Appendix J: Additional Sensitivity Analyses Related to the Benefits Analysis

The analysis presented in Chapter 5 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). 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.

J.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 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).

[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.

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

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

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., $5.5 million per incidence) 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 15 percent when a 7 percent discount rate is used. If no lag is assumed, benefits are increased by around 10 percent relative to the segmented lag with a 3 percent discount rate and 30 percent with a 7 percent discount rate.

Table J-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		*15/35*		*14/35*	
		Value (billion 1999$)[a,b]	*Percent Difference from Base Estimate*	*Value (billion 1999$)*[a,b]	*Percent Difference from Base Estimate*
None	Incidences all occur in the first year				
	3% discount rate	$16.5	10.4%	$29.1	10.4%
	7% discount rate	$16.5	31.2%	$29.1	31.2%
8-year	Incidences all occur in the 8th year				
	3% discount rate	$13.4	–10.3%	$23.6	–10.3%
	7% discount rate	$10.3	–18.3%	$18.1	–18.3%
15-year	Incidences all occur in the 15th year				
	3% discount rate	$10.9	–27.0%	$19.2	–27.0%
	7% discount rate	$6.4	–49.1%	$11.3	–49.1%
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	$14.5	–3.2%	$25.5	–3.2%
	7% discount rate	$11.5	–8.7%	$20.2	–8.7%
5-Year Distributed	50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5				
	3% discount rate	$15.7	4.9%	$27.6	4.9%
	7% discount rate	$14.7	17.1%	$25.9	17.1%
Exponential	Incidences occur at an exponentially declining rate following year of change in exposure				
	3% discount rate	$15.8	5.6%	$27.8	5.6%
	7% discount rate	$14.4	14.8%	$25.4	14.8%

[a] Dollar values rounded to two significant digits.

J.2 Visibility Benefits in Additional Class I Areas

The Chestnut and Rowe (1990a) study from which the primary valuation estimates are derived only examined WTP for visibility changes in Class I areas (national parks and wilderness areas) in the southeast, southwest, and California. To obtain estimates of WTP for visibility changes at national parks and wilderness areas in the northeast, northwest, and central regions of the U.S., we have to transfer WTP values from the studied regions. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility improvement in parks in one region is not necessarily the same

environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

Based on this benefits transfer methodology (implemented within the preference calibration framework discussed in Chapter 5 and Appendix I), estimated additional visibility benefits in the northwest, central, and northeastern U.S. are provided in Table J-2.

Table J-2: Monetary Benefits Associated with Improvements in Visibility in Additional Federal Class I Areas in 2020 Incremental to 15/65 Attainment Strategy (in millions of 1999$)[a]

Suite of Standards	*Northwest[b]*	*Central[c]*	*Northeast[d]*	*Total*
15/35	$96	$130	$6	$240
14/35	$67	$140	$44	$250

[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

[b] Northwest Class I areas include Crater Lake, Mount Rainier, North Cascades, and Olympic national parks, and Alpine Lakes, Diamond Peak, Eagle Cap, Gearhart Mountain, Glacier Peak, Goat Rocks, Hells Canyon, Kalmiopsis, Mount Adams, Mount Hood, Mount Jefferson, Mount Washington, Mountain Lakes, Pasayten, Strawberry Mountain, and Three Sisters wilderness areas.

[c] Central Class I areas include Craters of the Moon, Glacier, Grand Teton, Theodore Roosevelt, Badlands, Wind Cave, and Yellowstone national parks, and Anaconda-Pintlar, Bob Marshall, Bridger, Cabinet Mountains, Fitzpatrick, Gates of the Mountain, Lostwood, Medicine Lake, Mission Mountain, North Absaroka, Red Rock Lakes, Sawtooth, Scapegoat, Selway-Bitterroot, Teton, U.L. Bend, and Washakie wilderness areas.

[d] Northeast Class I areas include Acadia, Big Bend, Guadalupe Mountains, Isle Royale, Voyageurs, and Boundary Waters Canoe national parks, and Brigantine, Caney Creek, Great Gulf, Hercules-Glades, Lye Brook, Mingo, Moosehorn, Presidential Range-Dry Roosevelt Campobello, Seney, Upper Buffalo, and Wichita Mountains wilderness areas.

J.3 Income Elasticity of Willingness to Pay

As discussed in Chapter 5, 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 J-3 lists the ranges of elasticity values used to calculate the income adjustment factors, while Table J-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 J-5.

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.

Table J-3: 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 J-4: Ranges of Adjustment Factors Used to Account for Projected Real Income Growth[a]Error! Bookmark not defined.

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 J-5: Sensitivity of Monetized Benefits to Alternative Income Elasticities[a]

	Benefits Incremental to 15/65 Attainment Strategy (Millions of 1999$)			
	15/35		*14/35*	
Benefit Category	*Lower Sensitivity Bound*	*Upper Sensitivity Bound*	*Lower Sensitivity Bound*	*Upper Sensitivity Bound*
Minor Health Effect	$130	$140	$210	$220
Severe and Chronic Health Effects	$1,400	$1,600	$2,500	$2,700
Premature Mortality[b]	$13,000	$20,000	$23,000	$34,000
Visibility and Other Welfare Effects[c]	$530	$530	$1,200	$1,200
Total Benefits[b]	$15,000	$22,000	$26,000	$37,000

[a] All estimates rounded to two significant digits.

[b] Using mortality effect estimate from Pope et al. (2002) and 3 percent discount rate.

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

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