health effects is judged to be either “suggestive but not sufficient to infer a causal relationship” (respiratory morbidity) or “inadequate to infer the presence or absence of a causal relationship” (mortality, cancer, cardiovascular effects, reproductive/developmental effects) (ISA, sections 5.3.2.4-5.3.2.6). In contrast, the evidence relating short-term (minutes to hours) NO<sub>2</sub> exposures to respiratory morbidity is judged to be “sufficient to infer a likely causal relationship” (ISA, section 5.3.2.1). This judgment is supported primarily by a large body of recent epidemiologic evidence that evaluates associations of short-term NO<sub>2</sub> concentrations with respiratory symptoms, ED visits, and hospital admissions. It suggests that, at a minimum, consideration of the adequacy of the current annual standard should take into account the extent to which that standard provides protection against respiratory effects associated with short-term NO<sub>2</sub> exposures. Such an emphasis on health endpoints for which evidence has been judged sufficient to infer a likely causal relationship would be consistent with other recent NAAQS reviews (e.g., EPA, 2005; EPA, 2007h; EPA, 2007i) and would ensure that decisions are based on endpoints for which a causal relationship with NO<sub>2</sub> is judged to be “more likely than not” (ISA, Table 1.3-2).

Because there was concern in the 1996 review of the NO<sub>2</sub> NAAQS about the potential for respiratory effects associated with short-term exposure to NO<sub>2</sub> concentrations around 0.2 ppm, the extent to which the then-current standard (which remains the current standard for purposes of this review) could be expected to afford protection from NO<sub>2</sub> concentrations at this level was considered. In that review, the issue was examined with an air quality analysis that evaluated 1-h NO<sub>2</sub> concentrations. The conclusion from that analysis was that locations meeting the current standard are unlikely

to experience 1-h concentrations exceeding levels (e.g., 0.2 ppm) that have been associated with respiratory effects in controlled human exposure studies (EPA, 1995).

In the current review, a larger number of epidemiologic studies are available. In considering these epidemiologic studies, we note that annual average NO<sub>2</sub> concentrations were below the level of the current annual NO<sub>2</sub> NAAQS in many of the locations where positive associations with respiratory morbidity endpoints have been detected (ISA, section 5.4). With regard to these studies, we note that the ISA characterizes the evidence for respiratory effects as consistent and coherent. The evidence is consistent in that associations are reported in studies conducted in numerous locations and with a variety of methodological approaches (ISA, section 5.3.2.1). It is coherent in the sense that the studies report associations with respiratory health outcomes that are logically linked together (ISA, section 5.3.2.1). When the epidemiologic literature is considered as a whole, there are generally positive associations between NO<sub>2</sub> and respiratory symptoms, hospital admissions, and ED visits. A number of these associations are statistically significant, particularly the more precise effect estimates (ISA, section 5.3.2.1).

Interpretation of these NO<sub>2</sub> epidemiologic studies is complicated by the fact that on-road vehicle exhaust emissions are a nearly ubiquitous source of combustion pollutant mixtures that include NO<sub>2</sub>. In recognition of this complication, the ISA notes that it is difficult to determine the extent to which NO<sub>2</sub> is independently associated with respiratory effects versus being a marker for the effects of another traffic-related pollutant or mix of pollutants (see section 5.4). This uncertainty calls into question the extent to which effect estimates from epidemiologic studies reflect the independent contributions of NO<sub>2</sub> to the adverse respiratory outcomes assessed in these studies.

In order to provide some perspective on this uncertainty, the ISA has evaluated epidemiologic studies that employed multi-pollutant models, epidemiologic studies of indoor NO<sub>2</sub> exposure, and experimental studies. Specifically, the ISA notes that a number of NO<sub>2</sub> epidemiologic studies have attempted to disentangle the effects of NO<sub>2</sub> from those of co-occurring pollutants by employing multi-pollutant models. When evaluated as a whole, NO<sub>2</sub> effect estimates in these models generally remained robust when co-pollutants were included. Therefore, despite uncertainties associated with

separating the effects of NO<sub>2</sub> from those of co-occurring pollutants, the ISA (section 5.4, p. 5-16) concludes that “the evidence summarized in this assessment indicates that NO<sub>2</sub> associations generally remain robust in multi-pollutant models and supports a direct effect of short-term NO<sub>2</sub> exposure on respiratory morbidity at ambient concentrations below the current NAAQS.” With regard to indoor studies, the ISA notes that these studies can test hypotheses related to NO<sub>2</sub> specifically (ISA, section 3.1.4.1). Although confounding by indoor combustion sources is a concern, indoor studies are not confounded by the same mix of co-pollutants present in the ambient air or by the contribution of NO<sub>2</sub> to the formation of secondary particles or O<sub>3</sub> (ISA, section 3.1.4.1). The ISA notes that the findings of indoor NO<sub>2</sub> studies are consistent with those of studies using ambient concentrations from central site monitors (also see chapter 4 of this document) and concludes that indoor studies provide evidence of coherence for respiratory effects (ISA, section 3.1.4.1). With regard to experimental studies, we note that they have the advantage of providing information on health effects that are specifically associated with exposure to NO<sub>2</sub> in the absence of co-pollutants. The ISA concludes that the NO<sub>2</sub> epidemiologic literature is supported by 1) evidence from controlled human exposure studies of airway hyperresponsiveness in asthmatics, 2) controlled human exposure and animal toxicological studies of impaired host-defense systems and increased risk of susceptibility to viral and bacterial infection, and 3) controlled human exposure and animal toxicological studies of airway inflammation (ISA, section 5.3.2.1 and 5.4). When taken together, the results of epidemiologic and experimental studies form a plausible and coherent data set that supports a relationship between NO<sub>2</sub> exposures and respiratory endpoints, including symptoms and ED visits (ISA, section 5.4), at ambient concentrations that are present in areas that meet the current NO<sub>2</sub> NAAQS.

### **10.3.2 Exposure- and risk-based considerations**

In addition to the evidence-based considerations described above, staff has considered the extent to which exposure- and risk-based information can inform decisions regarding the adequacy of the current annual NO<sub>2</sub> standard, taking into account key uncertainties associated with the estimated exposures and risks. For this review,

exposures have been addressed in two ways. In the first, NO<sub>2</sub> air quality in 18 locations around the country has been used as a surrogate for exposure. In the second, exposures have been estimated for all asthmatics and for asthmatic children considering time spent in different microenvironments in one urban area, Atlanta, GA,. For both of these analyses, health risks have been characterized by comparing estimates of air quality or exposure to potential health benchmark levels (see chapters 4 and 6). The benchmarks are based on controlled human exposure studies involving known NO<sub>2</sub> exposure levels and measured airway hyperresponsiveness in asthmatics. The outputs of these analyses are estimates of the occurrence of exposures greater than or equal to benchmark levels, which provide some perspective on the NO<sub>2</sub>-related health risks that could exist. In another approach to characterizing NO<sub>2</sub>-related health risks, we have estimated the occurrences of NO<sub>2</sub>-related respiratory ED visits in Atlanta. This quantitative risk assessment is based on NO<sub>2</sub> concentration-response relationships identified in an epidemiologic study of air pollution-related ED visits in Atlanta. We have selected these endpoints because they are considered adverse to the health of individuals and because the data necessary for the assessment are available.

In making judgments as to whether NO<sub>2</sub>-related effects should be regarded as adverse to the health of individuals, staff has relied upon the guidelines published by the American Thoracic Society (ATS) (2000) and conclusions from the ISA. Of the morbidity endpoints used to characterize risks, ED visits are clearly indicative of effects that are adverse to the health of the individual. The ATS notes that detectable effects of air pollution on clinical measures, including ED visits, should be considered adverse. In addition, regarding airway responsiveness, we recognize the following:

- NO<sub>2</sub>-related airway hyperresponsiveness has the potential to increase asthma symptoms and worsen asthma control (ISA, sections 5.3.2.1 and 5.4).
- The majority of asthmatics may experience NO<sub>2</sub>-related airway hyperresponsiveness following short-term NO<sub>2</sub> exposures between 0.1 ppm and 0.3 ppm (ISA, table 3.1-3).
- Over 20 million people in the U.S. have asthma (ISA, table 4.4-1).

Despite uncertainty as to the magnitude of NO<sub>2</sub>-related airway hyperresponsiveness in any single individual (see below) and despite the fact that not all asthmatics are expected

to respond to NO<sub>2</sub> concentrations between 0.1 and 0.3 ppm, these considerations suggest that NO<sub>2</sub>-related airway hyperresponsiveness is an adverse effect when viewed from the perspective of the asthmatic population as a whole.

#### ***10.3.2.1 Key uncertainties***

The way in which exposure and risk results will inform ultimate decisions regarding the NO<sub>2</sub> standard will depend upon the weight placed on each of the analyses when uncertainties associated with those analyses are taken into consideration. The uncertainties associated with each of the analyses (air quality, Atlanta exposure, and Atlanta risk) are summarized below and are described in more detail in chapters 7-9 of this document. Although we are discussing these uncertainties within the context of the adequacy of the current standard, they apply equally to consideration of alternative standards.

##### *Air Quality Analyses*

A number of key uncertainties should be considered when interpreting these results with regard to decisions on the standard. These uncertainties are discussed briefly below and in more detail in chapter 7.

- In order to simulate just meeting the current annual standard and many of the alternative 1-h standards analyzed, an upward adjustment of recent ambient NO<sub>2</sub> concentrations was required. We note that this adjustment does not reflect a judgment that levels of NO<sub>2</sub> are likely to increase under the current standard or any of the potential alternative standards under consideration. Rather, these adjustments reflect the fact that the current standard, as well as some of the alternatives under consideration, could allow for such increases in ambient NO<sub>2</sub> concentrations. In adjusting air quality to simulate just meeting these standards, we have assumed that the overall shape of the distribution of NO<sub>2</sub> concentrations would not change. While we believe this is a reasonable assumption in the absence of evidence supporting a different distribution and we note that available analyses support this approach (Rizzo, 2008), we recognize this as an important uncertainty. It may be an especially important uncertainty for those scenarios where considerable upward adjustment is required to simulate just meeting one or more of the standards.
- In order to estimate NO<sub>2</sub> concentrations on roadways, empirically-derived relationships between ambient concentrations measured at fixed-site monitors and on-road concentrations were used. We have judged this to be an appropriate approach to estimating on-road NO<sub>2</sub> concentrations given that these

concentrations have been shown to be correlated with concentrations measured at fixed-site monitors (Cape et al., 2004). However, the data used to develop the relationships were likely collected under different conditions (e.g., with regard to meteorology, rate of transformation of NO to NO<sub>2</sub>). We do not know the extent to which it is appropriate to assume that these conditions are representative of the times and places included in our analyses. Therefore, there is uncertainty in the degree to which the relationships used to estimate on-road NO<sub>2</sub> concentrations reflect the actual relationship in the locations and over the time periods of interest.

- The potential health benchmark levels introduce sources of uncertainty including the following:
  - The meta-analysis that formed a large part of the basis for potential health benchmark levels included primarily mild asthmatics. For ethical reasons, more severely affected asthmatics were not included in the studies that formed the basis for the meta-analysis. Severe asthmatics may be more susceptible than mildly asthmatic individuals to the effects of NO<sub>2</sub> exposure (ISA, section 3.1.3.2). Therefore, the potential health effect benchmarks based on these studies could underestimate risks in populations with greater susceptibility. Although approaches to classifying asthma severity differ, some estimates indicate that over half of asthmatics could be classified as moderate/severe (Fuhlbrigge et al., 2002; Stout et al., 2006).
  - This meta-analysis provides information on the direction of the NO<sub>2</sub>-induced airway response, but not on the magnitude of the response. Therefore, although the ISA does conclude that increased airway responsiveness associated with NO<sub>2</sub> exposure could increase symptoms and worsen asthma control (ISA, section 5.4), the full public health implications of benchmark exceedances are uncertain.

### *Atlanta Exposure Assessment*

For our Atlanta exposure assessment, we have considered the occurrence of NO<sub>2</sub> exposures, in asthmatics, that exceed potential health benchmark levels. As with the air quality analyses, these exposures are considered for each of the air quality scenarios evaluated. A number of key uncertainties should be considered when interpreting these results with regard to decisions on the standard. Some of these uncertainties, including the approach used to adjust air quality to simulate just meeting different standards and uncertainties associated with benchmark levels, are shared with the air quality analyses. Additional uncertainties associated with the Atlanta exposure assessment are discussed briefly below. A more extensive discussion of uncertainties is provided in chapter 8.

- A number of uncertainties are associated with exposure modeling, many of them with the activity data used in APEX.
- When compared to ambient measurement data, predicted upper percentile NO<sub>2</sub> concentrations from AERMOD may be 10-50% higher. Because these AERMOD outputs are used as inputs for our exposure modeling, this suggests the possibility that we are over-predicting upper percentile NO<sub>2</sub> exposures. Other approaches used to evaluate our exposure results (i.e., comparison to personal exposure monitoring results and comparison of exposure-to-ambient concentration ratios with those identified in the ISA) suggest that exposure estimates are reasonable. However, we cannot rule out the possibility that we are over-predicting benchmark exceedances with our Atlanta exposure analysis.
- The exposure assessment is limited to Atlanta and the extent to which these results are representative of other locations in the U.S. is uncertain. As noted in section 8.11 above, staff has judged that the Atlanta exposure estimates are likely representative of other moderate to large urban areas. However, staff also recognizes that, given the greater proximity of the population to mobile sources in large urban areas such as Los Angeles, New York, and Chicago (see Tables 8-14 and 8-15), the Atlanta exposure estimates likely underestimate the fraction of asthmatics in these cities that is exposed to NO<sub>2</sub> concentrations greater than or equal to potential health benchmark levels.

#### *Atlanta Risk Assessment*

For our risk assessment, we have considered the prevalence of NO<sub>2</sub>-related respiratory ED visits in Atlanta. As with the air quality and Atlanta exposure analyses, ED visits are considered for each of the air quality scenarios evaluated. A number of key uncertainties should be considered when interpreting these results with regard to decisions on the standard. Some of these, including the approach used to adjust air quality to simulate just meeting different standards and the appropriateness of generalizing results from Atlanta, are uncertainties shared with the air quality and/or Atlanta exposure analyses. Additional uncertainties associated with the Atlanta risk assessment are discussed briefly below. A more extensive discussion of uncertainties is provided in chapter 9.

- There is uncertainty about whether the association between NO<sub>2</sub> and ED visits actually reflects a causal relationship. Our judgment, that there exists at least a likely causal relationship with either short-term NO<sub>2</sub> itself or with NO<sub>2</sub> serving as an indicator for itself and other components of ambient air, draws on the conclusions in the ISA and is discussed in more detail in chapter 4.

- The statistical uncertainty associated with the estimated NO<sub>2</sub> coefficient in the concentration-response function has been characterized by confidence intervals reflecting sample size. However, these confidence intervals do not reflect all of the uncertainties related to the concentration-response functions, such as whether or not the model used in the epidemiologic study is the correct model form.
- Concerning the possible role of co-pollutants in the Tolbert et al. (2007) study, single-pollutant models may produce overestimates of the NO<sub>2</sub> effects if some of those effects are really due to one or more of the other pollutants. On the other hand, effect estimates based on multi-pollutant models can be uncertain, and can even result in statistically non-significant estimates where a true relationship exists, if the co-pollutants included in the model are highly correlated with NO<sub>2</sub>. As a result of these considerations, we report risk estimates based on both the single- and multi-pollutant models from Tolbert et al. (2007).

#### ***10.3.2.2 Assessment results***

As noted previously, the current annual NO<sub>2</sub> standard was retained in 1996 based largely on an evaluation of short-term NO<sub>2</sub> air quality. In that review, an air quality analysis demonstrated that locations meeting the current annual standard were unlikely to experience short-term ambient NO<sub>2</sub> concentrations at central site monitors that have been associated with respiratory effects (i.e., airway hyperresponsiveness) in controlled human exposure studies (i.e., around 0.2 ppm). Therefore, the current annual standard was considered requisite to protect the public health against potential effects associated with short-term (as well as long-term) exposures. We note that a similar analysis of air quality in the current review produced similar results. That is, 1-h NO<sub>2</sub> concentrations greater than or equal to 0.20 ppm are unlikely to occur in locations around the U.S., all of which meet the current annual standard based on recent ambient air quality as measured at central site monitors (i.e., see tables 7-14 to 7-19).

However, in the current review, in addition to evaluating the adequacy of the current standard with ambient air quality as measured at central site monitors, we consider the results of additional analyses that provide perspective on potential NO<sub>2</sub>-associated health risks. For example, in our exposure analyses, we have evaluated NO<sub>2</sub> concentrations on roadways which are, on average, 80% higher than concentrations measured at central site monitors (section 7.3.2). Staff notes that high concentrations of NO<sub>2</sub> on or near roadways could impact asthmatics living or walking nearby (e.g., as

would be common in an urban environment) or commuting in cars. In addition, we have adjusted NO<sub>2</sub> concentrations to simulate NO<sub>2</sub> air quality that could occur upon just meeting the current and potential alternative standards. As noted above (section 10.3.2.1), these adjustments provide information on potential health risks that could be allowed to occur under different standard options. For our exposure analyses, we have compared NO<sub>2</sub> concentrations to potential health benchmark levels from 100 ppb to 300 ppb, a range that extends beyond that considered in the 1996 review. We have also evaluated NO<sub>2</sub>-related ED visits in the current review. Epidemiological studies that form the basis for this analysis were not available in the 1996 review. When taken together, these analyses provide additional information, not available in the 1996 review, on which to base a decision regarding the adequacy of the current annual standard (and potential alternative standards) to protect the public health. The uncertainties associated with these analyses (see 10.3.2.1 and chapter 7) should be carefully considered when interpreting the results of these assessments.

#### *Air Quality and Exposure Results*

The results of our air quality and exposure assessments provide some perspective on the public health impacts of effects that we cannot currently evaluate in a quantitative risk assessment. As noted previously, we have addressed potential exposures with two approaches. In the first, we have estimated air quality exceedances of health benchmark levels in 18 locations across the U.S. In the second, we have estimated exposure exceedances for asthmatics in Atlanta, GA. Results of these analyses, as they relate to the adequacy of the current standard, are discussed below.

When considering the air quality-based results, where air quality serves as a surrogate for exposure, as they relate to the adequacy of the current standard, we note the number of benchmark exceedances estimated to occur given air quality that just meets that standard. As noted above (section 10.3.2.1), this adjustment does not reflect a judgment that levels of NO<sub>2</sub> are likely to increase under the current standard. Rather, it reflects the fact that ambient NO<sub>2</sub> concentrations could increase under the current standard. In situations where annual NO<sub>2</sub> concentrations are adjusted upward to simulate just meeting the current standard, 1-h NO<sub>2</sub> concentrations measured at fixed-site monitors in locations across the U.S. could exceed concentrations that have been associated with

increased airway responsiveness. Most locations are estimated to experience at least 50 days per year with 1-h ambient NO<sub>2</sub> concentrations at fixed-site monitors greater than or equal to 100 ppb (Figures 7-2 and 7-3) under this hypothetical scenario. Far fewer ambient exceedances are predicted for the higher benchmark levels. For example, only 5 areas are estimated to experience any days with 1-h ambient NO<sub>2</sub> concentrations at central site monitors greater than or equal to 300 ppb, and none of those locations are estimated to experience more than 2 such days per year, on average (Appendix A).

However, as noted above on-road NO<sub>2</sub> concentrations are estimated to be 80% higher (on average) than concentrations at fixed-site monitors. In the majority of locations, roadway exceedances of the 100 ppb benchmark level could occur on most days of the year when air quality is adjusted upward to simulate just meeting the current standard (Figure 7-6). Even for higher benchmark levels, most locations are estimated to have exceedances on roadways. All locations evaluated except one (Boston) are estimated to experience on-road NO<sub>2</sub> concentrations greater than or equal to 300 ppb (Appendix A). Four of these locations are estimated to experience an average of greater than 20 days per year with on-road NO<sub>2</sub> concentrations greater than or equal to 300 ppb (Appendix A).

When considering the Atlanta exposure results as they relate to the adequacy of the current standard, we note the number of benchmark exceedances estimated to occur given air quality that is adjusted upward to simulate just meeting the current standard. If NO<sub>2</sub> concentrations were such that the Atlanta area just meets the current standard, nearly all asthmatics in Atlanta (>97%) would be estimated to experience six or more days per year with 1-h NO<sub>2</sub> exposure concentrations greater than or equal to our highest benchmark level (0.3 ppm) (Figure 8-22).

#### *Risk Results*

When considering the Atlanta risk assessment results as they relate to the adequacy of the current standard, there was a range of central estimates since a two year period (2006-2007) was included in the assessment. We note that the central estimates of incidence of NO<sub>2</sub>-related respiratory ED visits in Atlanta ranged from about 8-9% of total respiratory-related ED visits per year based on single pollutant models (or 9,800-10,900 NO<sub>2</sub>-related incidences) when air quality is adjusted upward to simulate a situation where

Atlanta just meets the current standard. Central estimates of incidence of NO<sub>2</sub>-related respiratory ED visits ranged from 2.9-7.7% of total respiratory-related ED visits per year based on two-pollutant models (or 3,600-9,400 NO<sub>2</sub>-related incidences) In addition, inclusion of O<sub>3</sub> and/or PM<sub>10</sub> in multi-pollutant models results in the inclusion of an estimate of zero NO<sub>2</sub>-related respiratory ED visits within the 95% confidence intervals.

### **10.3.3 Conclusions regarding the adequacy of the current standard**

As noted above, several lines of evidence are relevant to consider when making a decision regarding the adequacy of the current standard to protect the public health. These include causality judgments made in the ISA regarding the level of support for effects associated with short-term and long-term exposures, the epidemiologic evidence described in the ISA (and summarized in chapter 4 of this document), the conclusions in the ISA regarding the robustness of this evidence, and the support provided for epidemiologic findings by experimental studies. To the extent that these considerations are emphasized, the adequacy of the current standard to protect the public health would clearly be called into question. Such a conclusion would provide support for consideration of an NO<sub>2</sub> standard that would provide increased health protection for sensitive groups, including asthmatics and individuals who spend time on or near major roadways (see chapter 3), against health effects ranging from increased asthma symptoms to respiratory-related ED visits and hospital admissions associated with short-term exposures, as well as potential effects associated with long-term exposures.

In examining the exposure- and risk-based information with regard to the adequacy of the current annual NO<sub>2</sub> standard to protect the public health, we note that the results described above (and in more detail in chapters 7-9) indicate risks associated with air quality adjusted upward to simulate just meeting the current standard that can reasonably be judged important from a public health perspective. Therefore, exposure- and risk-based considerations reinforce the scientific evidence in supporting the conclusion that consideration should be given to revising the current standard so as to provide increased public health protection, especially for sensitive groups, from NO<sub>2</sub>-related adverse health effects associated with short-term, and potential long-term, exposures.

## **10.4 POTENTIAL ALTERNATIVE STANDARDS**

### **10.4.1 Indicator**

In the last review, EPA focused on NO<sub>2</sub> as the most appropriate indicator for ambient NO<sub>x</sub>. In this review, while the presence of gaseous NO<sub>x</sub> species other than NO<sub>2</sub> has been recognized (e.g., see section 1.3 of this document), no alternative to NO<sub>2</sub> has been advanced as being a more appropriate surrogate for ambient gaseous NO<sub>x</sub>. Controlled human exposure studies and animal toxicology studies provide specific evidence for health effects following exposure to NO<sub>2</sub>. Epidemiologic studies also typically report levels of NO<sub>2</sub>, as opposed to other gaseous NO<sub>x</sub>, though the degree to which monitored NO<sub>2</sub> reflects actual NO<sub>2</sub> levels, as opposed to NO<sub>2</sub> plus other gaseous NO<sub>x</sub>, can vary (e.g., see section 2.2.3 of this document). Because emissions that lead to the formation of NO<sub>2</sub> generally also lead to the formation of other NO<sub>x</sub> oxidation products, measures leading to reductions in population exposures to NO<sub>2</sub> can generally be expected to lead to reductions in population exposures to other gaseous NO<sub>x</sub>. Therefore, meeting an NO<sub>2</sub> standard that protects the public health can also be expected to provide some degree of protection against potential health effects that may be independently associated with other gaseous NO<sub>x</sub> even though such effects are not discernable from currently available studies indexed by NO<sub>2</sub> alone. Given these key points, staff judges that the available evidence supports the retention of NO<sub>2</sub> as the indicator in the current review.

### **10.4.2 Averaging Time**

The current annual averaging time for the NO<sub>2</sub> NAAQS was originally set in 1971, based on epidemiologic studies that supported a link between adverse respiratory effects and long-term exposure to low-levels of NO<sub>2</sub>. As noted in section 10.3.2.2, that annual standard was retained in subsequent reviews in part because an air quality assessment conducted by EPA concluded that areas that meet the annual standard would be unlikely to experience short-term ambient peaks above levels that had been shown in

controlled human exposure studies to impact endpoints of potential concern (see section 10.3.2.2). Based on currently available evidence, the issue of averaging time is being considered in the current review, as discussed below. In order to inform judgments on averaging time, staff has considered causality judgments from the ISA, results from experimental and epidemiologic studies, and NO<sub>2</sub> air quality correlations. These considerations are described in more detail below.

To inform general decisions regarding averaging time (e.g., short-term versus long-term), we note the causality judgments made in the ISA regarding different health endpoints. As described in chapter 4 of this document, the evidence relating short-term (minutes to hours) NO<sub>2</sub> exposures to respiratory morbidity is judged in the ISA to be “sufficient to infer a likely causal relationship” (ISA, section 5.3.2.1) while the evidence relating long-term (weeks to years) NO<sub>2</sub> exposures to adverse health effects is judged to be either “suggestive but not sufficient to infer a causal relationship” (respiratory morbidity) or “inadequate to infer the presence or absence of a causal relationship” (mortality, cancer, cardiovascular effects, reproductive/developmental effects) (ISA, sections 5.3.2.4-5.3.2.6). These judgments most directly support an averaging time that focuses protection on short-term exposures to NO<sub>2</sub>.

As has been done in past reviews, it is instructive to evaluate the potential for a standard based on annual average NO<sub>2</sub> concentrations, as is the current standard, to provide protection against short-term NO<sub>2</sub> exposures. To this end, Table 10-1 reports the ratios of short term to annual average NO<sub>2</sub> concentrations. Ratios of 1-h daily maximum concentrations (98<sup>th</sup> and 99<sup>th</sup> percentile) to annual average concentrations range from 2.5 to 8.7 while ratios of 24-h average concentrations to annual average concentrations range from 1.6 to 3.8 (see Thompson, 2008a for more details). The variability in these ratios across locations, particularly those for 1-h to annual average concentrations, suggests that a standard based on annual average NO<sub>2</sub> concentrations would not likely be an effective or efficient approach to focus protection on short-term NO<sub>2</sub> exposures.

**Table 10-1. Ratios of short-term to annual average NO<sub>2</sub> concentrations**

	<b>1-h Daily Max(99<sup>th</sup>):Annual</b>	<b>1-h Daily Max(98<sup>th</sup>):Annual</b>	<b>24-h Avg(99<sup>th</sup>):Annual</b>	<b>24-h Avg(98<sup>th</sup>):Annual</b>
<b>Location</b>				
Atlanta	4.36	4.00	2.37	2.13
Boston	2.73	2.50	1.81	1.66
Chicago	3.03	2.86	1.68	1.62
Cleveland	3.35	3.03	1.99	1.78
Denver	4.22	3.78	2.44	2.25
El Paso	4.09	3.65	2.05	1.91
Las Vegas	8.65	8.21	2.84	2.65
Los Angeles	3.06	2.70	1.97	1.79
Miami	7.41	7.03	3.76	3.42
New York	2.90	2.60	1.88	1.75
Philadelphia	3.62	3.34	2.40	2.07
Phoenix	2.95	2.70	1.81	1.69
St. Louis	3.82	3.74	1.99	1.84
Washington DC	3.70	3.02	2.10	1.88

For example, in an area with a relatively high ratio (e.g., 8), the current annual standard (0.053 ppm) would be expected to allow 1-h daily maximum NO<sub>2</sub> concentrations of about 0.4 ppm. In contrast, in an area with a relatively low ratio (e.g., 3), the current standard would be expected to allow 1-h daily maximum NO<sub>2</sub> concentrations of about 0.15 ppm. Thus, for purposes of protecting against the range of 1-h NO<sub>2</sub> exposures considered in this review (i.e., 0.1 to 0.3 ppm), a standard based on annual average concentrations would likely require more control than necessary in some areas and less control than necessary in others, depending on the standard level selected.

In considering the level of support available for specific short-term averaging times, we take note of evidence from both experimental and epidemiologic studies. Controlled human exposure studies and animal toxicological studies provide evidence that NO<sub>2</sub> exposures with exposure durations from less than 1-h up to 3-h can result in respiratory effects such as increased airway responsiveness and inflammation (ISA, section 5.3.2.7). Specifically, the ISA concludes that NO<sub>2</sub> exposures of 0.1 ppm for 1-h (or 0.2-0.3 ppm for 30-min) can result in small but significant increases in nonspecific airway responsiveness (ISA, section 5.3.2.1). In contrast, the epidemiologic literature does not provide clear support for one short-term averaging time versus another (ISA, section 5.3.2.7). A number of epidemiologic studies detect positive associations between respiratory morbidity and 1-h (daily maximum) and/or 24-h NO<sub>2</sub> concentrations. A few

epidemiologic studies have considered both 1-h and 24-h averaging times, allowing comparisons to be made. The ISA reports that such comparisons in studies that evaluate asthma ED visits fail to reveal differences between effect estimates based on a 1-h averaging time and those based on a 24-h averaging time (ISA, section 5.3.2.7). Therefore, the ISA concludes that it is not possible, from the available epidemiologic evidence, to discern whether effects observed are attributable to average daily (or multi-day) concentrations (24-h average) or high, peak exposures (1-h maximum) (ISA, section 5.3.2.7).

Given the above conclusions, the experimental evidence provides support for an averaging time of shorter duration than 24 hours (e.g., 1-h) while the epidemiologic evidence provides support for both 1-h and 24-h averaging times. At a minimum, this suggests that a primary concern with regard to averaging time is the level of protection provided against 1-h daily maximum NO<sub>2</sub> concentrations. However, it is also worthwhile to consider the ability of averaging times under consideration to protect against 24-h average NO<sub>2</sub> concentrations. To this end, Table 10-2 presents correlations between 1-h daily maximum NO<sub>2</sub> concentrations and 24-h average NO<sub>2</sub> concentrations (98<sup>th</sup> and 99<sup>th</sup> percentile) across 14 locations (see Thompson, 2008a for more detail). Typical ratios range from a 1.5 to 2.0, though one ratio (Las Vegas) is 3.1. These ratios are far less variable than those discussed above for annual average concentrations, suggesting that a standard based on 1-h daily maximum NO<sub>2</sub> concentrations could also be effective at providing adequate protection against 24-h NO<sub>2</sub> concentrations.

**Table 10-2. Ratios of 1-h daily maximum NO<sub>2</sub> concentrations to 24-h average concentrations (ppm)**

	<b>1-h</b>	<b>24 h</b>	<b>Ratio</b>	<b>1-h</b>	<b>24-h</b>	<b>Ratio</b>
<b>Location</b>	<b>99<sup>th</sup></b>	<b>99<sup>th</sup></b>		<b>98<sup>th</sup></b>	<b>98<sup>th</sup></b>	
Atlanta	0.078	0.042	1.84	0.071	0.038	1.88
Boston	0.064	0.043	1.50	0.059	0.039	1.50
Chicago	0.093	0.052	1.80	0.088	0.050	1.77
Cleveland	0.072	0.043	1.68	0.065	0.038	1.70
Denver	0.086	0.050	1.73	0.077	0.046	1.68
El Paso	0.075	0.038	1.99	0.067	0.035	1.91
Las Vegas	0.039	0.013	3.04	0.037	0.012	3.10
Los Angeles	0.095	0.061	1.56	0.083	0.055	1.50
Miami	0.059	0.030	1.97	0.056	0.027	2.06
New York	0.093	0.060	1.55	0.083	0.056	1.48
Philadelphia	0.060	0.040	1.51	0.056	0.035	1.61
Phoenix	0.093	0.057	1.63	0.085	0.053	1.60
St. Louis	0.064	0.034	1.91	0.063	0.031	2.03
Washington DC	0.079	0.045	1.76	0.065	0.040	1.61

As an additional matter, we note that a short-term standard (i.e., 1-h daily maximum) within the lower part of the range of standards considered in this risk and exposure assessment document could have the effect of maintaining annual average NO<sub>2</sub> concentrations below the level of the current standard (0.053 ppm). For example, in all locations evaluated, a 1-h standard with a level of 0.05 ppm is estimated to result in annual average NO<sub>2</sub> concentrations less than or equal to approximately 0.02 ppm. A 1-h standard with a level of 0.10 ppm is estimated to result in annual average NO<sub>2</sub> concentrations less than or equal to approximately 0.04 ppm. However, a 1-h standard with a level of 0.15 ppm could result in annual average NO<sub>2</sub> concentrations up to approximately 0.06 ppm and a 1-h standard with a level of 0.20 ppm could result in annual average NO<sub>2</sub> concentrations up to approximately 0.07 ppm (Table 10-3).

**Table 10-3. Mean annual NO<sub>2</sub> concentrations for 2004-2006 given just meeting alternative 1-h standards (98<sup>th</sup> percentile)**

Location	Monitor Distance from Major Roadway	Annual NO <sub>2</sub> Concentrations (ppm) for Different Standard Levels			
		0.05	0.10	0.15	0.20
Atlanta	>= 100 m	0.01	0.02	0.02	0.03
Boston	<= 20 m	0.02	0.03	0.05	0.07
Boston	> 20 and < 100 m	0.01	0.02	0.03	0.04
Boston	>= 100 m	0.01	0.01	0.02	0.02
Chicago	<= 20 m	0.01	0.02	0.03	0.04
Chicago	> 20 and < 100 m	0.02	0.03	0.05	0.07
Chicago	>= 100 m	0.01	0.02	0.03	0.04
Cleveland	<= 20 m	0.02	0.03	0.05	0.06
Cleveland	> 20 and < 100 m	0.01	0.02	0.04	0.05
Denver	<= 20 m	0.02	0.04	0.06	0.07
Denver	>= 100 m	0.01	0.03	0.04	0.05
Detroit	>= 100 m	0.02	0.03	0.05	0.06
El Paso	> 20 and < 100 m	0.01	0.02	0.03	0.04
El Paso	>= 100 m	0.01	0.02	0.03	0.04
Jacksonville	>= 100 m	0.01	0.03	0.04	0.05
Las Vegas	<= 20 m	0.02	0.03	0.05	0.06
Las Vegas	>= 100 m	0.01	0.01	0.02	0.03
Los Angeles	<= 20 m	0.02	0.03	0.05	0.06
Los Angeles	> 20 and < 100 m	0.01	0.03	0.04	0.05
Los Angeles	>= 100 m	0.01	0.02	0.03	0.04
Miami	<= 20 m	0.01	0.01	0.02	0.02
Miami	> 20 and < 100 m	0.01	0.02	0.03	0.05
Miami	>= 100 m	0.01	0.01	0.02	0.03
New York	<= 20 m	0.02	0.03	0.05	0.07
New York	> 20 and < 100 m	0.02	0.03	0.05	0.07
New York	>= 100 m	0.01	0.02	0.04	0.05
Philadelphia	> 20 and < 100 m	0.02	0.03	0.05	0.07
Philadelphia	>= 100 m	0.01	0.03	0.04	0.05
Phoenix	<= 20 m	0.01	0.03	0.04	0.06
Phoenix	> 20 and < 100 m	0.01	0.02	0.03	0.05
Phoenix	>= 100 m	0.01	0.03	0.04	0.06
Provo	>= 100 m	0.01	0.03	0.04	0.05
St Louis	<= 20 m	0.01	0.03	0.04	0.05
St Louis	> 20 and < 100 m	0.01	0.02	0.03	0.04
St Louis	>= 100 m	0.01	0.02	0.04	0.05
Washington	<= 20 m	0.01	0.03	0.04	0.06
Washington	> 20 and < 100 m	0.01	0.03	0.04	0.06
Washington	>= 100 m	0.01	0.02	0.03	0.05

### 10.4.3 Form

When evaluating alternative forms in conjunction with specific levels, staff considers the adequacy of the public health protection provided by the combination of level and form to be the foremost consideration. In addition, we recognize that it is

important to have a form that is reasonably stable and relatively insulated from the impacts of extreme meteorological events. A standard set with a high degree of instability could have the effect of reducing public health protection because shifting in and out of attainment due to meteorological conditions could disrupt an area's ongoing implementation plans and associated control programs.

Therefore, as noted in chapter 5 and consistent with recent reviews of the O<sub>3</sub> and PM NAAQS, we have focused in the current review on concentration-based forms averaged over 3 years. As noted in the review of the O<sub>3</sub> NAAQS (EPA, 2007h), concentration-based forms better reflect pollutant-associated health risks than forms based on expected exceedances because concentration-based forms give proportionally greater weight to periods of time when pollutant concentrations are well above the level of the standard than to times when the concentrations are just above the standard, while an expected exceedance form would give the same weight to periods of time with concentrations that just exceed the standard as to times when concentrations greatly exceed the standard. Concentration-based forms also provide greater regulatory stability than a form based on allowing only a single expected exceedance.

In considering specific concentration-based forms on which to focus the current review, we note the need to minimize the number of days per year that an area could exceed the standard level and still attain the standard. Given this, we have focused on 98<sup>th</sup> and 99<sup>th</sup> percentile forms averaged over 3 years. With regard to these alternative forms, staff notes that a 99<sup>th</sup> percentile form for a 1-h daily maximum standard would correspond to the 4<sup>th</sup> highest daily maximum concentration in a year while a 98<sup>th</sup> percentile form would correspond approximately to the 7<sup>th</sup> to 8<sup>th</sup> highest daily maximum concentration in a year (Table 10-4; see Thompson, 2008 for methods). As noted in chapter 5, staff has judged that these forms would provide an appropriate balance between limiting peak NO<sub>2</sub> concentrations and providing a stable regulatory target. This is consistent with judgments made in the 2006 review of the PM NAAQS (EPA, 2005).

**Table 10-4. NO<sub>2</sub> concentrations (ppm) corresponding to 2<sup>nd</sup>-9<sup>th</sup> daily maximum and 98<sup>th</sup>/99<sup>th</sup> percentile forms (2004-2006)**

2004-2006	NO <sub>2</sub> Daily Maximums								Percentiles	
Location	2nd	3rd	4th	5th	6th	7th	8th	9th	99th	98th
Atlanta	0.083	0.079	0.078	0.074	0.073	0.072	0.070	0.070	0.078	0.071
Boston	0.069	0.067	0.064	0.063	0.062	0.060	0.059	0.059	0.064	0.059
Chicago	0.103	0.094	0.093	0.090	0.090	0.088	0.088	0.088	0.093	0.088
Cleveland	0.075	0.074	0.072	0.070	0.069	0.066	0.065	0.064	0.072	0.065
Denver	0.094	0.089	0.086	0.082	0.079	0.077	0.073	0.072	0.086	0.077
El Paso	0.085	0.080	0.075	0.072	0.071	0.068	0.067	0.066	0.075	0.067
Las Vegas	0.042	0.040	0.039	0.039	0.038	0.038	0.037	0.037	0.039	0.037
Los Angeles	0.110	0.095	0.095	0.089	0.088	0.084	0.083	0.081	0.095	0.083
Miami	0.065	0.060	0.059	0.058	0.057	0.056	0.054	0.053	0.059	0.056
New York	0.112	0.099	0.093	0.090	0.086	0.084	0.082	0.081	0.093	0.083
Philadelphia	0.065	0.062	0.060	0.059	0.058	0.057	0.056	0.054	0.060	0.056
Phoenix	0.107	0.097	0.093	0.090	0.089	0.086	0.084	0.083	0.093	0.085
St. Louis	0.066	0.065	0.064	0.064	0.063	0.063	0.063	0.063	0.064	0.063
Washington DC	0.102	0.088	0.079	0.075	0.072	0.066	0.065	0.063	0.079	0.065

When considering the extent to which exposure and risk analyses inform judgments on standard form, staff notes that a 99<sup>th</sup> percentile form could be appreciably more protective than a 98<sup>th</sup> percentile form in some locations, as judged by the results of our air quality analyses. For example, in Boston, Philadelphia, and Washington, D.C. a 99<sup>th</sup> percentile standard of 0.20 ppm is estimated to decrease benchmark exceedances, relative to a 98<sup>th</sup> percentile form, by approximately 50-70% (Table 10-5). However, in St. Louis, Detroit, and Las Vegas a 99<sup>th</sup> percentile form could decrease benchmark exceedances by only approximately 10% (Table 10-5). For most locations, the difference is estimated to be between approximately 10 and 50% (Table 10-5). With regard to the Atlanta exposure assessment, we note that adoption of a 98<sup>th</sup> percentile form versus a 99<sup>th</sup> percentile form would have virtually no effect on exposure concentrations at or above 0.1 ppm. However, choice of form could make a difference of approximately 5-10% on the number of exposure concentrations at or above the 0.2 and 0.3 ppm benchmark levels (Figure 8-22). With regard to the Atlanta risk assessment, a 99<sup>th</sup> percentile form is estimated to be associated with approximately 6% to 8% fewer NO<sub>2</sub>-related ED visits than a 98<sup>th</sup> percentile form, across the levels of the potential 1-h standards examined (Tables 9-2 to 9-4).

**Table 10-5. Mean number of days per year (averaged over the 2004-2006 time period) estimated to have ambient (central site monitor) 1-h daily maximum NO<sub>2</sub> concentrations ≥ 0.10 ppm assuming 98<sup>th</sup> and 99<sup>th</sup> percentile forms of a 0.20 ppm standard**

Location Name	Exceedances	
	98 <sup>th</sup>	99 <sup>th</sup>
Atlanta	81	66
Boston	29	9
Chicago	84	62
Denver	211	159
Detroit	209	194
El Paso	159	116
Jacksonville	178	53
Las Vegas	108	97
Los Angeles	70	50
Miami	74	64
New York	108	63
Philadelphia	163	77
Phoenix	224	163
Provo	84	74
St. Louis	146	134
Washington	133	69

When considering these results as they relate to standard form, we note that a decision on form must be made in conjunction with selection of a particular standard level. The primary emphasis in such a decision will be on the level of public health protection provided by the combination of form and level. With regard to a decision on form, we note that the geographic heterogeneity of the impact of form, as indicated in the air quality analysis, suggests that caution should be exercised when using the Atlanta results as a basis for selecting the most appropriate form. To the extent that a decision regarding form emphasizes this geographic uncertainty, it would likely place more weight on the air quality results.

#### **10.4.4 Level**

##### ***10.4.4.1 Evidence-based considerations***

In considering alternative standard levels that would provide greater protection than that afforded by the current standard against NO<sub>2</sub>-related adverse health effects, staff has taken into account scientific evidence from both experimental and epidemiologic studies, as well as the uncertainties and limitations in that evidence. In particular, we have

considered the extent to which controlled human exposure studies provide evidence for a lowest-observed-effects level and the extent to which epidemiologic studies provide evidence for potential effect thresholds and/or for positive associations that extend down to the lower levels of NO<sub>2</sub> concentrations observed in studies. We note that the scientific evidence can provide insights into alternative standard levels only within the context of specific averaging times and forms. Therefore, while this section considers the evidence as it relates to alternative levels, such considerations assume particular averaging times and forms. Additional discussion of averaging time can be found in sections 5.3 and 10.5 of this document. Additional discussion of form can be found in sections 5.4 and 10.6 of this document.

When considering the scientific evidence as it relates to alternative levels, we note that NO<sub>2</sub> concentrations represent different metrics when reported in experimental studies versus epidemiologic studies. Concentrations of NO<sub>2</sub> reported in epidemiologic studies are typically based on ambient monitoring data while NO<sub>2</sub> levels reported in controlled human exposure studies represent the concentration of NO<sub>2</sub> in the breathing zone of the individual. In some locations and at some points in time, individuals are likely exposed to NO<sub>2</sub> concentrations that are higher than those measured at ambient monitors. The ISA concludes that elevated NO<sub>2</sub> monitors (e.g., monitors sited on building roofs), particularly in inner cities, likely underestimate concentrations and personal exposures occurring at lower elevations, closer to motor vehicle emissions (ISA, section 5.2.2). In this situation, average NO<sub>2</sub> concentrations measured at an elevated ambient monitor could be well below concentrations associated with effects in controlled human exposure studies while actual NO<sub>2</sub> exposures on and/or near roadways could be comparable to, or higher than, these concentrations. We note that in this situation, where personal exposure concentrations to ambient NO<sub>2</sub> are higher than ambient levels measured at a fixed-site monitor, ambient standards based principally on controlled exposure studies could be less health-protective than standards primarily based on concentrations reported in epidemiologic studies at ambient monitors. However, the ISA also concludes that, in exposure measurement field studies where personal exposures have been measured, personal exposures to NO<sub>2</sub> of ambient origin were generally lower than ambient NO<sub>2</sub> concentrations based on fixed-site monitors (ISA, tables 2.5-4 and 2.5-5; section 5.2.2).

In this type of situation, where personal exposure concentrations to ambient NO<sub>2</sub> are lower than the levels measured at ambient monitors, we note that an ambient standard based principally on effects levels observed in controlled exposure studies could be more health-protective than a standard based primarily on concentrations reported in epidemiologic studies at ambient monitors.

#### *Controlled Human Exposure Studies*

In considering the available controlled human exposure studies (see chapter 4 for more detail), we note that these studies have addressed the consequences of short-term (e.g., 30-minutes to several hours) NO<sub>2</sub> exposures for a number of health endpoints including airway responsiveness, host defense and immunity, inflammation, and lung function (ISA, section 3.1). In identifying health endpoints on which to focus for purposes of informing decisions about potential alternative standard levels, staff judges it appropriate to focus on those endpoints that occur at or near ambient levels of NO<sub>2</sub> and endpoints that are of clinical significance. As described in more detail in section 4.5.3, the only endpoint to meet these criteria is increased airway responsiveness in asthmatics. The ISA concludes that NO<sub>2</sub> exposures between 0.2 and 0.3 ppm for 30 minutes or 0.1 ppm for 60-minutes can result in small but significant increases in nonspecific airway responsiveness (ISA, section 5.3.2.1) and that “transient increases in airway responsiveness following NO<sub>2</sub> exposure have the potential to increase symptoms and worsen asthma control” (ISA, sections 3.1.3 and 5.4). This effect could have important public health implications due to the large size of the asthmatic population in the United States (ISA, Table 4.4-1). In addition, NO<sub>2</sub> effects on airway responsiveness in asthmatics are part of the body of experimental evidence that provides plausibility and coherence for the observed NO<sub>2</sub>-related increased in hospital admissions and ED visits in epidemiologic studies (ISA, section 5.3.2.1). For all of these reasons, we have focused on increased airway responsiveness in asthmatics when considering the controlled human exposure literature in terms of its ability to inform decisions on alternative standard levels.

With regard to controlled human exposure studies of airway responsiveness, we note that a meta-analysis of individual level data from 19 studies (section 3.1.3.2 and table 4-5 in chapter 4 of this document) indicates that 66% of resting asthmatics

experienced increased airway responsiveness following exposure to 0.1 ppm NO<sub>2</sub>, 67% experienced an increase following exposure to NO<sub>2</sub> concentrations between 0.1 and 0.15 ppm (inclusively), 75% experienced an increase following exposure to NO<sub>2</sub> concentrations between 0.2 and 0.3 ppm (inclusively), and 73% experienced an increase following exposure to NO<sub>2</sub> concentrations above 0.3 ppm. Effects of NO<sub>2</sub> exposure on the direction of airway responsiveness are statistically-significant at all of these levels. As noted in section 10.3.2.1, one of the important uncertainties associated with these results is that, because the meta-analysis evaluated only the direction of the change in airway responsiveness, it is not possible to discern the magnitude of the change from these data. This limitation makes it particularly difficult to quantify the public health implications of these results.

#### *Epidemiologic Studies*

When evaluating the epidemiologic literature for its potential to inform decisions on standard level, we note that the ISA concludes that NO<sub>2</sub> epidemiologic studies provide “little evidence of any effect threshold” (section 5.3.2.9, p. 5-15). In studies that have evaluated concentration-response relationships, they appear linear within the observed range of data (ISA, section 5.3.2.9). In the absence of an apparent threshold, we are focusing on the range of levels that have been associated with key U.S. studies for purposes of identifying the range of standard levels supported by the epidemiologic literature. When identifying this range, we focus on the higher percentiles of NO<sub>2</sub> concentrations measured at the ambient monitors used in the study (i.e., 98<sup>th</sup> and 99<sup>th</sup> percentiles), as these percentiles are likely most relevant for the health effects observed in epidemiologic studies.

Figures 5-1 and 5-2 (see chapter 5) show standardized effect estimates and the 98<sup>th</sup> and 99<sup>th</sup> percentile concentrations of daily 1-h maximum NO<sub>2</sub> for locations and time periods that correspond to key U.S. epidemiologic studies identified in the ISA (see table 5.4-1 in ISA for a list of key studies). These key studies are associated with a range of 98<sup>th</sup>/99<sup>th</sup> percentile 1-h daily maximum levels from 0.05 ppm to 0.21 ppm. In considering this information, we note that, toward the lower end of the range of NO<sub>2</sub> concentrations observed in epidemiologic studies, there is increasing uncertainty as to

whether observed health effects remain plausibly related to exposures to ambient NO<sub>2</sub>, as opposed to the broader mix of air pollutants present in the ambient air.

When considering an appropriate lower end of the range of levels that are supported by the evidence, staff has considered two primary factors. First, the study by Delfino et al., (2002) provides evidence for associations between short-term ambient NO<sub>2</sub> concentrations and respiratory morbidity in a location where NO<sub>2</sub> concentrations were well below levels in most other key U.S. epidemiologic studies. This study reports positive associations between 1-h and 8-h (only 8-h associations were statistically-significant) levels of NO<sub>2</sub> and asthma symptoms in a location where the 98<sup>th</sup> and 99<sup>th</sup> percentile 1-h daily maximum NO<sub>2</sub> concentrations were 0.05 and 0.053 ppm, respectively. Second, the controlled human exposure studies of airway responsiveness that formed the basis for the meta-analysis detected a lowest-observable-effect level of 0.1 ppm NO<sub>2</sub>. However, these studies did not evaluate severe asthmatics. Most of the subjects included in these studies were mild asthmatics. More severely affected asthmatics may be more susceptible than mild asthmatics to the effects of NO<sub>2</sub> exposure (ISA, section 3.1.3.2). As a result, staff judges that it is appropriate to base the lower end of the range of alternative standard levels on the epidemiologic study by Delfino et al. (2002) and on providing increased protection relative to the lowest-observed-effects level for airway hyperresponsiveness in asthmatics. Therefore, staff concludes that the lower end of the range of potential alternative 1-h daily maximum standards that is reasonably supported by the evidence is 0.05 ppm (50 ppb).

When considering an appropriate upper end of the range of 1-h daily maximum standard levels that is supported by the evidence, we note the following:

- Positive and statistically-significant associations were observed in several key U.S. epidemiologic studies associated with 1-h daily maximum levels of NO<sub>2</sub> close to 0.1 ppm (Peel et al., 2005; NYDOH, 2006; Ito et al., 2007; Tolbert et al., 2007) (see Figure 5-1). In multi-pollutant models, effect estimates remained statistically-significant in the study by Ito and positive, but non-significant, in the other studies.
- Positive and statistically-significant NO<sub>2</sub> effect estimates were also observed in the two key U.S. studies associated with the highest 1-h NO<sub>2</sub> concentrations (Linn et al., 2000; Ostro et al., 2001). These studies were associated with 98<sup>th</sup> and 99<sup>th</sup> percentile 1-h daily maximum NO<sub>2</sub> concentrations from 0.18 ppm to 0.21 ppm. These studies did not evaluate multi-pollutant models. Therefore, they do not

provide additional support for an independent association between NO<sub>2</sub> and respiratory morbidity beyond that provided by the studies noted above.

- The meta-analysis of airway responsiveness presented in the ISA reports statistically-significant effects on the direction of airway responsiveness following short-term NO<sub>2</sub> exposures from 0.1 ppm to 0.3 ppm. The ISA does not draw distinctions between levels within this range with regard to the likely magnitude of the response or the percent of asthmatics expected to respond.

Given these observations, staff notes that the scientific evidence provides strong support for a standard at or below 0.1 ppm (100 ppb). However, to the extent that a decision regarding standard level emphasizes the general uncertainties associated with quantifying the contributions of NO<sub>2</sub> to respiratory effects in epidemiologic studies and uncertainties regarding the public health significance of NO<sub>2</sub>-associated airway hyperresponsiveness (particularly at 0.1 ppm), a level as high as 0.2 could be supported. Therefore, staff concludes that the upper end of the range of 1-h daily maximum standard levels that is reasonably supported by the evidence is 0.2 ppm (200 ppb).

#### ***10.4.4.2 Exposure- and risk-based considerations***

Staff's consideration of exposure- and risk-based information as it relates to alternative levels for the primary NO<sub>2</sub> NAAQS builds upon our conclusion, discussed above in section 10.3, that the overall body of scientific evidence clearly calls into question the adequacy of the current standard to protect the public health. Therefore, we have judged it appropriate to consider a range of alternative levels that would improve upon the level of protection provided by the current standard. As noted in chapter 5, this range of levels (0.05-0.20 ppm) is based on results from controlled human exposure and epidemiologic studies. When considering this range of levels, we note that, given recent air quality, only a level of 0.05 ppm would be estimated to result in any counties in the U.S. that are above the level of the standard (Table 10-6; Thompson, 2008).

**Table 10-6. Percent of counties that may be above the level of the standard, given different levels (based on years 2004-2006)**

Alternative Standards and Levels (ppm)	Percent of counties, total and by region, (and total population) not likely to meet standard and level								
	Total counties (population in millions)	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern CA	Outside Regions**
Number of counties with monitors (population in 1000s)	138 (93.9)	31	32	20	12	9	20	13	1
3 year 99 <sup>th</sup> percentile daily 1 hour max:									
0.20	0	0	0	0	0	0	0	0	0
0.15	0	0	0	0	0	0	0	0	0
0.10	0	0	0	0	0	0	0	0	0
0.05	59 (79.6)	77	41	80	17	56	55	77	0
3 year 98 <sup>th</sup> percentile daily 1 hour max:									
0.20	0	0	0	0	0	0	0	0	0
0.15	0	0	0	0	0	0	0	0	0
0.10	0	0	0	0	0	0	0	0	0
0.05	46 (63.4)	55	34	60	17	44	45	69	0

The results of the air quality analysis are presented in chapter 7 of this document. In that chapter, we present estimates of the number of days per year with ambient (based on fixed-site ambient monitors) and on-road (based on on-road adjustment) NO<sub>2</sub> concentrations at or above our potential health benchmark levels for the years 2001-2006. These estimates are based on as-is air quality and air quality that has been adjusted to simulate just meeting the current and potential alternative standards. In considering the results presented chapter 7, we note the following key points:

- Given unadjusted air quality for the years 2001-2006, it is estimated that 1-h NO<sub>2</sub> concentrations would not exceed 0.1 ppm more than 14 days per year, on average, at fixed-site monitors in any location evaluated (Tables 7-13 through 7-18).
- Given air quality for the years 2001-2003 adjusted to simulate just meeting the current standard, it is estimated that that an average of up to 211 days per year (most locations between 20 and 100 days per year) could occur with ambient NO<sub>2</sub> concentrations at fixed-site monitors that exceed 0.1 ppm (Table 7-26).
- Given air quality for the years 2001-2003 adjusted to simulate just meeting a standard level of 200 ppb (98<sup>th</sup> percentile), it is estimated that an average of up to 327 days per year (most locations between 50 and 200 days per year) could occur with ambient NO<sub>2</sub> concentrations at fixed-site monitors that exceed 0.1 ppm (Table 7-26).
- Given air quality for the years 2001-2003 adjusted to simulate just meeting a standard level of 150 ppb (98<sup>th</sup> percentile), it is estimated that an average of up to 200 days per year (most locations between 20 and 100 days per year) could occur with ambient NO<sub>2</sub> concentrations at fixed-site monitors that exceed 0.1 ppm (Table 7-26).
- Given air quality for the years 2001-2003 adjusted to simulate just meeting a standard level of 100 ppb (98<sup>th</sup> percentile), it is estimated that an average of up to 8 days per year could occur with ambient NO<sub>2</sub> concentrations at fixed-site monitors that exceed 0.1 ppm (Table 7-26).
- Given air quality for the years 2001-2003 adjusted to simulate just meeting a standard level of 50 ppb (98<sup>th</sup> percentile), it is estimated that an average of up to 1 day per year could occur with 1-h ambient NO<sub>2</sub> concentrations at fixed-site monitors greater than or equal to 0.1 ppm. All of the locations evaluated except for 2 would be expected to experience 0 days per year with ambient NO<sub>2</sub> concentrations at fixed-site monitors that exceed this benchmark (Table 7-26).
- Mean estimates of days per year with on-road exceedances are higher than estimates of ambient exceedances at fixed-site monitors (Up to 18 days per year for a standard of 50 ppb, 257 for a standard of 100 ppb, 343 for a standard of 150 ppb, and 351 for a

standard of 200 ppb based on the years 2001 to 2006 and 98<sup>th</sup> percentile standards) (Table 7-29).

The results of the Atlanta exposure assessment are presented in chapter 8 of this document. In Figures 8-19 through 8-26, we present estimates of the percent of asthmatics in Atlanta expected to experience NO<sub>2</sub> exposure concentrations at or above our potential health benchmark levels for the year 2002 (estimates were similar for the years 2001-2003 (Figure 8-5)), given unadjusted air quality and air quality that has been adjusted to simulate just meeting the current and potential alternative standards. In considering the results presented in those figures, we note the following key points:

- Given unadjusted air quality, it is estimated that virtually all asthmatics in Atlanta could experience 6 or more exposures to NO<sub>2</sub> concentrations greater than or equal to 0.1 ppm. It is estimated that just under 60% of Atlanta asthmatics could experience at least one exposure to NO<sub>2</sub> concentrations greater than or equal to 0.3 ppm and that fewer than 10% could experience 6 or more exposures to NO<sub>2</sub> concentrations greater than or equal to 0.3 ppm (Figure 8-19).
- Given air quality adjusted to simulate just meeting the current standard, it is estimated that virtually all Atlanta asthmatics could experience 6 or more days per year with NO<sub>2</sub> exposure concentrations greater than or equal to 0.3 ppm (Figure 8-22).
- Given air quality adjusted to simulate just meeting a standard level of either 150 or 200 ppb (98<sup>th</sup> or 99<sup>th</sup> percentile standard), it is estimated that more than 95% of Atlanta asthmatics could experience 1 or more days per year with NO<sub>2</sub> exposure concentrations greater than or equal to 0.3 ppm (Figure 8-23).
- Given air quality adjusted to simulate just meeting a standard level of 100 ppb (99<sup>th</sup> percentile standard), it is estimated that approximately 70% of Atlanta asthmatics could experience 1 or more days per year with NO<sub>2</sub> concentrations of 0.3 ppm or above. With a 99<sup>th</sup> percentile standard, approximately 10% of Atlanta asthmatics could experience 6 or more days per year with NO<sub>2</sub> concentrations of 0.3 ppm or above (Figure 8-26).
- Given air quality adjusted to simulate just meeting a standard level of 50 ppb (99<sup>th</sup> percentile standard), it is estimated that fewer than 10% of asthmatics could experience 1 or more days per year with NO<sub>2</sub> concentrations of 0.3 ppm or above. With a 99<sup>th</sup> percentile standard, virtually none are estimated to be exposed 6 or more times per year. However, this standard is estimated to result in over 90% of asthmatics being exposed 6 or more times per year to NO<sub>2</sub> concentrations of 0.1 ppm or above (Figure 8-24).

The results of the Atlanta risk assessment are presented in chapter 9 of this document. In Tables 9-2 through 9-4, we present estimates, for the years 2005-2007, of the percent of total annual respiratory ED visits in Atlanta associated with NO<sub>2</sub>. These results are also presented as incidence of NO<sub>2</sub>-associated respiratory ED visits in Appendix C. In considering the results presented in Tables 9-2 through 9-4, we note the following key points:

- Based on single-pollutant models, central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with recent air quality for the years 2005-2007 range from 2.8 to 3.1% (or 3,400 to 3,800 NO<sub>2</sub>-related incidences per year). Based on multi-pollutant models, central estimates for this same time period range from 0.6 to 2.6% (or 700 to 3,200 NO<sub>2</sub>-related incidences per year).
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted upward to simulate just meeting the current annual standard (based on 2006-2007) range from 8.1 to 9.0% (or 9,800 to 10,900 NO<sub>2</sub>-related incidences per year) based on single-pollutant models and from 1.7 to 7.7% (or 3,100 to 9,400 NO<sub>2</sub>-related incidences per year) based on multi-pollutant models. .
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted upward to simulate just meeting a 200 ppb, 1-h daily maximum, 98<sup>th</sup> percentile standard (based on 2005-2007) ranges from 7.6 to 8.5% based on single-pollutant models and from 1.6 to 7.3% based on multi-pollutant models.
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted upward to simulate just meeting a 150 ppb, 1-h daily maximum, 98<sup>th</sup> percentile standard (based on 2005-2007) ranges from 5.8 to 6.4% based on single-pollutant models and from 1.2 to 5.5% based on multi-pollutant models.
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted to simulate just meeting a 100 ppb, 1-h daily maximum, 98<sup>th</sup> percentile standard (based on 2005-2007) ranges from 3.9 to 4.3% based on single-pollutant models and from 0.8 to 3.7% based on multi-pollutant models.
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted to simulate just meeting a 50 ppb, 1-h daily maximum, 98<sup>th</sup> percentile standard (based on 2005-2007) ranges from 2.0 to 2.2% based on single-pollutant models and from 0.4 to 1.9% based on multi-pollutant models.
- Central estimates of annual NO<sub>2</sub>-related respiratory ED visits associated with air quality adjusted to simulate 99<sup>th</sup> percentile 1-h daily maximum standards in the range of 50 to 200 ppb are generally on the order of 10% lower than the estimates summarized above for standards with a 98<sup>th</sup> percentile form.

#### ***10.4.4.3 Conclusions regarding level***

As noted in section 10.7.1, staff concludes that the scientific evidence reasonably supports a range of standard levels from 50 ppb to 200 ppb, with strong support for a level at or below 100 ppb. In also considering the exposure-based information, we note that standard levels of 150 and 200 ppb are generally estimated to be associated with a similar or greater number of benchmark exceedances than are associated with just meeting the current standard, with standard levels of 100 and 50 ppb providing appreciable reductions in estimated benchmark exceedances. In considering the risk-based information, we note that all of the standard levels evaluated are estimated to be associated with fewer NO<sub>2</sub>-related ED visits, on average, than are associated with just meeting the current standard, though the reduction associated with a standard level of 200 ppb is relatively small, with reductions notably increasing with standard levels going from 150 ppb down to 50 ppb. When the scientific evidence is considered in conjunction with exposure and risk results, the strongest support is for standard levels between 50 and 100 ppb. This represents a range of levels that is consistent with the scientific evidence and that would be expected to provide improved public health protection relative to that provided by the current annual standard.

## 11. References

- Abt Associates Inc. (2005). Particulate Matter Health Risk Assessment for Selected Urban Areas. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. June 2005. Available online at: [http://www.epa.gov/ttn/naaqs/standards/pm/s\\_pm\\_cr\\_td.html](http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_cr_td.html).
- Abt Associates Inc. (2007). Ozone Health Risk Assessment for Selected Urban Areas. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC., July 2007, Under Contract No. 68-D-03-002, Work Assignment 3-39 and 4-56. Available online at: [http://www.epa.gov/ttn/naaqs/standards/ozone/s\\_o3\\_cr\\_td.html](http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html).
- AHS. (2003). Source and Accuracy Statement for the 2003 AHS-N Data Chart. Available at: <http://www.census.gov/hhes/www/housing/ahs/03dtchrt/source.html>
- AHS. (2004). US Census Bureau's American Housing Survey for the for the Atlanta Metropolitan Area: 2004. Available at: <http://www.census.gov/prod/2005pubs/h170-04-21.pdf>.
- AHS. (2008). US Census Bureau's American Housing Survey, Metropolitan Data. <http://www.census.gov/hhes/www/housing/ahs/metropolitandata.html>.
- American Thoracic Society (2000). What Constitutes an Adverse Health Effect of Air Pollution? *American Journal of Respiratory and Critical Care Medicine*. 161:665-673.
- Barck, C, Sandström T, Lundahl J, Halldén G, Svartengren M, Strand V, Rak S, Bylin G. (2002). Ambient level of NO<sub>2</sub> augments the inflammatory response to inhaled allergen in asthmatics. *Respir Med*. 96:907-917.
- Barck, C, Lundahl J, Halldén G, Bylin G. (2005). Brief exposures to NO<sub>2</sub> augment the allergic inflammation in asthmatics. *Environ Res*. 97:58-66.
- Beckerman, B, Jerrett M, Brook JR, Verma DK, Arain MA, Finkelstein MM. (2008). Correlation of nitrogen dioxide with other traffic pollutants near a major expressway. *Atmos Environ*. 42:275-290.
- Bell, S and Ashenden TW. (1997). Spatial and temporal variation in nitrogen dioxide pollution adjacent to rural roads. *Water Air Soil Pollut*. 95:87-98.

Blackwell A and Kanny D. (2007). Georgia Asthma Surveillance Report. Georgia Department of Human Resources, Division of Public Health, Chronic Disease, Injury, and Environmental Epidemiology Section, February 2007. Publication Number: DPH07/049HW. Available at: <http://health.state.ga.us/epi/cdiee/asthma.asp>.

Blackwell AD, Wu M, Mertz KJ, Powell KE, Williams CP, Chowdhury P. (2003). *The Burden of Asthma in Georgia 2003*. Georgia Department of Human Resources; Division of Public Health; Chronic Disease, Injury, and Environmental Epidemiology Section, December 2003. Publication number DPH03/127HW. Available at: <http://health.state.ga.us/pdfs/epi/cdiee/burdenofasthma.03.pdf>.

Brauer, M, Hoek G, Smit HA, De Jongste JC, Gerritsen J, Postma DS, Kerkhof M, Brunekreef B. (2007). Air pollution and development of asthma, allergy and infections in a birth cohort. *Eur. Respir. J.* 29:879-888.

Bylin, G, Lindvall T, Rehn T, Sundin B. (1985) Effects of short-term exposure to ambient nitrogen dioxide concentrations on human bronchial reactivity and lung function. *Eur. J. Respir. Dis.* 66:205-217.

Cape, JN, Tang YS, van Dijk N, Love L, Sutton MA, Palmer SCF. (2004). Concentrations of ammonia and nitrogen dioxide at roadside verges, and their contribution to nitrogen deposition. *Environ Pollut.* 132:469-478.

CARB. (2001). Indoor air quality: residential cooking exposures. Final report. California Air Resources Board, Sacramento, California. Available at: <http://www.arb.ca.gov/research/indoor/cooking/cooking.htm>.

CARB (2003). Characterizing the Range of Children's Pollutant Exposure during School Bus Commutes. Prepared for the California Air Resources Board under Contract No. 00-322. October 10, 2003. Available at: <http://www.arb.ca.gov/research/apr/past/00-322.pdf>.

CARB. (2006). Diesel Particulate Matter Exposure Assessment Study for the Ports of Los Angeles and Long Beach, California Air Resources Board. Available at: <http://www.arb.ca.gov/regact/marine2005/portstudy0406.pdf>

CDC. (2007). National Center for Health Statistics. National Health Interview Survey (NHIS) Public Use Data Release (2003). Available at: [http://www.cdc.gov/nchs/about/major/nhis/quest\\_data\\_related\\_1997\\_forward.htm](http://www.cdc.gov/nchs/about/major/nhis/quest_data_related_1997_forward.htm).

Chan, AT and Chung MW. (2003). Indoor-outdoor air quality relationships in vehicle: effect of driving environment and ventilation modes. *Atmos Environ.* 37:3795-3808.

- Chauhan, AJ, Inskip HM, Linaker CH, Smith S, Schreiber J, Johnston SL, Holgate ST (2003). Personal exposure to nitrogen dioxide (NO<sub>2</sub>) and the severity of virus-induced asthma in children. *Lancet* 361:1939-1944.
- Clougherty, JE, Levy JI, Kubzansky LD, Ryan PB, Suglia SF, Canner MJ, and Wright RJ. (2007). Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. *Environ. Health Perspect.* 115:1140-1146.
- Cohen J, Cook R, Bailey C, Carr E. (2005). Relationship between motor vehicle emissions of hazardous pollutants, roadway proximity, and ambient concentrations in Portland, OR. *Environmental Modeling and Software.* 20:7-12.
- Delfino, RJ, Gone H, Linn WS, Pellizzari ED, Hu Y. (2003). Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environ. Health Perspect.* 111:647-656.
- Delfino, RJ, Zeiger RS, Seltzer JM, Street DH, McLaren CE. (2002). Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. *Environ. Health Perspect.* 110:A607-A617.
- Devalia, JL, Rusznak C, Herdman MJ, Trigg CJ, Tarraf H, Davies RJ. (1994) Effect of nitrogen dioxide and sulphur dioxide on airway response of mild asthmatic patients to allergen inhalation. *Lancet* 344:1668-1671.
- Dietert, RR, Etzel RA, Chen D, Halonen M, Holladay SD, Jarabek AM, Landreth K, Peden DB, Pinkerton K, Smialowicz RJ, Zoetis T. (2000). Workshop to identify critical window of exposure for children's health: immune and respiratory systems work group summary. *Environ Health Perspect.* Suppl. 108(3):483-490.
- EIA. (2001). Residential Energy Consumption Survey of 2001. Energy Information Administration. Available at: [http://www.eia.doe.gov/emeu/recs/recs2001\\_hc/2001tblhp.html](http://www.eia.doe.gov/emeu/recs/recs2001_hc/2001tblhp.html).
- EPA. (1982). Air Quality Criteria for Oxides of Nitrogen. National Center for Environmental Assessment, Research Triangle Park, NC. EPA-600/8-82-026. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=44436>.
- EPA (1983). Guideline on the Meaning and Use of Precision and Accuracy Data Required by 40 CFR Parts 58, Appendices A and B. June 1983. PB83-238949.
- EPA. (1993). Air Quality Criteria Document for the Oxides of Nitrogen. National Center for Environmental Assessment, Research Triangle Park, NC. EPA-600/8-91/049F. Available at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=40179>.

- EPA. (1995). Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information. OAQPS Staff Paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-95-005. September 1995.
- EPA. (2002). Consolidated Human Activities Database (CHAD) Users Guide. Database and documentation available at: <http://www.epa.gov/chadnet1/>.
- EPA. (2003). AERMOD: Latest Features and Evaluation Results. EPA-454/R-03-003. U.S. Environmental Protection Agency, Research Triangle Park, NC. Available at: [http://www.epa.gov/scram001/7thconf/aermod/aermod\\_mep.pdf](http://www.epa.gov/scram001/7thconf/aermod/aermod_mep.pdf)
- EPA. (2004). AERMOD: Description of Model Formulation. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-454/R-03-004. Available at: [http://www.epa.gov/scram001/7thconf/aermod/aermod\\_mfd.pdf](http://www.epa.gov/scram001/7thconf/aermod/aermod_mfd.pdf).
- EPA (2005). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at: [http://www.epa.gov/ttn/naaqs/standards/pm/data/pmstaffpaper\\_20051221.pdf](http://www.epa.gov/ttn/naaqs/standards/pm/data/pmstaffpaper_20051221.pdf).
- EPA (2005). National Ambient Air Monitoring Strategy. Draft. Available at: <http://www.epa.gov/ttn/amtic/files/ambient/monitorstrat/naamstrat2005.pdf>.
- EPA. (2006a). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4) Volume I: User's Guide. Office of Air Quality Planning and Standards, Research Triangle Park, NC. June 2006. Available at: [http://www.epa.gov/ttn/fera/human\\_apex.html](http://www.epa.gov/ttn/fera/human_apex.html).
- EPA. (2006b). Total Risk Integrated Methodology (TRIM) - Air Pollutants Exposure Model Documentation (TRIM.Expo / APEX, Version 4) Volume II: Technical Support Document. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at: [http://www.epa.gov/ttn/fera/human\\_apex.html](http://www.epa.gov/ttn/fera/human_apex.html).
- EPA. (2006c). User's Guide for the AMS/EPA Regulatory Model – AERMOD (EPA-454/B-03-001, September 2004), Addendum. U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711.
- EPA. (2007a). Plan for Review of the Primary National Ambient Air Quality Standard for Nitrogen Dioxide. Available at: [http://www.epa.gov/ttn/naaqs/standards/nox/s\\_nox\\_cr\\_pd.html](http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_cr_pd.html).

- EPA. (2007b). Nitrogen Dioxide Health Assessment Plan: Scope and Methods for Exposure and Risk Assessment. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at: [http://www.epa.gov/ttn/naaqs/standards/nox/s\\_nox\\_cr\\_pd.html](http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_cr_pd.html).
- EPA. (2007c). US EPA Air Quality System (AQS). Download Detailed AQS Data. Available at: <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm>.
- EPA (2007d). Field Guide to Air Quality Data (v1.0.0). February 28, 2007. Available at: <http://www.epa.gov/ttn/airs/aqsdatamart/documentation/index.htm>.
- EPA. (2007e). 2002 National Emissions Inventory Data & Documentation. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at: <http://www.epa.gov/ttn/chief/net/2002inventory.html>.
- EPA. (2007f). Clean Air Markets - Data and Maps. Office of Atmospheric Programs, Washington, DC. Emissions Prepackaged Data Sets. Available at: <http://camddataandmaps.epa.gov/gdm/index.cfm?fuseaction=emissions.wizard>.
- EPA. (2007g). Ozone Population Exposure Analysis for Selected Urban Areas (July 2007). Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-010. Available at: [http://epa.gov/ttn/naaqs/standards/ozone/s\\_o3\\_cr\\_td.html](http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html).
- EPA. (2007h). Review of the National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-007a. Available at: [http://epa.gov/ttn/naaqs/standards/ozone/s\\_o3\\_cr\\_sp.html](http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html).
- EPA (2007i). Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information. OAQPS Staff paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-013. Available at: [http://www.epa.gov/ttn/naaqs/standards/pb/data/20071101\\_pb\\_staff.pdf](http://www.epa.gov/ttn/naaqs/standards/pb/data/20071101_pb_staff.pdf).
- EPA. (2008a). ISA for Oxides of Nitrogen - Health Criteria. National Center for Environmental Assessment, Research Triangle Park, NC. Available at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=194645>.
- EPA (2008b). Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard: First Draft. Office of Air Quality Planning and Standards, Research Triangle Park, NC.

- EPA (2008c). Risk and Exposure Assessment to Support the Review of the NO<sub>2</sub> Primary National Ambient Air Quality Standard: Second Draft. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- ESRI. (2008). *ESRI Demographic Update Methodology: 2008/2013 (page 8)*. Available at: <http://www.esri.com/library/whitepapers/pdfs/demographic-update-methodology-2008.pdf>
- Finlayson-Pitts, BJ and Pitts JN. (2000). Chemistry of the Upper and Lower Atmosphere. Academic Press, San Diego CA. Page 17.
- Folinsbee, LJ. (1992). Does nitrogen dioxide exposure increase airways responsiveness? *Toxicol Ind Health*. 8:273-283.
- Fuhlbrigge, AL, Adams RJ, Guilbert TW, Grant E, Lozano P, Janson SL, Martinez F, Weiss KB, Weiss ST. (2002). The Burden of Asthma in the United States: Level and Distribution Are Dependent on Interpretation of the National Asthma Education and Prevention Program Guidelines. *Am J Respir Crit Care Med*. 166:144–1049.
- FHWA. (2002). Highway Statistics 2002, Section V: Roadway Extent, Characteristics and Performance. United States Department of Transportation, Federal Highway Administration. Available at: <http://www.fhwa.dot.gov/policy/ohim/hs02/re.htm>.
- FHWA. (2007). Draft Environmental Impact Statement (DEIS) for the Northwest Corridor Project. Produced by the Georgia Department of Transportation (GDOT) and Georgia Regional Transportation Authority (GRTA), in collaboration with the Federal Highway Administration and Federal Transit Administration. Available at: <http://www.nwhovbrt.com/pages/deis.htm#trs>.
- Gauderman, W J, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. (2004). The effect of air pollution on lung development from 10 to 18 years of age. *N. Engl. J. Med*. 351:1057-1067.
- Gauderman, WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R. (2005). Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology* 16:737-743.
- Ghenu, A, Rosant J-M, Sini J-F. (2007). Dispersion of pollutants and estimation of emissions in a street canyon in Rouen, France. *Environmental Modeling & Software*. 23:314-321.
- Gilbert, NL, Woodhouse S, Stieb DM, Brook JR. (2003). Ambient nitrogen dioxide and distance from a major highway. *Sci Total Environ*. 312:43-46.

- Gilles, JA, Etyemezian V, Kuhns H, Nikolic D, Gillette DA. (2005). Effect of vehicle characteristics on unpaved road dust emissions. *Atmos Environ.* 39:2341-2347.
- Graham, SE and McCurdy T. (2004). Developing meaningful cohorts for human exposure models. *J Expos Anal Environ Epidemiol.* 14(1)23-43.
- Hanrahan, PL. (1999a). The plume volume molar ratio method for determining NO<sub>2</sub>/NO<sub>x</sub> ratios in modeling. Part I: Methodology. *J Air & Waste Manage Assoc.* 49:1324-1331.
- Hanrahan, PL. (1999b). The plume volume molar ratio method for determining NO<sub>2</sub>/NO<sub>x</sub> ratios in modeling. Part II: Evaluation Studies. *J Air & Waste Manage Assoc.* 49:1332-1338.
- Heeb, NV, Saxer CJ, Forss AM, Bruhlmann S. (2008). [Trends of NO-, NO<sub>2</sub>-, and NH<sub>3</sub>-emissions from gasoline-fueled Euro-3- to Euro-4-passenger cars.](#) *Atmos Environ.* 42:2543-2554
- Hu PS and Reuscher TR. (2004). Summary of travel trends. 2001 National Household Travel Survey. US Department of Transportation, Federal Highway Administration. Available at: <http://nhts.ornl.gov/2001/pub/STT.pdf>
- Ito, K, Thurston, GD, Silverman, RA. (2007). Characterization of PM<sub>2.5</sub>, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. *J. of Expos. Science and Environ. Epidemiology.* 17:S45-S60.
- Jaffe, DH, Singer ME, Rimm AA. (2003). Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. *Environ. Res.* 91:21-28.
- Jaffe, DH. (2008). Personal Communication to Scott Jenkins. See NO<sub>2</sub> Docket.
- Jenkins, HS, Devalia JL, Mister RL, Bevan AM, Rusznak C, Davies RJ. (1999). The effect of exposure to ozone and nitrogen dioxide on the airway response of atopic asthmatics to inhaled allergen: dose- and time-dependent effects. *Am. J. Respir. Crit. Care Med.* 160:33-39.
- Johnson, T, Mihlan G, LaPointe J, Fletcher K, Capel J. (1999). Estimation Of Carbon Monoxide Exposures and Associated Carboxyhemoglobin Levels In Denver Residents Using pNEM/CO (version 2.0). Prepared by ICF Kaiser Consulting Group for the U.S. Environmental Protection Agency under Contract No. 68-D6-0064, March 1999.

- Jörres, R, Magnussen H. (1990). Airways response of asthmatics after a 30 min exposure, at resting ventilation, to 0.25 ppm NO<sub>2</sub> or 0.5 ppm SO<sub>2</sub>. *Eur. Respir. J.* 3:132-137.
- Jörres, R, Magnussen H. (1991). Effect of 0.25 ppm nitrogen dioxide on the airway response to methacholine in asymptomatic asthmatic patients. *Lung* 169:77-85.
- Jörres, R, Nowak D, Grimminger F, Seeger W, Oldigs M, Magnussen H. (1995). The effect of 1 ppm nitrogen dioxide on bronchoalveolar lavage cells and inflammatory mediators in normal and asthmatic subjects. *Eur Respir J.* 8:416-424.
- Kim, S-Y, O'Neill M, Lee J-T, Cho Y, Kim J, and Kim H. (2007). Air pollution, socioeconomic position, and emergency hospital visits for asthma in Seoul, Korea. *Int. Arch. Occup. Environ. Health* 80:701-710.
- Koontz, MD, Mehegan LL, and Nagda NL. (1992). Distribution and Use of Cooking Appliances That Can Affect Indoor Air Quality, Report No. GRI-93/0013. Gas Research Institute, Chicago, IL.
- Kwon J. (2005). Development of a RIOPA database and evaluation of the effect of proximity on the potential residential exposure to VOCs from ambient sources. PhD. dissertation. Graduate School, New Brunswick, Rutgers, the State University of New Jersey and the University of Medicine and Dentistry of New Jersey.
- Langstaff, JE. (2007). OAQPS Staff Memorandum to Ozone NAAQS Review Docket (OAR-2005-0172). Subject: Analysis of Uncertainty in Ozone Population Exposure Modeling. [January 31, 2007]. Available at: [http://www.epa.gov/ttn/naaqs/standards/ozone/s\\_o3\\_cr\\_td.html](http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html).
- Linn, WS, Shamoo DA, Anderson KR, Peng RC, Avol EL, Hackney JD, Gong, H Jr. (1996). Short-term air pollution exposures and responses in Los Angeles area schoolchildren. *J. Exposure Anal. Environ. Epidemiol.* 6:449-472.
- Linn, WS, Szlachcic Y, Gong H, Kinney PL, Berhane KT. (2000). Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ. Health Perspect.* 108:427-434.
- MACTEC (2005). Evaluation of Bias in AERMOD-PVMRM, Final Report. Alaska DEC Contract No. 18-9010-12. MACTEC Federal Programs, Inc., Research Triangle Park, NC.
- McCurdy, TR. (1994). Analysis of high 1 hour NO<sub>2</sub> values and associated annual averages using 1988-1992 data. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available in Docket A-93-06.

- McCurdy, T, Glen G, Smith L, Lakkadi Y. (2000). The National Exposure Research Laboratory's Consolidated Human Activity Database. *J Exp Anal Environ Epidemiol.* 10: 566-578.
- McCurdy T and Graham SE. (2003). Using human activity data in exposure models: analysis of discriminating factors. *J Expos Anal Environ Epidemiol.* 13:294-317.
- Meng QY, Turpin BJ, Korn L, Weisel CP, Morandi M, Colome S, Zhang JJ, Stock T, Spektor D, Winer A, Zhang L, Lee JH, Giovanetti R, Cui W, Kwon J, Alimokhtari S, Shendell D, Jones J, Farrar C, Maberti S. (2004). Influence of ambient (outdoor) sources on residential indoor and personal PM<sub>2.5</sub> concentrations: Analyses of RIOPA data. *J Exp Anal Environ Epidemiol.* 15:17-28.
- Mohsenin, V. (1987) Airway responses to nitrogen dioxide in asthmatic subjects. *J. Toxicol. Environ. Health* 22:371-380.
- Mortimer, KM, Neas LM, Dockery DW, Redline S, Tager IB. (2002). The effect of air pollution on inner-city children with asthma. *Eur Respir J.* 19:699-705.
- National Cooperative Highway Research Program (NCHRP) (1997). Recommended Procedure for Long-Range Transportation Planning and Sketch Planning, NCHRP Report 387, National Academy Press. 151 pp., ISBN No: 0-309-060-58-3.
- National Heart, Lung, and Blood Institute (NHLBI). (2007) Expert panel report 3: guidelines for the diagnosis and management of asthma. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Asthma Education and Prevention Program; NIH Publication No. 07-4051.
- NCDC. (2007). 2007 Local Climatological Data Annual Summary with Comparative Data. Atlanta, Georgia (Katl). National Climate Data Center. ISSN 0198-1560.
- New York Department of Health. (2006). A study of ambient air contaminants and asthma in New York City, Final Report Part B: Air contaminants and emergency department visits for asthma in the Bronx and Manhattan. Prepared for: The U.S. Department of Health and Human Services, Agency for Toxic Substance and Disease Registry.
- Oftedal, B, Brunekreef B, Nystad W, Madsen C, Walker SE, Nafstad P. (2008). Residential outdoor air pollution and lung function in schoolchildren. *Epidemiology* 19:129-137.

- Ostro, B, Lipsett M, Mann J, Braxton-Owens H, White M. (2001). Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology* 12:200-208.
- Peel, JL, Tolbert PE, Klein M, Metzger KB, Flanders WD, Knox T, Mulholland JA, Ryan PB, Frumkin H. (2005). Ambient air pollution and respiratory emergency department visits. *Epidemiology*. 16:164-174.
- Perry SG, Cimorelli AJ, Paine RJ, Brode RW, Weil JC, Venkatram A, Wilson RB, Lee RF, Peters WD. (2005). AERMOD: A Dispersion Model for Industrial Source Applications. Part II: Model Performance against 17 Field Study Databases. *J. Appl. Meteor.* 44:694-708.
- Persily, A, Gorfain J, Brunner G. (2005). Ventilation design and performance in U.S. office buildings. *ASHRAE Journal*. April 2005, 30-35.
- Pilotto, LS, Douglas RM, Attewell RG, Wilson SR (1997) Respiratory effects associated with indoor nitrogen dioxide exposure in children. *Int. J. Epidemiol.* 26: 788-796.
- Pilotto, LS, Nitschke M, Smith BJ, Pisaniello D, Ruffin RE, McElroy HJ, Martin J, Hiller JE. (2004). Randomized controlled trial of unflued gas heater replacement on respiratory health of asthmatic schoolchildren. *Int J Epidemiol.* 33:208-214.
- Pleijel, H, Karlsson GP, Gerdin EB. (2004). On the logarithmic relationship between NO<sub>2</sub> concentration and the distance from a highroad. *Sci Total Environ.* 332:261-264.
- Riediker M, Williams R, Devlin R, Griggs T, Bromberg P. (2003). Exposure to particulate matter, volatile organic compounds, and other air pollutants inside patrol cars. *Environ Sci Technol.* 37(10):2084-2093.
- Rizzo (2008). Investigation of how distributions of hourly nitrogen dioxide concentrations have changed over time in six cities. Nitrogen Dioxide NAAQS Review Docket (EPA-HQ-OAR-2006-0922 ). Available at [http://www.epa.gov/ttn/naaqs/standards/nox/s\\_nox\\_cr\\_rea.html](http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_cr_rea.html)
- Rodes, C, Sheldon L, Whitaker D, Clayton A, Fitzgerald K, Flanagan J, DiGenova F, Hering S, Frazier C. (1998). Measuring Concentrations of Selected Air Pollutants Inside California Vehicles. California Environmental Protection Agency, Air Resources Board. Final Report, December 1998.

- Rodes, CE and Holland DM. (1981). Variations of NO, NO<sub>2</sub> and O<sub>3</sub> concentrations downwind of a Los Angeles freeway. *Atmos Environ.* 15:243-250.
- Roger, LJ, Horstman DH, McDonnell W, Kehrl H, Ives PJ, Seal E, Chapman R, Massaro E. (1990). Pulmonary function, airway responsiveness, and respiratory symptoms in asthmatics following exercise in NO<sub>2</sub>. *Toxicol. Ind. Health* 6:155-171.
- Romieu, I, Ramírez-Aguilar M., Sienna-Monge JJ, Moreno-Macías, H, Del Rio-Navarro BE, David G, Marzec J, Hernández-Avila M, London S. (2006). GSTM1 and GSTP1 and respiratory health in asthmatic children exposed to ozone. *Eur. Respir. J.* 28:953-959.
- Rojas-Martinez, R, Perez-Padilla R, Olaiz-Fernandez G, Mendoza-Alvarado L, Moreno-Macias H, Fortoul T, McDonnell W, Loomis D, Romieu I. (2007a) Lung function growth in children with long-term exposure to air pollutants in Mexico City. *Am. J. Respir. Crit. Care Med.* 176:377-384.
- Rojas-Martinez, R, Perez-Padilla R, Olaiz-Fernandez G, Mendoza-Alvarado L, Moreno-Macias H, Fortoul T, McDonnell W, Loomis D, Romieu I. (2007b) Lung function growth in children with long-term exposure to air pollutants in Mexico City. Online data supplement. Available: <http://ajrccm.atsjournals.org/cgi/data/176/4/377/DC1/1> [3 October, 2007].
- Roorda-Knape, MC, Janssen NAH, De Hartog JJ, Van Vliet PHN, Harssema H, Brunekreef B. (1998). Air Pollution from traffic in city districts near major roadways. *Atmos Environ.* 32(11)1921-1930.
- Rubenstein, I, Bigby BG, Reiss TF, Boushey HA Jr. (1990) Short-term exposure to 0.3 ppm nitrogen dioxide does not potentiate airway responsiveness to sulfur dioxide in asthmatic subjects. *Am. Rev. Respir. Dis.* 141:381-385.
- Schildcrout, JS, Sheppard L, Lumley T, Slaughter JC, Koenig JQ, Shapiro GG. (2006). Ambient air pollution and asthma exacerbations in children: an eight-city analysis. *Am J Epidemiol.* 164:505-517.
- Schwartz, J, Dockery DW, Neas LM, Wypij D, Ware JH, Spengler JD, Koutrakis P, Speizer FE, Ferris BG, Jr. (1994). Acute effects of summer air pollution on respiratory symptom reporting in children. *Am J Respir Crit Care Med.* 150:1234-1242.

Shorter, JH, Herndon S, Zahniser MS, Nelson DD, Wormhoudt J, Demerjian KL, Kolb CE. (2005). Real-Time Measurements of Nitrogen Oxide Emissions from In-Use New York City Transit Buses Using a Chase Vehicle. *Environ Sci Technol.* 39:7991-8000.

Skuta and Wombold. (2008). *The Secret Life of Polygons: Understanding the relative accuracy of user-defined areas*. Available at: <http://www.esri.com/news/arcuser/1008/polygons.html>.

Spicer, CW, Kenny DV, Ward GF, Billick IH. (1993). Transformations, lifetimes, and sources of NO<sub>2</sub>, HONO, and HNO<sub>3</sub> in indoor environments. *JAWMA.* 43(11):1479-1485.

Stout, JW, Visness CM, Enright P, Lamm, C, Shapiro G, Gan, VN, Adams K, Mitchell HE. (2006). Classification of asthma severity in children. *Arch Pediatr Adolesc Med.* 160:844-850.

Strand, V, Salomonsson P, Lundahl J, Bylin G. (1996). Immediate and delayed effects of nitrogen dioxide exposure at an ambient level on bronchial responsiveness to histamine in subjects with asthma. *Eur Respir J.* 9:733-740.

Strand, V, Rak S, Svartengren M, Bylin G. (1997). Nitrogen dioxide exposure enhances asthmatic reaction to inhaled allergen in subjects with asthma. *Am J Respir Crit Care Med.* 155:881-887.

Strand, V, Svartengren M, Rak S, Barck C, Bylin G. (1998). Repeated exposure to an ambient level of NO<sub>2</sub> enhances asthmatic response to nonsymptomatic allergen dose. *Eur Respir J.* 12:6-12.

Suh, H. (2008). Personal Communication, August 27, 2008, regarding personal exposure data for Atlanta.

[Thompson, R. \(2008\)](#). Nitrogen Dioxide (NO<sub>2</sub>) Descriptive Statistics Tables. Memo to the NO<sub>2</sub> NAAQS docket. Available at [http://www.epa.gov/ttn/naaqs/standards/nox/s\\_nox\\_cr\\_rea.html](http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_cr_rea.html)

[Thompson, R. and Jenkins SM \(2008\)](#). Air Quality Statistics Associated with Nitrogen Dioxide Epidemiologic Studies. Memo to the NO<sub>2</sub> NAAQS docket. Available at [http://www.epa.gov/ttn/naaqs/standards/nox/s\\_nox\\_cr\\_rea.html](http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_cr_rea.html)

Tolbert, P. (2008a). Personal communication (email) to H. Richmond, U.S. EPA – “Atlanta Emergency Department Visit and Air Quality Data used in Tolbert et al. (2007),” May 30.

Tolbert, P. (2008b). Personal communication (email) to H. Richmond, U.S. EPA – “Response to Harvey Richmond regarding CASAC comments on SOPHIA ED study incidence data for the NO<sub>2</sub> risk and exposure assessment,” November 20.

[Tolbert, PE, Klein M, Peel JL, Sarnat SE, Sarnat JA.](#) (2007). Multipollutant modeling issues in a study of ambient air quality and emergency department visits in Atlanta. *J Expos Sci Environ Epidemiol.* 17S2:S29-35.

Tunnicliffe, WS, Burge PS, Ayres JG. (1994) Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 344:1733-1736.

US DOT. (2007). Part 3-The Journey To Work files. Bureau of Transportation Statistics (BTS). Available at: <http://transtats.bts.gov/>.

van Gent, R, van der Ent K, van Essen-Zandvliet LEM, Rovers MM, Kimpen JLL, de Meer G, Klijn PHC. (2007). No difference in physical activity in (un)diagnosed asthma and healthy controls. *Pediatric Pulmonology.* 42:1018-1023.

Vardoulakis, S, Gonzalez-Flesca N, Fisher BEA, Pericleous K. (2004). Spatial variability of air pollution in the vicinity of a permanent monitoring station in central Paris. *Atmos Environ.* 39:2725-2736.

Watkins, N. and Thompson, R. (2008). NO<sub>x</sub> Network Review and Background. Memo to the NO<sub>2</sub> NAAQS docket.

Weisel CP, Zhang JJ, Turpin BJ, Morandi MT, Colome S, Stock TH, Spektor DM, Korn L, Winer A, Alimokhtari S, Kwon J, Mohan K, Harrington R, Giovanetti R, Cui W, Afshar M, Maberti S, Shendell D. (2004). Relationship of Indoor, Outdoor and Personal Air (RIOPA) study; study design, methods and quality assurance / control results. *J Exp Anal Environ Epidemiol.* 15:123–137.

Westerdahl, D, Fruin S, Sax T, Fine PM, Sioutas C. (2005). Mobile platform measurements of ultrafine particles and associated pollutant concentrations on freeways and residential streets in Los Angeles. *Atmos Environ.* 39:3597-3610.

Wheeler, A, Zanobetti A, Gold DR, Schwartz J, Stone P, Suh HH. (2006). The relationship between ambient air pollution and heart rate variability differs for individuals with heart and pulmonary disease. *Environ Health Perspect.* 114(4):560-6.

- WHO (2008). Harmonization Project Document No. 6. Part 1: Guidance document on characterizing and communicating uncertainty in exposure assessment. Available at: <http://www.who.int/ipcs/methods/harmonization/areas/exposure/en/>.
- Witten, A, Solomon C, Abbritti E, Arjomandi M, Zhai W, Kleinman M, Balmes J. (2005). Effects of nitrogen dioxide on allergic airway responses in subjects with asthma. *J. Occup. Environ. Med.* 47:1250-1259.
- Wolff, GT. (1993). Letter to EPA Administrator Carol Browner: "CASAC Closure on the Air Quality Criteria Document for Oxides of Nitrogen." EPA-SAB-CASAC-LTR-93-015, September 30.
- Wolff, GT. (1995). Letter to EPA Administrator Carol Browner: "CASAC Review of the Staff Paper for the Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information." EPA-SAB-CASAC-LTR-95-004, August 22.
- Yao, X, Lau NT, Chan CK, Fang M. (2005). The use of tunnel concentration profile data to determine the ratio of NO<sub>2</sub>/NO<sub>x</sub> directly emitted from vehicles. *Atmos Chem Phys Discuss.* 5:12723–12740. Available at: <http://www.atmos-chem-phys-discuss.net/5/12723/2005/acpd-5-12723-2005.pdf>.
- Zhu Y, Hinds WC, Kim S, Shen S, Sioutas C. (2002). Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ.* 36:4323-4335.

---

United States  
Environmental Protection  
Agency

Office of Air Quality Planning and Standards  
Health and Environmental Impacts Division  
Research Triangle Park, NC

EPA-452/R-08-008a  
November 2008

---