Appendix 4a - NO_2 Benefits Methodology

4a.1 Introduction

This appendix documents the methodology for estimating and monetizing the health benefits expected from reducing exposure to NO_2. In addition, this appendix includes a brief discussion regarding the key findings from the NO_2 benefits analysis as well as the limitations and areas of uncertainty in our approach. Although this approach was incorporated into the NO_2 NAAQS proposal RIA for the area-wide analysis (U.S. EPA, 2009), this approach was not deemed appropriate for estimating NO_2 exposure at near roadway monitors that do not yet exist. [1] Therefore, this appendix documents a methodological approach for estimating direct NO_2 benefits, and we do not include these results in the NO_2 NAAQS final RIA.

4a.2 Primary Benefits Approach

This section presents our approach for estimating avoided adverse health effects due to NO_2 exposure in humans resulting from achieving alternative scenarios, relative to a baseline concentration of ambient NO_2. First, we summarize the scientific evidence concerning potential health effects of NO_2 exposure, and then we present the health endpoints we selected for our primary benefits estimate. Next, we describe our benefits model, including the key input data and assumptions. Finally, we describe our approach for assigning an economic value to the NO_2 health benefits.

Benefits Scenario

We estimated the economic benefits from annual avoided health effects expected to result from achieving alternative scenarios (the "control scenarios"). We estimated benefits in the control scenarios relative to the incidence of health effects consistent with the ambient NO_2 concentration expected (the "baseline"). Note that this "baseline" reflects emissions reductions and ambient air quality improvements that we anticipate will result from implementation of other air quality rules, including compliance with all relevant rules already promulgated

We compared benefits across three alternative scenarios. Consistent with EPA's approach for RIA benefits assessments, we estimated the health effects associated with an

[1] $PM_{2.5}$ co-benefits of reducing NO_2 emissions to meet alternate standard levels are quantified and monetized in Chapter 4 of this RIA.

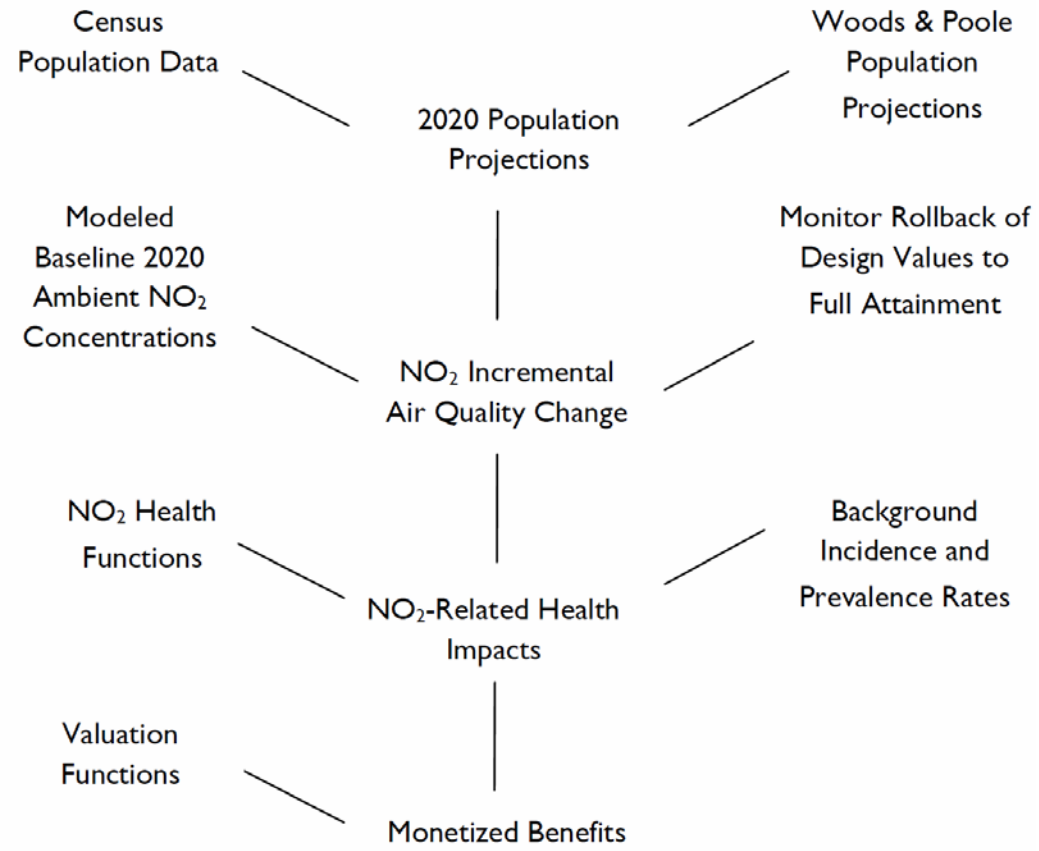
incremental difference in ambient concentrations between a baseline scenario and a pollution control strategy.

4a.3 Overview of analytical framework for benefits analysis

Benefits Model

For the primary benefits analysis, we use the Environmental Benefits Mapping and Analysis Program (BenMAP) to estimate the health benefits occurring as a result of implementing alternative NO_2 NAAQS levels. Although BenMAP has been used extensively in previous RIAs to estimate the health benefits of reducing exposure to $PM_{2.5}$ and ozone, this is the first RIA to use BenMAP to estimate the health benefits of reducing exposure to NO_2. Figure 4a-1 shows the major components of and inputs to the BenMAP model.

Figure 4a-1: Diagram of Inputs to BenMAP model for NO_2 Analysis

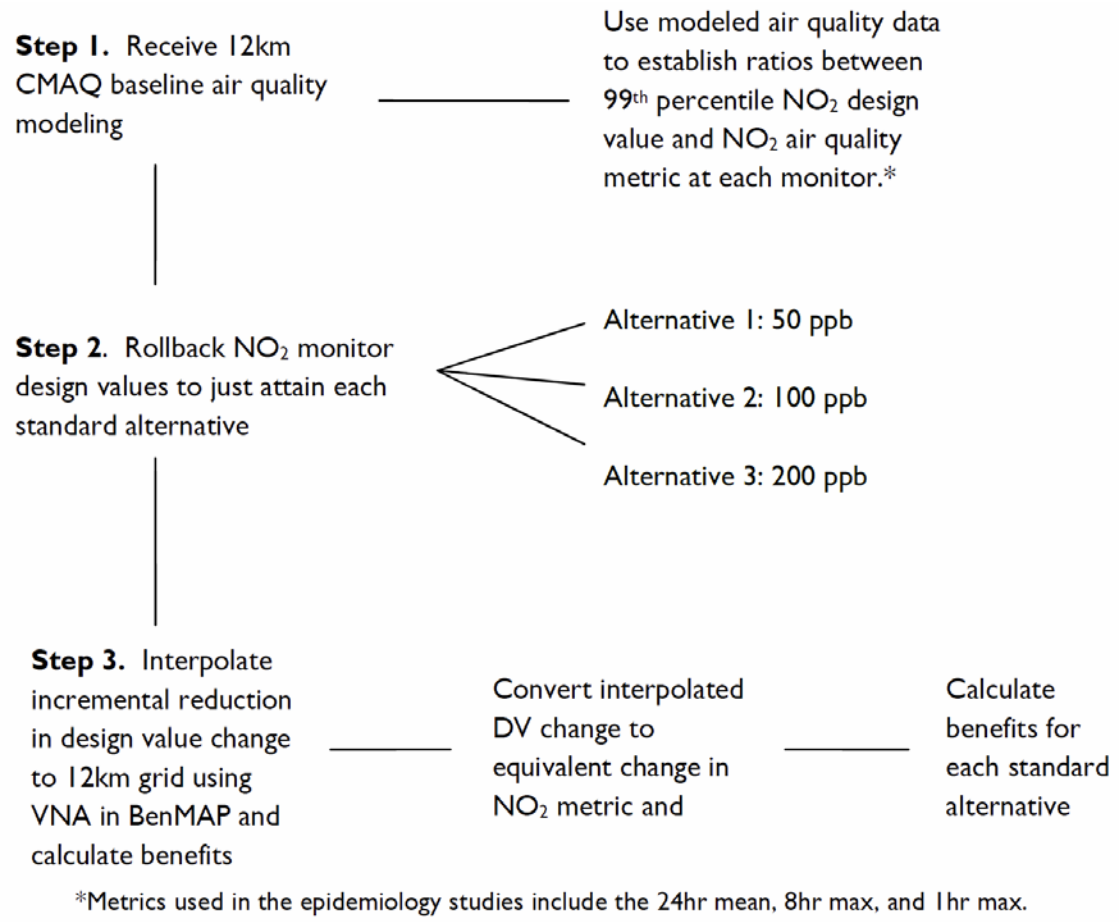


Air Quality Estimates

As shown in Figure 4a-1, the primary input to any benefits assessment is the estimated changes in ambient air quality expected to result from a simulated control strategy or attainment of a particular standard.

The CMAQ air quality model provides projects both design values at NO_2 monitors and air quality concentrations at 12km grid cells. To estimate the benefits of fully attaining the standards in all areas, EPA employed the "monitor rollback" approach to approximate the air quality change resulting from just attaining alternative scenarios at each design value monitor. Figure 4a-2 depicts the steps in the rollback process. The approach described here aims to estimate the change in population exposure associated with attaining an alternate NAAQS. This approach relies on data from the existing NO_2 monitoring network and the inverse distance squared variant of the Veronoi Neighborhood Averaging (VNA) interpolation method to adjust the CMAQ-modeled NO_2 concentrations such that each area just attains each alternative scenario. We believe that the interpolation method using inverse distance squared most appropriately reflects the steep exposure gradient for NO_2 around each monitor (see: EPA, 2008b). A sensitivity analysis for the NO_2 NAAQS proposal RIA (U.S. EPA, 2009) showed that the results are not very sensitive to the interpolation method.

Figure 4a-2: Diagram of Rollback Method



Because the VNA rollback approach interpolates monitor values, it is most reliable in areas with a denser monitoring network. In areas with a sparser monitoring network, there is less observed monitoring data to support the VNA interpolation and we have less confidence in the predicted air quality values further away from the monitors. For this reason, we interpolated air quality values—and estimated health impacts—within the CMAQ grid cells that are located within 30 km of the monitor, assuming that emission changes within this radius would affect the NO_2 concentration at each monitor. Limiting the interpolation to this radius attempts to account for the limitations of the VNA approach and ensures that the benefits and costs analyses consider a consistent geographic area.[2] Therefore, the primary benefits analysis assesses health impacts occurring to populations living in the CMAQ grid cells located within the 30km buffer for the specific geographic areas assumed to not attain the alternate standard levels.

[2] Please see Chapter 3 for more information regarding the technical basis for the 30 km assumption.

4a.4 Estimating Avoided Health Effects from NO_2 Exposure

Selection of Health Endpoints for NO_2

Epidemiological researchers have associated NO_2 exposure with adverse health effects in numerous toxicological, clinical and epidemiological studies, as described in the Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Final Report) (U.S. EPA, 2008a; hereafter, "NO_2 ISA"). The NO_2 ISA provides a comprehensive review of the current evidence of health and environmental effects of NO_2. The Risk and Exposure Assessment for NO_2 summarizes the NO_2 ISA conclusions regarding health effects from NO_2 exposure as follows (U.S. EPA, 2008b; Section 4.2.1):

> "The ISA concludes that, taken together, recent studies provide scientific evidence that is sufficient to infer a likely causal relationship between short-term NO_2 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 [Emergency Department (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% increase in risks for ED visits and hospital admissions and higher risks for respiratory symptoms (ISA, section 5.4)."

Previous reviews of the NO_2 primary NAAQS, completed in 1985 and 1996, did not include a quantitative benefits assessment for NO_2 exposure. As the first health benefits assessment for NO_2 exposure, we build on the methodology and lessons learned from the NO_2 risk and exposure assessment (U.S. EPA, 2008b) and the benefits assessments for the recent $PM_{2.5}$ and O_3 NAAQS (U.S. EPA, 2006a; U.S. EPA, 2008a).

We selected the health endpoints to be consistent with the conclusions of the NO_2 ISA. In general, we follow a weight of evidence approach, based on the biological plausibility of effects, availability of concentration-response functions from well conducted peer-reviewed epidemiological studies, cohesiveness of results across studies, and a focus on endpoints reflecting public health impacts (like hospital admissions) rather than physiological responses (such as changes in clinical measures like Forced Expiratory Volume (FEV1)). The differing evidence and associated strength of the evidence for these different effects is described in detail in the NO_2 ISA.

Although a number of adverse health effects have been found to be associated with NO_2 exposure, this benefits analysis only includes a subset due to limitations in understanding and quantifying the dose-response relationship for some of these health endpoints. In this analysis, we only estimated the benefits for those endpoints with sufficient evidence to support a quantified concentration-response relationship using the information presented in the NO_2 ISA, which contains an extensive literature review for several health endpoints related to NO_2 exposure. Because the ISA only included studies published or accepted for publication through December 2007, we also performed supplemental literature searches in the online search engine PubMed® to identify relevant studies published between January 2008, and the present.[3] Based on our review of this information, we quantified four short-term morbidity endpoints that the NO_2 ISA identified as "sufficient to infer a likely causal relationship": asthma exacerbation, respiratory-related emergency department visits, and respiratory-related hospitalizations.

Table 4a-1 presents the health effects related to NO_2 exposure quantified in this benefits analysis. In addition, the table includes other endpoints potentially linked to NO_2 exposure, but which we are not yet ready to quantify with concentration-response functions.

The NO_2 ISA concluded that the relationship between short-term NO_2 exposure and premature mortality was "suggestive but not sufficient to infer a causal relationship" because it is difficult to attribute the mortality risk effects to NO_2 alone. Therefore, we decided not to quantify premature mortality from NO_2 exposure in this analysis despite evidence suggesting a positive association (U.S. EPA, 2008a, Section 3.3.2). Although the NO_2 ISA stated that studies consistently reported a relationship between NO_2 exposure and mortality, the effect was generally smaller than that for other pollutants such as PM. We may revisit this decision in future benefits assessment for NO_2.

[3] The O'Conner et al. study (2008) is the only study included in this analysis that was published after the cut-off date for inclusion in the NO_2 ISA.

Table 4a-1: Human Health and Welfare Effects of NO_2

Pollutant / Effect	Quantified and Monetized in Primary Estimates [a]	Unquantified Effects [b, c] Changes in:
NO_2/Health	Asthma Hospital Admissions	Premature mortality
	Chronic Lung Disease Hospital Admissions	Pulmonary function
		Other respiratory emergency department visits
	Asthma ER visits	Other respiratory hospital admissions
	Asthma exacerbation	
	Acute Respiratory symptoms	
NO_2/Welfare		Visibility
		Commercial fishing and forestry from acidic deposition
		Recreation in terrestrial and aquatic ecosystems from acid deposition
		Commercial fishing, agriculture, and forestry from nutrient deposition
		Recreation in terrestrial and estuarine ecosystems from nutrient deposition
		Other ecosystem services and existence values for currently healthy ecosystems

[a] Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the alternative standards.
[b] The categorization of unquantified toxic health and welfare effects is not exhaustive.
[c] Health endpoints in the unquantified benefits column include both a) those for which there is not consensus on causality and those for which causality has been determined but empirical data are not available to allow calculation of benefits.

Selection of Concentration-Response Functions

After identifying the health endpoints to quantify in this analysis, we then selected concentration-response functions drawn from the epidemiological literature identified in the NO_2 ISA. We considered several factors in selecting the appropriate epidemiological studies and concentration-response functions for this benefits assessment.

- First, we considered ambient NO_2 studies that were identified as key studies in the NO_2 ISA (or a more recent study), excluding those affected by the general additive model (GAM) S-Plus issue.[4]
- Second, we 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 affected by factors such as the ambient pollutant mix, the

[4] The S-Plus statistical software is widely used for nonlinear regression analysis in time-series research of health effects. However, in 2002, a problem was discovered with the software's default conversion criteria in the general additive model (GAM), which resulted in biased relative risk estimates in many studies. This analysis does not include any studies that encountered this problem. For more information on this issue, please see U.S. EPA (2002).

placement of monitors, activity patterns of the population, and characteristics of the healthcare system especially for hospital admissions and emergency department visits. We include Canadian studies in sensitivity analyses, when available.

- Third, we only incorporated concentration-response functions for which there was a corresponding valuation function. Currently, we only have a valuation function for asthma-related emergency department visits, but we do not have a valuation function for all-respiratory-related emergency department visits.
- Fourth, we preferred concentration-response functions that correspond to the age ranges most relevant to the specific health endpoint, with non-overlapping ICD-9 codes. We preferred completeness when selecting functions that correspond to particular age ranges and ICD codes. Age ranges and ICD codes associated with the selected functions are identified in Table 4a.2.
- Fifth, we preferred multi-city studies or combined multiple single city studies, when available.
- Sixth, when available, we judged that effect estimates with distributed or cumulative lag structures were most appropriate for this analysis.
- Seventh, when available, we selected NO_2 concentration-response functions based on multi-pollutant models. Studies with multi-pollutant models are identified in Table 4a.2.

These criteria reflect our preferences for study selection, and it was possible to satisfy many of these, but not all. There are trade-offs inherent in selecting among a range of studies, as not all studies met all criteria outlined above. At minimum, we ensured that none of the studies were GAM affected, we selected only U.S. based studies, and we quantified health endpoints for which there was a corresponding valuation function.

We believe that U.S.-based studies are most appropriate studies to use in this analysis to estimate the number of hospital admissions associated with NO_2 exposure because of the characteristics of the ambient air, population, and healthcare system. Using only U.S.-based studies, we are limited to estimating the hospital admissions for asthma (ICD-9 493) and chronic lung disease (ICD-9 490-496) rather than all respiratory-related hospital admission, which is a more complete measure of health impacts. However, there are several Canada-based epidemiology studies that provide a more complete estimate of respiratory hospital admissions (Fung, 2006; Luginaah, 2005; Yang, 2003). Compared to the U.S. based studies, the Canadian studies produce a larger estimate of hospital admissions associated with NO_2 exposure.

When selecting concentration-response functions to use in this analysis, we reviewed the scientific evidence regarding the presence of thresholds in the concentration-response functions for NO_2-related health effects to determine whether the function is approximately linear across the relevant concentration range. The NO_2 ISA concluded that, "[t]hese results do not provide adequate evidence to suggest that nonlinear departures exist along any part of this range of NO_2 exposure concentrations." Therefore, we have not incorporated thresholds in the concentration-response function for NO_2-related health effects in this analysis.

Table 4a-2 shows the studies and health endpoints that we selected for this analysis. Table 4a-3 shows the baseline health data used in combination with these health functions. Following these tables is a description of each of the epidemiology studies used in this analysis.

Table 4a-2: NO_2-Related Health Endpoints Quantified, Studies Used to Develop Health Impact Functions and Sub-Populations to which They Apply

Endpoint	Study	Study Population
Hospital Admissions [b]		
Asthma	Linn et al. (2000)—ICD-9 493	All ages
Chronic Lung Disease	Moolgavkar (2003) —ICD-9 490-496	> 65
Emergency Department Visits		
Asthma	Pooled Estimate: Ito et al. (2007)—ICD-9 493 NYDOH (2006)[c]—ICD-9 493 Peel et al. (2005)—ICD-9 493	All ages
Other Health Endpoints		
Asthma exacerbations	Pooled estimate: O'Connor et al. (2008) (slow play, missed school days, nighttime asthma) [c] Ostro et al. (2001) (cough, cough (new cases), shortness of breath, shortness of breath (new cases), wheeze, wheeze (new cases) [a] Schildcrout et al. (2006) (one or more symptoms)	4 - 12
	Delfino et al. (2002) (one or more symptoms)	13 - 18[a]
Acute Respiratory Symptoms	Schwartz et al. (1994)[c]	7 - 14

[a] The original study populations were 9 to 18 for the Delfino et al. (2002) study, and 8-13 for the Ostro et al. (2001) study. We extended the applied population to facilitate the pooling process, recognizing the common biological basis for the effect in children in the broader age group. See: National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press, pg 117.

[b] We recognize that the ICD codes for asthma and chronic lung disease overlap partially, suggesting that our combined estimate of respiratory hospital admissions may be overstated to a small degree. However, we believe that using the other available health impact functions to quantify this endpoint would have resulted in a more biased and uncertain estimate, as these functions failed to meet key selection criteria.

[C] Study specifies a multipollutant model

Table 4a-3: National Average Baseline Incidence Rates used to Calculate NO_2-Related Health Impacts [a]

Endpoint	Source	Notes	Rate per 100 people per year by Age Group						
			<18	18–24	25–34	35–44	45–54	55–64	65+
Respiratory Hospital Admissions	1999 NHDS public use data files [b]	incidence	0.043	0.084	0.206	0.678	1.926	4.389	11.629
Asthma ER visits	2000 NHAMCS public use data files [c]; 1999 NHDS public use data files [b]	incidence	1.011	1.087	0.751	0.438	0.352	0.425	0.232
Minor Restricted Activity Days (MRADs)	Schwartz (1994, table 2)	incidence	0.416	—	—	—	—	—	—

Endpoint	Source	Notes		
Asthma Exacerbations	Delfino et al. (2002)	Incidence (and prevalence) among asthmatic children	Asthma symptoms	0.157 (0.0567)
	O'Connor et al. (2008)	Incidence (and prevalence) among asthmatic children	Missed school One or more symptoms Slow play Nighttime asthma	0.057 (0.0567) 0.207 (0.0567) 0.157 (0.0567) 0.121 (0.0567)
	Ostro et al. (2001)	Incidence (and prevalence) among asthmatic African American children	Cough Cough (new cases) Shortness of breath Shortness of breath (new cases) Wheeze Wheeze (new cases)	0.145 (0.0726) 0.067 (0.0726) 0.074 (0. 0726) 0.037 (0. 0726) 0.173 (0.0726) 0.076 (0.0726)
	Schildcrout et al. (2006)	Incidence (and prevalence) among asthmatic children	One or more symptoms	0.52 (0.0567)

[a] The following abbreviations are used to describe the national surveys conducted by the National Center for Health Statistics: HIS refers to the National Health Interview Survey; NHDS—National Hospital Discharge Survey; NHAMCS—National Hospital Ambulatory Medical Care Survey.
[b] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHDS/
[c] See ftp://ftp.cdc.gov/pub/Health_Statistics/NCHS/Datasets/NHAMCS/

Linn et al. (2000)

Linn et al. (2000) evaluated associations between air pollution and hospital admissions for cardiopulmonary illnesses in metropolitan Los Angeles during 1992-1995. In a single-pollutant Poisson regression model, daily average of NO_2 (year-round) was found significantly

associated with same-day asthma hospital admissions for both age groups (i.e., 0-29 and 30-99). The results for winter and autumn were also reported but insignificant.

Moolgavkar (2003)

Moolgavkar (2003) presented re-analyses of Moolgavkar(2000a; 2000b; 2000c) of the associations between air pollution and daily deaths and hospital admissions in Los Angeles and Cook counties in the United States.[5] The author also reported the results of generalized linear model (GLM) analyses using natural splines with the same degree of freedom as the smoothing splines he used in the generalized additive model (GAM) analyses. In single-pollutant Poisson regression models, hospital admissions for chronic obstructive disorder (COPD) (ICD-9 code 490-496) were associated with daily average of NO_2 levels at lags of 0, 1, 2, 3, 4 and 5 days for individuals 65 and older. The association was strongest at lag 0 using both GAM (stringent convergence) and GLM.

Ito et al. (2007)

Ito et al. (2007) assessed associations between air pollution and asthma emergency department visits in New York City for all ages. Specifically they examined the temporal relationships among air pollution and weather variables in the context of air pollution health effects models. The authors compiled daily data for $PM_{2.5,}$ O_3, NO_2, SO_2, CO, temperature, dew point, relative humidity, wind speed, and barometric pressure for New York City for the years 1999-2002.The authors evaluated the relationship between the various pollutants' risk estimates and their respective concurvities, and discuss the limitations that the results imply about the interpretability of multi-pollutant health effects models.

NYDOH (2006)

New York State Department of Health (NYDOH) investigated whether day-to-day variations in air pollution were associated with asthma emergency department (ED) visits in Manhattan and Bronx, NYC and compared the magnitude of the air pollution effect between the two communities. NYDOH (2006) used Poisson regression to test for effects of 14 key air contaminants on daily ED visits, with control for temporal cycles, temperature, and day-of-week effects. The core analysis utilized the average exposure for the zero- to four-day lags. Mean daily NO_2 was found significantly associated with asthma ED visits in Bronx but not Manhattan. Their findings of more significant air pollution effects in the Bronx are likely to relate in part to

[5] The principal reason for conducting these re-analyses was to assess the impact of using convergence criteria that are more stringent than the default criteria used in the S-Plus software package.

greater statistical power for identifying effects in the Bronx where baseline ED visits were greater, but they may also reflect greater sensitivity to air pollution effects in the Bronx.

Peel et al. (2005)

Peel et al. (2005) examined the associations between air pollution and respiratory emergency department visits (i.e., asthma (ICD-9 code 493, 786.09), COPD (491,492,496), upper respiratory infection (URI) (460-466, 477), pneumonia (480-486), and an all respiratory-disease group) in Atlanta, GA from 1 January 1993 to 31 August 2000. They used 3-Day Moving Average (Lags of 0, 1, and 2 Days) and unconstrained distributed lag (Lags of 0 to 13 Days) in the Poisson regression analyses. In single-pollutant models, the authors found that positive associations persisted beyond 3 days for several outcomes, and over a week for asthma. Standard deviation increases of O_3, NO_2, CO, and PM_{10} were associated with 1-3% increases in URI visits; a 2 $\mu g/m^3$ increase of $PM_{2.5}$ organic carbon was associated with a 3% increase in pneumonia visits; and standard deviation increases of NO_2 and CO were associated with 2-3% increases in chronic obstructive pulmonary disease visits.

Delfino et al. (2002)

Delfino et al. (2002) examined the association between air pollution and asthma symptoms among 22 asthmatic children (9-19 years of age) followed March through April 1996 (1,248 person-days) in Southern California. Air quality data for PM_{10}, NO_2, O_3, fungi and pollen were used in a logistic model with control for temperature, relative humidity, day-of-week trends and linear time trends. The odds ratio (95% confidence interval) for asthma episodes in relation to lag0 (i.e. immediate) 20 ppb changes in 8-hr max NO_2 is 1.49 (0.95-2.33). The authors also considered subgroups of asthmatic children who were on versus not on regularly scheduled anti-inflammatory medications and found that pollutant associations were stronger during respiratory infections in subjects not on anti-inflammatory medications.

O'Connor et al. (2008)

O'Connor et al. (2008) investigated the association between fluctuations in outdoor air pollution and asthma exacerbation among 861 inner-city children (5-12 years of age) with asthma in seven US urban communities. Asthma symptom data were collected every two months during the 2-year study period. Daily pollution measurements were obtained from the Aerometric Information Retrieval System between August 1998 and July 2001. The relationship of symptoms to fluctuations in pollutant concentrations was examined by using logistic models. In single-pollutant models, significant or nearly significant positive associations were observed between higher NO_2 concentrations and each of the health outcomes. Significant positive

associations with symptoms but not school absence were observed in the single-pollutant model for CO. The O_3, $PM_{2.5,}$ and SO_2 concentrations did not appear significantly associated with symptoms or school absence except for a significant association between $PM_{2.5}$ and school absence. The authors concluded that the associations with NO_2 suggest that motor vehicle emissions may be causing excess morbidity in this population. This study is not included in the NO_2 ISA only because it was published after the cut-off date, but it met all of the other criteria for inclusion in this analysis.

Ostro et al. (2001)

Ostro et al. (2001) examined relations between several air pollutants and asthma exacerbation in African-Americans children (8 to 13 years old) in central Los Angeles from August to November 1993. Air quality data for PM_{10}, $PM_{2.5,}$ NO_2, and O_3 were used in a logistic regression model with control for age, income, time trends, and temperature-related weather effects. Asthma symptom endpoints were defined in two ways: "probability of a day with symptoms" and "onset of symptom episodes". New onset of a symptom episode was defined as a day without symptoms followed by a day with symptoms. The authors found cough prevalence associated with PM_{10} and $PM_{2.5}$ and cough incidence associated with $PM_{2.5,}$ PM_{10}, and NO_2. Ozone was not significantly associated with cough among asthmatics. The authors found that both the prevalent and incident episodes of shortness of breath were associated with $PM_{2.5}$ and PM_{10}. Neither ozone nor NO_2 were significantly associated with shortness of breath among asthmatics. The authors found both the prevalence and incidence of wheeze associated with $PM_{2.5,}$ PM_{10}, and NO_2. Ozone was not significantly associated with wheeze among asthmatics.

Schildcrout et al. (2006)

Schildcrout et al. (2006) investigated the relation between ambient concentrations of the five criteria pollutants (PM_{10}, O_3, NO_2, SO_2, and CO) and asthma exacerbations (daily symptoms and use of rescue inhalers) among 990 children in eight North American cities during the 22-month prerandomization phase (November 1993-September 1995) of the Childhood Asthma Management Program. Short-term effects of CO, NO_2, PM_{10}, SO_2, and warm-season O_3 were examined in both one-pollutant and two-pollutant models, using lags of up to 2 days in logistic and Poisson regressions. Lags in CO and NO_2 were positively associated with both measures of asthma exacerbation, and the 3-day moving sum of SO_2 levels was marginally related to asthma symptoms. PM_{10} and O_3 were unrelated to exacerbations. The strongest effects tended to be seen with 2-day lags, where a 1-parts-per-million change in CO and a 20-parts-per-billion change in NO_2 were associated with symptom odds ratios of 1.08 (95% confidence interval (CI): 1.02, 1.15) and 1.09 (95% CI: 1.03, 1.15), respectively.

Schwartz et al. (1994)

Schwartz et al. (1994) studied the association between ambient air pollution exposures and respiratory illness among 1,844 schoolchildren (7-14 years of age) in six U.S. cities during five warm season months between April and August. Daily measurements of ambient SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$, light scattering, and sulfate particles were made, along with integrated 24-h measures of aerosol strong acidity. Significant associations in single pollutant models were found between SO_2, NO_2, or $PM_{2.5}$ and incidence of cough, and between sulfur dioxide and incidence of lower respiratory symptoms. Significant associations were also found between incidence of coughing symptoms and incidence of lower respiratory symptoms and PM_{10}, and a marginally significant association between upper respiratory symptoms and PM_{10}.

Pooling Multiple Health Studies

After selecting which health endpoints to analyze and which epidemiology studies provide appropriate effect estimates, we then selected a method to combine the multiple health studies to provide a single benefits estimate for each health endpoint. The purpose of pooling multiple studies together is to generate a more robust estimate by combining the evidence across multiple studies and cities. Because we used a single study for acute respiratory symptoms and a single study for hospital admission for asthma, there was no pooling necessary for those endpoints.

For the hospital admission studies for chronic lung disease, we pooled the effect estimates reported for two counties (Los Angeles, CA, and Cook, IL) from Moolgavkar (2003) using random/fixed effects.[6] For the emergency department visit studies, we pooled the three studies (Ito et al., 2007; NYDOH, 2003; Peel et al., 2005) using random/fixed effects. For the asthma studies, we pooled the three studies (O'Conner et al, 2008; Ostro et al, 2001; Schildcrout et al, 2006) using random/fixed effects for ages 4 to 12, and then we summed this results with the Delfino study (2002) for ages 13 to 18. Because asthma represents the largest benefits category in this analysis, we tested the sensitivity of the NO_2 benefits to alternate pooling choices. In general, the estimate using the Ostro study is much lower than the estimate that combines Ostro with the new studies, and the estimate for one-or-more asthma symptoms is much higher than the estimate that combines all of the asthma endpoints.

[6] Random/fixed effects pooling allows for the possibility that the effect estimates reported among different studies may in fact be estimates of different parameters, rather than just different estimates of the same underlying parameter. For additional information regarding BenMAP pooling techniques, please consult the BenMAP technical appendices available at http://www.epa.gov/air/benmap/models/BenMAPappendicesSept08.pdf .

4a.5 Valuation of Avoided Health Effects from NO_2 Exposure

The selection of valuation functions is largely consistent with the $PM_{2.5}$ NAAQS (U.S. EPA, 2006a) with two exceptions. First, in this analysis, we only estimate chronic lung disease and asthma, two types of hospital admissions, whereas the $PM_{2.5}$ NAAQS estimated changes in all respiratory hospital admissions, which generated a larger monetized value. Second, we use the any-of-19 symptoms valuation for acute respiratory symptoms instead of the "minor-restricted activity day" (MRADs) estimated for the $PM_{2.5}$ NAAQS. The valuation for any-of-19-symptoms is approximately 50% of the valuation for MRADs. Consistent with economic theory, these valuation functions include adjustments for inflation (2006$) and income growth over time (2020 income levels). Table 4a-4 describes the valuation functions used to monetize the benefits of reduced exposure to NO_2.

Table 4a-4: Central Unit Values NO_2 Health Endpoints (2006$)*

Health Endpoint	Central Unit Value Per Statistical Incidence (2020 income level)	Derivation of Distributions of Estimates
Hospital Admissions and ER Visits		
Asthma Admissions	$10,000	No distributional information available. The cost-of-illness (COI) estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Chronic Lung Disease Admissions	$16,000	No distributions available. The COI point estimates (lost earnings plus direct medical costs) are based on ICD-9 code level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality, 2000 (www.ahrq.gov).
Asthma Emergency Room Visits	$370	No distributional information available. Simple average of two unit COI values: (1) $400 (2006$), from Smith et al. (1997) and (2) $340 (2006$), from Stanford et al. (1999).
Respiratory Ailments Not Requiring Hospitalization		
Asthma Exacerbation	$53	Asthma exacerbations are valued at $49 (2006$) per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986). This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma exacerbation is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. The value is assumed have a uniform distribution between $19 and $83 (2006$).
Acute Respiratory Symptoms	$30	The valuation estimate for "any of 19 acute respiratory symptoms" is derived from Krupnick et al. (1990) assuming that this health endpoint consists either of upper respiratory symptoms (URS) or lower respiratory symptoms (LRS), or both. We assumed the following probabilities for a day of "any of 19 acute respiratory symptoms": URS with 40 percent probability, LRS with 40 percent probability, and both with 20 percent probability. The point estimate of WTP to avoid a day of "the presence of any of 19 acute respiratory symptoms" is $28 (2006$). The value is assumed have a uniform distribution between $0 and $56 (2006$).

* All estimates rounded to two significant figures. All values have been inflated to reflect values in 2006 dollars and income levels in 2020.

4a.6 Limitations and Uncertainty

Our approach incorporates methods to assess two aspects of uncertainty quantitatively: Monte Carlo analysis and sensitivity analysis. We also provide a qualitative assessment for those aspects that we are unable to address quantitatively in this analysis. Each of these analyses is described in detail in the following sections.

This analysis includes many data sources as inputs, including emission inventories, air quality data from models (with their associated parameters and inputs), population data, health effect estimates from epidemiology studies, and economic data for monetizing benefits. Each of these inputs may be uncertain and would affect the benefits estimate. When the uncertainties from each stage of the analysis are compounded, small uncertainties can have large effects on the total quantified benefits. In this analysis, we are unable to quantify the cumulative effect of all of these uncertainties, but we provide the following analyses to characterize many of the largest sources of uncertainty.

Monte Carlo analysis

Similar to other recent RIAs, we used Monte Carlo methods for estimating characterizing random sampling error associated with the concentration response functions and economic valuation functions. Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables, such as incidence of morbidity. Specifically, we used Monte Carlo methods to generate confidence intervals around the estimated health impact and dollar benefits. In Table 4a-5, we present the results of this Monte Carlo analysis conducted in the area-wide analysis for the NO_2 NAAQS proposal RIA as an illustrative example of the random sampling error and 95th percentile confidence intervals.

Table 4a-5: NO_2 Benefits of Attaining 50 ppb Standard (95th percentile confidence interval) [a]

		Incidence		Valuation	
Total	Asthma Exacerbation	87,000	(250 -- 220,000)	$4,700,000	($240,000 -- $13,000,000)
	Hospital Admissions, Chronic Lung Disease	28	(23 -- 35)	$490,000	($400,000 -- $560,000)
	Hospital Admissions, Asthma	27	(11 -- 50)	$300,000	($130,000 -- $460,000)
	Emergency Room Visits, Respiratory	160	(32 -- 330)	$61,000	($14,000 -- $110,000)
	Acute Respiratory Symptoms	27,000	(-7,900 -- 75,000)	$820,000	(-$220,000 -- $2,700,000)
Grand Total				**$6,300,000**	**($570,000 -- $16,000,000)**

[a] This table shows the results of the Monte Carlo analysis conducted for the area-wide analysis in the NO_2 NAAQS proposal RIA as an illustrative example of the sensitivity of the random sampling error and 95th percentile confidence intervals.

Sensitivity analyses

We performed a variety of sensitivity analyses on the benefits results to assess the sensitivity of the primary results to various data inputs and assumptions. We then changed each default input one at a time and recalculated the total monetized benefits to assess the percent change from the default. In Table 4a-6, we present the results of this sensitivity analysis conducted in the area-wide analysis for the NO_2 NAAQS proposal RIA as an illustrative example of the sensitivity of various parameters. We indicate each input parameter, the value used as the default, and the values for the sensitivity analyses, and then we provide the total monetary benefits for each input and the percent change from the default value. Descriptions of the sensitivity analyses are provided in the relevant sections of this appendix.

Table 4a-6: Sensitivity Analyses for NO_2 Health Benefits to Fully Attain the 50 ppb Standard (Area-wide analysis) [a]

		Total NO_2 Benefits (millions of 2006$)	% Change from Default
Exposure Estimation Method	30km radius	$6.3	N/A
	12km grid cell	$1.4	-77%
	15km radius	$5.1	-19%
	CBSA	$6.3	0.6%
	Unconstrained	$8.9	42%
Location of Hospital Admission Studies	w/US-based studies only	$6.3	N/A
	w/Canada-based studies only[b]	$11	79%
Simulated Attainment	Just attainment	$6.3	N/A
	Over-control attainment	$6.8	10%
	Partial Attainment (El Paso)	$5.8	-6.2%
	Partial Attainment (El Paso and Los Angeles)	$4.6	-27%
Asthma Pooling Method	Pool all endpoints together	$6.3	N/A
	Ostro et al only	$2.1	-66%
	One or more symptoms only	$6.9	11%
Interpolation Method	Inverse Distance Squared	$6.3	N/A
	Inverse Distance	$5.8	-6.2%

[a] This table shows the results of the sensitivity analysis conducted for the area-wide analysis in the NO_2 NAAQS proposal RIA as an illustrative example of the sensitivity of various parameters of this methodology.
[b] Using Canadian studies is not a direct comparison because it includes a more complete endpoint (all respiratory hospital admissions, ages 65+), whereas the US-based studies only include hospital admissions for asthma (all ages) and chronic lung disease (ages 65+).

Qualitative assessment of uncertainty and other analysis limitations

Although we strive to incorporate as many quantitative assessments of uncertainty, there are several aspects for which we are only able to address qualitatively. These aspects are important factors to consider when evaluating the relative benefits of the attainment strategies for each of the alternative standards:

1. The gradient of ambient NO_2 concentrations is difficult to estimate due to the sparsity of the monitoring network. The 12km CMAQ grid, which is the air quality modeling resolution, may be too coarse to accurately estimate the potential near-field health benefits of reducing NO_2 emissions. These uncertainties may under- or over-estimate benefits.
2. The interpolation techniques used to estimate the full attainment benefits of the alternative standards contributed some uncertainty to the analysis. The great majority of benefits estimated for the most stringent standard alternative were derived through interpolation. As noted previously in this appendix, these benefits are likely to be more uncertain than if we had modeled the air quality scenario for both NO_2 and $PM_{2.5}$. In general, the VNA interpolation approach will under-estimate benefits because it does not account for the broader spatial distribution of air quality changes that may occur due to the implementation of a regional emission control program.
3. There are many uncertainties associated with the health impact functions used in this modeling effort. These include: within study variability (the precision with which a given study estimates the relationship between air quality changes and health effects); across study variation (different published studies of the same pollutant/health effect relationship typically do not report identical findings and in some instances the differences are substantial); the application of C-R functions nationwide (does not account for any relationship between region and health effect, to the extent that such a relationship exists); the possibility of exposure misclassification in the study due to unmeasured variability in NO_2 concentrations near roadways; extrapolation of impact functions across population (we assumed that certain health impact functions applied to age ranges broader than that considered in the original epidemiological study); and various uncertainties in the C-R function, including causality and thresholds. These uncertainties may under- or over-estimate benefits.
4. Co-pollutants present in the ambient air may have contributed to the health effects attributed to NO_2 in single pollutant models. Risks attributed to NO_2 might be overestimated where concentration-response functions are based on single pollutant models. If co-pollutants are highly correlated with NO_2, their inclusion in an NO_2 health effects model can lead to misleading conclusions in identifying a specific causal

pollutant. Because this collinearity exists, many of the studies reported statistically insignificant effect estimates for both NO_2 and the co-pollutants; this is due in part to the loss of statistical power as these models control for co-pollutants. Where available, we have selected multipollutant effect estimates to control for the potential confounding effects of co-pollutants; these include NYDOH (2006), Schwartz et al. (1994) and O'Conner et al. (2007). The remaining studies include single pollutant models.

5. This analysis is for the year 2020, and projecting key variables introduces uncertainty. Inherent in any analysis of future regulatory programs are uncertainties in projecting atmospheric conditions and source level emissions, as well as population, health baselines, incomes, technology, and other factors.
6. This analysis omits certain unquantified effects due to lack of data, time and resources. These unquantified endpoints include other health effects, ecosystem effects, and visibility. EPA will continue to evaluate new methods and models and select those most appropriate for estimating the benefits of reductions in air pollution. Enhanced collaboration between air quality modelers, epidemiologists, toxicologists, ecologists, and economists should result in a more tightly integrated analytical framework for measuring benefits of air pollution policies.

4a.7 Discussion

The benefits methodology described in this appendix suggests that reducing NO_2 emissions would produce substantial health benefits in the form of fewer respiratory hospitalizations, respiratory emergency department visits and cases of acute respiratory symptoms from reduced NO_2 exposure.

This methodology is the first time that EPA has estimated the monetized human health benefits of reducing exposure to NO_2 to support a proposed change in the NAAQS. In contrast to recent $PM_{2.5}$ and ozone-related benefits assessments, there was far less analytical precedent on which to base this assessment. For this reason, we developed entirely new components of the health impact analysis, including the identification of health endpoints to be quantified and the selection of relevant effect estimates within the epidemiology literature. As the NO_2 health literature continues to evolve, EPA will reassess the health endpoints and risk estimates used in this analysis.

While monetized NO_2 benefits may appear small when compared to recent analyses for $PM_{2.5}$ benefits or ozone benefits, readers should not necessarily infer that the total monetized benefits of NO_2 emission reductions are small. The methodology described in this appendix

only captures NO_2 health benefits, not the significant monetized co-benefits from reductions in $PM_{2.5}$ or ozone. Further, the size of the benefits is related to three principle factors. As demonstrated in previous RIAs, the magnitude and geographic extent of emission reductions in the control strategy necessary to bring an area into attainment are well correlated with the size of the monetized health benefits of that standard. Second, the size of monetized benefits is correlated with both the severity of those health effects correlated of NO_2 exposure. Third, the monetized benefits are in part a function of the health endpoints quantified in the analysis. Compared to the $PM_{2.5}$ co-benefits, the benefits from reduced NO_2 exposure appear small. This is primary due to the decision not to quantify NO_2-related premature mortality and other morbidity endpoints due to the uncertainties associated with estimating this endpoint. Because premature mortality generally comprises over 90% of the total monetized benefits, this decision may underestimate the monetized health benefits of reduced NO_2 exposure. Studies have shown that there is a relationship between NO_2 exposure and premature mortality, but that relationship is generally weaker than the PM-mortality relationship and efforts to quantify that relationship have been hampered by confounding with other pollutants. For most scenarios, $PM_{2.5}$ co-benefits would represent over 95% of the total monetized benefits. This result is consistent with recent RIAs, where the $PM_{2.5}$ co-benefits represent a large proportion of total monetized benefits.

It is important to note that this analysis does not attempt to estimate the benefits in any area of the country other than those counties currently served by one of the 409 monitors in the current monitoring network. We recognize that once a network of near-roadway monitors is in place, more areas could exceed the new NO_2 NAAQS and require emission reductions. However for this analysis, we lack sufficient data to predict NO_2 exposure after implementation of a near-roadway monitoring network. Therefore, we are unable to estimate the NO_2 benefits of that scenario.

4a.8 References

American Lung Association. 2002. Trends in Asthma Morbidity and Mortality. American Lung Association, Best Practices and Program Services, Epidemiology and Statistics Unit. Available on the Internet at <http://www.lungusa.org/data/asthma/ASTHMAdt.pdf>.

Delfino, R. J., R. S. Zeiger, J. M. Seltzer, D. H. Street and C. E. McLaren. 2002. Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. Environ Health Perspect. Vol. 110 (10): A607-17.

Fung, K. Y., S. Khan, D. Krewski and Y. Chen. 2006. Association between air pollution and multiple respiratory hospitalizations among the elderly in Vancouver, Canada. Inhal Toxicol. Vol. 18 (13): 1005-11.

Ito, K., G. D. Thurston and R. A. Silverman. 2007. Characterization of $PM_{2.5,}$ gaseous pollutants, and meteorological interactions in the context of time-series health effects models. J Expo Sci Environ Epidemiol. Vol. 17 Suppl 2: S45-60.

Linn, W. S., Y. Szlachcic, H. Gong, Jr., P. L. Kinney and K. T. Berhane. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. Environ Health Perspect. Vol. 108 (5): 427-34.

Luginaah, I. N., K. Y. Fung, K. M. Gorey, G. Webster and C. Wills. 2005. Association of ambient air pollution with respiratory hospitalization in a government-designated "area of concern": the case of Windsor, Ontario. Environ Health Perspect. Vol. 113 (3): 290-6.

Moolgavkar, S. H. 2000a. Air Pollution and Daily Mortality in Three U.S. Counties. Environ Health Perspect. Vol. 108 (8): 777-784.

Moolgavkar, S. H. 2000b. Air Pollution and Hospital Admissions for Chronic Obstructive Pulmonary Disease in Three Metropolitan Areas in the United States. Inhalation Toxicology. Vol. 12 (Supplement 4): 75-90.

Moolgavkar, S. H. 2000c. Air pollution and hospital admissions for diseases of the circulatory system in three U.S. metropolitan areas. J Air Waste Manag Assoc. Vol. 50 (7): 1199-206.

Moolgavkar, S. H. 2003. Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties. In: Revised Analyses of Time-Series Studies of Air Pollution and Health. Health Effects Institute. Boston, MA. May.

Mortimer, K. M., L. M. Neas, D. W. Dockery, S. Redline and I. B. Tager. 2002. The effect of air pollution on inner-city children with asthma. Eur Respir J. Vol. 19 (4): 699-705.

National Research Council (NRC). 2002. Estimating the Public Health Benefits of Proposed Air Pollution Regulations. Washington, DC: The National Academies Press.

NYDOH. 2006. A Study of Ambient Air Contaminants and Asthma in New York City. New York State Department of Health Center for Environmental Health.

O'Connor, G. T., L. Neas, B. Vaughn, M. Kattan, H. Mitchell, E. F. Crain, R. Evans, 3rd, R. Gruchalla, W. Morgan, J. Stout, G. K. Adams and M. Lippmann. 2008. Acute respiratory health effects of air pollution on children with asthma in US inner cities. J Allergy Clin Immunol. Vol. 121 (5): 1133-1139 e1.

Ostro, B., M. Lipsett, J. Mann, H. Braxton-Owens and M. White. 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. Epidemiology. Vol. 12 (2): 200-8.

Peel, J. L., P. E. Tolbert, M. Klein, K. B. Metzger, W. D. Flanders, K. Todd, J. A. Mulholland, P. B. Ryan and H. Frumkin. 2005. Ambient air pollution and respiratory emergency department visits. Epidemiology. Vol. 16 (2): 164-74.

Schildcrout, J. S., L. Sheppard, T. Lumley, J. C. Slaughter, J. Q. Koenig and G. G. Shapiro. 2006. Ambient air pollution and asthma exacerbations in children: an eight-city analysis. Am J Epidemiol. Vol. 164 (6): 505-17.

Schwartz, J., D. W. Dockery, L. M. Neas, D. Wypij, J. H. Ware, J. D. Spengler, P. Koutrakis, F. E. Speizer and B. G. Ferris. 1994. Acute Effects of Summer Air Pollution on Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. Vol. 150 (5): 1234-1242.

Smith, D. H., D. C. Malone, K. A. Lawson, L. J. Okamoto, C. Battista and W. B. Saunders. 1997. A national estimate of the economic costs of asthma. Am J Respir Crit Care Med. Vol. 156 (3 Pt 1): 787-93.

Stanford, R., T. McLaughlin and L. J. Okamoto. 1999. The cost of asthma in the emergency department and hospital. Am J Respir Crit Care Med. Vol. 160 (1): 211-5.

U.S. Environmental Protection Agency (U.S. EPA). 2008a. Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Final Report). National Center for Environmental Assessment, Research Triangle Park, NC. July. Available on the Internet at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=194645>.

U.S. Environmental Protection Agency (U.S. EPA). 2008b Risk and Exposure Assessment to Support the Review of the NO_2 Primary National Ambient Air Quality Standard (Final Report). Office of Air Quality Planning and Standards, Research Triangle Park, NC.

November. Available on the Internet at <http://www.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf>.

U.S. Environmental Protection Agency (U.S. EPA). 2009. Proposed NO_2 NAAQS Regulatory Impact Analysis (RIA). Office of Air Quality Planning and Standards, Research Triangle Park, NC. June. Available on the Internet at < http://www.epa.gov/ttn/ecas/regdata/RIAs/proposedno2ria.pdf>.

Yang, Q., Y. Chen, Y. Shi, R. T. Burnett, K. M. McGrail and D. Krewski. 2003. Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada. Inhal Toxicol. Vol. 15 (13): 1297-308.