

AN EVALUATION OF THE PM_{2.5} HEALTH BENEFITS ESTIMATES IN REGULATORY IMPACT ANALYSES FOR RECENT AIR REGULATIONS



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Abstract.

When preparing its Regulatory Impact Analyses (RIAs) for regulations under the Clean Air Act (CAA) that are not intended to control ambient fine particulate matter (PM_{2.5}), the US Environmental Protection Agency (EPA) often predicts reductions of ambient PM_{2.5} that may occur coincidentally, and attributes so-called “PM_{2.5} co-benefits” to those coincidental reductions. This paper reviews and evaluates EPA’s practice of including PM_{2.5} co-benefits in its RIAs for non-PM rules. It is based on review of 57 individual CAA-related RIAs released since EPA promulgated its first PM_{2.5} national ambient air quality standard (NAAQS) and finds that EPA has been relying on PM_{2.5} co-benefits estimates to create an apparent benefit-cost justification for almost all of its non-PM CAA rules. This paper then evaluates that practice from multiple perspectives: theoretical, practical, scientific, and analytical. It concludes that co-benefits from separately-regulated pollutants, such as PM_{2.5}, should not be reported as part of the total benefits estimates in an RIA, nor should they be included in public announcements of the benefits of a new regulation. EPA should reform the manner in which it defines its baselines of emissions for each RIA, and provide more temporal information on benefits and costs to eliminate problems of double-counting. This paper also concludes that EPA should reform its current methods of calculating benefits from reductions in ambient PM_{2.5} even in its PM-related rules, because it finds that as EPA’s reliance on co-benefits has increased, EPA has shifted to less credible methods of estimating PM_{2.5} benefits.

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1. Introduction and Synopsis

Regulatory Impact Analyses (RIAs) are documents required by an Executive Order (EO) of the President to be submitted to the Office of Management and Budget (OMB) by all agencies proposing new major regulations. RIAs' quality and usefulness have been a substantial interest for the past thirty years. Policy analysts have written many papers and reports identifying the ways RIAs provide value to policy making and offering suggestions for improvement.² This paper reviews and evaluates the US Environmental Protection Agency's (EPA's) practice of estimating benefits from reducing ambient fine particulate matter (PM_{2.5}) in its RIAs for rulemakings under the Clean Air Act (CAA).

In recent years, EPA has relied on reductions of ambient PM_{2.5} as the primary source of benefits in most of its RIAs for CAA-related regulations – even for regulations not specifically to protect the public health from exposures to ambient PM_{2.5}. When the regulation is not targeting PM_{2.5}, they are called “co-benefits” because they result from changes in ambient PM_{2.5} levels projected to follow coincidentally from efforts to reduce other types of air pollutants. Questions and concerns have been raised by many in policy making and policy analysis communities about EPA's reliance on such co-benefits.

Based on review of CAA-related RIAs since 1997, this paper identifies the degree of EPA's reliance on PM_{2.5} co-benefits. It then examines EPA's co-benefits practice from multiple perspectives: theoretical, practical, scientific, and analytical. It finds that the theoretical formulation of benefit-cost analysis (BCA) – a key underpinning of RIAs – does not support inclusion of co-benefits from pollutants subject to their own, separate regulation. Also, allowing such co-benefits to dominate RIAs detracts from RIAs' most valuable practical role, which is to help guide us toward regulations that provide cost-effective, minimally-complex management of societal resources. From a scientific perspective, this review finds EPA's estimates of the risks of PM_{2.5} have become less and less credible as EPA has come to rely more and more heavily on them to justify regulation of other pollutants. It also finds that use of co-benefits in many RIAs being prepared simultaneously degrades the analytical rigor of benefits accounting across the body of RIAs as a group, with double-counting and related analytical maladies resulting.

Accordingly, this paper recommends changes in how RIA baselines are set in order to eliminate problems of double-counting and inappropriate benefit-cost comparisons. It also concludes that:

- Public announcements about the benefits of a new regulation should not include co-benefits of pollutants that are already directly regulated; nor should such co-benefits be included in the total benefits reported in RIA Executive Summaries.
- EPA should reform its practice of calculating benefits from reductions in ambient PM_{2.5} by using more credible sets of risk analysis assumptions, and eliminating extrapolations.

² Examples include Morgenstern (1997), Hahn and Dudley (2007), and Harrington *et al.* (2009).

This paper is organized as follows. The next section provides background on the history and purpose of RIAs as the general requirement under which EPA is producing its CAA-related RIAs. It is followed by a summary of EPA's growing reliance on PM_{2.5} co-benefits identified through a review of RIAs dating back to 1997, when EPA released its first regulation of ambient PM_{2.5}. The four sections after that examine EPA's co-benefits practice from four different perspectives: theoretical, practical, scientific, and analytical, and conclude the practice is problematic from each of these perspectives. The last section recaps findings and recommendations made throughout the paper and draws further conclusions.

2. History and Purpose of RIAs

The practice of using BCA for assessing the appropriateness of public policies dates back well before RIAs were required, but was relatively sporadic. This changed in 1981 when President Ronald Reagan issued EO 12291. EO 12291 required that a BCA be prepared and submitted to the OMB for each major regulation issued by the Federal government. That EO required that each new major rule be demonstrated to provide greater benefits than its costs,³ using the term “regulatory impact analysis” for the document making this demonstration.⁴ The requirement that benefits be greater than costs is certainly a prerequisite for passing any BCA test; nevertheless, the economically-proper definition of a benefit-cost optimum – the BCA basis for determining the appropriate stringency of a standard – is that the incremental or “marginal” cost of making a standard tighter is equal to the marginal benefits that such tightening would provide. This is because net benefits are at their maximum level when marginal costs equal marginal benefits. Subsequent guidance for conducting RIAs requires that several alternative standards be evaluated in the RIA. This is to help steer the selected alternative to the one that would offer the highest net benefits without requiring a precise optimization using marginal analysis.⁵

EO 12291's requirement that benefits exceed costs was at odds with the fact that many standards, including National Ambient Air Quality Standards (NAAQSs) for criteria pollutants, and National Emissions Standard for Hazardous Air Pollutants (NESHAPs) for air toxics, must be set without regard to costs. The CAA requires that each NAAQS

³ Section 2 of EO 12291 specifically required, *inter alia*, that “(b) Regulatory action shall not be undertaken unless the potential benefits to society for the regulation outweigh the potential costs to society; (c) Regulatory objectives shall be chosen to maximize the net benefits to society; (d) Among alternative approaches to any given regulatory objective, the alternative involving the least net cost to society shall be chosen.” These are overtly the requirements of standard BCA-based decision making.

⁴ EO 12291, Section 3.

⁵ Guidance has been provided over the years. The most current version of guidance, known as “Circular A-4,” states that: “In general, both the benefits and costs associated with a regulation will increase with the level of stringency (although marginal costs generally increase with stringency, whereas marginal benefits may decrease). You should study alternative levels of stringency to understand more fully the relationship between stringency and the size and distribution of benefits and costs among different groups.” (OMB, 2003, p.8).

be set at a level that protects the public health with “an adequate margin of safety,”⁶ and has been interpreted to require that this level be set without regard for their costs. The CAA’s requirements for NESHAP rules are more complex, but the most commonly required provision used under a NESHAP rule is maximum achievable control technology (MACT). The least stringent emissions level for a MACT (the “MACT floor”) is determined based solely on the average performance achieved by the best 12% of existing technologies for a given production process, regardless of what cost achieving that MACT floor may impose on facilities that do not already have that emissions rate.⁷ Thus the requirement of EO 12291 that all major regulations demonstrate they could pass the most basic requirement of BCA was instantly at odds with the legal framework for CAA rules. Resolution of this inconsistency was addressed by a provision in EO 12291 that the BCA provisions be applied “to the extent permitted by law.”⁸

An executive order requirement for RIAs for all major regulations has remained in effect since 1981, but has evolved. In particular, President Bill Clinton issued EO 12866 in 1993 which revokes EO 12291, but replaces it with very similar requirements for assessment of regulatory impacts of major rulemakings.⁹ Other than instituting some procedural changes, EO 12866 primarily moderates the emphasis of EO 12291 on meeting benefit-cost criteria. However, the change is one of degree only and the fundamental underpinnings of RIAs in BCA remains apparent. For example, EO 12866 states:

In deciding whether and how to regulate, agencies should assess all costs and benefits of available regulatory alternatives, including the alternative of not regulating. Costs and benefits shall be understood to include both quantifiable measures (to the fullest extent that these can be usefully estimated) and qualitative measures of costs and benefits that are difficult to quantify, but nevertheless essential to consider. Further, in choosing among alternative regulatory approaches, agencies should select those approaches that maximize net benefits (including potential economic, environmental, public health and safety, and other advantages; distributive impacts; and equity), unless a statute requires another regulatory approach.¹⁰

⁶ 42 USC §7409(b)(1).

⁷ 42 USC §7412(d)(3)(A).

⁸ EO 12291, Section 2.

⁹ Interestingly, EO 12866 did not use the term “regulatory impact assessment” despite the similarity of its requirements. Apparently as a result, after the issuance of Clinton’s EO 12866, and for the remainder of his years in office, the term RIA was largely replaced by “Economic Impact Analysis” (EIA) as the name of these EO-mandated documents. After about 2002, the term RIA came back into use, and remains the term used today. This temporary change in the common name for the regulatory assessment documents submitted to OMB can be confusing when performing reviews of RIAs and the methodological practices associated with them. This paper will refer to all of them as RIAs, although the titles of some of the documents cited do not use that term.

¹⁰ EO 12866, Section 1(a).

As the excerpt above reveals, EO 12866 no longer requires that benefits be greater than costs. Also, it broadens the criteria to be considered to include more allowance for qualitative and non-quantifiable benefits. This is reinforced by the following language:

*Each agency shall assess both the costs and the benefits of the intended regulation and, recognizing that some costs and benefits are difficult to quantify, propose or adopt a regulation only upon a reasoned determination that the benefits of the intended regulation justify its costs.*¹¹

As a result, the purpose of RIAs has been more clearly defined as one of providing decision-relevant information in a structured, coherent and transparent format. Costs and benefits still play a central role, but are not treated as if they should be determinative, even in situations where the law allows consideration of costs. Most policy analysts, including most economists, have been supportive of this broader purpose of RIAs.

In 2011, President Barack Obama issued EO 13563 to supplement EO 12866: “to improve regulation and regulatory review.” EO 13563 requires that “equity, human dignity, fairness, and distributive impacts” be considered, in addition to the requirements of EO 12866.¹² This last modification does not alter the fundamental view of the role and purpose of RIAs: EO 13563 states that the purpose of RIAs is that regulations will be adopted based “upon a reasoned determination that its benefits justify its costs” and also that each agency “tailor its regulations to impose the least burden on society” and “select, in choosing among regulatory approaches, those approaches that maximize net benefits.”¹³ It also, importantly, notes that agencies should strive to reduce regulatory requirements that are “redundant, inconsistent, or overlapping.”¹⁴

Thus, RIAs have an original foundation in BCA, but they rarely have been used strictly as BCA-based decision documents. Their practical value in policy making is that they can provide a structured assessment of the merits of individual regulations, so that policymakers and the public can have a clear understanding of the overall burden and positive contributions of individual regulations.¹⁵ This practical role is quite clearly stated in OMB’s “Circular A-4,” which is the present OMB guidance for conducting RIAs:

Regulatory analysis is a tool regulatory agencies use to anticipate and evaluate the likely consequences of rules. It provides a formal way of organizing the evidence on the key effects, good and bad, of the various alternatives that should be considered in developing regulations. The

¹¹ EO 12866, Section 1(b)(6).

¹² EO 13563, Section 1(c).

¹³ EO 13563, Section 1(b).

¹⁴ EO 13563, Section 3.

¹⁵ See, for example, Arrow *et al.* (1996).

*motivation is to (1) learn if the benefits of an action are likely to justify the costs or (2) discover which of various possible alternatives would be the most cost-effective.*¹⁶

Similarly, as RIAs are prepared for multiple different rules required by certain sections of law, such as a NESHAP under the CAA, the combined set of RIAs can help create an understanding of the overall merits of the way the controlling laws have been written. With time, good RIA practice should help identify laws that are poorly written, or regulatory approaches that implement laws in an ineffective manner. Political pressure to revise ineffective laws may result in better long-term legislation or regulatory practice. The fact that this role is recognized as valuable may be one reason why the requirement for RIAs has endured for thirty years and through Administrations with differing political perspectives. Whatever the merits of RIAs may be, they do face one problem with respect to serving their most valuable public policy objective. They are not subject to any formal public or peer review process other than review by OMB's Office of Information and Regulatory Affairs (OIRA).¹⁷

3. Evidence of the Predominance of PM_{2.5} Co-Benefits in RIAs for Air Regulations

The research for this paper sought to identify and obtain all of the RIAs released for rulemakings under the CAA since 1997, which was the year in which EPA released its first NAAQS for PM_{2.5}. EPA's first estimates of mortality benefits from reducing ambient PM_{2.5} were developed as part of that 1997 NAAQS policy decision, which makes 1997 an appropriate starting point for tracing the history of the use of PM_{2.5} co-benefits in CAA-related RIAs. Creation of a list of RIAs to include in this study started with the identification of major regulations that the US EPA Air Office has submitted to OMB since 1997.¹⁸ The list was then expanded to include RIAs for major air regulations still in the proposal stage.¹⁹ This process identified 57 rules, 54 of which are final and 3 of which have been recently proposed and not yet been finalized.

¹⁶ OMB (2003), pp. 1-2.

¹⁷ The importance of extensive external review is highlighted in the sixth principle of the appropriate use of BCA in RIAs in Arrow *et al.* (1996), p. 221.

¹⁸ This was done by going to <http://www.reginfo.gov/public/do/eoAdvancedSearch#> on the OMB website, and filtering for rules that met the following selection criteria: submitted by the EPA Air Office, concluded, final rule, major, and completed in any year since 1997. One rule that appeared on the resulting list was excluded (the renewable fuels standard known as "RFS2") because it is required under the Energy Independence and Security Act of 2007, not the CAA. One rule found on the OMB website fit the selection criteria, but did not appear when the filter was applied. This rule (the Petroleum Refineries NSPS rule concluded 12/10/08) was included.

¹⁹ These were identified from RIAs posted on the EPA RIA website as of October 31, 2011 (at <http://www.epa.gov/ttnecas1/ria.html>). During initial research, the final RIA for the proposed air toxics rule for electricity generating units (EGUs) was all that was available. This rule, which is called the "EGU MACT rule" in this paper, was finalized in December 2011. This paper relies on results from the RIA for the final EGU MACT rule for its conclusions, but the paper quotes statements made by EPA and others that

A greater challenge was obtaining copies of the RIAs for all of the rules on this list. Neither OMB nor EPA maintains a complete, publicly-available record of RIAs. Also, an RIA is apparently not usually placed in its associated rulemaking docket. This situation may be a limitation in the completeness of dockets, or may be because RIAs are not required by the legal rulemaking procedures, but only by executive order.²⁰ Although *Federal Register* Notices of Rulemakings often summarize information from the RIAs, these sources were not always sufficient, because they may report total benefits estimated, but without the details needed to disaggregate those totals into direct benefits, PM_{2.5} health-related co-benefits, and any other types of co-benefits.

At this point in time, this study has found either the final RIA itself, or sufficient information in other rulemaking documents to understand what EPA did to analyze the benefits and associated costs for 51 of the 57 rules identified, 48 of which are final rules and 3 of which are recently proposed rules. References for the source documents used are provided in Appendix A for all 57, with, where possible, page references for the relevant information on benefit and cost estimates found for each rule. Summary tables in the body of this paper are based on the references in Appendix A. Any RIAs that are quoted or described in greater detail in this paper are also listed in the References section of this paper.

This review of CAA RIAs since 1997 found that as EPA releases each of its proposed and final air quality rules, it typically emphasizes that the rule will generate health benefits that exceed its costs. However, close inspection of the associated RIAs reveals that a majority of those benefits – sometimes *all* of them – are from reductions in PM_{2.5}, even for air regulations that are targeting clean air objectives other than PM_{2.5}. For many of those regulations, the bulk of the benefits estimates in their RIAs are attributable to reductions in already-low concentrations of ambient PM_{2.5} that EPA has predicted will occur *coincidentally* as a result of regulation of those non-PM pollutant(s).

Figure 1 provides a summary of the role of PM_{2.5} co-benefits in all of the air regulation RIAs that were for rules not targeting PM_{2.5}-related health risks and which provided any quantified benefits estimates at all. The rulemakings are listed in chronological order (based on the date the rules were finalized), so that one can observe the trend over time since 1997 in how frequently EPA has relied on PM_{2.5} co-benefits, and the general degree of importance those co-benefits played in lending EPA a benefit-cost case for each rule. Figure 1 shows that a trend towards almost complete reliance on PM_{2.5}-related health co-benefits has grown over time. The main exceptions in recent years have been rules addressing greenhouse gases (GHGs) under the CAA. (The GHG-related RIAs all report negative costs, so PM_{2.5} co-benefits are not necessary to make a benefit-cost justification in any case.)

used numbers from the final RIA for the proposed EGU MACT rule. Care is taken to clarify where information in this paper refers to information released with the proposed EGU MACT rule.

²⁰ Although EO 12866 requires that all communications between OIRA and the agencies, White House and public be added to the docket, our experience has been that the RIAs themselves cannot be found in all the respective dockets.

Figure 1. Summary of Degree of Reliance on PM_{2.5}-Related Co-Benefits in RIAs Since 1997 for Major Non-PM Rulemakings under the CAA

(RIAs with no quantified benefits at all are not in this table. Where ranges of benefit and/or cost estimates are provided, percentages are based on upper bound of both the benefits and cost estimates. Estimates using the 7% discount rates are used in all cases.)

Year	RIAs for Rules Not Targeting Ambient PM 2.5	PM Co-Benefits Are >50% of Total	PM Co-Benefits Are Only Benefits Quantified
1997	Ozone NAAQS (.12 1hr=>.08 8hr)	x	
1997	Pulp&Paper NESHAP		
1998	NOx SIP Call & Section 126 Petitions		
1999	Regional Haze Rule	x	
1999	Final Section 126 Petition Rule	x	
2004	Stationary Reciprocating Internal Combustion Engine NESHAP	x	
2004	Industrial Boilers & Process Heaters NESHAP	x	x
2005	Clean Air Mercury Rule	x	
2005	Clean Air Visibility Rule/BART Guidelines	x	
2006	Stationary Compression Ignition Internal Combustion Engine NSPS		
2007	Control of HAP from mobile sources	x	x
2008	Ozone NAAQS (.08 8hr =>.075 8hr)	x	
2008	Lead (Pb) NAAQS	x	
2009	New Marine Compress'n-Ign Engines >30 L per Cylinder	x	
2010	Reciprocating Internal Combustion Engines NESHAP – Comp. Ignit.	x	x
2010	EPA/NHTSA Joint Light-Duty GHG & CAFES		
2010	SO2 NAAQS (1-hr, 75 ppb)	x	> 99.9%
2010	Existing Stationary Compression Ignition Engines NESHAP	x	x
2011	Industrial, Comm, and Institutional Boilers NESHAP	x	x
2011	Indus'l, Comm'l, and Institutional Boilers & Process Heaters NESHAP	x	x
2011	Comm'l & Indus'l Solid Waste Incin. Units NSPS & Emission G'lines	x	x
2011	Control of GHG from Medium & Heavy-Duty Vehicles		
2011	Ozone Reconsideration NAAQS	x	
2011	Utility Boiler MACT NESHAP (<i>Final Rule's RIA</i>)	x	≥ 99%
2011	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	x	
2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	x	x

The practice of including co-benefits in RIAs is not new. Examples where co-benefits of one sort or another played a significant role in the evaluation of an RIA can be found as early as 1984.²¹ However, the current situation is dramatically different from past examples. First, the pollutant for which co-benefits are being claimed is already stringently regulated – in fact, the CAA language defining the required stringency implies stringency greater than the BCA optimum (this point is explained in the next section). Second, these PM_{2.5} co-benefits not only dominate the majority of RIAs for EPA’s non-PM rules, but in many cases they are the only benefit that is being quantified at all.

Section 6 will discuss a number of reasons why these PM_{2.5} co-benefits are overstated. That discussion also implies that the direct benefits that EPA is estimating in its RIAs for rules that do directly address PM_{2.5} risks are also overstated. However, the intervening two sections first address the reasons why reliance on PM_{2.5} co-benefits in non-PM rulemakings is inappropriate theoretically, inconsistent with the stated objectives of requiring RIAs to be prepared, and promotes excessively complex, duplicative and cost-ineffective regulation for the nation.

4. EPA’s Use of PM_{2.5} Co-Benefits in RIAs Is Inconsistent with Theoretical Underpinnings of Benefit-Cost Analysis

Although RIAs are no longer intended to serve solely as BCAs, BCA is clearly a core tenet of the exercise of producing RIAs. For this reason, it is important to explore from a theoretical perspective what BCA implies about the use of “co-benefits.” As this section will demonstrate, the theoretical underpinnings of BCA do not support EPA’s practice of adding co-benefits of separately-regulated pollutants into the total benefits estimates in RIAs, even when there are significant interrelationships between the pollutants in terms of the technologies that control them, their atmospheric formation, or health or welfare responses. In fact, the analysis in this section demonstrates that EPA’s co-benefits practice actually moves the resulting benefit-cost comparison *in the wrong* direction from what BCA would advise if the pollutants for which co-benefits are being calculated are criteria pollutants already regulated under a NAAQS.

The purpose of BCA when applied to a policy choice, such as determining the level of an emissions standard, is to identify the level that would provide the highest net benefits (and also to ensure that those net benefits would be positive). The optimization objective for choosing a required amount of reduction of a single pollutant is mathematically stated as:

$$\text{Maximize } B(P)-C(P) \quad [i]$$

{P}

where “P” stands for the amount of reduction in the pollutant in question, B(P) represents the benefits achieved given the amount of emissions reduction P, and C(P) represents the

²¹ See, for example, the case study on the RIA for the lead phase-out regulations by A. Nichols in Chapter 4 of Morgenstern (1997).

costs of achieving the emissions reduction P. The BCA-optimal degree of reduction is identified by mathematically differentiating equation (i) with respect to P and setting it equal to zero, as in equation (ii):

$$dB/dP - dC/dP = 0 \quad [ii]$$

This is known as the “first-order condition” for the optimization. One can see from equation (ii) that in the single-pollutant situation the BCA-optimal reduction for P is where the marginal benefit of P equals the marginal cost of P.

Consider now the somewhat more complex situation of performing a BCA for multiple pollutants simultaneously. For example, if two pollutants are to be controlled so that the net benefits of the combined regulations are optimized, the optimization objective is written as:

$$\begin{aligned} &\text{Maximize } B(P,H)-C(P,H) && [iii] \\ &\{P, H\} \end{aligned}$$

where P stands for reduction in the first pollutant and H for reduction in the second pollutant.²² Optimization of equation (iii) requires the simultaneous solution of two equations. The two equations are the first-order conditions for each pollutant separately, which requires differentiating the above objective function first with respect to P and second with respect to H and setting both equal to zero:

$$\partial B/\partial P - \partial C/\partial P = 0 \quad [iv-1]$$

$$\partial B/\partial H - \partial C/\partial H = 0 \quad [iv-2]$$

Equations (iv-1) and (iv-2) show that if the standards for multiple pollutants can be selected simultaneously, then the level at which to set each one is decided without any reference to the changes in benefits or costs that would come from any other of the simultaneously-regulated pollutants. In other words, there is no term in the joint optimality conditions that represents co-benefits from any of the other pollutants. The decision about levels for H must be made assuming that P will be set at its optimal level, and vice versa.

In reality, the CAA does not give EPA the ability to actually set regulations in this simultaneous manner. The optimality rules become more complex when one must choose the level of one pollutant given that levels of other, interrelated pollutants have been *separately* established. This more realistic BCA situation can be represented by building on the two-pollutant BCA optimization of equation [iii]. Assume now that the

²² For a person who wishes to read through this section with a specific emissions control policy example in mind, think of P as representing PM_{2.5}, and think of H as representing an air toxic such as mercury. As PM_{2.5} precursors are reduced, so too is mercury, and vice versa. Total benefits, B, are a function of the amount of reduction in both PM_{2.5} and mercury. Similarly, changes in policy costs, C, are a function of the reduction in both PM_{2.5} and mercury that must be achieved.

policymaker can only control one pollutant, say by choosing H, while accepting that P has already been regulated to the level $P=P^N$. Now, the optimization objective is stated as:

$$\text{Maximize } B(P^N, H) - C(P^N, H) \quad [v]$$

$$\{H\}$$

This gives rise to a single first-order condition for H which replaces the first-order condition for H in equation (iv-2):

$$(\partial B/\partial H - \partial C/\partial H) = (\partial C/\partial P - \partial B/\partial P) * dP/dH, \text{ evaluated at } P=P^N \quad [vi]$$

Equation (vi) is similar to equation (iv-2), except that instead of choosing H make the left-hand side (LHS) equal to zero, the LHS now should be made equal to the complex term that appears on the right-hand side (RHS) of equation (vi). This RHS term includes the marginal cost and marginal benefit of P (evaluated at $P=P^N$), as well as a term dP/dH . These elements of the RHS term clearly relate to co-benefits from the other separately-regulated pollutant, P, but the RHS term is not equal to P's co-benefits, specifically.²³ Thus, equation (vi) reveals how interactions of P with H would properly be incorporated into a BCA for choosing the optimal level of H. One cannot conclude that there is a theoretical rationale for including co-benefits of separately-regulated pollutants in a BCA for setting a standard on another pollutant unless the term on the RHS of equation (vi) were equal to what EPA calculates as co-benefits (*i.e.*, $\partial B/\partial P * dP/dH$), which it clearly is not. In fact, as will be explained below, equation (vi) actually implies that adding a separately-regulated pollutant's co-benefits into a BCA-based evaluation of any other pollutant regulation can drive the resulting regulation in the opposite direction from what BCA would advise.

The first thing to note about the RHS term of equation (vi) is that if P^N were to be set at the BCA-optimal level for P, then the RHS would equal zero.²⁴ In that case, equation (vi) would be identical to equation (iv-2), thus indicating that H should be selected without any reference to any co-effects from P. In this case, co-benefits of a separately-regulated pollutant clearly should not play any role in the BCA for another pollutant.

Consider now the alternative case in which P^N is *not* set at the BCA-optimal level for P. Although the RHS of equation (vi) is non-zero in this case, it still indicates that EPA's method of including co-benefits from P in a BCA for H is inappropriate. The non-zero RHS term can be interpreted as a measure of the degree to which the non-optimized choice of P *diverges* from its own first-best optimality condition, multiplied by dP/dH . If

²³ For example, dP/dH , which stands for the change in the level of P when H is changed, reflects coincidental additional reduction in P beyond its existing reduction level for the given reduction requirement on H. In other words, dP/dH is the physical *co-reduction* in P when a standard is set on H. Thus, the *co-benefit* from coincidental changes in P that result from choosing a level for H, which is what EPA includes in its RIAs, would be equal to $\partial B/\partial P * dP/dH$.

²⁴ This is because the portion of the RHS term in parentheses is consistent with the LHS of equation (iv-1), and thus equals zero if P^N is set at its optimal level where its own marginal costs equal its own marginal benefits, as prescribed by equation (iv-1).

co-benefits are positive, and P^N is over-controlled with respect to its BCA-optimal level, then the RHS of equation (vi) will be positive, because over-control occurs when the marginal costs of P have exceeded its marginal benefits, which in turn means that $(\partial C/\partial H - \partial B/\partial H) > 0$.²⁵ Thus, if P has been *over*-controlled with respect of its BCA optimum, equation (vi) tells us that H should then be *under*-controlled with respect to its BCA optimum. That is, the LHS of equation (vi) will need to be positive too, which means that H should not be controlled beyond a point where its own marginal benefits become equal to or less than its own marginal costs.²⁶ Similarly, if P is under-controlled, then equation (vi) indicates that H should be controlled to a level beyond its own BCA-optimum.

The case of P being *over*-controlled is of particular interest and relevance to $PM_{2.5}$ co-benefits. This is because the legal framework that determines what the EPA Administrator must select as a standard for ambient $PM_{2.5}$ clearly drives towards over-control of $PM_{2.5}$ with respect to its own BCA optimum.²⁷ The key implication is that if $PM_{2.5}$ co-benefits should be considered at all in a BCA for another pollutant (such as air toxics or ozone), they should enter as a *negative* term on the benefits side of the ledger, and not as a positive one. Thus, estimates of the co-benefits from $PM_{2.5}$ specifically should not be incorporated into BCAs for any other pollutants being controlled under the CAA, and when they are included, this only serves to drive other policies *away* from what would be their appropriate level, based on BCA principles.

In fact, this conclusion can be stated more broadly, that co-benefits from any pollutant that is regulated as a criteria pollutant with a NAAQS that conforms with the requirements of CAA Section 109 should not be included in the BCA of any other pollutant. This is because Section 109 requires over-control of all criteria pollutants with respect to their own BCA optima, due to Section 109's requirement for an adequate margin of safety when setting a NAAQS.

²⁵ Because co-benefits are positive, dP/dH is positive (*i.e.*, if pollutant H is reduced, then pollutant P is also reduced), so the sign of the term on the RHS is equal to the sign of the term in parentheses.

²⁶ The degree of under-control of H is not easily inferred without more situation-specific technical information, but the term that determines how much to over- or under-control H is not determined by simply adding the monetary co-benefits of P to the BCA for H. One can determine however that the amount of adjustment is *less* than the monetary co-benefits of P: the adjustment should be equal to $(\partial C/\partial P - \partial B/\partial P) * dP/dH$, and this is less than $\partial B/\partial P * dP/dH$, which is the monetary measure of co-benefits of P.

²⁷ The requirement that a NAAQS be set at a level that protects the public health with a margin of safety is equivalent to saying that it should be set at a point where no further material public health improvements would be expected to be gained by tightening the standard any further. In fact, the "margin of safety" implies that the standard be set a notch beyond that point of no further expected gains. In terms of a benefits curve, this would be a point where the benefits curve (as a function of ambient pollutant reduction) becomes flat, or at least that there is a significant probability in the mind of the Administrator that it is flat by that point. When the benefits curve is flat, marginal benefit equals zero. However, since marginal costs will always be increasing for all levels of reduction up to 100% reduction, the point where the Administrator must set the NAAQS is a point where marginal costs are greater than marginal benefits. Thus the point where marginal cost *equals* marginal benefit, which defines the BCA-optimal degree of reduction, is at a lesser level of than what the CAA requires for a NAAQS determination.

The fact that the CAA results in over-control of criteria pollutants *with respect to their BCA-optima* can be no surprise to analysts familiar with BCA. However, it might be useful to bring this point into the open in the way RIAs are conducted for criteria pollutants (and any other pollutants that must be regulated without regard to costs). If a NAAQS has indeed been set to a point where it provides an adequate margin of safety, its RIA *should* show that it fails the marginal BCA test. If RIAs are to more effectively serve in the role of providing sound and policy-relevant information to policymakers and the public about the impacts of new regulations, they should strive to actually quantify the net cost that our society is paying for that margin of safety in its air pollution rules. The purpose of recrafting CAA-related RIAs in this manner would not be to demonstrate that NAAQS rules fail a benefit-cost test that the CAA does not allow in the first place; rather, it would be to inform ourselves about the *degree* to which we are accepting higher costs in order to have the extra degree of public health safety that is mandated by the CAA, and apparently desired by the public.

To summarize, the analysis in this section establishes that EPA's practice of adding co-benefits of pollutants that are separately subject to policymaker control into the total benefits reported in RIAs for other pollutants is not supported by BCA theory. This conclusion holds whether or not the other pollutants are being regulated to their own BCA-optimal levels. This analysis has also demonstrated that EPA's practice of including co-benefits of criteria pollutants in its RIAs actually intensifies the pressure towards over-control relative to what BCA would prescribe that already exists in the standard-setting requirements of the CAA.

5. EPA's Use of PM_{2.5} Co-Benefits Subverts the Practical Purpose of RIAs as an Informational Device for Improving Policy Making

As noted in Section 2, RIAs are no longer viewed as having to justify policy decisions based solely on BCA principles. They are now viewed as assessments that should help inform the public and policymakers on a variety of impacts that can be anticipated to result from each new major regulation, all of which are viewed as having relevance to societal objectives. People who feel that this structured informational role of RIAs is more important than its BCA-related role *per se* may argue that there is merit in understanding each rule's co-benefits, even those that come from separately-regulated co-pollutants such as PM_{2.5}. Unfortunately, EPA's use of PM_{2.5} benefits as the predominant (often the *only*) quantified benefit in RIAs undercuts the objective of providing the public with a transparent understanding of the relative merits of each type of regulation.

Quantification of PM_{2.5} co-benefits creates particular problems when they are reported as part of the total benefits of a rule in the Executive Summary of an RIA. Including them in the summary of the RIA, which is where the benefits of a rule are compared to its costs, and then also including them in public announcements about net benefits and benefit-cost ratios of new rules creates confusion for the public and other audiences who have little time to study the details of the underlying analysis in each RIA. An example of the confusion that can be created is in the following quote from the EPA Administrator

regarding the benefits case for controlling air toxics from electricity generating units (EGUs), known as the “Proposed EGU MACT” rule:

When these new standards are finalized, they will assist in preventing 11,000 heart attacks, 17,000 premature deaths, 120,000 cases of childhood asthma symptoms and approximately 11,000 fewer cases of acute bronchitis among children each year. Hospital visits will be reduced and nearly 850,000 fewer days of work will be missed due to illness.²⁸

The fact is that every one of the benefits in the quote above comes from EPA’s predicted PM_{2.5} co-benefits, and not from any of the reductions in air toxics that are the purpose of that rule.²⁹ Anyone with the time and interest can read the RIA from which these numbers came, and may realize that these are PM_{2.5}-related benefits. However, not every such motivated reader would also realize that PM_{2.5} is not an air toxic, and that those PM_{2.5}-related benefits would be more certain and more cost-effectively obtained through a different regulation altogether than an air toxics rule (*i.e.*, through the PM_{2.5} NAAQS). By including those PM_{2.5} co-benefits as part of the “total benefits” reported in the RIA for the Proposed EGU MACT rule, EPA encourages this misunderstanding.³⁰ The statements can become even more misleading in the hands of advocates outside of EPA. For example, the following statement in testimony by an advocate outside of EPA before the House Energy and Commerce Committee on the Proposed EGU MACT rule gives the distinct impression to readers that air toxics from power plants are killing and otherwise harming massive numbers of people:

EPA’s proposed mercury and air toxics standards for power plants that burn coal and oil are projected to save as many as 17,000 American lives every year by 2015. These standards also will prevent up to 11,000 cases of heart attacks, 120,000 cases of asthma attacks, 11,000 cases of acute bronchitis among children, 12,000 emergency room and hospital visits and 850,000 lost work days every year.³¹

²⁸ Quote from Administrator Jackson in EPA Air News Release (HQ), “EPA Extends Public Comment on Mercury and Air Toxics Standards,” June 21, 2011. (In the RIA for the final EGU MACT rule the “17,000 premature deaths” has been reduced to 11,000, but this quote was made before the RIA for the final EGU MACT was released in December 2011.)

²⁹ This can be confirmed by reviewing the RIA for the Proposed EGU MACT rule, which is EPA (2011a).

³⁰ Any counter-argument that co-benefits are essential to add into an RIA’s statement of a new rule’s total benefits would require one to argue that RIAs have a BCA function. As has been demonstrated above, the principles of BCA, when scrutinized, prescribe that co-benefits of an already-regulated pollutant not be added in the comparison of benefits and costs of a regulation for a different pollutant.

³¹ John D. Walke, Natural Resources Defense Council, Testimony at Hearing on “Recent EPA Rulemakings Relating To Boilers, Cement Manufacturing Plants, And Utilities,” before the Subcommittee on Energy and Power, Committee on Energy and Commerce, U. S. House of Representatives, April 15, 2011.

EPA's reliance on co-benefits estimates thus undercuts the transparency that RIAs are supposed to bring to assessments of the impacts of new rules. Lack of transparency enables misleading advocacy.

Reliance on co-benefits creates another problem that undercuts the practical value of RIAs: it shields EPA from pressure to improve its ability to describe, characterize and even quantify the health and welfare benefits for the other pollutants that it is charged with regulating. The EGU MACT RIA is not the worst case in this regard. As Figure 1 shows, during the period 2009-2011, EPA released 13 CAA-related RIAs that provided quantitative estimates of benefits and in which PM_{2.5} was a co-benefit rather than a direct benefit. PM_{2.5} co-benefits accounted for more than half of the total benefits in all but 2 of the 13.³² EPA did not even attempt to quantify the direct benefits in 6 of those RIAs: PM_{2.5} co-benefits accounted for 100% of the total benefits identified in those RIAs. In two more of those 13 RIAs, although some direct benefits estimates were provided, PM_{2.5} co-benefits accounted for more than 99% of the total reported benefits. In essence, EPA has been abdicating its responsibility to make a clear direct benefits case for its air rules, particularly those for air toxics. Furthermore, although EPA has quantified direct benefits for the new standards it has set for other criteria pollutants, it is relying on its PM_{2.5} co-benefits estimates to create its case that those other NAAQS revisions will produce benefits greater than their costs, when in fact their direct benefits are often miniscule compared to their costs.

Clearly, EPA's PM_{2.5} co-benefits habit is allowing EPA to avoid grappling with the important task of making a case that all of these other pollutants really require tighter controls. It may be possible for that case to be made for some of those pollutants, but a high degree of complacency and analytical laziness has instead taken root as EPA has found it can more easily rely on simplistically-derived estimates of co-benefits from a pollutant that it has every authority it needs, and indeed the legal *requirement*, to directly regulate to levels that are safe for the public health. This situation is completely at odds with the purpose of RIAs, which is to provide a consistent, credible and thoughtful evaluation of the societal value gained with the increased regulatory burden that new rulemakings create. It also stymies scientific progress in risk assessment techniques and associated knowledge.

In summary, PM_{2.5} co-benefits have become a device for keeping some regulations of dubious public policy value from transparent scrutiny. Although many of those regulations may be mandated by law, the degree of stringency imposed requires judgment by the Administrator, and co-benefits may be masking judgments that would not otherwise pass scrutiny. On a longer-term basis, this practice is also preventing RIAs from playing their most meaningful practical role, which is to help the policymaking

³² The two RIAs out of the 13 rulemakings during 2009-2011 in which PM_{2.5} co-benefits were not the predominant form of quantified benefits were rules to reduce mobile source greenhouse gases. EPA estimated negative costs for both of those greenhouse gas rules, so their benefits would have exceeded their costs even with zero direct benefits. EPA has developed a method for quantifying benefits from reductions in greenhouse gases, so while it does also report PM_{2.5} co-benefits in those two RIAs, the emphasis on them as the benefit-cost justification for the rule is less pronounced.

community identify those frameworks for regulation that may not be providing the value that they were originally expected to provide when enacted or otherwise established. Including PM_{2.5} co-benefits in all air-related RIAs thus is preventing any meaningful identification of ways to reduce regulatory burdens while still meeting our national air quality objectives.

6. PM_{2.5} Risk Estimates Have Become Increasingly Less Credible as EPA's Use of Them as Co-Benefits in Non-PM RIAs Has Grown

This paper has demonstrated that BCA theory does not support the role that EPA gives to PM_{2.5} co-benefits, and that excessive reliance on them in its regulatory justifications is undercutting the more general purposes of RIAs for guiding policy making. This review of EPA's RIAs has also found that as EPA has used PM_{2.5} co-benefits to justify more and more of its non-PM_{2.5} rules, it has also moved to less and less scientifically-credible methods for estimating those co-benefits. These changes in methodology and assumptions have inflated the PM_{2.5} co-benefits estimates dramatically (and also the direct PM_{2.5} benefits estimates in rulemakings targeting PM_{2.5}).

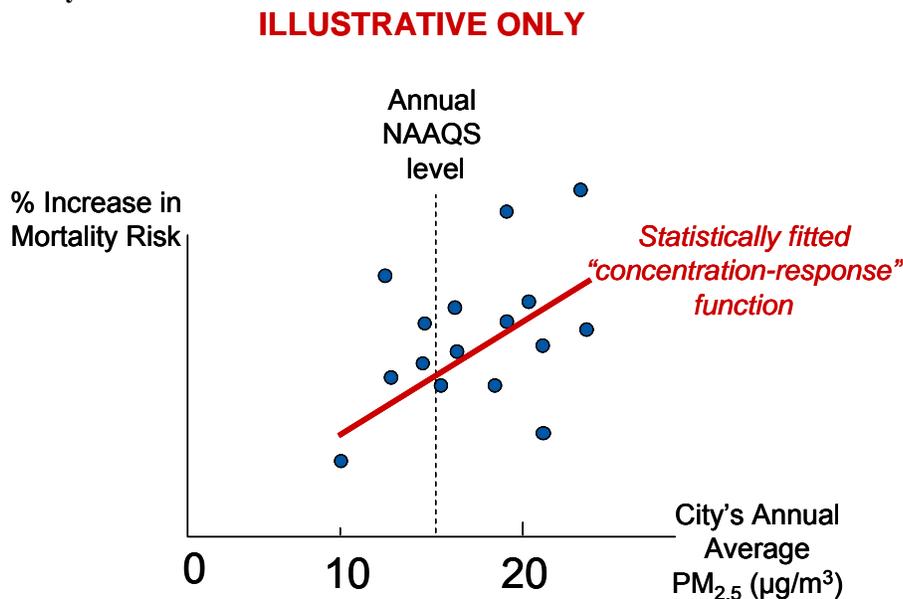
To explain the methodological issues, it is necessary that the reader first understand the general elements of the scientific basis for EPA's PM_{2.5} risk calculations. These calculations are all based on the presumption that statistical correlations between health effects levels and ambient air quality are causal in nature. The illustrative example of how these studies are performed and then used to calculate risk changes from hypothetical changes in ambient PM_{2.5} levels will be based on the "chronic exposure" studies that are the starting point for EPA's estimates of mortality risks from PM_{2.5}. The focus of this discussion will be on mortality because PM_{2.5} mortality benefits estimates account for over 90% (and as much as 97%) of the total PM_{2.5}-health benefits estimates in EPA's RIAs. All of the categories of PM_{2.5} morbidity benefits account for as little as 3% of the estimated PM_{2.5} co-benefits estimates, particularly when the upper-bound mortality estimates are reported.³³

For the chronic exposure mortality-risk studies, a database is created of many individuals living in multiple cities across the US. Researchers then track the survival outcomes of those individuals over time to build up estimates of the relative mortality risk at each age level in each of the different cities where these individuals, or "cohorts" live. After enough deaths have been observed (which can require as much as a decade), the researchers assess whether a statistical correlation exists between the estimated relative mortality risk in each city and the cities' average ambient PM_{2.5} concentrations. This statistical analysis also attempts to control for all the other major factors that contribute to mortality risk, which is a daunting and perhaps elusive goal, given limitations in the availability of the relevant data.

³³ A single morbidity category, chronic bronchitis, accounts for about half of all the morbidity benefits value. EPA's estimates of chronic bronchitis risks are also based on a "chronic exposure" type of study. The rest of the morbidity benefits (as little as 1.5% of the total PM_{2.5} co-benefits) are based on "acute exposure" types of studies that differ in a number of ways from the illustrative example that this section provides.

Figure 2 provides a simplified illustration of the way that population risk information from the chronic exposure studies produces an equation that EPA uses to calculate mortality risks from current and changed levels of PM_{2.5}. Each dot in the figure represents the percent increase in mortality risk for an entire city,³⁴ plotted against each city's respective annual average monitored ambient PM_{2.5} concentration. The heights of the dots on the vertical axis should be viewed as the percent differences in mortality risks that remain across the cities after first controlling for and removing other risk factors for which data can be obtained (*e.g.*, age, income level, smoking status, weight, local climate, *etc.*). The placement of each dot on the horizontal axis reflects that city's average concentration of ambient PM_{2.5} as measured at central monitoring stations. The statistical analysis then estimates the line through these data points that provides the most likely explanation of their scatter.³⁵ The most important attribute of this line is its slope, *i.e.*, the percent risk increase per additional $\mu\text{g}/\text{m}^3$ of ambient PM_{2.5}. The estimated slope is a single constant percent per $\mu\text{g}/\text{m}^3$ from the city with the lowest measured PM_{2.5} concentration to that with the highest measured concentration. This statistically-fitted curve is called the “concentration-response” function, because it associates risk with city-wide concentrations of ambient PM_{2.5} measured at monitoring stations. Monitored concentrations serve as rough proxies for individuals' exposures to PM_{2.5}, which certainly vary among the individuals within a city, but are not known.

Figure 2. Illustration of Basis of Concentration-Response Functions in Cross-City Comparisons of Relative Mortality Risks



³⁴ The absolute risk varies by age, but the statistical estimation method used in the PM_{2.5} chronic exposure mortality studies (the “Cox Proportional Hazards model”) assumes that any increase in risk due to a city's average ambient PM_{2.5} level increases risk by the same proportion for all age groups. Thus, relative risks can be summarized as a single dot for each city in the study.

³⁵ The formula for the fitted relationship is in a “log-linear” form. It appears as a line in the illustrative figure because the y-axis units are the *percentage* increase in risk per increment of PM_{2.5} concentration, which is stated in absolute units.

The ambient concentrations observed in each study fall within a range. For example, in the illustrative figure, the range is roughly from 10 $\mu\text{g}/\text{m}^3$ to 25 $\mu\text{g}/\text{m}^3$. There is no factual evidence to indicate the shape of the concentration-response function below or above this range, which is why the red line in Figure 2 does not extend beyond the range of the observed data. A term that will figure prominently in the discussion below is the *lowest measured level* (LML) of ambient average $\text{PM}_{2.5}$. This term refers to the average city-wide $\text{PM}_{2.5}$ concentration of the “cleanest” city in the dataset from which a concentration-response function has been estimated. In the illustrative figure, the LML is about 10 $\mu\text{g}/\text{m}^3$.

Each chronic exposure study has a different number of cities, or data points. One widely-cited database is called the American Cancer Society (ACS) cohort. This database includes people (the “cohort”) residing in over a hundred cities. Statistical estimates of a $\text{PM}_{2.5}$ -mortality slope based on the ACS cohort usually include from 50 to 160 cities, or data points as shown in Figure 2. A commonly-cited slope estimate based on the ACS cohort is from Pope *et al.* (2002), which is often used by EPA to estimate its lower bound $\text{PM}_{2.5}$ mortality benefits estimates. Another widely-cited database is the Harvard “Six-Cities” cohort. As its name implies, it offers a slope estimated from only six data points. One slope estimate based on the Six Cities cohort is reported in Laden *et al.* (2006), which is notable here as the study on which EPA presently bases its upper bound $\text{PM}_{2.5}$ mortality benefits estimates.

Most (but not all) $\text{PM}_{2.5}$ researchers studying the ACS, Six-Cities, and several other cohort datasets have reported that the estimated slope of the concentration-response curve is positive, and statistically significant. However, this fact does not eliminate uncertainties about the size of the risk, nor about whether the association is causal. A discussion of the many uncertainties that remain is provided before turning to how the estimated relationship is being used by EPA to predict benefits from regulations that would reduce ambient concentrations of $\text{PM}_{2.5}$ in certain areas of the US.

First, there are usually relatively few data points near the upper and lower ends of the range of ambient $\text{PM}_{2.5}$ data. This causes the confidence interval on the slope estimate to widen progressively as one moves from concentrations near the average among the cities studied out to the extremes of the observed data. The confidence interval on the relative risk associated with cities with concentrations at the LML may be very wide. This means that the slope may be lower or higher than that which has been estimated over the full range of data. One may not even be able to statistically assert that the $\text{PM}_{2.5}$ -risk relationship is non-zero for concentrations at or near the LML, even when the average slope estimated over the full range of $\text{PM}_{2.5}$ levels is statistically significant. Thus, there is much greater uncertainty about the size of the $\text{PM}_{2.5}$ effect at lower ambient concentrations, such as at the LML, than is usually acknowledged.

Second, data point “scatter” lies beneath the average relationship that the fitted line summarizes. This implies that the estimated concentration-response curve will be a poorer predictor of the change in risk that will be experienced in any specific city than it may be for predicting average risks over many different cities. It also follows that

uncertainties in predicted responses to reductions in PM_{2.5} levels in cities that were not in the original dataset will be even greater than the statistical confidence intervals imply.

Third, there is great uncertainty on the true shape of the concentration-response relationship. Researchers report they have not been able to identify any shape that is statistically superior to the log-linear form, but this does not mean that the actual relationship is log-linear. There are numerous problems in the quality of the data being used that can undercut the ability to detect shape. Thus, even within the observed data range, uncertainty remains about the shape of the estimated concentration-response function. However, there is no ability at all to determine statistically whether or not the slope of the curve continues unchanged below the LML, as ambient PM_{2.5} concentrations approach near-zero levels, because there are no observations in that range at all.³⁶ In situations such as this, researchers usually attempt to use mechanistic understanding of the phenomenon being estimated to guide shape assumptions. However, EPA has not been able to provide any mechanistic explanation of how current ambient levels of PM_{2.5} may increase risk of death, and so there can be no help from mechanistic reasoning. Any extrapolation of the concentration-response relationship below the LML is therefore subject to much greater uncertainty than the statistical confidence intervals might suggest.

Fourth, there is substantial uncertainty in defining the appropriate concentration to serve as the best proxy for levels of PM_{2.5} to which the individuals in the study cohort have been exposed.³⁷ EPA now states that the LML for the ACS cohort is 7.5 µg/m³, and 10 µg/m³ for the Six-Cities cohort. However, the LML for the ACS cohort averaged about 10 µg/m³ during 1979-1983, which spans the time that cohort was recruited (in 1982). The LML for the Six-Cities cohort averaged about 11 µg/m³ during 1979-1985, although that cohort was recruited earlier still, in 1974-1977. But even relying on these earlier, higher concentration levels as estimates of the levels that might account for observed differences in mortality risk levels is open to question. Recall that the estimates of differences in mortality risk across cities are built up by following the survival outcomes of the people in each city over many years. This means that the observations of their mortality risks at each age, if attributable to air pollution at all, could be a result of exposures they experienced many years in the past, or that they accumulated over a long period of time.

Take the ACS cohort as an example. The ACS cohort was first established in 1982. At the time that the individuals were recruited for the ACS study, they had to be at least 30 years old and their average age in 1982 was 56 years. Thus all of the individuals in the ACS database had been exposed to US pollution levels since at least 1952 (*i.e.*, 30 years before 1982), and the average individual in the database experienced US pollution levels

³⁶ Efforts to explore shape near the LML have produced inconsistent results. Some researchers report finding upward curvature and others report finding downward curvature. At present, no consensus or weight of evidence can be said to exist on this matter.

³⁷ Recall also that the term “concentration-response function” is used to remind us that none of these studies actually measure what *exposures* the individuals tracked were receiving. An assumption is made that the average exposure across individuals in each city can be approximated by the readings at that city’s ambient monitors. All individuals in a city are assumed to be exposed to the same concentrations.

dating back to 1926. As researchers using the ACS database have stated “In the 1950s, levels of air pollution in most North American and European cities were 10 to 50 times higher than those found today.”³⁸ Since the mortality risk estimated for each city is based on many years of tracking these people, recent average PM_{2.5} concentrations such as those in 2000 cannot be viewed as indicative of the PM_{2.5} exposure level that most affected their observed survival outcomes. Those individuals who had not already died by 2000 would have already lived at least 44 years of their lives while being exposed to earlier, higher PM_{2.5} levels. To say that the estimated mortality-risk relationship has been observed down to the level of the lowest PM_{2.5} concentration most recently measured in any of these cities is close to assuming that recent lower levels of PM_{2.5} accounted for the health outcomes of people who died as much as several decades ago. The same issues are present with the Six-Cities and all other cohorts being used in PM_{2.5} epidemiological studies of risks due to chronic exposures to PM_{2.5}.

Fifth, none of the PM_{2.5}-risk estimates that EPA relies on for a concentration-response function slope assumption has been estimated while also accounting for the relative levels of pollutants other than PM_{2.5}. The presumption is being made that PM_{2.5} is the sole air pollutant contributing to observations of an increased average mortality risk associated with higher average ambient pollution. Nevertheless, some studies have controlled for other pollutants. For example, the ACS cohort’s slope with respect to PM_{2.5} was found in 2000 to be much smaller and statistically insignificant when another pollutant (SO₂) was included in the analysis.³⁹ Since 2000, not a single study based on that ACS cohort has reported an estimate of the PM_{2.5} slope that came from a model that also accounted for SO₂.

Sixth, unlike other pollutants, the chemical and physical composition of PM_{2.5} varies over space and time,⁴⁰ but none of these statistical studies have sufficient data yet to try to determine the degree to which some PM_{2.5} constituents account for more of the observed associations than other constituents. The concentration-response functions that EPA constructs from these types of epidemiological studies all assume that every one of the multiple types of PM_{2.5} is equally potent. This assumption is not realistic when one considers the wide variety in the chemical properties of the many major components of ambient PM_{2.5} concentrations. This fact creates substantial unquantified uncertainty in estimates of benefits from future decreases in PM_{2.5} concentrations except in the unlikely case there all the individual constituents would be reduced by the same percentage. This source of uncertainty in benefits estimates becomes particularly extreme for regulations would only reduce one type of PM_{2.5} constituent. Reduction of that single type of PM_{2.5}

³⁸ Krewski *et al.* (2000), p. 33.

³⁹ Krewski *et al.* (2000).

⁴⁰ PM_{2.5} comprises all compounds in the ambient air that are not in the form of a gas; it includes compounds that are as physically different as solid particles and very fine liquid droplets. Chemically, the constituents that may be found in the ambient mass that counts as PM_{2.5} include a diversity of compounds including dust, soot (elemental carbon), sulfates, nitrates, and secondary organic compounds. Some are soluble and some are insoluble, and each has its own distinct physiological impact when inhaled. Not only is PM_{2.5} a hodge-podge of compounds, but the mix differs dramatically from location to location and temporally at any given location.

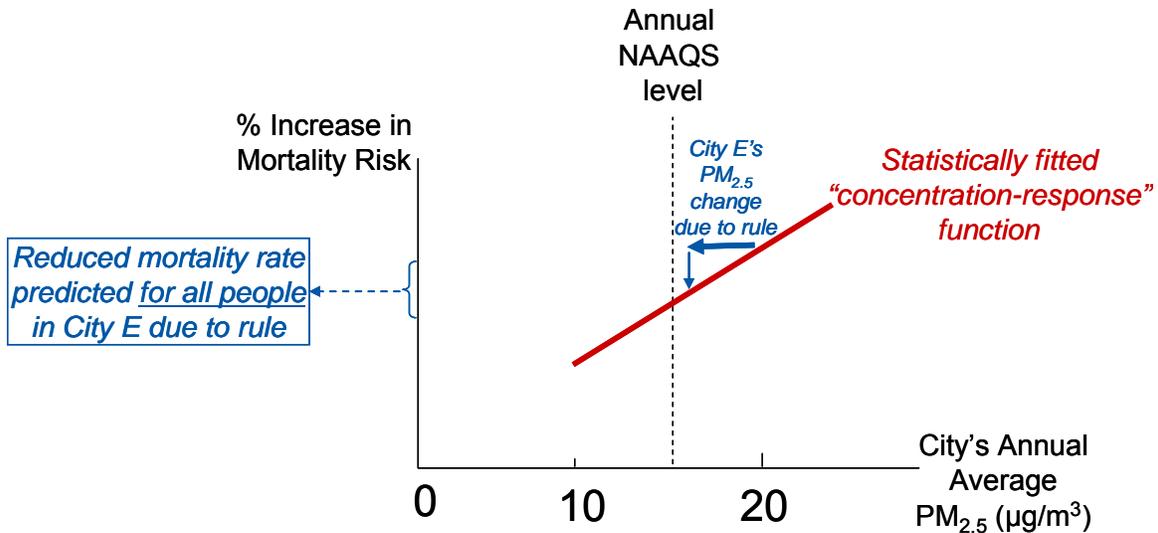
could have no effect at all on health, or it could have even greater effect than EPA predicts using its equal-toxicity assumption. EPA has never attempted to quantify this uncertainty in any of its benefits estimates. This is a particular concern for co-benefits estimates in RIAs for non-PM rules, because co-benefits are often based on changes in a single constituent that happens to be linked with reductions of the non-PM pollutant. For example, almost all of the 11,000 deaths attributable to PM_{2.5} co-benefits in the Final EGU MACT rule RIA are due to reductions in sulfates alone.

These six uncertainties represent just a few of the uncertainties that exist for the “concentration-response” function’s ability to predict how much mortality will be reduced if national ambient PM_{2.5} is decreased. At present, the only statement of statistical uncertainty that EPA provides for an estimate based on any individual epidemiological study reflects only the statistical confidence of the overall slope of the estimate. As explained above, this is not an appropriate measure of the uncertainty of predictions of risk at concentrations at the extreme ends of the observed dataset, such as those at and just above the LML. But these six types of technical problems imply larger uncertainties than even the expanded confidence bounds would imply, and undercut confidence in interpreting the statistical association as causal in nature. Nevertheless, EPA uses the slope estimates from these studies to predict risk from changes in PM_{2.5} that will occur in the future, and in many locations that were not even studied, as explained next.

The next figure, Figure 3, illustrates how EPA uses the slope that is estimated from cohort studies to project PM_{2.5} deaths due to changes in baseline PM_{2.5} levels. *First, and foremost, EPA starts by presuming that the statistically-estimated concentration-response slope represents a causal relationship with PM_{2.5} and that pollutant alone.* As the figure shows, EPA just takes the average slope from one of the studies, and then determines how much the mortality rate in a given city (not necessarily one in the original study) will be reduced if its ambient PM_{2.5} concentrations are reduced. Consider, for example, “City E” in the illustrative figure, which has a baseline annual average PM_{2.5} concentration of 20 µg/m³ that is projected to decline to about 16 µg/m³ under an hypothetical regulation. EPA’s risk assessment calculation for that regulation would assume that every person residing in City E will experience a drop in mortality risk equal to the vertical drop along the concentration-response function, as indicated by the blue arrows in Figure 3.

Figure 3. Illustration of Calculation of Reduced Premature Deaths Due to Changes in Predicted Ambient Average PM_{2.5} Levels in a City (“City E”)

ILLUSTRATIVE ONLY



In its national benefits analyses, EPA performs the same computation as shown for illustrative “City E” for the changes in PM_{2.5} concentrations that EPA projects for every county in the US. All those changes – assumed to benefit every resident of any county or city in which a change in PM_{2.5} is projected to occur – are added up to produce EPA’s estimate of the national reduction in deaths due to PM_{2.5} from a regulation. Very small changes in PM_{2.5} (and therefore in PM_{2.5}-related mortality risk) thus can produce very large changes in estimated premature deaths, if spread over a population of about 300 million people. For example, the 11,000 deaths that EPA attributes as co-benefits in the RIA for the Final EGU MACT rule involve median changes in PM_{2.5} concentrations of 0.36 µg/m³ at simulated monitors.⁴¹ Further, (as will be shown later in this paper) almost all of those small changes in ambient concentrations occur at very low levels of baseline PM_{2.5} concentrations – levels for which no observed concentration-response function exists.

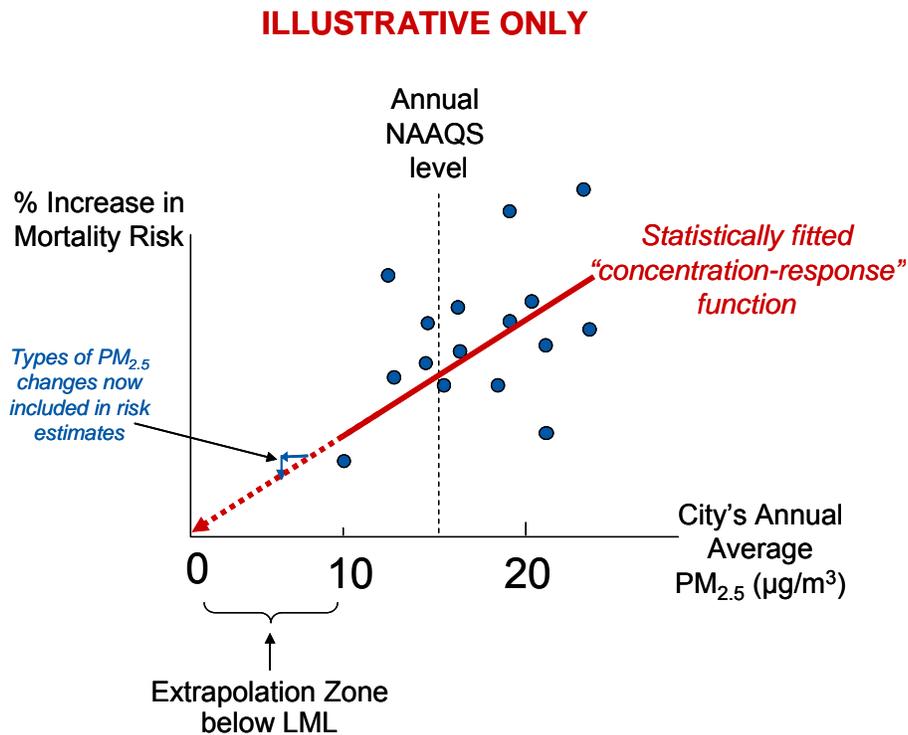
It should be apparent from the discussion above that EPA’s estimates of the benefits from regulations that will reduce concentrations of PM_{2.5} in certain locations are fraught with uncertainties, even for changes in PM_{2.5} concentrations that occur above the most recently measured LML. However, in 2009, EPA modified its PM_{2.5}-mortality risk formula in a way that greatly increased its benefits estimates. In the illustrative examples above, risks were not computed for changes below the LML in the underlying epidemiological studies. That is, if ambient PM_{2.5} in a location was already below the end of the curve (e.g., at 10 µg/m³ in the figures), then prior to 2009, EPA did not assume

⁴¹ See EPA (2011c), p. 5B-4.

there was any further potential for PM_{2.5}-related mortality risk reduction in that location. However, starting in 2009, EPA decided that it would calculate risks to the lowest level projected by its air quality models, even though no observed or empirical evidence exists for what the slope of the concentration-response may be in that low-concentration zone.

Figure 4 shows this methodological change, building on the graphical illustrations of Figure 2 and Figure 3. Instead of calculating risks only in areas with PM_{2.5} down to the LML of the study – the point at which all scientific evidence of a statistical association ends – EPA now assumes risks continue *at the same rates* to levels well below the range in which there is any scientific evidence to support those calculations. “Extrapolation” is the use of quantitative relationships outside of the range of evidence on which it was based.⁴²

Figure 4. Illustration of the Extrapolation EPA Is Now Using to Calculate Risks in Areas with Ambient PM_{2.5} Concentrations below the Lowest Measured Level in the Original Statistical Study



⁴² Most elementary lectures on statistical methods such as regression warn the students that extrapolation of any statistically-derived relationships is a highly dubious exercise. For example, a summary of statistical regression methods carries the following warning: “After computing the regression line, you must not use it to predict values of the response for values of the explanatory variable outside the range of the data used to compute the line in the first place. This practice, called extrapolation, is dangerous because the original data can only produce a formula that describes the association for values found in the original data” (see http://emp.byui.edu/BrownD/Stats-intro/dscrptv/dscrptv_2_qunt_vars/smpl_lnr_rgrsn_ref.htm, accessed November 22, 2011, emphasis in original.)

In other words, in 2009, EPA suddenly started including an entirely new set of presumed risks in its RIAs, based entirely on an extrapolation that has little to no scientific support and without assessing the statistical confidence for predictions of risk changes even at the LMLs of the studies that EPA started from. This created a major change in the level of national mortality estimated to be due to PM_{2.5} that EPA is assuming, because the majority of the US population resides in locations where ambient PM_{2.5} concentrations are below 10 µg/m³. Where EPA previously did not presume any risk for those people, EPA is now attributing as much health benefit per person in those areas from very small changes in PM_{2.5} (e.g., 0.36 µg/m³) as it attributes per person for the same size change in areas that have ambient levels above the LML, and even in areas with PM_{2.5} exceeding the “safe” PM_{2.5} NAAQS level of 15 µg/m³.

EPA’s change in its risk analysis assumptions also dramatically inflated its estimates of baseline mortality due to PM_{2.5} in areas with PM_{2.5} above the LML. Prior to 2009, EPA assumed their risk was elevated only in proportion to the degree that their location’s ambient PM_{2.5} concentration exceeded the LML, but after 2009, EPA started to assume their risk was elevated in proportion to the degree that their location’s ambient PM_{2.5} exceeded background levels (which EPA assumes is about 1 µg/m³).⁴³ For example, in an area with PM_{2.5} equal to 16 µg/m³, EPA used to calculate risks for an excess exposure of 6 µg/m³ (i.e., 16 minus an LML of about 10). Now, for that same population, EPA is assuming an excess exposure of 15 µg/m³ (i.e., 16 minus a background of about 1). This decision to calculate risks below the LML increased the estimated mortality risk in that illustrative type of location (i.e., one that is above the LML) by 250%.

Overall, the decision in 2009 to extrapolate risks below the LML caused EPA’s estimates of total US deaths due to PM_{2.5} to nearly quadruple. Prior to 2009, EPA was calculating (for its upper bound RIA benefits estimates) that PM_{2.5} caused up to 88,000 deaths nationwide in the relatively clean year of 2005; then, overnight in 2009, EPA changed that number to 320,000 deaths – an increase of a factor of 3.6.⁴⁴

The fact that EPA’s methodological change would increase EPA’s estimates of deaths due to PM_{2.5} in the year 2005 by a factor of 3.6 (or by about 232,000 more deaths) was never reported or peer reviewed. Although EPA points to concurrence from a committee

⁴³ “Background” level is supposed to represent the ambient concentration that would still remain if all manmade sources of emissions were to be eliminated. EPA formally uses an estimate it calls “policy-relevant background,” which is supposed to represent US ambient concentrations if all US, Canadian and Mexican manmade emissions were to be eliminated, but does allow for contributions to US ambient concentrations from emissions in other locations such as Asia, Europe and South America.

⁴⁴ The fact that this inflation from 88,000 to over 320,000 was due solely to the decision to extrapolate below the LML is directly observable in EPA’s *Quantitative Health Risk Assessment for PM_{2.5}* (EPA, 2010b). Appendix G of that document shows the 2005 national risk estimates based on the epidemiological study by Laden *et al.* when calculated just down to the LML, and also when calculated down to zero concentrations (EPA, 2010b, Table G-1, pp. G-6 to G-7). Although a risk estimate that extrapolates below the LML appears in this appendix, none of the CASAC-approved risk estimates in the main body of EPA (2010b) includes risks below the LML. The fact that EPA is actually using an estimate of 320,000 deaths due to PM_{2.5} in 2005 is explicitly stated in EPA’s RIA for the Cross-State Air Pollution Rule (EPA, 2011b, pp. 2-3).

of its Science Advisory Board (SAB) that it could extrapolate risks below the LML in its RIAs, there was no public discussion about that decision's implications for a huge inflation in the estimated number of lives that could still be saved through yet-more reductions of PM_{2.5}. There is also no evidence that the SAB committee that was asked to opine on this decision was offered any information on its quantitative implications. (Notably, that SAB committee was *not* CASAC. As a result, EPA is now using these inconsistent estimates of baseline PM_{2.5}-related deaths simultaneously in different contexts – EPA is using the smaller number of baseline deaths in its CASAC-reviewed risk analyses for the PM_{2.5} NAAQS review, and it is using the larger number of baseline deaths in its RIAs that are generating the large co-benefits for non-PM_{2.5} regulations, such as for air toxics regulations and for non-PM NAAQS, such as ozone.)

The quantitative inflation in PM_{2.5}-related mortality benefits through the non-scientific process of extrapolation below the LML is dramatic in its own right, but its lack of credibility becomes more clear when one considers what it means about the fraction of all deaths in the US that are due to PM_{2.5}. EPA's presumption that 320,000 deaths in 2005 in the US were "due to PM_{2.5}" means that over 13% of all deaths in the US *on average* were due to PM_{2.5}.⁴⁵ The estimate of 13% of all deaths may seem implausible, but the fractions at the regional level are what gives one pause. These can be seen in Figure 5, which is found in EPA's final RIA for the Proposed EGU MACT rule.⁴⁶ Its legend has been adapted here to be consistent with the upper bound PM_{2.5} mortality co-benefits estimates in the Final EGU MACT and other post-2009 RIAs. In other words, the scale shown in red font on Figure 5 is the scale that is consistent with 320,000 deaths due to PM_{2.5} in 2005. It shows that EPA is assuming as a starting point for its benefits calculations that 16% to 22% of all deaths in 2005 were due to PM_{2.5} in large expanses of the Eastern US (*i.e.*, in all of the red-colored counties on the map).

Another inference can be made from EPA's post-2009 method of extrapolating PM_{2.5}-related mortality risks below the LML. It implies that about 25% of all deaths *nationwide* were due to PM_{2.5} as recently as 1980.⁴⁷ These assumptions, which underpin EPA's co-benefits calculations, stretch the bounds of credibility, and thus undercut the credibility of all of EPA's PM_{2.5}-related mortality benefits estimates.

EPA's post-2009 baseline risks are so large because EPA now assumes that there is no tapering off of relative risk as PM_{2.5} exposure approaches zero. For years there has been a debate about whether the concentration-response relationship can truly be linear down

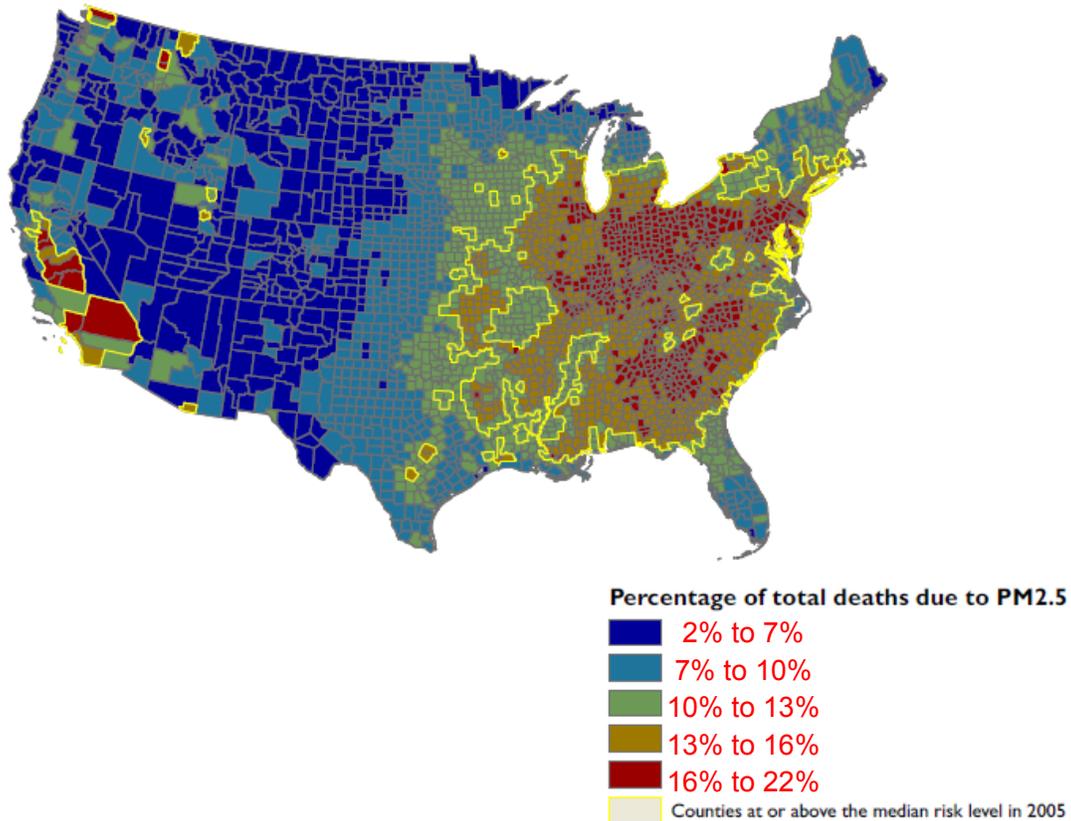
⁴⁵ In contrast, the estimate of 88,000 deaths is 4% of all US deaths. Although this starts to seem like a small number in comparison to the 13% that EPA now endorses, it is most likely also an overstatement of the true risks, for reasons discussed above, such as the difficulty in identifying the correct concentration to which elevated mortality risks should be attributed, the uncertainty in the appropriate LML to apply, and the presumption of causality itself in these risk calculations.

⁴⁶ Although this figure comes from the final RIA for the Proposed EGU MACT rule (EPA, 2011a), it is still applicable to the Final EGU MACT rule because it reports EPA's estimates of historical (*i.e.*, 2005) levels of mortality risk, which have not been affected by any of the changes in baselines or MACT-related co-reductions of ambient PM_{2.5} that occurred between the proposed and final EGU MACT rule.

⁴⁷ See Smith (2011), pp. 14-16 for how this calculation is done.

to zero, but this debate has been focused on questions of statistical power and on basic principles of toxicology. The implication of the linear-to-zero/no-threshold assumption has never been debated in terms of its implication that an implausible proportion of total deaths in the US would be due to PM_{2.5} – but perhaps now it should be debated that way too.

Figure 5. EPA-Produced Map Showing Percentage of Total Deaths due to PM_{2.5} in the Year 2005, with Legend Adjusted by Author to Represent the PM_{2.5} Risk Slope that EPA Uses for its Upper Bound PM_{2.5} Risk Calculations.⁴⁸



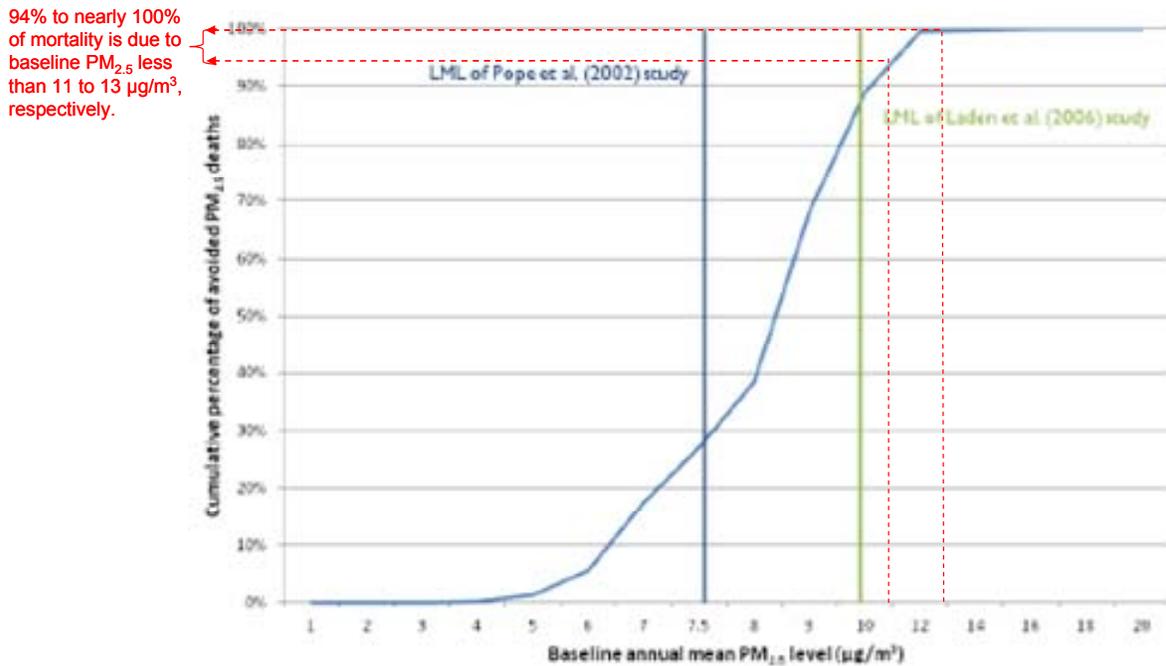
EPA’s 2009 inflation in the number of estimated “deaths due to PM_{2.5}” has its greatest impact on risks calculated for very low PM_{2.5} levels. Thus, its primary impact has been to increase co-benefits estimates for regulations that are not related to attaining the PM_{2.5} NAAQS, such as the EGU MACT standard. That is, where EPA previously estimated

⁴⁸ Figure copied from EPA (2011a), Figure C-2. However, the figure in the RIA is presented for a PM_{2.5} concentration-response slope that is not the one EPA uses to calculate its upper bound estimate of lives saved from the EGU MACT due to PM_{2.5} co-benefits. That is, the text in EPA (2011a) explaining the derivation of the figure indicates that it is based on a PM_{2.5} concentration-response slope from Krewski *et al.* (2009). EPA’s current upper bound estimates of lives saved from PM_{2.5} is based a concentration-response slope from Laden *et al.* (2006). Since the 2005 PM_{2.5} levels in each county in the map would not change (they are historical data), the risk range for the scale can readily be recalculated for the Laden *et al.* slope, as done in this paper. Smith (2011) explains how this adjustment is made.

zero co-benefits from PM_{2.5} reductions in areas already below the LML, EPA has created a reservoir of perhaps over 100,000 deaths that it can tap into as co-benefits from new non-PM regulations.

The extent to which this inflationary extrapolation enhances co-benefits estimates can be seen in Figure 6, which is taken from the RIA for the Final EGU MACT RIA.⁴⁹ This figure reports (on the vertical axis) the percentage of EPA’s estimate of the EGU MACT’s total PM_{2.5} mortality co-benefits (*i.e.*, the 11,000 lives saved) that is attributable to ambient PM_{2.5} concentrations at or below the level reported on the x-axis. It shows that nearly all of those 11,000 deaths are in populations that are in areas that are already in attainment with the current PM_{2.5} annual NAAQS of 15 µg/m³.⁵⁰ Under current EPA policy, all of those estimated deaths would be deaths of people living in areas that are protected with an “adequate margin of safety” from PM_{2.5} risks.

Figure 6. Copy of Figure 5-15 from EPA’s RIA for the Final EGU MACT Rule Showing that 94% to Nearly 100% of the PM_{2.5} Co-Benefits in that RIA Are Due to Changes in Exposures to Annual Average Ambient PM_{2.5} that Will Still Be Deemed Safe by EPA after Revising the PM_{2.5} NAAQS.



⁴⁹ EPA (2011c), Figure 5-15.

⁵⁰ This fact can be inferred from the figure in the following way. The blue S-shaped curve in Figure 6 indicates on the vertical axis the percent of the RIA’s PM_{2.5} co-benefits estimate that is attributable to baseline PM_{2.5} exposures at or below the PM_{2.5} concentration on the horizontal axis. This is known as a “cumulative distribution.” The point on the horizontal axis where the S-shaped curve just reaches 100% indicates the level of baseline PM_{2.5} at or below which *all* (*i.e.*, “100%”) of the estimated PM_{2.5} co-benefits occur. As one can see, the vertical reading on the blue S-shaped curve is about 100% at 15 µg/m³, which means that about 100% of EPA’s estimated PM_{2.5} co-benefits from the EGU MACT would be based on reductions in annual average PM_{2.5} exposures that are already below the health-protective level of the current PM_{2.5} standard.

Figure 6 also shows that if EPA had not extrapolated below the LML, about 89% of the estimated upper bound co-benefits of the EGU MACT would have been estimated as zero.⁵¹ This is confirmed in the RIA, which reports that of the 11,000 estimated avoided premature deaths, only 1,200 are in areas where the baseline PM_{2.5} concentrations are above the LML.⁵²

The 15 µg/m³ annual PM_{2.5} NAAQS is under review now, and EPA staff (with CASAC's concurrence) has stated that it will consider revising the annual PM_{2.5} NAAQS to somewhere in the range of 11 to 13 µg/m³.⁵³ EPA's reluctance to set the annual PM_{2.5} NAAQS anywhere below 11 to 13 µg/m³ would appear to reveal the extent to which EPA does not itself feel that risk estimates below that range are credible; if it did view them as credible estimates, surely EPA and CASAC would be compelled to propose a lower PM_{2.5} NAAQS.

Dotted red lines have been added to Figure 6 to show that between 94% and nearly 100% of the 11,000 PM_{2.5} mortality benefits that EPA has estimated from the Final EGU MACT are attributed to estimated PM_{2.5} concentrations below levels that will be deemed protective of the public health with an adequate margin of safety even if EPA revises the annual PM_{2.5} NAAQS to a level within its recommended range of 11 µg/m³ to 13 µg/m³.^{**} If those concentrations are safe, then it is not appropriate for EPA to be calculating them as co-benefits justifying non-PM regulations such as the EGU MACT rule. Thus those estimates are non-credible from a scientific standpoint.

Further, the remaining <1% to 6% of estimated mortality reductions (*i.e.*, ~0 to ~660 avoided premature deaths out of EPA's estimated 11,000) that are attributable to baseline concentrations between whatever the new PM_{2.5} NAAQS level may be and the upper end of the x-axis (*i.e.*, at about 15 µg/m³) should, if anything, be counted as direct benefits of the revised PM_{2.5} NAAQS. They are overstated due to issues discussed above concerning the use in chronic exposure studies of recent ambient data rather than average ambient concentrations experienced over the cohort's lifetime, and due to EPA's presumption that there is no uncertainty in the causality of the statistical associations. However, even a more appropriately calculated lower estimate should not be considered a co-benefit for the EGU MACT or other non-PM regulation; it should be counted as a benefit of the PM_{2.5} NAAQS. Placing them in the co-benefits category is tantamount to double-counting them, will be explained in Section 7.

⁵¹ The LML for the upper bound is at the green vertical line in the figure.

⁵² EPA (2011c), Table 5-20, p. 5-101.

⁵³ EPA, (2010a), p. 2-106.

^{**} Note: A previous version of this report erroneously stated that the lower bound of the range was 84%.

7. EPA's Baselines and Reporting of Benefit and Costs Estimates for a Single Year Cause Double-Counting

This paper has already shown that:

- Co-benefits have no support in BCA theory,
- Use of PM_{2.5} co-benefits in RIAs is undercutting the more general, practical objectives of RIAs,
- EPA's current calculations of PM_{2.5} risks are unsupported by data or scientific principles, and
- The resulting magnitude of EPA's risk estimates are *prima facie* non-credible.

These points should give any thoughtful person reason to call for a stop to the practice of using co-benefits as the primary benefit justification for new rules, and to call for a stop to the estimation of PM_{2.5} risks below the range of observed associations. However, there is yet another significant concern that merits discussion in this paper: EPA may be double-counting many of the PM_{2.5} benefits as it moves from one RIA to the next.

EPA has argued that it does not double-count the PM_{2.5} benefits because it includes all existing regulations in the baseline of emissions for each of its RIAs for another rule. If EPA were doing so thoroughly and consistently, double-counting would not be a concern. However, this is not the actual case.

First, many RIAs are being prepared simultaneously. In 2010, 6 final major CAA-related RIAs and at least 7 proposed RIAs were released for CAA-related rulemakings. In 2011, 7 final and at least 4 proposed CAA-related RIAs were released. This creates a constant source of confusion and potential for double-counting. For example, the RIA for the Proposed EGU MACT rule applied the Proposed CATR rule in *its* baseline, while the RIA for the Final EGU MACT rule applied the Final CSAPR rule in *its* baseline (simply because the CSAPR rule was finalized in the interim between the proposal and finalization of the EGU MACT rule). This change of baseline appears to be the primary reason why the EGU MACT rule's estimated PM_{2.5}-related co-benefits for mortality fell from 17,000 to 11,000 when the RIA for the final rule was released. This reveals the extent to which double-counting can occur due to seemingly small differences in what specific rules are included in an RIA's baseline. Moreover, *neither* of the EGU MACT RIAs' baselines included compliance with other existing regulations that have yet to be fully implemented, such as the new 1-hour SO₂ NAAQS, the new NO₂ NAAQS or even the 2006 daily or annual PM_{2.5} NAAQS themselves. In fact, there is a very small amount of co-benefit in the RIA for the Final EGU MACT that is due to reduction of baseline PM_{2.5} exceeding the 15 µg/m³ annual NAAQS level.⁵⁴ Small as that amount is, it is direct evidence that double-counting can and does occur across all CAA RIAs as a group.

⁵⁴ See EPA (2011c), Figure 5-14, p. 5-100.

Clearly the development of an RIA's baseline plays a critical role in the estimates of benefits (and costs) of a rule. The significance of the baseline in determining the benefit and cost comparisons of an RIA has been pointed out by other reviewers of RIAs.⁵⁵ However, only one of the previous RIA critiques reviewed in this study has addressed the question of how a baseline affects PM_{2.5} co-benefits. In a review of proposed and final RIAs for the first mercury rule, which was under development during the period 2001-2005, O'Neill takes issue with the fact that coincidental PM_{2.5} reductions from mercury regulation did not appear in the RIA for the mercury rule proposal released in 2004 (*i.e.*, the proposal for what became the Clean Air Mercury Rule, CAMR, which was finalized in 2005). The reason the co-benefits did not appear in the proposed CAMR's RIA was because by the time that rule had been proposed, another new rule that was designed specifically to reduce those same PM_{2.5} levels also was in its final stages (*i.e.*, the Clean Air Interstate Rule, CAIR). As a result, the CAIR rule was incorporated into the baseline for the final CAMR rule's RIA, and what might have once appeared to be co-benefits of CAMR were actually recognized as direct benefits of CAIR. O'Neill also criticizes the fact that EPA chose the single year 2020 to assess the incremental benefits of CAMR, which was the point in time at which the CAIR rule would have been fully implemented. The choice of year as well as the choice of placing CAIR in the baseline had the effect of attributing all of CAIR's benefits to CAIR, rather than allowing any of them to appear as co-benefits from CAMR.

One reasonable response to O'Neill's criticism is that since CAIR was a rule specifically designed to control PM_{2.5}, any PM_{2.5}-related benefits that might be derived from CAIR should rightfully appear as direct benefits in the RIA for CAIR. In fact, one could contend that they never should have been viewed as co-benefits of the CAMR (or any other possible non-PM rule) as long as the PM_{2.5} NAAQS was in place, because throughout the period when the first mercury rule was being crafted, one could fully anticipate that the PM_{2.5} NAAQS would require implementation. Even if CAIR had not been in development stages at that same time, any non-PM RIA, such as a mercury RIA, should have assumed full implementation of the PM_{2.5} NAAQS anyway. That would have had the same effect as the simple and appropriate act of moving the PM_{2.5}-related benefits estimates off of the mercury rule's co-benefits ledger and onto the direct benefits ledger of a PM_{2.5} rulemaking.

The point raised by O'Neill does highlight how the baseline can alter whether PM_{2.5} changes will be counted as co-benefits to justify a non-PM rule or not. However, it does not address double-counting, which is another concern that arises from EPA's choices of RIA baselines. It is nearly impossible to keep the baselines straight when multiple regulations are in the proposal stage at the same time. However, a simple prescription can be applied to EPA's current practice that would help minimize the problem. If any RIA will be accounting for co-benefits from a pollutant that it does not directly address, such as those from PM_{2.5} in a NESHAP rulemaking, then the baseline for that RIA should include "existing" rules, even if not fully implemented yet. It should also explicitly

⁵⁵ See, for example, Morgenstern in Chapter 3 of Morgenstern (1997) and O'Neill in Chapter 6 of Harrington *et al.* (2009).

incorporate any reasonably anticipated future standards and/or rulemakings that will deal with that pollutant before allowing any co-benefits from that pollutant to be counted in some unrelated RIA. This may be an uncertain task, but it can certainly be handled by at least considering two baselines:

Baseline A: Include only the present level of current standards, but ensure that all of them are simulated as attained at their respective attainment deadlines.

Baseline B: Incorporate reasoned assumptions regarding levels of new regulations that are known to be on the verge of modification, even if not yet promulgated or even proposed, and accounting for their future attainment deadlines. (For example, Baseline B would incorporate a reasoned estimate of the most stringent potential level of a tightened PM_{2.5} NAAQS level that may be implemented within the next decade.)

This recommendation conforms with OMB guidance for performing RIAs, which states “When more than one baseline is reasonable and the choice of baseline will significantly affect estimated benefits and costs, you should consider measuring benefits and costs against alternative baselines.”⁵⁶ Morgenstern also has highlighted the value of considering multiple baselines to highlight the role it plays in RIA findings.⁵⁷

In neither of the alternative baselines should PM_{2.5} co-benefits be calculated based on extrapolation of the concentration-response relationship below the data range over which its slope has been estimated. Indeed, the limit for extrapolation should not be based on the *most recent* LML among the cities in the database; the calculation of risks should be curtailed at a level reflective of the concentrations that the individuals in the cohort experienced on average across their lives.

Another change that is required in order to mitigate double-counting is that EPA stop reporting its benefits and cost estimates for a single year. Regulatory compliance costs and benefits should be considered on a present value basis. EPA’s practice of reporting the costs and benefits for a single year can be misleading, especially if the baseline of emissions is declining after the single year selected. For example, PM_{2.5}, SO₂, and NO₂ can all be expected to keep declining after 2016 even in the absence of an EGU MACT rule because there are specific standards for each of those pollutants that will take effect between now and 2020. However, in the RIA for the EGU MACT, EPA reports its PM_{2.5} co-benefits only for 2016, at a point in time where PM_{2.5} levels should be on a steady decline through 2019 (which is the latest attainment date for the 2006 PM_{2.5} NAAQS). Thus, there must be a declining trend in baseline risks, and hence the EGU MACT’s PM_{2.5} co-benefits soon will be much smaller than EPA reports in the RIA for the single year, 2016. In contrast, the annual costs that EPA reports for that rule will not be declining. Choosing 2016 as the single year for reporting the benefits and costs from the EGU MACT gives an overstated impression of the size of the benefits relative to their

⁵⁶ OMB (2003), p. 15.

⁵⁷ Morgenstern in Chapter 3 of Morgenstern (1997), p. 35.

costs. In fact, it is tantamount to double-counting of co-benefits, because the reported “annual co-benefits” in 2016 includes mortality and morbidity risks that will be gained (and attributed to) the PM_{2.5} and other existing rules just a couple of years later. If benefits and costs are reported for only a single year, that year should be selected as one in which all other regulations in the baseline will be fully implemented.

8. Summary and Conclusions

The key findings of this paper, which is based on review of the benefit and cost evidence in RIAs for major CAA-based rules dating back to 1997, are:

- EPA is relying heavily on coincidental “co-benefits” from PM_{2.5} reductions to create the impression of a benefit-cost justification for many air regulations that are not intended to address PM_{2.5}.
- Consideration of co-benefits for a separately-regulated pollutant is not supported by benefit-cost analysis (BCA) theory, and EPA’s excessive reliance on them undercuts the broader practical value of RIAs, which is to provide structured and transparent information to help avoid and reduce redundant and ineffective regulations.
- In 2009, EPA vastly increased the levels of mortality risks that it attributes to PM_{2.5} (and hence inflated its estimates of PM_{2.5} benefits and co-benefits) simply by starting to assign risks down to background levels of PM_{2.5}, below the most recent of the lowest measured levels (LMLs) in the epidemiological studies. This created non-credible estimates of risks from ambient exposures that are well within the safe range established by the PM_{2.5} NAAQS.
- Identifying an appropriate lower bound below which risk estimates are not scientifically supported is not as simple as identifying the most recently-observed LML among cities in a chronic exposure epidemiological study. The exposure level to attribute to the observed mortality differences could be much earlier in time, given that such studies track mortality outcomes dating several decades back, based on people whose lifetime exposures date back to well before 1950.
- The decision to inflate the PM_{2.5} risk estimates by presuming risks continue at an unchanged rate down to background has its greatest impact on co-benefits estimates because – for rules that do not address PM_{2.5} directly – a much greater share of their incremental reduction of PM_{2.5} will occur in areas that are already in attainment with the PM_{2.5} NAAQS (and thus have PM_{2.5} levels that EPA has deemed safe).
- Poor choices of baselines and EPA’s practice of reporting benefits and costs for only a single year leads to double-counting of the PM_{2.5}-related benefits and co-benefits.

Based on the above observations, several recommendations and conclusions follow for aligning RIA methods with BCA principles, and for improving the quality and usefulness of RIAs that EPA produces. These include:

- Baselines in RIAs should incorporate implementation of all reasonably anticipated standards, even if formal rules to implement them are not yet in place. Estimates of benefits from PM_{2.5} reductions will thus remain the direct benefits of PM_{2.5}-specific rules, and double-counting will be avoided. Any temporary benefits from early introduction of PM_{2.5} reductions via a non-PM_{2.5} rule should be identified as temporary only, and not reported as the co-benefits in a single, “snapshot” year, which implies those benefits would be permanent.
- Co-benefits from a pollutant that EPA already regulates under separate rulemakings should not be allowed to serve as a component of the total benefits reported in the Executive Summary of RIAs for rules that target different public health or welfare concerns. The current practice of doing so subverts the practical values of preparing RIAs, leads to unnecessary regulatory complexity, and incentivizes use of less credible methods of risk estimation. Co-benefits should not be reported as part of the total benefits estimates in an RIA, nor should they be included in public announcements of the benefits of a new regulation.
- EPA should stop using its scientifically non-credible method of extrapolating PM_{2.5} risks below the LML. If EPA does persist in producing estimates of benefits or co-benefits from changes in concentrations below the LML, those estimates should be kept clearly separated from all other PM_{2.5}-related mortality benefits estimates, not be added to any other PM-related benefits estimates, and should be accompanied by a clear statement that there is no scientific evidence about the shape or existence of any concentration-response function in that range of ambient PM_{2.5} concentrations.
- For benefits estimates based on PM_{2.5} concentrations above the LML, EPA should be offering quantitative estimates of the uncertainties associated with its risk estimates, *taking account of the expanded confidence interval for estimates nearing the LML*. Confidence ranges based only on the statistical error of the slope estimate are not an appropriate measure of the statistical confidence of its predicted changes in health risk for reductions in PM_{2.5}, especially those well below the average PM_{2.5} in the underlying epidemiological study.

In all, EPA’s use of co-benefits in its RIAs should end for several reasons. It scares the public into believing that large numbers of people die prematurely were it not for implementation of new rules on pollutants for which EPA has not actually identified any current public health risk. EPA’s use of co-benefits also gives EPA a shield to justify building a complex web of rules when EPA could (and is already obligated to) provide almost all of those purported health-protective benefits with just a single rule, if warranted: the PM_{2.5} NAAQS. If large effects below the level of the PM_{2.5} NAAQS were deemed credible, the appropriate policy remedy would be to tighten the PM_{2.5} standard. The fact that EPA does not take this simple, streamlined approach hints at the degree to which the Agency realizes that its co-benefits calculations do not reflect true public health risks. But finally, promoting the goal of further PM_{2.5} risk reductions by way of rules for totally different categories of emissions is just bad policy. This cannot possibly result in a cost-effective path to addressing a nation’s clean air needs.

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Appendix A.

Sources of Cost and Benefit Information on CAA RIAs since 1997 that Were Reviewed in this Study

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
7/12/1997	1997	Ozone NAAQS (To revise the 0.12 1hr standard to a 0.08 8hr standard)	Ozone	Final RIA. See pp.13-2 for cost, pp. 12-64 for other co-benefits, pp. 12-1 for total benefits. Full attainment (F/A) numbers were estimated by scaling partial attainment (P/A) numbers for target benefits, PM co-benefits and PM mortality rates.	http://www.epa.gov/ttn/oarpg/naaqsfm/ria.html
7/16/1997	1997	PM _{2.5} NAAQS (To implement the first PM _{2.5} standard at 15/65 annual/daily averages, supplementing the PM ₁₀ standard)	PM _{2.5}	Final RIA. F/A numbers provided. Target benefit estimate comes from: low- 19.8-4.3 = 15.5; high- 109.7-8.1 = 101.6. Cost: pp.13-2. Other co-benefits: pp.12-64. Total benefits: pp.13-2. Mortality rates: see pp.12-43 for P/A numbers. F/A estimated by 15.5/14.5 * 3300 = 3528 for low; 101.6/96.1 * 15,600 = 16493 for high.	http://www.epa.gov/ttn/oarpg/naaqsfm/ria.html
8/14/1997	1997	Hospital/ Medical/ Infectious Waste Incinerators NSPS and EG	Many pollutants	Final rule FR. See pp. 29 for cost and benefits. All quantified benefits are PM.	http://www.gpo.gov/fdsys/pkg/FR-1997-09-15/pdf/97-23835.pdf#page=1
9/22/1997	1997	Highway Heavy-Duty Engines and Diesel Engines	NO _x , HC	Final RIA. See pp. 97 for cost.	http://www.regulations.gov/#!searchResults:rpp=10;po=0;s=EPA-HQ-OAR-2003-0012-0949
10/27/1997	1997	Pulp & Paper NESHAP	HAPs, VOC, TRS	Final rule FR. See pp. 120 for cost, pp. 126 for benefits. Included air related numbers only. Identified negative co-benefits (due to SO ₂ , CO, PM, NO _x increases).	http://www.gpo.gov/fdsys/pkg/FR-1998-04-15/pdf/98-9613.pdf#page=1
12/16/1997	1997	Locomotive Emission Standards	NO _x , PM	1998 Regulatory support document. See pp. 120 for NPV (7% discount rate) of the total cost, no annualized figure provided.	http://www.regulations.gov/#!searchResults:rpp=10;po=0;s=EPA-R03-OAR-2009-0956-0038

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/14/1998	1998	National VOC Standards for Architectural Coatings	VOC	Proposed rule and notice of public hearing FR. See pp. 6 for cost.	http://www.gpo.gov/fdsys/pkg/FR-1996-06-25/pdf/96-16009.pdf#page=1
8/27/1998	1998	Non-Road Diesel Engines	NO _x , HC	Final RIA. See pp. 72 for cost.	http://www.epa.gov/nonroaddiesel/fm1998/nr-ria.pdf
9/2/1998	1998	Revised NO _x Std: New Fossil-Fuel-Fired Steam Generating Units	NO _x	Insufficient information.	http://www.gpo.gov/fdsys/pkg/FR-1997-07-09/pdf/97-17950.pdf#page=1
9/23/1998	1998	NO _x SIP Call & Section 126 Petitions	NO _x	RIA. Ranges quoted are for low to high assumption sets. See pp. ES-3 for cost, pp. ES-6 for total benefits, pp. 4-50 for a breakdown of benefits, pp. 4-23 for mortality rates. All ozone related benefits are target benefits.	http://www.epa.gov/ttn/oarpg/otag/sipriav2.zip
3/1/1999	1999	Phase II Emission Stds for New Nonroad Spark-Ignition Non-Handheld Engines <19 kW	HC, NO _x	Final RIA. See pp. 7-15 for fuel savings (0.2 billion per year), pp. 7-13 for cost (0.132 billion) for a net cost savings of 0.0907.	http://www.epa.gov/otag/equip-ld.htm
4/22/1999	1999	Regional Haze Rule	Visibility	RIA. See pp. 9-48 and 9-51 for benefits, pp. 10-20 for cost, pp. 9-55 and 9-61 for mortality rates. Reported 1.0 dv/10years levels, ranges quoted for benefits and mortality rates are the low and high ends across Case A and Case B. Total benefit is calculated accordingly.	http://www.epa.gov/ttnecas1/regdata/RIAs/rhria.zip
4/30/1999	1999	Final Section 126 Petition Rule	NO _x	RIA. See pp. ES-3 for cost, pp. ES-11 for benefits, pp. ES-7 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/126fn0.zip
12/21/1999	1999	Gasoline Sulfur Control Requirements	NO _x , PM	Final FR. See pp. 88 for benefits and mortality rate, pp. 86 for cost.	http://www.gpo.gov/fdsys/pkg/FR-2000-02-10/pdf/00-19.pdf#page=1

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
3/1/2000	2000	Phase 2 Emission Stds for New Nonroad Small Spark Ignition Handheld Engines <19 kW	HC, NO _x	Final RIA. See pp. 110 for annualized cost (.234-.284) and fuel savings (0.094).	http://www.epa.gov/otaq/equip-ld.htm
7/28/2000	2000	Control of Emissions from 2004 and Later Model Year Highway Heavy-Duty Engines	Many pollutants	RIA. See pp. 89 and 106 for cost.	http://www.regulations.gov/#!searchResults;rpp=10;po=0;s=EPA-HQ-OAR-2003-0012-0950
10/19/2000	2000	Protection of Stratospheric Ozone Reductions	Stratospheric O ₃	RIA. See pp. 4 for cost.	http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2008-0009-0012
12/21/2000	2000	Heavy-Duty Engine & Diesel Fuel Sulfur Control Reqm 2007	NO _x , PM	Final RIA. See pp. xvi for cost and benefits, pp. xvii for mortality rates.	http://www.epa.gov/oms/highway-diesel/regs/exec-sum.pdf
9/13/2002	2002	Emissions from Nonroad & Recreational Spark-Ignition Engines	NO _x , VOC, CO, PM	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2001-10-05/pdf/01-23591.pdf#page=1
8/27/2003	2003	PSD & NSR: Routine Maintenance and Repair	all	Insufficient Information.	http://www.epa.gov/fedrgstr/EPA-AIR/2003/October/Day-27/a26320.htm
2/26/2004	2004	Stationary Reciprocating Internal Combustion Engine NESHAP	HAPs	RIA. See pp. ES-5 for cost, pp.8-40 for mortality rates (50% NO _x emission reduction), pp. 8-45 for benefits. Assuming the social cost of 0.255 on pp. ES-7 is a typo. Mortality rates are prorated assuming 25% NO _x emission reduction. Ozone and PM ₁₀ benefits are regarded as "other benefits".	http://www.epa.gov/ttnecas1/regdata/RIAs/RICERIA-finalrule.pdf
2/26/2004	2004	Plywood & Composite Wood Products NESHAP	organic HAPs	RIA. See pp. ES-1 for cost.	http://www.epa.gov/ttnecas1/regdata/RIAs/pcwp-finalruleRIA.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
2/26/2004	2004	Automobile & Light-Duty Vehicle Manufacturing NESHAP	HAPs	RIA. See pp. ES-5 for cost. Did mention ozone and PM co-benefits from VOC reduction, but did not quantify.	http://www.epa.gov/ttnecas1/regdata/RIAs/autolightdutyRIAFinaltotal.pdf
2/26/2004	2004	Industrial Boilers & Process Heaters NESHAP	HAPs, HCl, metals	RIA. See pp.ES-1 for cost, pp. 10-45 for mortality rates and benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/indboilprocheatfinalruleRIA.pdf
5/7/2004	2004	Non-Road Diesel Rule	PM, NO _x , HAPs	RIA. See pp.9-42 to 43 for mortality rates and benefits, pp. 9-52 for costs. Other co-benefits include 2.5-3.4 (pp. 9-27) reductions in unpleasant odors, and 2.15 PM welfare benefits.	http://www.epa.gov/nonroad-diesel/2004fr.htm#ria
3/10/2005	2005	Clean Air Interstate Rule	NO _x ,SO ₂ (SO ₂ as precursor of amb. PM _{2.5})	RIA. See pp. 1-2 for benefits and cost, pp. 1-4 for mortality rates.	http://www.epa.gov/cair/pdfs/finaltech08.pdf
3/15/2005	2005	Clean Air Mercury Rule	Hg	RIA. See pp. 11-14 for target benefits, pp.7-13 for cost, pp.12-8 for PM _{2.5} co-benefits and mortality rates.	http://www.epa.gov/ttn/ecas/regdata/RIAs/mercury_ria_final.pdf
6/15/2005	2005	Clean Air Visibility Rule/BART Guidelines	Visibility	RIA. See pp. 1-3 for cost and benefits, pp. 1-5 for mortality rates.	http://www.epa.gov/oar/visibility/pdfs/bart_ria_2005_6_15.pdf
3/15/2006	2006	Inclusion of Delaware and New Jersey in CAIR	NO _x ,SO ₂ (SO ₂ as precursor of amb. PM _{2.5})	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-05-12/pdf/05-5520.pdf#page=1
3/15/2006	2006	Sec. 126 from NC to Reduce Interstate Transport of PM & O ₃ ; FIPs to Reduce Interstate Transport of PM & O ₃ ; Revisions to CAIR; Revisions to Acid Rain Program	NO _x ,SO ₂ (SO ₂ as precursor of amb. PM _{2.5})	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-08-24/pdf/05-15529.pdf#page=1

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
6/28/2006	2006	Stationary Compression Ignition Internal Combustion Engine NSPS	many	RIA. See pp. 1-2 for cost, pp. 6-4 for benefits. Direct PM benefit is recorded as target benefit. Benefits from NO _x and SO ₂ are recorded as PM co-benefits. Premature mortality prevention accounts for 90% of the total benefit, but cannot be quantified (pp.6-5). Benefits are quoted at 3% discount rate (pp.6-4).	http://www.epa.gov/ttnecas1/regdata/RIAs/ci_nsps_ria_reportfinal06.pdf
9/21/2006	2006	PM _{2.5} NAAQS (To revise from 15/65 annual/daily averages, to 15/35)	PM _{2.5}	RIA. See pp. ES-9 to 10 for costs and benefits, pp. 5-100 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/Executive%20Summary.pdf http://www.epa.gov/ttnecas1/regdata/RIAs/Chapter%205--Benefits.pdf
2/8/2007	2007	Control of HAP from mobile sources	HAPs	Final RIA. See pp. 12-20 for mortality rate (Pope et al. only), pp. ES-10 for PM benefits, pp. ES-11 for cost.	http://www.epa.gov/otaq/regs/toxics/fr-ria-sections.htm
3/28/2007	2007	Clean Air Fine Particle Implementation Rule	PM _{2.5}	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-11-01/pdf/05-20455.pdf#page=1
2/14/2008	2008	Control of Emissions form New Locomotives & Marine Diesel Engines <30 L per Cylinder	PM, NO _x	RIA. See pp. 6-52 for cost and benefits, pp. 6-44 for PM mortality, pp. 6-46 for a breakdown of benefits.	http://www.epa.gov/oms/regs/nonroad/420r08001a.pdf
3/12/2008	2008	Ozone NAAQS (To revise the 0.08 8hr standard to 0.075 8hr standard)	Ozone	RIA. See pp.ES-3 for cost and visibility benefits, pp. 6-62 for target benefits, pp. 6-48 for mortality rates, pp. 6-64 for PM co-benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/452_R_08_003.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/18/2008	2008	Control of Emissions from Non-road Spark-Ignition Engines & Equipment	VOC, NO _x , PM, CO	Final rule FR. See pp. 122 for mortality rates, pp. 124 for a breakdown of benefits, pp. 128 for cost and total benefits. Target benefit is all PM; other benefits include visibility and ozone benefit.	http://www.gpo.gov/fdsys/pkg/FR-2008-10-08/pdf/E8-21093.pdf#page=1
10/15/2008	2008	Lead (Pb) NAAQS	Pb	RIA. See pp. ES-11 for cost and benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/finalpbria.pdf
12/10/2008	2008	Petroleum Refineries NSPS	SO ₂ , NO _x , PM, VOC	RIA. See pp. 7-6 for benefits, pp. 7-12 for cost. Direct benefit is PM benefit, PM 2.5 co-benefits include benefits from reductions in PM precursors.	http://www.epa.gov/ttnecas1/regdata/RIAs/finalpetroleumrefineriesnspsria43008.pdf
9/16/2009	2009	GHG Mandatory Reporting Rule	GHGs	Final rule FR. See pp. 105 for cost.	http://www.epa.gov/climatechange/emissions/notices.html
12/17/2009	2009	Control of Emissions from New Marine Compress'n-Ign Engines >30 L per Cylinder	NO _x	Final rule FR. See pp. 69 for benefits and cost, pp. 67 for PM and Ozone ("other") benefit, pp. 65 for mortality rates.	http://edocket.access.gpo.gov/2010/pdf/2010-2534.pdf
2/17/2010	2010	Reciprocating Internal Combustion Engines NESHAP - Compression Ignition	Organic, metal HAPs	RIA. See pp.1-1 for cost, pp.7-1 for PM _{2.5} co-benefits, and pp.7-8 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/CIRICENESHAPRIA2-17-10cleanpublication.pdf
3/31/2010	2010	EPA/NHTSA Joint Light-Duty GHG Emission Stds & CAFES	GHGs	Final FR. See pp. 213 for benefits and cost, pp. 206 for PM benefits, pp. 205 for mortality rates,	http://www.gpo.gov/fdsys/pkg/FR-2010-05-07/pdf/2010-8159.pdf#page=1
5/12/2010	2010	Greenhouse Gases PSD and Tailoring Rule	GHGs	RIA. See pp.18 for cost. No benefits quantified.	http://www.epa.gov/ttnecas1/regdata/RIAs/riatailoring.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
6/2/2010	2010	SO ₂ NAAQS (To set a 75 ppb 1-hr standard)	SO ₂	See pp. ES-9 for cost and benefits, pp. 5-30 for mortality rates. Negative cost indicates regulatory relief benefit. Numbers quoted are for Step 1, 25000 tpy.	http://www.epa.gov/ttnecas1/regdata/RIAs/fso2ria100602full.pdf
8/6/2010	2010	Portland Cement Manufacturing Industry NSPS & NESHAP Amendment	HC, HAPs, PM	RIA. See pp. 1-2 for cost, pp. 6-1 for PM _{2.5} co-benefits, pp. 6-15 for mortality rates. All benefits of the rule are from NESHAP portion. NESHAP does not target PM _{2.5} or PM ₁₀ mass, but all the benefits of the NESHAP (and of the NESHAP + NSPS) are PM _{2.5} ambient concentration. Therefore, all are co-benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/portlandcementfinalria.pdf
8/10/2010	2010	Existing Stationary Compression Ignition Engines NESHAP	HAPs	Final RIA. See pp. 7-10 for mortality rates, pp. 7-9 for benefits, pp. 1-1 for costs.	http://www.regulations.gov/#!documentDetail:D=EPA-HQ-OAR-2008-0708-0571
2/21/2011	2011	Industrial, Comm'l, and Institutional Boilers NESHAP	HAPs	Final FR. See pp. 27 for benefits and mortality rates, pp.29 for cost.	http://www.gpo.gov/fdsys/pkg/FR-2011-03-21/pdf/2011-4493.pdf#page=1
2/21/2011	2010	Indus'l, Comm'l, and Institutional Boilers & Process Heaters NESHAP	HAPs	RIA. See pp.1-1 for cost, pp.6-1 for PM _{2.5} co-benefits pp. 6-8 for mortality rates.	http://www.epa.gov/airquality/combustion/docs/boilerria20100429.pdf
2/21/2011	2011	Comm'l & Indus'l Solid Waste Incineration Units NSPS and Emission Guidelines	CO, Pb, HAPs	RIA. See pp. 1-1 for cost, pp. 1-2 for benefits, pp. 5-10 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/CISWIRIAfinal110221_psg2.pdf
7/1/2011	2011	Cross State Air Pollution Rule (CSAPR)	NO _x , SO ₂ (SO ₂ as precursor of amb. PM _{2.5})	RIA. See pp.1 for mortality rates, pp.2 for cost, table 1-3 on pp. 6 to 7 for benefits. Other co-benefits include visibility (4.1) + social cost of carbon (0.6) = 4.7. Sum up all the remaining items to get target benefits.	http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/8/2011	2011	Control of GHG from Medium & Heavy-Duty Vehicles	GHGs	Final RIA. See pp. 9-45 for benefits and cost, net cost = 24.7 (technology cost) - 166.5 (fuel savings), pp. 8-86 for PM benefit and pp.8-81 for mortality rates. Benefit range is derived from different assumptions for social cost of carbon in 2012.	http://www.epa.gov/otaq/climate/documents/420r11901.pdf
9/6/2011	2010	Reconsideration of Ozone NAAQS	Ozone	RIA. F/A (75 ppb) numbers quoted. See pp. S1-4 for cost, pp.3-11 for mortality rates and benefits, pp.3-6 for visibility. Target benefit refers to ozone benefit here.	http://www.epa.gov/ttnecas1/regdata/RIAs/s1-supplemental_analysis_full.pdf
12/16/2011	2011	EGU MACT Rule	Hg, HAPs	RIA. See p. ES-2 for cost; pp. ES-6/7 for direct benefits, PM _{2.5} co-benefits, & social cost of carbon. SCC is quoted at 3% discount rate. See p. ES-5 for mortality counts.	http://www.epa.gov/ttnecas1/regdata/RIAs/matsriafinal.pdf
N/A	2011	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	Hg	RIA. See pp.1-2 for cost and total benefit, pp. 5-1 for PM _{2.5} co-benefits, pp. 5-11 for mortality rates, and pp. 5-16 for the social cost of carbon (other co-benefits).	http://www.epa.gov/ttnecas1/regdata/RIAs/mercurycell.pdf
N/A	2011	Oil and Natural Gas Industry NSPS & NESHAP Amendment	VOC, SO ₂ , HAPs, Methane	RIA. See pp.1-4 and pp. 1-6 for costs.	http://www.epa.gov/ttnecas1/regdata/RIAs/oilnaturalgasfinalria.pdf
N/A	2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	Hg	RIA. See pp. 3 for cost and benefit, pp. 5-11 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/ssiria110201.pdf