Appendix 6c: Additional Sensitivity Analyses Related To the Benefits Analysis

The analysis presented in Chapter 6 is based on our current interpretation of the scientific and economic literature. That interpretation requires judgments regarding the best available data, models, and modeling methodologies and the assumptions that are most appropriate to adopt in the face of important uncertainties. The majority of the analytical assumptions used to develop the primary estimates of benefits have been reviewed and approved by EPA's SAB. Both EPA and the SAB recognize that data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that alternative choices exist for some inputs to the analysis, such as the mortality C-R functions.

This appendix supplements our primary analysis of benefits with three additional sensitivity calculations. These supplemental estimates examine sensitivity to both valuation issues (e.g., the appropriate income elasticity) and for physical effects issues (e.g., the structure of the cessation lag and the sensitivity of the premature mortality estimate to the presence of a presumed threshold). These supplemental estimates are not meant to be comprehensive. Rather, they reflect some of the key issues identified by EPA or commentors as likely to have a significant impact on total benefits. The individual adjustments in the tables should not simply be added together because 1) there may be overlap among the alternative assumptions and 2) the joint probability among certain sets of alternative assumptions may be low.

6c.1 Premature Mortality Cessation Lag Structure

Over the last ten years, there has been a continuing discussion and evolving advice regarding the timing of changes in health effects following changes in ambient air pollution. It has been hypothesized that some reductions in premature mortality from exposure to ambient $PM_{2.5}$ will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question. The SAB-HES has recognized this lack of direct evidence. However, in early advice, they also note that "although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population" (EPA-SAB-COUNCIL-ADV-00-001, 1999, p. 9). In recent advice, the SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate (EPA-SAB-COUNCIL-ADV-04-002, 2004). They suggest that diseases with longer progressions should be characterized by longer-term lag structures, while air pollution impacts occurring in populations with existing disease may be characterized by shorter-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the long-term cohort studies. Although it may be reasonable to assume the cessation lag for lung cancer deaths mirrors the long latency of the disease, it is not at all clear what the appropriate lag structure should be for cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags. The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including using disease-specific lag models and constructing a segmented lag distribution to combine differential lags across causes of death (EPA-SAB-COUNCIL-ADV-04-002, 2004). The SAB-HES indicated support for using "a Weibull distribution or a simpler distributional form made up of several segments to cover the response mechanisms outlined above, given our lack of knowledge on the specific form of the distributions" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24). However, they noted that "an important question to be resolved is what the relative magnitudes of these segments should be, and how many of the acute effects are assumed to be included in the cohort effect estimate" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24-25). Since the publication of that report in March 2004, EPA has sought additional clarification from this committee. In its followup advice provided in December 2004, this SAB suggested that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$ (EPA-COUNCIL-LTR-05-001, 2004). The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Furthermore, in their advisory letter, the SAB-HES recommended that EPA include sensitivity analyses on other possible lag structures. In this appendix, we investigate the sensitivity of premature mortality-reduction related benefits to alternative cessation lag structures, noting that ongoing and future research may result in changes to the lag structure used for the primary analysis.

In previous advice from the SAB-HES, they recommended an analysis of 0-, 8-, and 15-year lags, as well as variations on the proportions of mortality allocated to each segment in the segmented lag structure (EPA-SAB-COUNCIL-ADV-00-001, 1999, (EPA-COUNCIL-LTR-05-001, 2004). The 0-year lag is representative of EPA's assumption in previous RIAs. The 8- and 15-year lags are based on the study periods from the Pope et al. (1995) and Dockery et al. (1993) studies, respectively.[1] However, neither the Pope et al. nor Dockery et al. studies assumed any lag structure when estimating the relative risks from PM exposure. In fact, the Pope et al. and Dockery et al. analyses do not supporting or refute the existence of a lag. Therefore, any lag structure applied to the avoided incidences estimated from either of these studies will be an

[1]Although these studies were conducted for 8 and 15 years, respectively, the choice of the duration of the study by the authors was not likely due to observations of a lag in effects but is more likely due to the expense of conducting long-term exposure studies or the amount of satisfactory data that could be collected during this time period.

assumed structure. The 8- and 15-year lags implicitly assume that all premature mortalities occur at the end of the study periods (i.e., at 8 and 15 years).

In addition to the simple 8- and 15-year lags, we have added three additional sensitivity analyses examining the impact of assuming different allocations of mortality to the segmented lag of the type suggested by the SAB-HES. The first sensitivity analysis assumes that more of the mortality impact is associated with chronic lung diseases or lung cancer and less with acute cardiopulmonary causes. This illustrative lag structure is characterized by 20 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 30 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The second sensitivity analysis assumes the 5-year distributed lag structure used in previous analyses, which is equivalent to a three-segment lag structure with 50 percent in the first 2-year segment, 50 percent in the second 3-year segment, and 0 percent in the 6- to 20-year segment. The third sensitivity analysis assumes a negative exponential relationship between reduction in exposure and reduction in mortality risk. This structure is based on an analysis by Röösli et al. (2004), which estimates the percentage of total mortality impact in each period t as

$$\%\ \text{Mortality Reduction(t)} = \frac{\left[(RR-1)e^{-0.5t}+1\right]-1}{\sum_{t=1}^{\infty}\left[(RR-1)e^{-0.5t}+1\right]-1} \qquad \text{(C.1)}$$

The Röösli et al. (2004) analysis derives the lag structure by calculating the rate constant (–0.5) for the exponential lag structure that is consistent with both the relative risk from the cohort studies and the change in mortality observed in intervention type studies (e.g., Pope et al. [1992] and Clancy et al. [2002]). This is the only lag structure examined that is based on empirical data on the relationship between changes in exposure and changes in mortality.

The estimated impacts of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Pope et al. ACS impact function) are presented in Table J-1. These estimates are based on the value of statistical lives saved approach (i.e., $6.6 million per incidence in 2006$) and are presented for both a 3 and 7 percent discount rate over the lag period.

The results of this sensitivity analyses demonstrate that because of discounting of delayed benefits, the lag structure may also have a large impact on monetized benefits, reducing benefits by 30 percent if an extreme assumption that no effects occur until after 15 years is applied. However, for most reasonable distributed lag structures, differences in the specific shape of the lag function have relatively small impacts on overall benefits. For example, the overall impact of moving from the previous 5-year distributed lag to the segmented lag recommended by the SAB-HES in 2004 in the primary estimate is relatively modest, reducing benefits by approximately 5 percent when a 3 percent discount rate is used and 17 percent when a 7 percent discount rate is used. If no lag is assumed, benefits are increased by approximately 10 percent relative to the segmented lag with a 3 percent discount rate and 22 percent with a 7 percent discount rate.

Table 6c-1: Sensitivity of Benefits of Premature Mortality Reductions to Alternative Cessation Lag Structures, Using Pope et al (2002) Effect Estimate

Alternative Lag Structures for PM-Related Premature Mortality		Value (billions of 2006$)[a]	Percent Difference from Base Estimate
None	Incidences all occur in the first year		
	3% discount rate	$3.4	10.4%
	7% discount rate	$3.4	22.5%
8-year	Incidences all occur in the 8th year		
	3% discount rate	$2.8	-10.3%
	7% discount rate	$2.1	-23.7%
15-year	Incidences all occur in the 15th year		
	3% discount rate	$2.2	-27.0%
	7% discount rate	$1.3	-52.5%
Alternative Segmented	20 percent of incidences occur in 1st year, 50 percent in years 2 to 5, and 30 percent in years 6 to 20		
	3% discount rate	$3.0	-3.2%
	7% discount rate	$2.6	-6.6%
5-Year Distributed	50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5		
	3% discount rate	$3.2	4.9%
	7% discount rate	$3.0	9.4%
Exponential	Incidences occur at an exponentially declining rate following year of change in exposure		
	3% discount rate	$3.2	5.6%
	7% discount rate	$3.1	11.3%

[a] All valuations rounded to two significant figures. This table reflects full attainment in all locations of the U.S. except two areas of California. These two areas, which have high levels of ozone, are not planning to meet the current standard until after 2020. The estimates in the table do not reflect benefits for the San Joaquin and South Coast Air Basins.

6c.2 Threshold Sensitivity Analysis

Chapter 6 presents the results of the $PM_{2.5}$ premature mortality benefits analysis based on an assumed cutpoint in the long-term mortality concentration-response function at 10 $\mu g/m^3$, and an assumed cutpoint in the short-term morbidity concentration-response functions at 10 $\mu g/m^3$. There is ongoing debate as to whether there exists a threshold below which there would be no benefit to further reductions in $PM_{2.5}$. Some researchers have hypothesized the presence of a threshold relationship. The nature of the hypothesized relationship is the possibility that there exists a PM concentration level below which further reductions no longer yield premature mortality reduction benefits. EPA's most recent $PM_{2.5}$ Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies" (U.S. EPA, 2004b, p. 9-44).

EPA's Science Advisory Board (SAB) that provides advice on benefits analysis methods[2] has been to model premature mortality associated with PM exposure as a non-threshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations.

For these reasons we provide the results of a sensitivity analysis in which we estimate the change in reduction in incidence of $PM_{2.5}$-related premature mortality resulting from changes in the presumed threshold. We also provide a corresponding estimate of the valuation of these changes in incidence.

Table 6c-2: Mortality Threshold Sensitivity Analysis for 0.070 ppm Ozone Scenario (Using Pope et al., 2002 Effect Estimate with Slope Adjustment for Thresholds Above 7.5 ug) 95th Percentile Confidence Intervals Provided in Parentheses [a]

		East	Western U.S. Excluding CA	California	Total
Less Certain That Benefits Are at Least as Large	No Threshold	580 (120--1,000)	56 (15--98)	12 (3.9--19)	640 (140--1,100)
	Threshold at 7.5 µg	570 (130--1,000)	49 (16--81)	11 (3.6--18)	630 (150--1,100)
	Threshold at 10 µg	420 (110--730)	6.3 (2.1--10)	5.4 (2--9)	430 (110--750)
	Threshold at 12 µg	46 (14--79)	0.00 (0.00--0.00)	3.7 (1.2--6.2)	50 (15--85)
More Certain That Benefits are at Least as Large	Threshold at 14 µg	1.0 (0.35--1.7)	0.00 (0.00--0.00)	2.9 (1.0--4.9)	4.0 (1.3--6.6)

[a] All estimates are rounded to 2 significant digits. All rounding occurs after final summing of unrounded estimates. As such, totals will not sum across columns. Estimates do not include South Coast and San Joaquin Air Basins.

[2] The advice from the 2004 SAB-HES (U.S. EPA-SAB, 2004b) is characterized by the following: "For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between $PM_{2.5}$ and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3, and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

Table 6c-3: Sensitivity of Monetized Benefits of Reductions in Mortality Risk to Assumed Thresholds for 0.070 ppm Partial Attainment Scenario (Using Pope et al., 2002 Effect Estimate with Slope Adjustment for Thresholds Above 7.5 ug, 95th Percentile Confidence Intervals Provided in Parentheses, in billions of 2006$)[a]

			Eastern U.S.	Western U.S. Excluding CA	California	Total Nationwide Attainment
Less Certain that Benefits Are at Least as Large	No Threshold	3%	4.0 ($0.49--$10)	0.40 ($0.05--$0.94)	0.08 ($0.01--$0.19)	4.5 ($0.55--$11)
		7%	3.6 ($0.44--$8.8)	0.36 ($0.05--$0.84)	0.02 ($0.01--$0.17)	4.1 ($0.49--$10)
	Threshold at 7.5 µg	3%	4.0 ($0.49--$10)	0.34 ($0.05--$0.78)	0.08 ($0.01--$0.17)	4.4 ($0.55--$11)
		7%	3.6 ($0.44--$8.6)	0.31 ($0.04--$0.70)	0.07 ($0.01--$0.16)	4.0 ($0.49--$9.5)
	Threshold at 10 µg	3%	3.0 ($0.38--$7.0)	0.04 ($0.01--$0.10)	0.04 ($0.01--$0.09)	3.0 ($0.39--$7.2)
		7%	2.7 ($0.35--$6.3)	0.04 ($0.01--$0.09)	0.03 ($0.00--$0.08)	2.7 ($0.36--$6.5)
	Threshold at 12 µg	3%	0.33 ($0.04--$0.76)	0.00 ($0.00--$0.0)	0.03 ($0.00--$0.06)	0.35 ($0.05--$0.82)
		7%	0.29 ($0.04--$0.68)	0.00 ($0.00--$0.00)	0.02 ($0.00--$0.05)	0.32 ($0.04--$0.73)
More Certain that Benefits Are at Least as Large	Threshold at 14 µg	3%	0.01 ($0.00--$0.02)	0.00 ($0.00--$0.0)	0.02 ($0.00--$0.05)	0.03 ($0.00--$0.06)
		7%	0.01 ($0.00--$0.01)	0.00 ($0.00--$0.00)	0.02 ($0.00--$0.04)	0.03 ($0.00--$0.06)

[a] All estimates are rounded to 2 significant digits. All rounding occurs after final summing of unrounded estimates. As such, totals will not sum across columns. Estimates do not include South Coast and San Joaquin Air Basins.

6c.3 Income Elasticity of Willingness to Pay

As discussed in Chapter 6, our estimates of monetized benefits account for growth in real GDP per capita by adjusting the WTP for individual endpoints based on the central estimate of the adjustment factor for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility). We examined how sensitive the estimate of total benefits is to alternative estimates of the income elasticities. Table 6c.3 lists the ranges of elasticity values used to calculate the income adjustment factors, while Table 6c.4 lists the ranges of corresponding adjustment factors. The results of this sensitivity analysis, giving the monetized benefit subtotals for the four benefit categories, are presented in Table 6c.5.

Table 6c-4. Ranges of Elasticity Values Used to Account for Projected Real Income Growth[a]

Benefit Category	Lower Sensitivity Bound	Upper Sensitivity Bound
Minor Health Effect	0.04	0.30
Severe and Chronic Health Effects	0.25	0.60
Premature Mortality	0.08	1.00
Visibility[b]	—	—

[a] Derivation of these ranges can be found in Kleckner and Neumann (1999) and Chestnut (1997). COI estimates are assigned an adjustment factor of 1.0.

[b] No range was applied for visibility because no ranges were available in the current published literature.

Table 6c-5. Ranges of Adjustment Factors Used to Account for Projected Real Income Growth[a]

Benefit Category	Lower Sensitivity Bound	Upper Sensitivity Bound
Minor Health Effect	1.018	1.147
Severe and Chronic Health Effects	1.121	1.317
Premature Mortality	1.037	1.591
Visibility[b]	—	—

[a] Based on elasticity values reported in Table C-4, U.S. Census population projections, and projections of real GDP per capita.

[b] No range was applied for visibility because no ranges were available in the current published literature.

Table 6c-6. Sensitivity of Monetized Benefits to Alternative Income Elasticities[a]

Benefit Category	Ozone Analysis		PM Analysis	
	Lower Sensitivity Bound	Upper Sensitivity Bound	Lower Sensitivity Bound	Upper Sensitivity Bound
Minor Health Effect	$48	$48	$8.3	$8.5
Severe and Chronic Health Effects	--	--	$170	$200
Premature Mortality[b]	$340	$520	$2,600	$4,000
Total Benefits[b]	$380	$560	$2,800	$4,200

[a] All estimates rounded to two significant digits. All Benefits Incremental to 080 ppm Partial Attainment Strategy (Millions of 2006$). This table reflects full attainment in all locations of the U.S. except two areas of California. These two areas, which have high levels of ozone, are not planning to meet the current standard until after 2020. The estimates in the table do not reflect benefits for the San Joaquin and South Coast Air Basins.

[b] Using mortality effect estimate from Bell (2004) and mortality effect estimate from Pope et al (2002) to estimate $PM_{2.5}$ mortality at a 3% discount rate.

[c] No range was applied for visibility because no ranges were available in the current published literature.

Consistent with the impact of mortality on total benefits, the adjustment factor for mortality has the largest impact on total benefits. The value of mortality in 2020 ranges from 90 percent to 130 percent of the primary estimate based on the lower and upper sensitivity bounds on the income adjustment factor. The effect on the value of minor and chronic health effects is much less pronounced, ranging from 98 percent to 105 percent of the primary estimate for minor effects and from 93 percent to 106 percent for chronic effects.

6c.4 References

Chestnut, L.G. 1997. "Draft Memorandum: Methodology for Estimating Values for Changes in Visibility at National Parks." April 15.

Chestnut, L.G., and R.D. Rowe. 1990a. *Preservation Values for Visibility Protection at the National Parks: Draft Final Report.* Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC and Air Quality Management Division, National Park Service, Denver, CO.

Chestnut, L.G., and R.D. Rowe. 1990b. "A New National Park Visibility Value Estimates." In *Visibility and Fine Particles*, Transactions of an AWMA/EPA International Specialty Conference, C.V. Mathai, ed. Air and Waste Management Association, Pittsburgh.

Clancy, L., P. Goodman, H. Sinclair, and D.W. Dockery. 2002. "Effect of Air-pollution Control on Death Rates in Dublin, Ireland: An Intervention Study." *Lancet* Oct 19;360(9341):1210-4.

Desvousges, W.H., F.R. Johnson, and H.S. Banzhaf. 1998. *Environmental Policy Analysis With Limited Information: Principles and Applications of the Transfer Method (New Horizons in Environmental Economics.)* Edward Elgar Pub: London.

EPA-SAB-COUNCIL-ADV-00-001. October 1999. *The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects*. Part 2.

EPA-SAB-COUNCIL-ADV-99-012. July 1999. *The Clean Air Act Amendments (CAAA) Section 812 Prospective Study of Costs and Benefits (1999): Advisory by the Health and Ecological Effects Subcommittee on Initial Assessments of Health and Ecological Effects*. Part 1.

EPA-SAB-COUNCIL-ADV-01-004. September 2001. *Review of the Draft Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act 1990-2020: An Advisory by a Special Panel of the Advisory Council on Clean Air Compliance Analysis.*

EPA-SAB-COUNCIL-ADV-04-002. March 2004. *Advisory on Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis—Benefits and Costs of the Clean Air Act, 1990-2020: Advisory by the Health Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis.*

Kleckner, N., and J. Neumann. June 3, 1999. "Recommended Approach to Adjusting WTP Estimates to Reflect Changes in Real Income." Memorandum to Jim Democker, US EPA/OPAR.

Roosli M, Kunzli N, Braun-Fahrlander C, Egger M. 2005. "Years of life lost attributable to air pollution in Switzerland: dynamic exposure-response model." *International Journal of Epidemiology* 34(5):1029-35.

U.S. Environmental Protection Agency (EPA). 2004. Air Quality Criteria for Particulate Matter, Volume II. Office of Research and Development. EPA/600/P-99/002bF, October 2004.