



Analysis

Air pollution, health and economic benefits—Lessons from 20 years of analysis

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ABSTRACT

This paper reviews and compares two air quality benefit assessments completed for California's South Coast Air Basin in 1989 and 2008. Specifically, we separate the influence of changes in population and air quality from that of newer health concentration–response relationships and changing economic values. The dynamic interaction of key variables, including health and economic, as well as changes in population and air quality, lead to significant changes in results over time. Results show dramatic reductions in exposures to ozone and particulate concentrations between the two time periods, a continually evolving health literature, and in contrast, fairly constant real economic unit values assigned to adverse health outcomes. Such research is important because highly technical analyses of the expected benefits of proposed air quality regulatory programs have become an increasingly important component of many decision-making processes.

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1. Introduction

Worldwide, highly technical analyses of the expected benefits of proposed regulatory programs have become an increasingly important component of regional and national decision-making. More specifically, the World Health Organization and many federal governments have set health-based air quality standards (AQS) for ozone and particulate matter (PM_{2.5} and PM₁₀)³ because there is extensive evidence that these pollutants pose the most serious risks to health. Adverse effects include a spectrum from symptoms that moderately limit normal daily activity to premature death. Requirements for compliance with AQS, in turn, drive complex regulatory schemes designed to curtail air pollution emissions. Such regulations often pose substantial costs, which are a concern for those who bear them. Elected and appointed public officials thus voice concern about whether or not regulation is “worth it.” What does society get for the significant sums spent to improve air quality? Many studies have considered the benefits within a timeframe (see, for example, EPA, 1999), but none has retrospectively compared benefits between two timeframes with the specific purpose of determining which variables dominate observed changes.

This research reviews and compares two large-scale air quality benefit assessments completed for California's South Coast Air Basin,⁴ which is a crucible for such analyses, in 1989 and 2008. Specifically, we separate the influence of changes in population and air quality from that of newer health concentration–response relationships and changing economic values. Results show dramatic reductions in population exposures to ozone and particulate concentrations between the two time periods, a continually evolving health literature, and in contrast, fairly constant real economic unit values assigned to adverse health outcomes.

2. Background

One of the first large-scale regional benefit assessments was completed for California's South Coast Air Basin (SoCAB) in 1989 (Hall et al., 1989, 1992; Kleinman et al., 1989; Winer et al., 1989). At the time, the 1-hour national ambient air quality standard (NAAQS) for ozone was exceeded on more than 150 days a year, and the annual average PM₁₀ concentration was nearly double the standard in the region. The impetus for the study was the need for quantitative benefits estimates to inform the policy debate. The regional regulatory agency was under increasing pressure to back away from aggressive and costly control measures needed to attain the NAAQS, on the grounds that the costs would wreak economic havoc. The benefit assessment essentially provided a counter weight to claims of catastrophic control costs, since the potential economic benefits of regulation had never been adequately quantified (Stammer, 1988; Jacobs and Kelly, 2008). For the SoCAB, the 1989 study concluded that

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E-mail addresses: jhall@fullerton.edu (J.V. Hall), vbrajer@fullerton.edu (V. Brajer), fred@sonomatech.com (F.W. Lurmann).¹ Tel.: +1 657 278 3818; fax: +1 657 278 3097.² Tel.: +1 707 665 9900; fax: +1 707 655 9800.³ Standards have been set for other contaminants, but because economic effects associated with ozone and fine particles dominate economic analysis, these are the focus here.⁴ This region includes Los Angeles and Orange Counties and the non-desert portions of Riverside and San Bernardino Counties.

the health-related benefits of meeting the NAAQS for ozone and particulates exceeded \$9 billion annually or \$750 per person (in 1987 dollars).

Over the past 20 years, the SoCAB's air quality control program has been extraordinarily successful, especially in the context of a regional population that increased by more than 40%, while the state's real income grew by over 50%. Although the NAAQS have not been attained, the number of days exceeding the 1-hour ozone standard decreased by more than 80%, and the annual average PM₁₀ concentration was halved. Moreover, during this period, the NAAQS became more stringent, increasing the technical difficulties and cost of attainment.

Still, the region is far from achieving healthful air, and it is at least as important now as it was in the late 1980s to understand the likely benefits from ultimately attaining the NAAQS. With this in mind, a recent study similar in scope to the 1989 work was undertaken (Hall et al., 2008). This 2008 study found the annual health cost of human exposure to ozone and PM_{2.5} levels above the federal standards in the SoCAB to be over \$1250 per person (in 2007 dollars), which translates into a total of nearly \$22 billion in benefits if the ozone and PM_{2.5} NAAQS were met.⁵

The core objective of this paper is to determine what factors explain the differences between the 1989 and 2008 results. As in previous studies, we use an integrated approach that calculates reductions in adverse health outcomes by linking the severity of pollutant exposure of the affected population to the resulting health outcomes, and then assigning dollar values to each adverse health outcome. This linkage relies on the Regional Human Exposure Model (REHEX), which was initially developed in 1989 to estimate a population's exposure to various concentrations of air pollution. The model accounts for spatial and temporal pollution patterns, and has been employed in numerous studies over the past two decades (see, for example, Lurmann et al., 1989, 1991a,b, 1999; Lurmann and Korc, 1994; Hall et al., 1994, 2008). Here, by using REHEX in targeted ways (for example, by applying 1989's health equations to 2008's pollution and population levels), we provide a breakdown of the factors that have changed over the two-decade period to ascertain not only by how much, but also why, results vary.

3. Air Quality

The NAAQS in effect in 1989 were for 1-hour daily maximum ozone concentrations, and both 24-hour average and annual average PM₁₀ concentrations. The PM₁₀ NAAQS was a relatively new standard, adopted in 1987 as a replacement for the total suspended particulate matter (TSP) standard. The specific standards in effect in 1989 were:

- The 1979 ozone standard, which was attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 parts per million (ppm) was less than one.
- The 1987 24-hour PM₁₀ standard, which was attained when there were fewer than two expected days per calendar year with a 24-hour average concentration above 150 micrograms per cubic meter (μg/m³).
- The 1987 annual PM₁₀ standard, which was attained when the annual arithmetic average concentration was equal to or less than 50 μg/m³.

Compliance with the standards was evaluated using three consecutive years of measured air quality data to smooth out year-to-year variations in meteorology, which can cause substantial variations in air quality.

In 1997, the 0.12 ppm 1-hour ozone standard was replaced with a more stringent 0.08 ppm 8-hour standard, which was revised in 2008 to 0.075 ppm. Also, new standards for fine particles (PM_{2.5}) were

⁵ In 1987 real dollars, this total translates to \$11.8 billion, an increase of roughly 30%. The per capita figure in real terms is about 10% less in 2008.

Table 1
Air quality conditions in California's South Coast Air Basin.

Air quality parameter	1984–1986	2005–2007
Number of days per year with 1-hour ozone >0.12 ppm	167 ^a	31 ^a
Number of days per year with 8-hour ozone >0.075 ppm	204 ^a	113 ^a
Number of days per year with 24-hour PM ₁₀ > 150 μg/m ³	48 ^a	47 ^a
Number of days per year with 24-hour PM _{2.5} > 35 μg/m ³	NA	45 ^{a,e}
Maximum 1-hour ozone concentration (ppm)	0.390	0.182
Maximum 8-hour ozone concentration (ppm)	0.288	0.145
Maximum 24-hour PM ₁₀ concentration (μg/m ³)	294 ^d	142 ^b
Maximum 24-hour PM _{2.5} concentration (μg/m ³)	NA	73.4 ^c
Annual average PM ₁₀ concentration (μg/m ³)	87 ^d	58 ^b
Annual average PM _{2.5} concentration (μg/m ³)	NA	21 ^e

a) 3-year average.

b) Based on 2005–2006; 2007 PM₁₀ strongly influenced by wild fires.

c) 98th percentile design value.

d) Based on 1985–1986 data.

e) PM_{2.5} is 43% of PM₁₀ on average in southern California (Peters et al., 1999).

adopted in 1997 and revised in 2006, and the annual standard for PM₁₀ was revoked in 2006. The standards in effect in 2008 were:

- The 2008 ozone standard, which is attained when the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year is equal to or less than 0.075 ppm.
- The 2006 24-hour PM_{2.5} standard, which is attained when the 3-year average of the 98th percentile of 24-hour concentrations at each population-oriented monitor within an area is less than or equal to 35 μg/m³.
- The 2006 annual PM_{2.5} standard, which is attained when the 3-year average of the annual arithmetic mean PM_{2.5} concentrations from single or multiple community-oriented monitors is equal to or less than 15 μg/m³.⁶

Air quality in 1984–1986 and 2005–2007 was used as the baseline in the 1989 and 2008 analyses, respectively. Table 1 summarizes the frequency and severity of the NAAQS exceedances in the two periods. Ozone statistics are derived from a network of 32 stations with continuous hourly measurements; the PM statistics are derived from 14 stations that measure PM₁₀ once every 6th day and PM_{2.5} either every day or every 3rd day (this varies by station). The data show that dramatic improvements in ozone and PM air quality occurred between the two periods. The number of days per year with one or more stations recording 1-hour ozone concentrations above the 0.12 ppm standard declined from 167 to 31 days, and the highest 1-hour ozone concentration declined from 0.390 to 0.182 ppm. The number of annual exceedances of the 8-hour ozone standard (0.075 ppm) declined from 204 to 113 days and the maximum 8-hour concentration fell from 0.288 to 0.145 ppm.

While the number of days per year with exceedances is smaller for PM than ozone, the recorded PM values are almost twice the level of the standards. For example, the maximum 24-hour and annual average PM₁₀ concentrations were 294 and 87 μg/m³ in 1985–1986, and declined to 142 μg/m³ and 58 μg/m³ in 2005–2007, respectively.⁷ Few, if any, urban areas with 40% population (and associated economic) growth made as much progress as the Los Angeles area during this period.

Though these large improvements are impressive and unique, current SoCAB air quality is still unhealthful. Moreover, spatial trends

⁶ The 1987 24-hour PM₁₀ standard was in effect in 2008, but was not included in this analysis because it is much less stringent than the 35 μg/m³ daily PM_{2.5} standard.

⁷ PM_{2.5} measurements were not implemented until 1998 so it is not possible to compare PM_{2.5} air quality for the two periods.

in population growth have impeded reductions in aggregate population exposure to ozone and PM. Population growth, on a percentage basis, was much larger in San Bernardino (96%) and Riverside (180%) counties than in the coastal counties of Los Angeles (27%) and Orange (35%). This is important because the highest air pollution levels and the highest frequency of standard exceedances occur in the inland areas, specifically western Riverside County and western San Bernardino County.

The baseline population exposure distributions for 1-hour daily maximum ozone and daily PM₁₀ concentrations for the two periods, measured by person-days per year with exposures above various thresholds, are shown in Tables 2 and 3. The distributions were estimated with the REHEX model using ambient (outdoor) exposure concentrations and residential (census) population data. The number of person-days per year with exposure to ozone concentrations above 0.12 ppm declined from 712 million in 1984–1986 to 38 million in 2005–2007. In the earlier period, 5, 16, and 46% of the exposures were to 1-hour daily maximum concentrations above 0.18, 0.12, and 0.06 ppm, respectively. In contrast, by 2005–2007, 0, 1, and 25% of the exposures were to 1-hour daily maximum concentrations above 0.18, 0.12, and 0.06 ppm. For PM₁₀, the number of person-days per year with exposure to daily concentrations above 150 µg/m³ declined from 67 million in 1984–1986 to 2 million in 2005–2007. Approximately 2, 8, and 51% of all exposures were to daily PM₁₀ concentrations above 150, 100, and 50 µg/m³, respectively, in 1984–1986 compared to 0, 1, and 14% in 2005–2007. These results illustrate the dramatic reductions in population exposures to higher ozone and PM₁₀ air pollutant concentrations between the periods.

A linear rollback model was then used to estimate future year air quality with attainment of the NAAQS in both the 1989 and 2008 analyses assuming background concentrations of 40 ppb ozone, 6 µg/m³ PM_{2.5}, and 15 µg/m³ PM₁₀ (Fiore et al., 2002; Kim et al., 2000). For each time period, as air quality improves, the resulting health benefits can be estimated and valued. We address these next two steps of the valuation process in Sections 4 and 5 below.

4. Health Concentration–Response Functions

In 1989, a review of the then-current particulate health literature indicated that the strongest evidence and most established quantitative results were for premature mortality and for restricted-activity days (RADs). These, therefore, were the outcomes for which we developed concentration–response relationships and calculated associated health and economic benefits. For PM and mortality, we used results derived by Evans et al. (1984), who reviewed 23 original cross-sectional mortality studies, along with 28 reviews and criticisms. The

Table 2
Population ozone exposure distributions in 1984–1986 and 2005–2007 in the SoCAB.

1-hour daily maximum ozone concentration threshold (ppm)	Person-days per year of exposure to concentrations above threshold (millions)		Percent of exposures	
	1984–1986	2005–2007	1984–1986	2005–2007
0.18	227	0	5.3	0.0
0.16	344	0	8.0	0.0
0.14	500	7	11.6	0.1
0.12	712	38	16.5	0.6
0.10	1003	156	23.3	2.5
0.08	1405	550	32.7	8.7
0.06	1976	1598	45.9	25.3
0.04	2814	4227	65.4	66.9
0.02	3859	6075	89.7	96.1
0	4303	6319	100.0	100.0

Table 3
Population PM₁₀ exposure distributions in 1984–1986 and 2005–2007 in the SoCAB.

Daily PM ₁₀ concentration threshold (µg/m ³)	Person-days per year of exposure to concentrations above threshold (millions)		Percent of exposures	
	1984–1986	2005–2007	1984–1986	2005–2007
175	27	0	0.6	0.0
150	67	2	1.6	0.0
125	143	8	3.3	0.1
100	357	29	8.3	0.5
75	896	152	20.8	2.4
50	2194	893	51.0	14.1
25	3957	4487	91.9	71.0
0	4303	6319	100.0	100.0

linear concentration–response function derived by Evans et al. was viewed as conservative relative to other functional forms reported in the literature at the time, and was given as the linear equation:

$$\Delta \text{Annual Deaths} / 100,000 = 6.15 \Delta \text{PM}_{10} \quad (1)$$

where $\Delta \text{Annual Deaths}/100,000$ denotes the change in premature deaths per 100,000 people and ΔPM_{10} is the change in annual average PM₁₀.

For PM₁₀-related RADs, we relied on results obtained from Ostro (1987), who found a significant association between particulate matter and RADs based on data covering 49 different metropolitan areas in the U.S. The resulting concentration–response relationship for RADs was expressed as:

$$\Delta \text{RAD} / \text{person} = 0.0556 \Delta \text{PM}_{10} \quad (2)$$

where $\Delta \text{RAD}/\text{person}$ is the per-person, or average, change in RADs, and again, ΔPM_{10} refers to the change in annual average PM₁₀ levels.

Over the past 20 years, however, a burgeoning and increasingly refined health literature has produced epidemiological studies that quantify links between fine particulates and a host of other adverse health outcomes (see Table 4). These include: acute bronchitis, asthma ER visits, and lower/upper respiratory symptoms in children; and cardiopulmonary (cardiovascular and respiratory) hospital admissions, work loss days, chronic bronchitis, non-fatal heart attacks and premature mortality in adults. The U.S. Environmental Protection Agency (USEPA), California Air Resources Board (CARB), and other agencies have all drawn from this literature for regulatory impact assessments (RIAs) and other studies over the past 10 to 15 years, in most cases focusing on the same underlying epidemiological studies

Table 4
Health endpoints included.

Ozone–1989	Ozone–2008
MRADs, 18–64	MRADs, 18–64
Symptoms: mild cough, headache, sore throat, chest discomfort, eye irritation	School absences, ER visits, respiratory hospital admissions, asthma attacks
PM ₁₀ –1989	PM _{2.5} –2008
Premature mortality	Premature mortality, ages 30+
RADs	Acute bronchitis
	Lower/upper respiratory symptoms, ages < 18
	Neo-natal mortality
	Respiratory hospital admissions, ages 65+
	Asthma ER visits, ages < 18
	MRADs
	Work loss days, ages 18–64
	Chronic bronchitis, ages 27+
	Non-fatal heart attacks, ages 18+
	Cardiovascular hospital admissions, ages 18+

to estimate health benefits. (For listings of these epidemiological studies, see EPA, 2005; or CARB, 2006, for example).

We also note that most modern concentration–response relationships are exponential rather than linear (in delta concentrations) because of the underlying relative risk relationships used in most epidemiological studies. The non-linear equation generates a response function whose slope decreases at higher pollutant concentrations, though in the pollutant ranges observed in most epidemiological studies (and for most actual human exposures), observed associations are probably not significantly different from linear (as noted by Pope and Dockery, 2006; EPA, 2005; NRC, 2002). This is especially true for the most studied sub-class of the C/R functions—those for premature mortality. Pope and Dockery (2006) note that over the past 20 years a variety of parametric and nonparametric smoothing approaches have been used to evaluate the shape of the C/R function for this health outcome. While not all of the work possesses the power to make strong statistical inferences regarding function shape, Pope and Dockery conclude that generally the association between particulates and premature deaths seems to be near linear. We therefore feel comfortable viewing the evolution of C/R functions, from linear to exponential, as a smooth one.

Specifically, the functional form currently used is as follows:

$$\Delta C = -C_0(e^{-\beta\Delta P} - 1) \tag{3}$$

where ΔC = the change in the number of cases (of a particular health outcome), C_0 = the number of baseline cases, ΔP = the change in ambient pollution concentrations, and β = an exponential “slope” factor derived from the health literature’s relative risk (RR) factors pertaining to that specific health outcome.⁸

In addition, there has been a shift in focus from PM₁₀ to PM_{2.5} due to epidemiological and toxicological evidence suggesting that the fine particles “may play the largest role in affecting human health” (Pope and Dockery, 2006, p. 711). Fine particles can remain suspended in the atmosphere longer than larger particles, travel greater distances, penetrate more easily into indoor settings, and reach more deeply into the lungs. This switch from PM₁₀ to PM_{2.5} has had a material impact on the estimation of premature mortality effects.

In addition, the early cross-sectional studies had several methodological limitations, in particular not controlling for the potentially important confounding effects of such factors as income, smoking, and diet. Over the past 20 years, the scientific literature that assesses associations between PM_{2.5} and premature mortality in adults has expanded rapidly, with the emergence of several large-scale, multicity, prospective cohort studies (in particular, Dockery et al., 1993; Pope et al., 1995). A key feature of these studies is their ability to incorporate information on confounding variables and subjects’ place of residence. Overall, a stronger concentration–response relationship between fine particles and premature mortality across multiple locations in the United States has been found. More recently, a group of health studies re-analyzed and extended this earlier work (Krewski et al., 2000; Pope et al., 2002; Laden et al., 2006; Jerrett et al., 2005), who focused specifically on California; and Krewski et al., 2009, who focused on multiple regions, including California, essentially substantiating and extending the basic results. Finally, in 2006 the USEPA sponsored an expert elicitation as part of the process of determining what risk factors should be used in risk assessments conducted to inform policy decisions. All of the twelve responding experts chose a central estimate relative risk (RR) factor higher than the central value derived directly from the American Cancer Society study of Pope et al. (2002), which had been widely cited and used in most EPA benefit assessments over the prior decade (Roman et al., 2008).

⁸ These risk factors are related to the β in the concentration–response functions in the following manner: $\beta = (1 + \text{RR factor}) / (\text{Change in Pollution})$.

Given the differing strengths of the primary underlying health studies, and the conclusions from the expert elicitation, we use a weighted average of Jerrett et al. (RR = 1.17), Laden et al. (RR = 1.16), and Pope et al., 2002 (RR = 1.06) in our 2008 study, resulting in a relative risk factor of 1.10 and a concentration–response β of 0.009531. We assign greater weight (two-thirds) to Pope et al. because of the national scope of the study, and the inclusion of California residents. Both of the other studies include smaller samples, in one case including only cities outside of California, and in the other including only Southern California. Greater weight was not given to Jerrett et al., even though it is Los Angeles region-specific, because it is unclear why their results were significantly different from the ACS study, which has been rigorously assessed (Deck and Chestnut, 2008). We demonstrate the increased strength of this PM_{2.5}-based, exponential concentration–response relationship in Section 6 below.

A complete listing of the PM-related health endpoints that can now be quantified appears in Table 4. We note that with the extended range of symptoms now in the health literature, the estimation of particulate-related RADs has essentially been discontinued. This is because RADs were to some degree a surrogate for adverse effects that are now quantified separately, such as respiratory symptoms, ER visits and work loss days. To include RADs in the analysis would likely lead to overestimation of overall effects.

For ozone, a similar evolution has occurred. In 1989 our analysis focused on minor restricted-activity days (MRADs) and dose-influenced symptoms (specifically sore throat, headache, mild cough, chest discomfort and eye irritation). To estimate MRADs, we turned to a study by Portney and Mullahy (1986), which utilized a nationwide 1979 Health Interview Survey (HIS) of over 100,000 individuals to quantify the ozone relationship. This relationship was expressed as:

$$\Delta \text{MRAD}_{ij} = 0.077 \times \Delta O_{3ij} \times \text{POP}_{ij} \tag{4}$$

In contrast, our dose-related symptoms were calculated by applying specific exposure and dose–response functions developed by Kleinman et al. (1989) to the distribution of exposure and dose generated by REHEX. The dose-based approach provided a more comprehensive assessment of exposure than relying solely on epidemiological functions, because it differentiated the population by age within three micro-environmental settings and five activity states. However, the informational requirements to generate dose estimates are prohibitive and costly, so this approach has generally not been followed subsequently.

Instead, over the past two decades, the growing health literature has provided exponential concentration–response relationships for school absences, ER visits, respiratory hospital admissions, asthma attacks, and premature mortality in recent years. MRADs continue to be featured as part of most health assessments, but the work of Ostro and Rothschild (1989) has become the standard with which O₃-related MRADs are estimated. They used a fixed effects model and six separate years of data to determine the statistical association between ozone and minor restrictions in activity. Using a weighted average of their coefficients, the USEPA (2003) developed a “best” estimate β coefficient of 0.0022, which can then be incorporated into the following exponential concentration/response relationship:

$$\Delta \text{MRADs} = -C_0 [e^{-0.0022\Delta O_3} - 1], \tag{5}$$

where C_0 equals the annual baseline MRAD rate of 7.8 per person. Finally, we note that the association developed here is actually several times less sensitive than the Portney and Mullahy results (which we explain further in Section 6 below).

A final technical issue relating both to ozone and PM is how to determine the threshold, a level of pollution below which effects are not expected to occur. Over the past 20 years, successive health

studies have not found a “safe” level for either pollutant. Given this, we have assumed that effects occur down to the *background level* of each pollutant—that is, the ambient concentration that would prevail naturally in the absence of anthropogenic pollution (40 ppb ozone, 6 $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$ and 15 $\mu\text{g}/\text{m}^3$ PM_{10}). For policy purposes this is an important variable, in part because as pollution levels are brought down to the AQS in the most polluted areas, levels will fall well below that in others. Even though these reductions are below the AQS, they still generate health and economic benefits.

5. Economic Valuation

Ideal measures of value would represent all of the losses that result from adverse health effects. They would also accurately reflect real preferences and decision-making processes similar to those we use to make basic choices every day. Generally accepted measures of the value of increased well-being due to reducing the adverse health effects of air pollution include the cost of illness (COI) measure and willingness to pay (WTP) or willingness to accept (WTA) measures. The cost of illness (COI) method requires calculating actual medical expenditures, plus indirect costs (usually lost wages), incurred due to illness. The more inclusive WTP and WTA measures are typically captured by either hedonic, or revealed preference, techniques, such as wage-risk or consumer product studies, or by stated preference studies, usually through the survey, or contingent valuation (CV), approach. While each of these value measures has limitations, collectively they provide a generally accepted range of values for the purpose of assessing the benefits of reducing pollution.⁹

Of course, of all the adverse effects of air pollution, people place the greatest value on reducing the risk of premature death (referred to as the value of a statistical life or VSL). Thus, considerable effort has been made over the years to improve estimation of this value, and it in fact has dominated the evolving discussion regarding the most appropriate way to characterize pollution-related risk, and the valuation of that risk. Here, important issues that must be considered include the effects of health, work status, and age on VSL.

Back in 1989, the range of estimates from early WTP and WTA studies (including wage-risk, consumer market, and contingent valuation studies) placed the value of a statistical life in a wide range from \$490,000 to \$9.2 million (in 1988 dollars). Values below \$1.7 million were questioned, however, because of significant downward biases in the studies from which they were derived. On the other hand, very high wage-risk values (above \$8.6 million) were also questioned because negative job attributes other than risk had not been accounted for adequately or because the values calculated from healthy working individuals would not be representative of the general population, especially elderly individuals (see Hall et al., 1989, for more details).

Consequently, in our 1989 study, we selected three values, representing a reasonable range identified in the literature: \$1.7 million, \$3.7 million, and \$8.6 million. The reasons for these selections were as follows. First, the value of \$1.7 million was the lowest supported by defensible empirical (wage-risk and CV) studies (for example, Gegax et al., 1991, and Jones-Lee et al., 1985). Second, \$8.6 million was within the range supported by some of the wage-risk studies (for example, Garen, 1988). Finally, our mid, or “best,” estimate of \$3.7 million was identified by Viscusi (1986) as both within the range of values attached to involuntary exposure of the public to small environmental risks (typical of the problems posed by urban air pollution) and a reasonable estimate of the representative worker's value of life from both wage-risk and CV studies.¹⁰

⁹ See Freeman, 2003, for example, for a more detailed discussion of these various measures and methods.

¹⁰ In their First Prospective Study of the Benefits and Costs of the Clean Air Act, the US EPA (1997) estimated the VSL to be \$4.8 million (\$4.26 million in 1988 dollars). Our early value is therefore about 87% of that EPA figure.

Table 5
Health outcomes and economic values—1989 and 2008.

Particulates	1989 study (1988 \$)	2008 study (1988 \$) ^a	2008 study (2007 \$)	1989 study (2007 \$) ^a
Premature deaths	1617		3000	
VSL	\$3.7 million	\$3.54 million	\$6.63 million	\$6.93 million
Total value	\$5.98 billion		\$19.88 billion	
Ozone				
MRADs	17.65 million		961,400	
Unit value	\$34.95 ^b	\$35.08	\$65.70	\$65.46
Total value	\$379.5 million		\$63.16 million	

^a Adjusted for price level (CPI) and income changes.

^b Commonly-used Tolley et al. (1986) value (our value was \$21.50).

Since 1990, there has been an ongoing expansion of the economics literature assessing the value of reduced workplace risk of death. Wage-risk studies conducted over the past 20 years now more carefully control for job attributes that are not related to differences in risk (Viscusi, 1992, 1993, 2004; Viscusi and Aldy, 2003). There is also a smaller, yet also evolving, literature that investigates differences in consumer expenditures relative to risk of injury or death associated with product use. The results from this work are generally consistent with the wage-risk studies (Atkinson and Halvorsen, 1990; Viscusi, 1992). The CV technique has increasingly become a significant source of values over the past two decades, as the methodology has matured and become more widely accepted, and as policy-makers have become more engaged with the application of economic values to decision-making (Carson et al., 2001). Finally, several “meta-analyses” have been completed that assess the value of reduced risk based on statistical amalgamation of multiple underlying studies (see Kochi et al., 2006; Mrozek and Taylor, 2002; and Viscusi and Aldy, 2003).

Recognizing that values adduced from workplace behavior of healthy younger and middle-aged adults might not accurately reflect the value to older adults of avoiding environmental risk, recent research has also focused on the effects of health status and older age on VSL (Alberini et al., 2004), finding no strong evidence that VSL declines significantly with age, and then only at age 70 and above. Further, those with underlying health conditions report little difference in VSL from those who are healthier. At the other end of life, there is evidence (Dickie and Messman, 2004; EPA, 2003 and the references therein) that families and society place a higher value on children's well-being, but there is no well-established basis to adjust adult values to account for this. Consistent with these findings and the recommendations of peer-review advisory groups, benefit assessments carried out for proposed federal and state rules and programs (EPA, 2003, 2005; CARB, 2005, 2006, 2008; SAB-EPA, 2007; NRC, 2008) do not make any adjustments for age or health status. Quality adjusted life years (QALYs) are a common basis for assessing the relative costs and benefits of medical treatment, suggesting that this method might also be appropriate for evaluating public policies directed at protecting health and saving life years, especially for valuing risks that might fall predominantly on the elderly. However, no consensus has emerged to recommend a shift to this approach.

Accordingly, we continue to rely heavily on VSL derived from wage-risk studies, supported by growing evidence from stated preference studies. There is presently no basis to give any single study greater weight than another, which argues for averaging over a group of studies. Also, it is preferable (EPA-SAB, 2007; NRC, 2008) to include both wage-risk and stated preference (CV) values. This is in part because the VSL should reflect in some way the age distribution of the population at greatest risk (primarily, the older population). CV studies include this population, whereas wage-risk studies largely do not.

For our 2008 analysis, we construct a value based on the recent meta-analyses of Mrozek and Taylor (2002), Viscusi and Aldy (2003), and Kochi et al. (2006). Further, we rely on the U.S.-only values reported

Table 6
Direction and magnitude of factors affecting health effects.

Pollutant	Population exposed	Pollution reduction	Strength of C/R relationship	Number of averted cases
PM (mortality)	27% larger in 1989	60% larger in 1989	3.78 times stronger in 2008	86% higher in 2008
O ₃ (MRADs)	23% larger in 2008	5 times larger in 1989	4.5 times stronger in 1989	18 times higher in 1989

by Viscusi and Aldy, and Kochi et al., and include the expanded revealed preference estimate (based on Kochi et al., developed by Deck and Chestnut, 2008). The mean of the Viscusi and Aldy U.S. values is \$7.6 million, which we average with \$2.5 million from Mrozek and Taylor and \$10.6 million from Kochi et al. This yields \$6.9 million based on hedonic wage-risk studies. Then, we give equal weight to the average wage-risk VSL and the CV value of \$6.3 million calculated by Deck and Chestnut, which they based on CV studies underlying the Kochi et al. meta-analysis, to determine a final VSL of \$6.63 million.¹¹ (All values are in 2007 dollars.)

To contrast this with the value used in 1989, we update the earlier VSL for income and inflation. As stated before, our VSL in 1989 = \$3.7 million (1988 dollars). Adjusting this VSL to 2007 dollars by the all-item CPI for LA/Riverside/Orange Counties moves the value to \$6.535 million. Then, adjusting for the 15.08% increase in median real income in California over the same interval (as reported by the California Department of Finance) and assuming an income elasticity of 0.4 (EPA, 2005), VSL becomes \$6.93 million. Strikingly, this adjusted 1989 value represents less than a 5% difference from the newer value which is derived from a much expanded literature, multiple meta-analyses, and repeated peer-reviews of federal, state and regional regulatory benefit analyses. (A summary of the contrasting values appears in Table 5). In short, twenty years of intense discussion, research and review have not led to any significant alteration of the value used to determine VSL.

5.1. The Value of other Health Endpoints—Ozone

The only non-mortality endpoint that is included in both the 1989 and 2008 analyses is ozone-related MRADs. Our MRAD value in 1989 was \$21.50 (1988 dollars). We derived this estimate from the widely-cited work of Tolley et al. (1986) and a number of other valuation studies (including Loehman et al., 1979). Most other health assessments used Tolley’s median value for a 3-symptom combination, which was equal to \$34.95. Taking this figure and updating it to 2007 dollars results in \$61.73. In contrast, our 2008 study used a value of \$65.70 (again, see Table 5). The difference can be easily explained, however, as MRAD values probably grow by more than the change in CPI. Again, real median household income in California grew by 15.08% from 1988 to 2007. An income elasticity with respect to MRAD valuation of 0.4264 explains the discrepancy (since \$65.70/\$61.73 = 1.0643, or 6.43%, and 0.4264 times 15.08 = 6.43). This income elasticity is consistent with other values in the literature.

6. Comparison of 1989 and 2008 Results

As discussed previously, the health outcomes estimated in our health valuation research have changed over the years, in large part due to the evolution of the health science. Two endpoints continue to dominate the analysis: PM-related premature mortality and MRADs associated with ozone. Clearly, the 1989 versus 2008 results for these two endpoints are quite different—1617 fewer PM-related premature deaths in 1989 compared to 3000 in 2008 (an increase of 86%

over 20 years, despite significant pollution reductions), valued at \$5.982 billion and \$19.88 billion; and 17.65 million fewer ozone-related MRADs in 1989 vs. 961,400 fewer in 2008 (here, the 1989 figure is over 18 times larger), valued at \$379.5 million and \$63.16 million, respectively (see Table 5).

The calculation of these reductions in adverse health outcomes can be visualized in the form of a chain, consisting of several links: reduction in pollutant levels, size of the affected population, resulting health effects (as captured by a concentration–response relationship) and dollar values assigned to the adverse health outcome. By using the REHEX model in targeted ways, we can provide a breakdown of the factors that have changed over the two-decade period. As we have noted, the economic unit values used to monetize these effects have remained nearly constant (in real terms). We therefore focus our temporal comparison on how the changes in exposure and concentration–response relationship have influenced the changes in adverse health outcomes (premature deaths and MRADs) for each of the two time periods.

6.1. PM and Premature Deaths

In our 1989 study, we reported that more than 1600 premature deaths would be averted in the SoCAB if the then NAAQS were attained; the comparable 2008 estimate is 3000. As noted previously, there are three factors that drive these estimates. Eq. (6) and Table 6 present the breakdown of how these factors changed over time. As seen in the equation and table, the susceptible SoCAB population in 1989 was actually 27% larger than 2008’s population. This is because in the earlier period we included the entire SoCAB population, while in 2008, we only considered adults over age 30 in the analysis. This is dictated by the recent health literature, which limits investigation of the pollutant/mortality link to this subset of adults.

Next, we consider the size of the PM reduction required to reach compliance. The REHEX model’s detailed use of monitoring data allows us to calculate that the change in PM needed in 1989 was 60% larger than what is required in the later period, reflecting 2008’s improved air quality.¹² Finally, we measure the strength of the concentration–response relationships used in the two time periods: the Evans et al. (1984) linear equation in the first study versus the exponential form used in the 2008 analysis. By running the REHEX model for 2008’s exposed population and pollution levels with the Evans et al. equation, we calculate that the newer concentration–response relationship is 3.78 times stronger. Thus, we can explain the change in overall premature death estimates in terms of three simultaneous changes as follows:

$$\begin{aligned} \Delta PM \text{ Mortality} &= (\Delta C / R \text{ Relationship}) / [(\Delta Population) * (\Delta PM \text{ level})] \\ &= (3.78) / [(1.27) * (1.60)] = 1.86. \end{aligned} \tag{6}$$

Our breakdown therefore accounts completely for the 86% increase in predicted premature mortality reduction between 1989 and 2008.

¹¹ A recent EPA (2005) estimate of \$7.36 million (adjusted to 2007 dollars and California income levels) places our value at about 90% of the EPA figure, comparable to the ratio seen for our 1989 work.

¹² As mentioned earlier, we have also taken into account the changing measure of fine particles used in the two time period – PM₁₀ in the first study, and PM_{2.5} in the second.

6.2. Ozone and MRADs

Our 1989 study of the SoCAB estimated that a reduction of over 17 million MRADs would result from meeting federal air quality standards, compared to a much more modest decline of about 1 million MRADs in 2008. Eq. (7) displays the breakdown of how the relevant factors changed over the 20-year study period. Here, the size of the ozone reduction necessary to meet the NAAQS was 5 times larger in 1989, as determined by the grid-by-grid concentrations estimated by the REHEX model. This again reflects the decline in overall ozone levels in the basin over the 20 year interval. In contrast to the PM results, however, the susceptible population (adults 18–64) is 23% larger in 2008, and the strength of the concentration/response relationship used in 1989 is 4.51 times greater than the equation used for 2008. Again, we determine this by running REHEX for the 2008 population and ozone levels, but with the concentration–response relationship used in 1989.

The large difference in ozone-related MRADs derives from using Portney and Mullahy (1986) in 1989, and Ostro and Rothschild (1989) in our later study. Most health valuation studies now use the Ostro and Rothschild results. These results are consistent with many other ozone studies of the time: Ozkaynak et al. (1984), Schwartz et al. (1988) and Krupnick and Cropper (1989), which generate a fairly tight range of ozone MRAD elasticities (0.06 to 0.13), despite being obtained from studies of different years, locations, and samples. In contrast, Portney and Mullahy's MRAD elasticity of 0.382 is over 4.5 times larger than the Ostro and Rothschild elasticity of 0.082, who provide a detailed explanation of the factors that can lead to such largely different results.

We therefore can explain the change in overall MRAD estimates in terms of the three simultaneous changes as follows:

$$\begin{aligned} \Delta \text{Ozone MRADs} &= [(\Delta \text{ Health Equation}) * (\Delta \text{ Ozone level})] / (\Delta \text{ Population}) \\ &= [(4.51) * (5.0)] / (1.23) = 18.33 \end{aligned} \quad (7)$$

where again, the 2008 concentration–response relationship is 4.51 times more sensitive, and the 2008 change in ozone is 5 times larger, than the corresponding 1989 levels. The susceptible population, in contrast, is 23% larger in 2008. Thus, our breakdown accurately captures the components contributing to the 18-fold greater MRAD reduction estimated for 1989 relative to 2008.

7. Discussion and Lessons Learned

Complex and highly technical analyses of the expected benefits and costs of proposed air quality regulatory programs have become an important component of many decision-making processes. They range from single rules with relatively narrow scope (but potentially large impact), to regional air quality management plans encompassing myriad source categories and millions of residents, to assessment of the entire U.S. Clean Air Act itself.¹³ Over time, some aspects of these studies have become rather standardized, while others remain the subject of debate. The central point is to understand what these studies add to the policy process in terms of improving decisions and ensuring that public health is protected.

The most basic role of such studies is providing context and perspective. For any proposed regulation or program there are entities (firms, industries, individuals and governments) whose operations will be impacted. They are frequently well funded and adept at communicating their estimate of the costs of any action. Interestingly, focusing on broad economic risk is a shift away from previous

¹³ While economic analyses are required for specific regulations and regulatory programs designed to attain the NAAQS, federal law proscribes the use of any economic test in setting the NAAQS themselves.

arguments that pollution was not truly a health risk or that it was technically not possible to reduce pollution sufficiently to meet stringent AQS. In essence, the issue would then be reduced to “is it worth it?” Benefit assessments are necessary to complete the picture. Apart from the economic activity directly generated by abatement itself, and by research and development into new technologies, cleaner air generates very valuable public health benefits, reduces medical costs and improves the lives of millions.

One of the clearest examples of this is the experience of the SoCAB over the past 20 years. During this time, air quality has improved substantially, and the economy has grown, while the standards defining healthful air quality have been lowered. Population has also increased. Reanalysis of the potential benefits of further improvements indicates that they are as large – or larger – than they were 20 years ago when the air was far more polluted. How can this be explained?

Since 1989, the health literature has evolved, both in the number of studies and the extension of methodologies and application of newer methods to expanded time periods and populations. Perhaps the most striking result of this evolution is the increased strength of the association that has been detected between fine particles and premature death. At the same time, some adverse health effects, such as eye irritation and cough, have been subsumed under newer categories, and new endpoints, such as school absences, can be quantified. As a result, there are many endpoints we can quantify in 2008 that we could not in 1989.

Interestingly, the economic values used in most benefit assessments have changed little, in spite of considerable debate about the best means to value risk to life and many new studies and meta-analyses. This could reflect the fact that more extensive research had already been conducted on VSL in the 1980s, relative to the body of research on air pollution and mortality at that time. Overall, revisiting the 1989 analysis in the context of advances in the health and economics literatures, as well as changes in air quality and population as reflected in more recent work (Hall et al., 2008), provides useful perspective regarding both the dimensions of the regulatory challenge and the likely health and economic benefits of seeking to meet that challenge. As policy-makers endeavor to develop regulatory approaches to attaining ever-more stringent air quality standards, it becomes even more important to evaluate the potential benefits of doing so, and to understand why the benefits of further reductions might be large even though substantial progress has already been made. Advancing science, along with economic growth and increased population, make this clear.

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