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An Evaluation of the Environmental and Health Effects of Vehicle Exhaust Catalysts in the United Kingdom

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Since 1993, all new gasoline-engine automobiles in the United Kingdom have been supplied with three-way vehicle exhaust catalytic converters (VECs) containing platinum, palladium, and rhodium, to comply with European Commission Stage I limits on emissions of regulated pollutants: carbon monoxide, hydrocarbons, and oxides of nitrogen. We conducted a physical and economic evaluation of the environmental and health benefits from a reduction in emissions through this mandated environmental technology against the costs, with reference to urban areas in Great Britain. We made both an *ex post* assessment—based on available data to 1998—and an *ex ante* assessment—projected to 2005, the year when full penetration of VECs into the fleet is expected. Substantial health benefits in excess of the costs of VECs were indicated: By 1998 the estimated net societal health benefits were approximately £500 million, and by 2005 they were estimated to rise to as much as £2 billion. We also found through environmental surveys that although lead in road dust has fallen by 50% in urban areas, platinum accumulations near roads have risen significantly, up to 90-fold higher than natural background levels. This rapid accumulation of platinum suggests further monitoring is warranted, although as yet there is no evidence of adverse health effects. **Key words:** air quality, economic evaluation, Great Britain, health risk assessment, pollution, pollution control, platinum, road transport, urban, vehicle exhaust catalysts. *Environ Health Perspect* 112:132–141 (2004). doi:10.1289/ehp.6349 available via <http://dx.doi.org/> [Online 22 October 2003]

In pursuit of efficient pollution control policies and the health benefits that flow from them, it can be useful to evaluate the costs and benefits of specific actions such as the mandated introduction of a technology like vehicle exhaust catalysts (VECs). *Ex post* evaluations allow realized benefits to be reviewed and the results to inform future policy decisions. *Ex ante* evaluations can also assist in the selection of appropriate policy interventions from competing alternatives. In this study we report on an evaluation of the short-term health benefits from the introduction of VECs to gasoline fueled vehicles in Great Britain. In addition to providing an evaluation of this particular technology, this paper illustrates the use of a relatively straightforward, low cost methodology that could be used more widely to address the evaluation of other, alternative technologies.

Road transport accounts for significant proportions of total United Kingdom emissions of oxides of nitrogen (NO_x), carbon monoxide (CO), volatile organic compounds (VOCs), carbon dioxide (CO₂), and particulate matter < 10 µg in diameter (PM₁₀), particularly NO_x (52%), VOCs (37%), and CO (90%) (Quality of Urban Air Review Group 1993). Gasoline-fueled automobiles are the main source of road transport's contribution, accounting for some 50–90% of the sector's share of U.K. emissions (Holman 1996), and in urban areas the contribution from road transport is even higher. From 1993, to comply with European Commission (EC) Stage I limits on emissions of CO, VOCs, and NO_x, all new gasoline-engine automobiles in the

United Kingdom have been supplied with three-way VECs (Salway et al. 1996). A catalyst increases the rate of a chemical reaction while itself not undergoing any permanent change (Heck and Farrauto 1995). VECs contain platinum, palladium, and rhodium, which catalyze the conversion of the incomplete products of gasoline combustion—NO_x, hydrocarbons, and CO—to N₂, H₂O, and CO₂. Automobile lead emissions have also been reduced effectively by the introduction of VECs because unleaded gasoline is required to avoid poisoning the catalysts. Surprisingly, although studies have addressed concerns over the release of platinum in particular (Helmert 1997; Rosner and Merget 2000; Zereini et al. 1998), there do not seem to have been any attempts specifically to assess the health benefits of the reduction in emissions from the introduction of VECs against their costs, either in Europe or the United States. This study appears to represent the first comprehensive attempt to quantify, in health and economic terms, the net benefits of this mandated environmental technology to Great Britain.

Methods

Using 1992 (the year before catalysts became compulsory for all new automobiles) as the base case, we made two evaluations: first, an *ex post* evaluation of the health effects arising from the action of VECs on pollutant levels in urban areas from 1993 to 1998 (the most recent year for which all the necessary data were available); second, an *ex ante* evaluation of the health benefits projected to 2005, since

full catalyst penetration in the car fleet is expected by then, assuming a 13-year car life span (Aguye M. Personal communication). The evaluation was constrained to short-term acute health effects in urban areas in the United Kingdom: These are the effects for which the most reliable epidemiologic evidence is available (with reference to the United Kingdom); most of the population [85%; Office for National Statistics (ONS) 2000] live in urban areas, and this is where public concern is highest.

Health Risk Assessment Model

To undertake these evaluations, we developed and applied a health risk assessment model.

The four principal steps in the modeling process are illustrated in the flow chart below (Figure 1), using PM₁₀ data as a worked example (for clarity, the example relates only to primary PM₁₀). In the first step (Figure 1), we developed a model to estimate reductions in emissions of NO_x, VOCs, CO, and PM₁₀ arising from the use of VECs. In the second step we estimated the resulting changes in ambient urban concentrations for the primary pollutants CO, VOCs, and primary PM₁₀, and for the secondary pollutants NO₂, O₃, and secondary PM₁₀. In the third step we quantified the health benefits from these changes in pollution, in terms of morbidity and mortality, using exposure–response functions from selected epidemiologic studies. Finally, in the fourth step we used established cost–benefit analysis methods to carry out a detailed economic valuation of the selected societal health benefits, which were then weighed against the costs of VECs.

Estimating reduction in emissions. In the first step (Figure 1), we developed a model to estimate reductions in urban emissions of NO_x, VOCs, CO, and PM₁₀ arising from the use of

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VECs. The fitting of VECs is estimated to result in the following reductions in emissions for gasoline vehicles: CO, 81%; hydrocarbons, 89%; NO_x, 85%; and PM₁₀, 50% (Salway et al. 1996). Salway et al. estimated decreases relative to the previous standard [United Nations Economic Commission for Europe (UNECE) regulation 15-04 noncatalyst equipped] by applying urban emission factors (based on experimental measurements of emissions and distance traveled of vehicles meeting each standard and driven under urban test cycle conditions) to urban emissions attributable to gasoline automobiles in the base year, 1992, then year on year to 1998, and then projected to 2005.

Estimates of national emissions of regulated pollutants and their source apportionment are available from the U.K. National Atmospheric Emissions Inventory (Salway et al. 1996). Recently a growing number of urban emissions inventories have also been produced for the United Kingdom (Hutchinson and Clewley 1996; LRC 1997a, 1997b, 1997c, 1998). On the basis of these inventories, the contribution of gasoline automobiles to total urban emissions in 1992 was estimated at 43% for NO_x, 87% for VOCs, 8% for primary PM₁₀, and 87% for CO.

Lead emissions from catalyst-equipped automobiles were assumed to be zero because of the use of unleaded fuel. Unfortunately, in the final analysis U.K. data were insufficient to quantify and value the environmental and health benefits of reduced lead emissions solely attributable to catalysts. This represents an area in which further research is needed.

We estimated increased platinum emissions arising from the use of vehicle exhaust catalysts in urban areas from 1993 to 1998 using traffic data and emissions factors [Artelt et al. 1999; Simoni S. Personal communication; U.K. Department of Environment, Transport and the Regions (DETR) 1999]. This is reported in the results section.

We estimated the penetration of catalyst-equipped automobiles into the fleet, year on year, using data on existing and new car registrations from the DETR (Aguye M. Personal communication). We allowed for increases in traffic growth on the basis of the U.K. government's published figures on traffic growth for cars and taxis for 1988–1998 and their central forecast for 1999 to 2005 (DETR 1999); increases in traffic of 13%, for 1992–1998, and 10% for 1999–2005 were thus derived. In the absence of emissions projections from sources other than traffic, the use of these estimates necessitated the assumption that all other emissions would stay constant over this time. In this manner, and through the use of relevant emission factors, we estimated a series of annual urban emissions from gasoline automobiles from 1992 to 1998 and further projected to 2005.

Estimating changes in concentrations. We then used these urban emissions reductions from gasoline automobiles to estimate the corresponding reductions in ambient urban concentrations from 1992 to 1998 and those projected from 1999 to 2005. Estimates of these changes (step 2) were made for the primary pollutants VOCs and primary PM₁₀ assuming direct relationships; for the secondary pollutants NO₂ and O₃ using relevant conversion formulae from air pollution models (Carlaw et al. 1998; Derwent 1999); and for primary and secondary PM₁₀ together using a simple source apportionment method [Airborne Particulate Expert Group (APEG) 1999]. We also estimated the reduction in carbon monoxide concentrations but excluded them from the health risk assessment because of current uncertainty over available exposure–response functions.

We estimated reductions in ambient urban concentrations of regulated pollutants

relative to average urban concentrations in 1992 [National Centre for Environmental Technology (NETCEN) 1999]. Because we considered only health end points for short-term acute effects in urban areas in this evaluation, for primary pollutants it seemed reasonable to assume direct relationships between emissions reductions and reductions in concentrations. Air pollution modeling has shown that in urban areas, where buildings prevent dispersion of emissions, low-level emissions disperse very slowly, so that over short distances concentrations are directly proportional to the emission rate of inert primary pollutants (Williams 1996).

For the secondary pollutants O₃ and NO₂, we estimated the effect of reducing precursor emissions on concentrations using relevant air pollution models developed by the U.K. Meteorologic Office and the South East Institute of Public Health (SEIPH) (Carlaw et al. 1998; Derwent 1999). We developed a

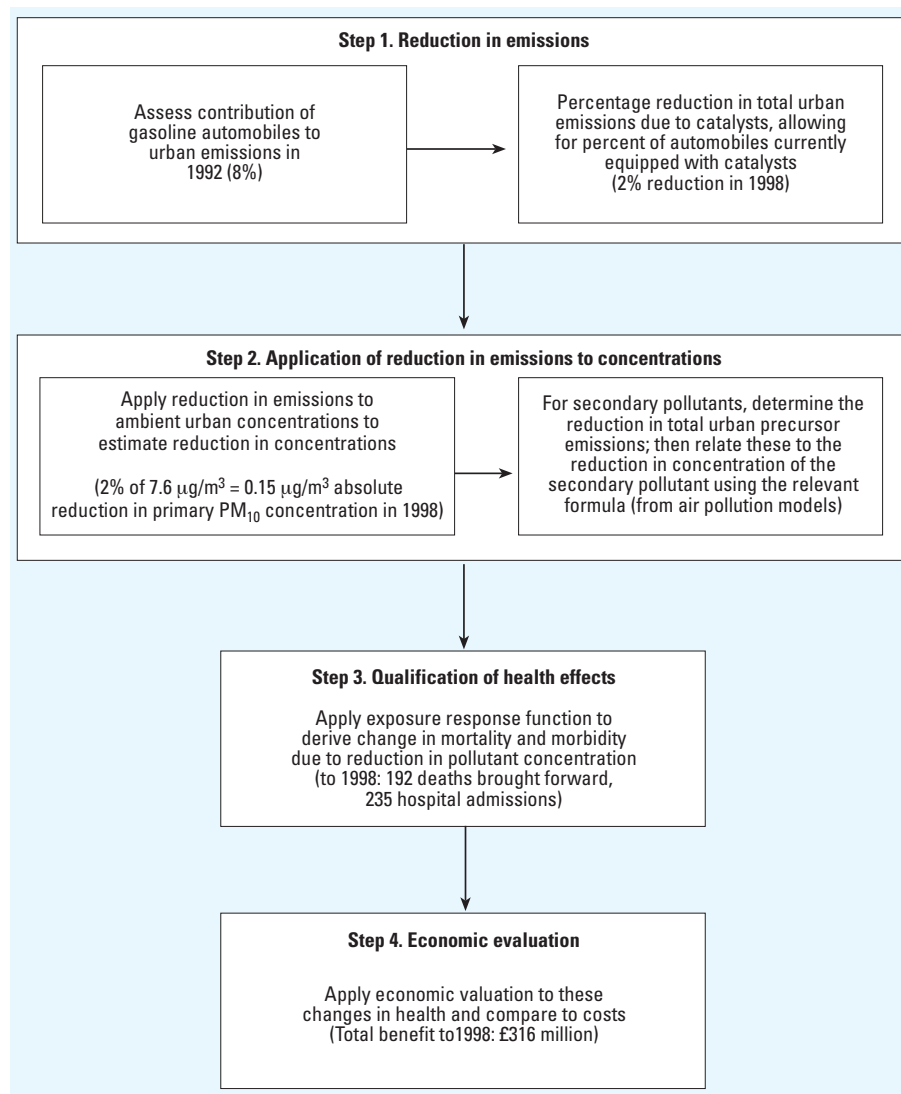


Figure 1. Method for evaluation of catalyst pollution abatement (illustrative figures for primary PM₁₀ to 1998).

simple source apportionment method for PM₁₀ using the latest data available from APEG (1999). A weighted concentration was used, based on the contribution of gasoline car emissions to each fraction of particulate matter, to take account of the reduction in both primary emissions and the secondary precursor emissions. The estimated change in urban concentrations of these pollutants attributable to the action of VECs is illustrated in Figure 2. We also estimated the absolute reduction in urban concentrations of PM₁₀ (primary and secondary), NO₂, and O₃ arising from the reduction in emissions by the use of VECs in urban areas for each of the years 1993–1998, as well as further projections to 2005.

Quantification of health effects. We analyzed the relationship between human health impacts and reductions in pollutant concentrations because of the spread of VECs according to the methods employed by Calthrop (Maddison et al. 1996). The relationship can be expressed as

$$\Delta H_{ij} = b_{ij} \times POP \times \Delta A_{jt},$$

where ΔH_i = change in health impact i per year; j = a particular pollutant j emitted from exhaust pipes; t = transport fuel type—diesel, gasoline, or unleaded gasoline; b_{ij} = slope of exposure–response function of health effect i per year with respect to pollutant j ; POP = population exposed to pollutant j ; and ΔA_{jt} = change in ambient concentration of pollutant j attributable to emissions of specific fuel types.

We quantified the health benefits from the estimated changes in pollutant concentrations in terms of effects on morbidity and mortality, using exposure–response functions from selected epidemiologic studies [Anderson et al. 1997; Committee on the Medical Effects of Air Pollution (COMEAP) 1998; Dab et al. 1996; Dockery and Pope 1997; Katsouyanni et al. 1997; Spix et al. 1998; Sunyer et al. 1997; Touloumi et al. 1997], combined with population data for urban Great Britain in step 3 (ONS 1998, 2000a, 2000b). These epidemiologic studies

were selected from the following reviews and meta-analyses and are applied to either the United Kingdom or Europe:

- The European Union Environment 1991–1994 Programme's Air Pollution and Health—A European Approach (APHEA) project, "Short Term Effects of Air Pollution on Health: A European Approach Using Epidemiological Time Series Data: The APHEA Protocol" (Katsouyanni et al. 1996)
- The U.K. Department of Health's COMEAP review, *Quantification of the Effects of Air Pollution on Health in the United Kingdom* (COMEAP 1998)
- The Royal Society of Chemistry's *Air Pollution and Health*, a review of current knowledge in their *Issues in Environmental Science and Technology* Series (Hester and Harrison 1998).

The APHEA project consists of time-series studies conducted in 15 cities in 11 European countries, including Greece, Germany, France, Italy, Poland, The Netherlands, Spain, Finland, France, Slovakia, and the United Kingdom. Because of the availability of these studies, we thought it most appropriate to assess critically and draw on the existing studies, rather than to conduct further separate meta-analyses. Available individual studies relevant to the United Kingdom were also appraised and used to inform selection of exposure–response functions from the reviews (Anderson et al. 1996, 1998; Ponce de Leon et al. 1996; Walters et al. 1995; Wordley et al. 1997). Available reviews and meta-analyses from the United States were also explored where available and used to produce an upper-bound estimate of health effects (Dockery and Pope 1997; Pope et al. 1995; Schwartz 1994).

General criteria set out by Wright et al. (1997) for selecting epidemiologic studies for exposure–response assessment were applied to the selection of studies used in this work; they were also applied in the APHEA studies, the World Health Organization study, and the U.S. studies that have been used as the main basis for estimating exposure–response functions in this work (Hutchinson 2001). These criteria include consistency of findings with other studies, quality of exposure data for the relevant period, statistical precision of the risk estimates, inclusion of data on exposure–response relationships, and inclusion of data concerning major confounding factors. Further discussion of the selection criteria employed in this study may be found in Hutchinson (2001).

These epidemiologic studies use time-series methods for investigating the association between air pollution levels and ill health in urban areas. All selected studies controlled for season and the effect of co-pollutants, and, where possible, studies conducted in European

cities were used. Thus exposure–response functions for mortality and hospital admissions for PM₁₀ and O₃ and for hospital admissions only for NO₂ were combined with population, mortality, and hospital admission data to produce estimates of the numbers of deaths brought forward and of respiratory hospital admissions (additional and brought forward) avoided by the use of VECs in urban areas. Although exposure–response functions for NO₂ are available for all cause mortality (Touloumi et al. 1997), there is a lack of consistency among studies, and some studies have found evidence of confounding with black smoke (Zmirou et al. 1998) and other vehicle-derived pollutants (Kinney and Özkaynak 1991). In light of this evidence, we judged that a robust exposure–response function could not readily be estimated for the effects of NO₂ on mortality. Therefore we used only exposure–response functions for hospital admissions for NO₂ in this work.

In line with current practice, we assumed the health benefits arising from each pollutant to be additive, and thus estimated total benefits by summing the benefits from the change in each of the three pollutants considered here. It is possible that their individual effects are not simply additive and may, for example, exacerbate one another. However, in the absence of estimates of the effects of pollutant mixes specific to gasoline-powered vehicles, a simple additive model was adopted. For carcinogenic mixtures, at least, this has been reported as a reasonable assumption at low levels (Krewski and Thomas 1992).

For each of the pollutants considered, linearity and the absence of a threshold were assumed. There has been some debate over the existence of a threshold for O₃, below which no adverse health effects occur. Although one study conducted in London for 1987–1992 produced evidence for the existence of a threshold at 40–60 ppb maximum 8-hr O₃ (Ponce de Leon et al. 1996), other studies have failed to show evidence for such a threshold for effects (COMEAP 1998). Recent work comparing seasonal ozone concentration has not contributed further to the identification of a threshold (COMEAP 2002). In this study we adopted a no-threshold approach and linearity of effects, following the approach of COMEAP (1998). In light of the debate over thresholds, our results should be interpreted with appropriate caution.

The most robust epidemiologic evidence exists for PM₁₀ and O₃, whereas less is available for NO₂ and CO. After stringent review, we decided not to estimate the health effects of CO, VOCs, and lead because of uncertainty about the shape of the exposure–response function for CO at the low concentrations that are found in the United Kingdom, and lack of exposure–response

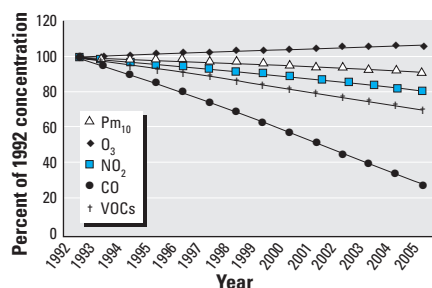


Figure 2. Estimated percent change in urban concentration of selected pollutants arising from the use of vehicle exhaust catalysts, using a model developed in this work.

functions for the relevant endpoints (mortality and hospital admissions) for VOCs and lead. For lead, it was also difficult to identify clearly the role of catalysts in reducing lead emissions.

Evaluations of other health end points, such as symptoms or chronic effects, were not included because of variations in data quality, diagnosis patterns, measurement error, difficulties in estimating retrospective exposure, and consequent uncertainty and lack of agreement over appropriate exposure–response functions. We selected exposure–response functions from time-series studies rather than cohort studies because the available and relevant time-series studies are more robust—in particular, they are not subject to as many confounding factors—and there is a greater availability of such studies conducted for the United Kingdom and Europe. Most of the available cohort studies have been conducted in the United States, thus introducing transferability issues. Our use of time-series studies is consistent with the methodology used by COMEAP (1998). However, also in line with this approach, similarity of baseline factors between the United States and United Kingdom has been assumed here in cases where short-term U.S.-based studies are used in central estimates of exposure–response functions.

To quantify the health effects arising from changes in air pollution because of the action of VECs, we applied exposure–response functions (estimated percentage changes in mortality or morbidity rates) to the underlying mortality and morbidity rates for the urban U.K. population for 1992. These changes can then be quantified by applying these percentage increases or decreases to the urban U.K. population for 1992. This methodology has been widely reported in the literature [COMEAP 1998; DETR 1998; Economic Appraisal of the Health Effects of Air Pollution (EAHEAP) 1999; Maddison et al. 1996].

Thus, data were required on the population and on the underlying mortality rate and the hospital admission rate for Great Britain. We obtained population statistics for Great Britain for 1992 (mid-year estimates) from the ONS, and extracted estimated population figures for urban areas on the basis of the 1998 ONS area classifications. This yielded an estimated urban Great Britain population of 47,704,322, which represented 84.6% of the total Great Britain population (ONS 2000).

The crude annual death rate for Great Britain for all causes was 1098.19 per 100,000 population in 1992 [1021.91, excluding deaths from external causes (*International Classification of Diseases*, 9th ed. [ICD9] codes > 800); ONS 1998]. Most cities have an approximately 10% higher mortality rate than the overall national U.K. rate. However, no

data are available for an overall urban mortality rate (ONS 1998), so in line with the COMEAP (1998) report, we assumed the Great Britain mortality rate to apply to both urban and rural areas. Consequently, this may have the effect of biasing the estimates of health benefits downward. Because data for monthly mortality were not readily available, the mortality rate for England and Wales for summer only (April–September), of 506.8 per 100,000 in 1995, quoted in COMEAP (1998), was used in the health risk assessment for O₃.

Data on total hospital admissions by diagnosis in National Health Service (NHS) hospitals are collected in England by the Hospital Episodes Statistics (HES; Collin L. Personal communication) service. Data for all respiratory (ICD9 codes 460–519) hospital admissions for 1992–1993 were obtained. By combining this with the mid-year population estimate for England in 1992, rates of 1245.83 respiratory hospital admissions per 100,000 population in 1992–1993 and of 770 emergency respiratory hospital admissions per 100,000 population in 1992, were derived. In line with the approach of the COMEAP (1998) report, we assumed all hospital admission rates for England to apply to all urban Great Britain. Because data by season were not available, the rate of 345 emergency respiratory hospital admissions per 100,000 for 1993, for the summer only (April to September), quoted in COMEAP, was used in the O₃ health risk assessment.

We obtained the estimated number of deaths not brought forward and the number of hospital admissions avoided and not brought forward, respectively, by making the following estimates for each year and then summing them over all the years considered. For example, for deaths not brought forward, the estimate was based on the product of the urban population and the change in the mortality rate each year (divided by 100,000), where the change in the mortality rate in each year was given by the underlying mortality rate in 1992 multiplied by the exposure–response function for mortality applied to the change in the

concentration in each year produced by the action of VECs.

Illustration for Primary PM₁₀

- Urban population in 1993 (84.6% of Great Britain population of 56,388,087) = 47,849,255
- Recorded 1992 urban PM₁₀ concentration = 7.57036 µg/m³.
- Estimated 1993 urban PM₁₀ concentration = 7.57000 µg/m³ (estimated from PM₁₀ emission factors and proportion of urban PM₁₀ from gasoline fueled automobiles).
- Hence, decrease in concentration because of VECs = 0.00036 µg/m³.
- Mortality exposure–response coefficient = 0.00074.
- Apply to decrease in concentration: proportional decrease in mortality = 0.00074 × 0.00036 = 0.000015.
- 1992 mortality rate = 1098.19.
- Therefore, 1993 decrease in mortality rate = 1098.19 × 0.000015 = 0.02
- Number of deaths not brought forward = (decrease in mortality rate × urban population) ÷ 100,000 = (0.02 × 47,849,255) ÷ 100,000 = 7.98

We followed the same procedure for hospital admissions, using changes in the underlying hospital admission rate as estimated from the relevant exposure–response functions selected above.

Economic evaluation. Using established cost–benefit analysis methods [U.S. Environmental Protection Agency (EPA) 2000], a detailed economic evaluation of the selected societal health benefits was conducted, summed for all pollutants, and then weighed against the costs of VECs (step 4). Economic valuations were applied to the estimated changes in morbidity (respiratory hospital admissions) and mortality (deaths brought forward). The former are willingness-to-pay (WTP) estimates that incorporate values for the pain, suffering, and lost time component associated with an increase in risk for hospital admissions—thus they represent the value of avoiding a stay in hospital. The WTP values

Table 1. Selected values for mortality, morbidity, and NHS costs to be used for transfer (central estimates).

Health effect	Monetary value (£ 1998)	Monetary value (\$US 1998)	Study
VOSL ^a	1,490,000	2,260,000	EAHEAP 1999
Hospital admission (WTP) ^b	565.40	856.67	EAHEAP 1999
Hospital admissions for respiratory disease (NHS cost) ^b	2124.10	3218.33	EAHEAP 1999
Cost per catalyst ^c	227.30	344.39	Thornton C. Personal communication; SMMT 1998

Abbreviations: NHS, National Health Service; SMMT, Society of Motor Manufacturers and Traders.

^aAdjusted for age to reflect the > 65 age group, which is most likely to be at risk for adverse health effects from air pollution. ^bEleven days. ^cBecause no officially published figures exist for the cost of catalysts in Europe (DETR 1998a), we estimated a weighted average excluding value-added tax retail price for the replacement cost of a catalyst, based on recommended retail prices for replacement catalysts for the most common makes and models of automobiles purchased in the United Kingdom (SMMT 1998). These prices are likely to be higher than the actual cost to car manufacturers for the original catalyst, and therefore will bias the cost component upward.

used in this work were selected from an array of recent contingent valuation methods (CVM) studies (EAHEAP 1999). Medical costs were represented by average U.K. National Health Service hospital admission costs for respiratory illness (EAHEAP 1999). Values for changes in mortality due to changes in pollution were estimated from recent value of a statistical life (VOSL) studies (Day 2000; Desvousges et al. 1998; EAHEAP 1999). VOSL represents the cumulative willingness to pay of a population to reduce the risk of death of a random member of that population, and is used to represent the value society places on the enjoyment of a longer, healthier life (Porter 1999). The central estimate used for VOSL in this study was based on results derived by EAHEAP (1999). It was adjusted for age to reflect the > 65 age group, which is most likely to be at risk of suffering adverse health effects from air pollution. The EAHEAP report took £2 million (1996 prices) as the baseline VOSL, representing the average value for people of average age (40 years) and average health in various contexts. Adjustments were made to this baseline value to allow for the older age and impaired health of those affected by air pollution: WTP among those > 65 years old was stated to be approximately 70% of WTP among those of 40 years old, whereas adjustment for life expectancy ranged from no adjustment (average 12 years at age 65) to 1 year to 1 month. Therefore WTP would be reduced by zero, one-twelfth, or one-twelfth of one-twelfth respectively. Those affected also often have a lower quality of life than average [0.76 using the EuroQual quality of life questionnaire instrument (EAHEAP 1999)] for their age of

between 0.2 and 0.7. Given that a lower quality of life may be expected to reduce WTP, we assumed WTP to decrease in proportion to quality of life, by a factor of 0.2/0.76 to 0.7/0.76. This produces a range of VOSL from £1.4 million to a WTP of £2,600. The age-adjusted-only value was used in this study, while the lower bound was used in a sensitivity analysis.

Costs for catalysis were based on the recommended retail price for a replacement catalyst because no officially published figure for the cost of VECs exists for Europe (DETR 1998a). Various motor manufacturers and organizations were approached but none agreed to disclose any data, citing restrictions placed by commercial confidentiality. The value used in this work was, therefore, a weighted average retail price for the replacement cost of a catalyst (excluding value-added tax), where the weighting took into consideration the makes and models of the most popular automobiles registered in the United Kingdom in 1994 (the closest year to 1992 for which data were available). A weighted average of £227.29 in 1998 prices was thus derived.

The capital costs of catalysis were “annualized” through the use of annuity rates given by the formula $A(n, r) = r / (1 - (1 + r)^{-n})$, where A = annuity, r = discount rate, and n = lifetime of converter (13 years). This enabled the study to ascribe an annual VEC charge for each relevant year of use, and so allows for the capital costs (principal plus interest) relating to catalysis still in use beyond the period of the study. Discounting is the process whereby the values of future effects are adjusted to make them comparable to the values placed

on current consumption, costs, and benefits, because a given amount of future consumption is worth less than the same amount of consumption today. Present values are obtained by multiplying the future values of a policy’s effects by discount factors that reflect both the amount of time between the present and the point at which these events occur and the degree to which current consumption is more highly valued than future consumption. A U.K. public sector discount rate of 6% was used in this work. For discounting the net benefits, we used a U.K. public sector discount rate of 6%. The monetary values used for the central estimate in this study, normalized to 1998 Great Britain pounds, are shown in Table 1. A 1998 \$US equivalent may be derived using a purchasing power parity adjusted exchange rate of 0.66 (i.e., £1998 divided by 0.66)

Environmental Surveys

Although no currently known health effects are associated with the platinum forms emitted from VECs (metal and oxide), adverse health effects have been observed in occupational studies of exposure to various platinum compounds, particularly halogenated platinum salts (HSE 1990; Merget 2000; Rosner and Merget 1990). Because the possibility remains of future transformation in the environment of platinum and platinum oxides, we investigated whether any changes had occurred in the levels of platinum and lead in soils and road dusts in the U.K. urban environment since the 1993 introduction of VECs as part of this study. To do this, environmental surveys were conducted in the cities of Nottingham and Birmingham.

Table 2. Estimated physical and health effects and economic valuations from reductions in U.K. urban pollution from VECs, 1993–1998 (actual) and 1993–2005 (projected).

Effects	PM ₁₀ ^a	PM ₁₀ ^a	O ₃ ^b	O ₃ ^b	NO ₂	NO ₂
	1993–1998	1993–2005	1993–1998	1993–2005	1993–1998	1993–2005
Physical effects (reductions in concentrations)						
Average ambient urban concentration in 1992	26.6 (µg/m ³)		15.28 (ppb)		29.58 (ppb)	
Estimated reduction in pollutant (unit)	0.92 (µg/m ³)	2.24 (µg/m ³)	−0.46 (ppb)	−1.04 (ppb)	2.45 (ppb)	5.59 (ppb)
Health effects (mortality and morbidity)						
e-r function mortality ^c	0.074	0.074	0.106	0.106		
	(0.044–0.075) ^d	(0.044–0.075) ^d	(0.051–0.153) ^e	(0.051–0.153) ^e		
Reduction in deaths brought forward ^f	1,186	5,859	−391	−1,832		
	(705–1,202)	(3,483–5,938)	(−188 to −565)	(−878 to −2,634)		
e-r function morbidity ^d	0.08	0.08	0.137	0.137	0.096	0.096
	(0.045–0.11) ^d	(0.045–0.11) ^d	(0.061–0.210) ^e	(0.061–0.210) ^e	(0.008–0.187) ^e	(0.008–0.187) ^e
Reduction in respiratory hospital admissions	1,454	7,185	−345	−1,609	2,833	13,050
	(818–2,000)	(4,042–9,879)	(−153 to −528)	(−713 to −2,459)	(227–5,553)	(1,044–25,578)
Economic evaluation of present values of net benefits (£1998)						
Present value of total benefits £million ^h	1953.86	7,378	−644.49	−2,306.30	8.39	29.72
	(1160.74–1983.58)	(4,383–7,490)	(−310.10–931.48)	(−1109.71 to −3333.29)	(0.43–21.32)	(1.51–75.54)
Present value of costs of VECs £million			808.15	2944.5		
Total benefits net of catalyst costs £million ⁱ			509.61	2156.9		

Results in Table 2 represent estimates of selected health benefits based on applying central, lower, and upper bound estimates for exposure response (e-r) functions to the inferred reduction in urban pollution arising from the use of vehicle exhaust catalysis. Where a range of exposure response functions was unavailable, the upper and lower 95% confidence intervals for the available function were used to present a lower and upper bound range.

^aPrimary and secondary. ^bSummer. ^cPercent change in mortality rate per unit pollutant; selected e-r functions were all adjusted for co-exposure to other pollutants. ^dLower and upper bounds selected from other studies. ^e95% CI when only one study available, except for NO₂ where two studies were combined and represents lowest and highest CI from the individual studies used. ^fAll causes. ^gPercent change in morbidity rate per unit pollutant; selected e-r functions were all adjusted for co-exposure to other pollutants. ^hFigures in parentheses represent low and high economic valuation estimates for morbidity respectively (comprising WTP to avoid hospital admission and NHS hospital costs). ⁱCentral estimate.

Detailed methods and results have been published elsewhere (Farago et al. 1996; Hall and Pelchat 1994; Hutchinson et al. 2000). Platinum was not, however, included in the health risk assessment model in this evaluation because of the absence of any clearly established health effect.

Results

Estimated reductions in urban concentrations of PM₁₀, O₃, NO₂, CO, and VOCs (relative to 1992 levels) associated with the use of VECs are illustrated in Figure 2. Table 2 shows the results of the health impacts valuation model, both for 1993–1998 and for the projections to 2005. The health effects avoided by the reduction in PM₁₀ dominate these valuation estimates, because of both the estimated large decrease in the number of deaths brought forward and the higher economic values used for reductions in mortality risk relative to morbidity risk. This large decrease in the number of deaths brought forward results from the relatively high exposure–response function associated with PM₁₀ and the comparatively high estimated reduction in ambient concentration associated with the use of VECs. In line with current protocol, we assumed the health effects of each pollutant to be additive and summed the values to produce an overall result. For urban Great Britain, on the health benefits quantified, net benefits of £510 million to 1998 and £2,157 million to 2005 were estimated.

Because nitric oxide is a scavenger of ozone, reductions in urban emissions of NO_x lead to increases in estimated urban ground level ozone concentrations (DETR 1998a). Derwent (1999) has suggested that as NO_x emissions from motor vehicles approach zero, urban NO_x levels would decrease and urban ozone levels would eventually rise to the levels in the rural areas surrounding them. We quantified health effects for the summer period only (April–September) because the exposure–response relationships show a stronger association between ozone and health effects for the summer months, when ozone concentrations are higher. Results of the present model (Table 2) show a considerable negative impact of NO_x reduction on ozone-related health effects, with an estimated 391 deaths brought forward and 345 additional or brought-forward hospital admissions by 1998, and an estimated 1,832 and 1,609 respectively by 2005; however, reductions in urban precursor emissions for ozone can be expected to contribute to reductions in rural ozone levels. The decreases in PM₁₀ and NO₂, on the other hand, suggest significant reductions in these health end points. The greatest benefit in terms of morbidity appears to be associated with NO₂ reductions [in line with results from the economic analysis of the

National Air Quality Strategy Objectives (DETR 1998a)].

Recent published estimates for the VOSL have been considerably higher than the central value used here; this has been historically the case for estimates derived in U.S. studies [£2.66 million (Desvousges et al. 1998); £1.99 million (Levy et al. 2001)], and has also been reflected in a recent European meta-analysis [£3.87 million (EC 2000)]. Estimates based on the latter two values provide a further sensitivity analysis, producing estimated net benefits three to five times larger than the figures in Table 2: They range from £1,533 million to £2,588 million by 1998, and from £6,108 million to £10,207 million by 2005, respectively. This is illustrated in Figures 3 and 4.

Because individuals with pre-existing disease are more susceptible to adverse effects from air pollution, and deaths are not solely additional but consist of deaths brought forward and additional deaths, valuing years of life lost would be preferable to estimating the VOSL per se. No robust estimates of the extent to which deaths are brought forward are currently available which can reasonably

be applied to time-series studies (COMEAP 2001), although the U.K. Department for Environment, Food and Rural Affairs has commissioned studies to investigate and value reductions in the quality and length of life—focusing specifically on risks from air pollutants (Maynard R. Personal communication). If these estimates were significantly less than the VOSL estimates, they would reduce the estimated net benefits below those shown in Table 2: Sensitivity analysis shows that the value of VOSL would have to fall below £1 million to result in a negative benefit.

A first approximation of the value of years of life lost, of £0.0028 million, is provided by EAHEAP (1999). This value is not, however, a true VOSL value but rather an adjusted estimate of VOSL allowing for an estimated minimum reduction in life expectancy (based on current value judgments) and possible maximum reduction in the quality of life because of the health effects of air pollutants. It is therefore not directly comparable with the other VOSL values used in this work; rather, it represents a possible minimum value for life years lost, and is used here only as an additional sensitivity analysis to represent the

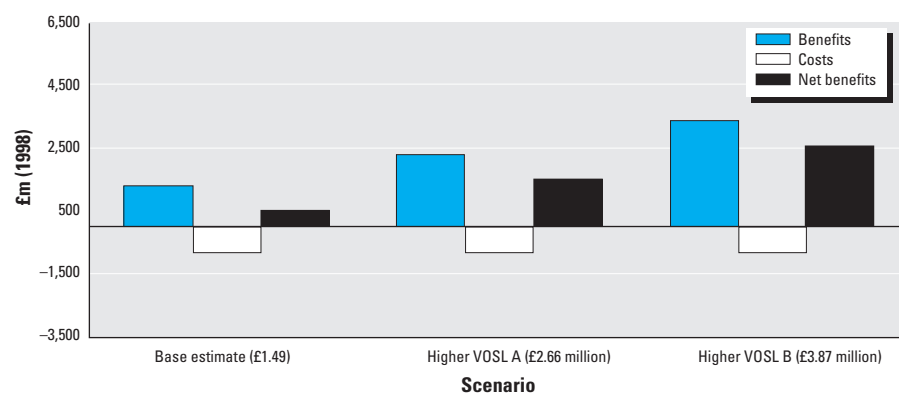


Figure 3. Benefits, costs, and net benefits for selected scenarios to 1998. For the results presented in Figures 3 and 4, VOSL estimates were taken from EAHEAP (1999; base estimate), Desvousges et al. (1998; higher VOSL A), and Day (2000; higher VOSL B). In all other respects, the same parameters were used for the three estimates presented above.

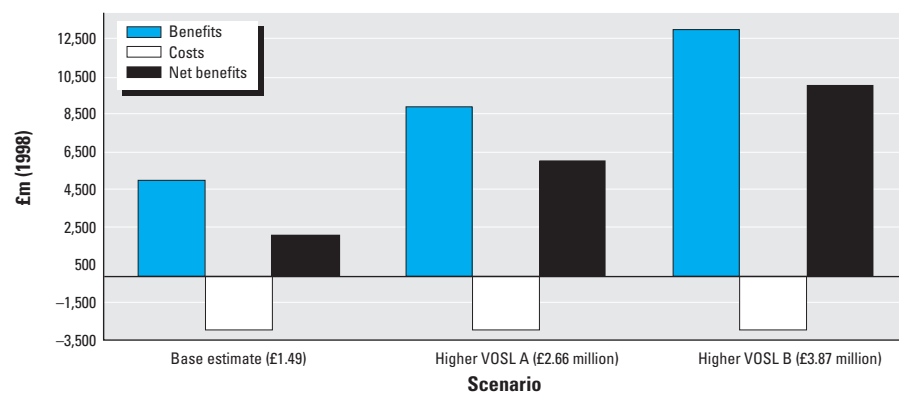


Figure 4. Benefits, costs, and net benefits for selected scenarios to 2005. For the results presented in Figures 3 and 4, VOSL estimates were taken from EAHEAP (1999; base estimate), Desvousges et al. (1998; higher VOSL A), and Day (2000; higher VOSL B). In all other respects, the same parameters were used for the three estimates presented above.

effects of using this type of estimate. Estimated net benefits based on this value were –£794.1 million to 1998 and –£2892.8 million to 2005.

Environmental Survey

The results of the environmental survey showed significant increases in concentrations of platinum in both soils and dusts since the early 1980s. Measurement of metal concentrations in road dust represents recent deposition from traffic sources, as road dust is washed away periodically by rain water. Platinum levels in Nottingham road dust in 1998 were correlated with traffic density: e.g., a geometric mean value of 160.3 ng/g Pt was observed in city center road dust, compared to 30 ng/g in that from residential roads, whereas overall levels in the city increased from 0.8 ng/g Pt in 1982 to 70 ng/g in 1998. An estimate of the increase in total platinum emissions in the United Kingdom was also made through the use of traffic data (DETR 1999; Simoni S. Personal communication) and emissions factors produced from test bench studies (Artelt et al. 1999). Total urban platinum emissions from catalysts for the United Kingdom were projected to be in the range of 13–45 kg to 1998, and 59–210 kg to 2005.

A large (50%) reduction in lead concentrations in road dust was found (from 1,100 µg/g in 1982 to 500 µg/g in 1998), but no statistically significant reduction in soil [195 µg/g in 1982 and 236 µg/g in 1998, respectively; Hutchinson (2001)].

Discussion

The cost–benefit analysis presented above suggests that by 1998, on the benefits quantified, a positive net health benefit had been reached from the use of VECs, and much larger net benefits are anticipated with full catalyst penetration by 2005. The projected estimates for health benefits and their valuation to 2005 were based on relatively simple modeling, and assumptions had to be made to produce these estimates within the scope of this study (e.g., although we allowed for the projected increase in traffic growth, we assumed that all nontraffic emissions remained constant, and no evidence was available on the performance of VECs with increasing mileage). However, with these caveats, by 2005 a further positive net benefit seems likely to be achieved with regard to reductions in deaths brought forward and reductions in additional or brought-forward respiratory hospital admissions.

The general approach in this study was to err on the side of caution, with the aim of reducing the risks of overestimating net health benefits. Consequently, estimates were excluded where there was significant uncertainty about relationships and/or parameters or other data.

Had other associated health effects been included, such as those for symptoms and chronic effects and for the omitted but potentially damaging pollutants [VOCs, polycyclic aromatic hydrocarbons (PAHs), lead], a further increase in health benefits would be expected. The particular exception was the effect on rural ozone of reducing urban precursor emissions. Any effects arising from changes in global or regional pollutants (except low-level ozone) also lay beyond the scope of this study, although they merit examination [for example, catalysts increase nitrous oxide emissions (Wade et al. 1994)]. Other factors, such as the possible health disbenefits from increased platinum exposure and the environmental impacts of increasing platinum production, were excluded because of insufficient evidence through which to quantify them. In our view, however, although there is no current evidence of adverse health effects arising from VEC platinum emissions, the rapid increase in platinum deposition warrants continued monitoring, as adverse health effects have been observed in occupational studies of exposure to various platinum compounds. The environmental survey results highlight the potential complexities of bringing in technologic solutions to pollution control—in this case although several pollutant emissions have decreased (e.g., CO, NO_x, PM₁₀, VOCs, lead), other pollutants, such as platinum, whose effects are not yet fully understood, have increased.

Because our current work is primarily a health risk assessment, we have constrained our analyses to the effects of local urban air pollution. The effects on rural ozone of reducing urban precursor emissions are complex and lie beyond the primarily urban focus of this work. Furthermore, with only 15% of the Great Britain population living in rural areas, we judged that any positive health effects arising from reducing urban precursor emissions would be unlikely to have a relatively large health impact on the overall population (although such effects would undeniably affect rural dwellers and other receptors, such as plants). Nevertheless, recent research gives some impression of current trends in rural ozone: A downward trend has been observed in annual maxima from national U.K. monitoring site data since the 1980s, although this was not significant for most sites [National Expert Group on Transboundary Air Pollution (NEG-TAP) 2001], whereas in an analysis of five rural sites over 1984–1998 (of meteorologically adjusted summer daily maxima), Gardner and Dorling (2000) found a downward trend of –0.7 to –2.3 ppb/year.

On the cost side, the catalyst costs used here are likely to overestimate those borne by car manufacturers: In the absence of quoted manufacturers' data, they were based on retail prices for replacement catalysts (excluding

value-added tax). Data on catalyst recycling were also not available. There is, however, the possibility that catalysts would have to be replaced during the time span considered (thus increasing costs) or might deteriorate over time (thus decreasing benefits). However, catalysts are designed to be operational for the automobile's lifetime and should not require replacement if they are appropriately maintained (Department of Transport 1995). The California Air Resources Board estimated a sales weighted average worldwide cost to manufacturers for "tailpipe emission control" of US\$324 in 1990 (Wang et al. 1993). Using purchasing power parity adjusted exchange rates and allowing for inflation, this is equivalent to £250 in 1998 prices. This is 10% more than the £227 used in this study, although advances in VEC production and volume since 1990 might have reduced U.S. costs.

Since this work was carried out, a new U.K. public sector discount rate of 3.5% was recommended in February 2003 (Her Majesty's Treasury 2003). This figure, closer to the rate often used in U.S. health evaluation studies [e.g., Grosse et al. (2002) used 3%, with sensitivities of 0 and 5%], would have the effect of increasing the estimated net benefits. A previously conducted sensitivity analysis for this work using discount rates of 3 and 0%, respectively, produced estimated net benefits of £615 million and £698 million to 1998 and projected net benefits of £2,886 million and £3,705 million to 2005 respectively. Use of the 6% discount rate in this article is consistent, therefore, with a relatively cautious approach to evaluation.

Carbon Monoxide, VOCs, and PAHs

We judged it not possible reliably to estimate the health impacts arising from the catalyst-induced reductions in emissions of Pb, VOCs, and PAHs at this time, although it seems likely that these reductions would yield further positive health benefits.

Although benefits were initially quantified for CO, the net benefit values presented here also omit the results for CO, because of uncertainty about how the exposure–response relationships derived from epidemiologic time-series studies conducted in the United States and in Athens, Greece, relate to the low concentrations of carbon monoxide found in the United Kingdom (Schwartz 1999; Touloumi et al. 1996). More research is needed in this area to determine whether and to what extent health effects associated with CO are likely at these low exposures. It has been suggested that exposure functions for CO may represent a proxy for the health effects of other pollutants associated with traffic pollution; potential pollutants that have been suggested as also possibly having acute cardiovascular toxicity are the VOCs

(Schwartz 1999). However, this remains untested at present. Additionally, current thinking attributes some cardiovascular toxicity to particulate matter (Medina et al. 2002).

In a program funded by the European Union, intended to evaluate the externalities associated with transport in Europe (EC 1999), effects on health were estimated as well as other external costs, such as accidents. Case studies quantifying damage estimates for health, crops, and buildings were conducted for countries including the United Kingdom. Health impacts dominated the damages quantified, particularly those associated with mortality from primary and secondary PM₁₀. Long-term health effects associated with carcinogens (such as PAHs and benzene) were also quantified but were of much lower importance than particles because the former are emitted only in small quantities. This suggests that, in this work, inclusion of chronic carcinogenic effects for PAHs would have been unlikely to alter the health benefits substantially.

The quantification of the potential health effects from reductions in lead emissions in this study was problematic for several reasons. First—and decisively—difficulties were experienced in identifying the role of catalysts in reducing lead emissions, caused by lack of appropriate data on the relationships between unleaded fuel consumption in the United Kingdom and the factors (in addition to VEC introduction) that have influenced them, including decreased permitted levels of lead in petrol in 1981 and 1985 and the tax-related price differential between leaded and unleaded petrol introduced in 1989 (DETR 1998b). Second, although exposure–response functions for end points other than those used in this work are available for lead (specifically for blood lead and IQ), there is some controversy over the causal nature of the IQ association: Although many studies support such an association, they have been criticized because of their inability to account for all confounding factors; hence, it remains possible that the relationship is not causal (Expert Panel on Air Quality Standards 1998). For instance, children of a lower average intelligence may be more likely to be exposed to lead as a result of their habits and the quality of housing in which they live. Nevertheless, recent studies with better control for social confounding have given more support to the relationship between blood lead and IQ particularly in the United States (DETR 1997; Grosse et al. 2002). Third, uncertainty has also been expressed about the relationship between changes in blood lead and health effects at normal levels (Institute for Environment and Health 1998).

Exposure–Response Functions

Since this research was carried out, further exposure–response functions have been estimated by

Air Pollution and Health: A European Information System (APHEIS) in their second-year report on the health impact assessment of air pollution in 26 cities (Medina et al. 2002). For a 10- $\mu\text{g}/\text{m}^3$ increase in PM₁₀, the following changes were estimated: 1.006 [95% confidence interval (CI), 1.004–1.008] change in mortality rate (all ages); 1.009 (95% CI, 1.006–1.013) change in respiratory hospital admissions rate in ≥ 65 age group; and 1.005 (95% CI, 1.002–1.008) in cardiac hospital admissions rate for all ages (ICD9 codes 410–414, 427, 428). Using these functions in this work would have the effect of slightly lowering the decrease in the estimated numbers of deaths brought forward, but slightly raising the decrease in respiratory hospital admissions. Cardiac admissions were not considered in the current evaluation, because of uncertainty and lack of existing agreement regarding this end point for PM₁₀. Inclusion of this end point would, of course, result in a further estimated decrease in hospital admissions and a consequent increase in estimated benefits. In addition to this, in the studies used for the selection of exposure–response functions in this work, lag times of 0–1 day were generally assumed. However, there is currently a move toward looking at longer lag times (≤ 40 days) to investigate further whether associated deaths are solely brought forward (and so a decrease in deaths would be expected a few days later) or might be additional. In a meta-analysis of daily deaths associated with PM₁₀ in 10 of the APHEA cities, Zanobetti et al. (2002) report an exposure–response function of 1.61% (95% CI, 1.02–2.20) increase in daily deaths (all cause mortality) using a 40-day lag. Substitution of this function in this work would produce a larger estimated decrease in deaths from PM₁₀ and a consequent increase in estimated benefits. Nevertheless, exposures on the day of death and the immediately preceding day still had the greatest impact on mortality in the Zanobetti study (2002).

Recently, Levy et al. (2001) reviewed exposure–response functions for ozone from studies conducted in the United States and produced a central estimate, using meta-analysis, relative to the United States of 0.5% (95% CI, 0.3–0.7%) increase in premature deaths per 10- $\mu\text{g}/\text{m}^3$ increase in 24-hr average ozone concentrations. Inclusion of two European studies produced a pooled estimate of 0.6% (95% CI, 0.4–0.9%). This estimate is compatible with that used in this study (0.05%/1 $\mu\text{g}/\text{m}^3$ O₃)—although the latter is based on an 8-hr average concentration.

We acknowledge that our use of time-series studies precluded consideration of long-term health effects. Had we felt it appropriate to use exposure–response functions from cohort studies, our estimates of

benefits probably would have risen. The exposure–response functions used in this work represent a cautious estimation of health effects, and it is likely that in future epidemiologic studies, health effects, including symptoms, of a greater magnitude may be associated with urban air pollution. Future research may also enable more precise identification and estimation of effects associated with particular size fractions of particulate matter.

Estimates from Related Studies

Few U.K. studies of the effects of air pollution on health have attempted to quantify health benefits from reductions in emissions. Most, such as the COMEAP (1998) and EAHEAP (1999) studies, present estimates of health effects associated with concurrent ambient pollution levels or per unit of pollution. However, the economic analysis of the National Air Quality Strategy objectives (DETR 1998a) estimates benefits for various emissions reduction scenarios with respect to London. Three scenarios were examined, including a “business as usual” scenario representing the expected effect of current policies.

In this scenario, numbers for reductions in deaths brought forward and reductions in respiratory hospital admissions (additional or brought forward) expected to result from the reduction in pollution levels from current policies by the years 2000, 2004, and 2005, relative to a baseline of 1995, were projected: From 1995 to 2005, 5,120 fewer deaths brought forward were estimated to follow from PM₁₀ reductions, whereas total reductions in emergency respiratory hospital admissions were estimated to be 4,225 for PM₁₀ and 8,255 for NO₂ reductions. Because these estimates represent pollution reductions from all current environmental policies, they would be expected to produce higher estimates of benefits than those in this article for the effects of reduced pollution from vehicle exhaust catalyst use alone. Whereas this is so for PM₁₀ (primary PM₁₀ only), for NO₂ the estimates in this article are higher. Because the quantification of health benefits in this article is based on the same exposure–response function, it is possible that the difference arises either from lower estimates of emissions reductions or from the estimation of the relationship between the reduction in emissions of NO_x and the corresponding reduction in NO₂ concentrations. In relation to the latter, in the economic analysis of the NAQS report, relatively sophisticated mapping techniques developed by NETCEN were employed, whereas in the current work we used a simpler relationship produced by the South East Institute of Public Health (Carlaw et al. 1998). Such techniques lay outside the scope and resources of the research reported here.

Ex Ante and Ex Post Cost-Benefit Evaluations

Although the estimates in Table 2 suggest that the introduction of this pollution abatement technology has produced substantial actual and projected positive net benefits—and this is a useful finding—these results do not establish that the mandating of VECs was necessarily the best policy option; alternative policies or actions might conceivably have led to larger benefits or a reduction in cost. This emphasizes the value of undertaking *ex ante* evaluations of sets of alternative environmental policy interventions, to assist in the development of effective, appropriate pollution control policy strategies, as well as *ex post* appraisals of the net benefits realized from the chosen instrument(s).

Conclusion

In an era when growing concern over the health effects of pollution has led governments to adopt increasingly stringent emission controls, this study shows how relatively straightforward techniques can be used to evaluate policy measures. The study represents the first major attempt to quantify and value net health benefits from the mandated introduction of VECs, both for the United Kingdom and elsewhere. For the pollutants and health effects quantified, the results suggest that between 1993 and 1998 there were substantial health benefits in excess of the costs of VECs. Projections through 2005, when full catalyst penetration is expected, suggest net benefits of more than £2 billion. An increase in platinum levels of up to 90 times the natural background was observed only 5 years after the introduction of VECs. Although no negative health effects from platinum emissions have yet been identified, in view of the apparent rapid accumulation rate of platinum into the environment, the situation warrants continued monitoring.

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