APPENDIX 2. ADDITIONAL NOTES ON METHODOLOGY

Emissions approach

The analysis has been undertaken over the period 1990 – 2010. We stress that the data from 1990 – 2000 represents the estimated actual emissions, as recorded and reported in the National Atmospheric Emissions Inventory (NAEI, 2003).

Modelling

The modelling approach used for the analysis of impacts arising from changes in incinerator emissions is consistent with that used for other Defra national air quality mapping studies and previously published work[85]. Contributions to ground level PM_{10} concentrations are influenced by emissions from point sources, area sources, secondary particles and coarse particles. Emissions from point sources specifically were modelled using two atmospheric dispersion modelling techniques. For large sources, (e.g. sources with >100 tonnes PM_{10} emission per annum) impacts of emissions were modelled using the ADMS 3.1 dispersion modelling package. For smaller point sources, (e.g. sources with <100 tonnes PM_{10} emission per annum), impacts were modelled using a dispersion matrix approach.

Health Benefits Additional endpoints for Sensitivity Analysis

COMEAP (1998) provided functions for particles, sulphur dioxide and ozone for two health endpoints - deaths brought forward and respiratory hospital admissions. The group did not quantify health effects from NO_2 due to doubts about the reliability of evidence, though a possible relationship for NO_2 and respiratory hospital admissions was included. The lack of UK studies and uncertainties about the independent effect of CO led COMEAP not to quantify the effects of this pollutant. Subsequent communications from COMEAP (2001) also added the effects of chronic mortality and cardiovascular admissions (as a sensitivity analysis) from particulates, as well as variations on the above functions.

The EC's ExternE Project (EC, 1995; 1999; 2000) also reviewed the relationships between air pollution and health and provided functions for quantification. As well as the functions included in COMEAP, ExternE recommended the quantification of additional health effects where good evidence existed, even if quantification was more uncertain. An example is the use of the US literature on chronic mortality[86]. The dose-response functions recommended by ExternE include mortality due to primary particles ($PM_{2.5}$ or PM_{10}), nitrates, sulphates, SO_2, ozone, benzene and butadiene. Functions for morbidity were recommended for particles, CO, SO_2 and ozone.

Both COMEAP and ExternE form similar judgements on which pollutants are causal (though ExternE has used the size fraction $PM_{2.5}$ and COMEAP PM_{10}). They also agree closely on those functions (e.g. deaths brought forward, respiratory hospital admissions) for which the

[85] Stedman JR, Bush T and Vincent K. UK air quality modelling for annual reporting 2001 on ambient air quality assessment under Council Directives 96/62/EC and 1999/30/EC. A Netcen report to the Department for Environment, Food and Rural Affairs, The Scottish Executive, Welsh Assembly Government and the Department of Environment for Northern Ireland. AEAT/ENV/R/1221 Issue 1 September 2002.

[86] Note chronic mortality was initially excluded from COMEAP, but was later included within the revised statement in 2001.

evidence is strongest. However, because of the additional endpoints considered in ExternE, the two studies provide very different sets of functions. The use of COMEAP functions provides a sub-total of possible health effects, but the confidence associated with this sub-total is high. The ExternE functions include the same sub-total but also quantify other effects for which quantification is more difficult or which necessitates the transfer of dose-response functions from the US. They therefore aim to quantify total effects. However, by including these additional impacts, the uncertainty in the results increases. Note that, even within the approach recommended by ExternE, there may remain unmeasured endpoints.

It is acknowledged that the ExternE functions as reported in 2001 are now out of date (by the ExternE authors[87]). The ExternE analysis was reviewed and updated by the Institute of Occupation Medicine for the Scottish Executive (2003)[88]. There was also a recent implementation of the approach recommended by COMEAP, that including chronic mortality presented in the IGCB Economic Analysis to Inform the Review of the Air Quality Strategy Objectives for Particles (2001).

As a sensitivity, the study has considered the additional functions used in the IOM (2003) analysis for particulates. This increased the morbidity endpoints considered. The functions are presented below.

Concentration-response functions IOM (% change per 10 µgm^{-3} pollutant) used in the study for sensitivity analysis on morbidity from PM_{10}.

	(% change per 10 µgm^{-3} pollutant)	**additional cases/person /year/µgm^{-3}**	**Degree of confidence / units**	**Baseline cases/year/100 000 population**
Cardio-vascular hospital admissions	0.8			
A&E visits for respiratory illness	1.0*		2 TEOM	1122 (inferred from London study)
GP visits: Asthma Lower respiratory symptoms	 3.6 0.4		 2 TEOM 2 TEOM	 4599 (inferred from London study) 20185
Restricted activity days		0.025/adult	4 GRAV	from US study used by ExternE
Respiratory symptoms in people with asthma Adults Children		 0.1676/asthmatic 0.1335/asthmatic child	3 GRAV	 5% adults asthmatic** 10% children asthmatic** (19% of population children)

** studies have reported that 20% of adults and 33% of children have symptoms of wheezing, one study found 21% of children have doctor diagnosed asthma

[87] For example, the PEACE study did not find any evidence of an association of particles with respiratory symptoms. There are also some issues of possible double counting of e.g. restricted activity days and symptoms. Finally, the work of Abbey et al for chronic bronchitis is based on only one study.

[88] Quantifying the Health Impacts of Pollutants Emitted in Central Scotland (P830). Report for the Scottish Executive by: The Institute of Occupational Medicine and AEA Technology plc. Alison Searl, Fintan Hurley, Mike Holland, Katie King, John Stedman, Keith Vincent. February 2003.

A full discussion of the choice of pollutants and functions, and their implementation, is presented in the Scottish Executive Report (The Institute of Occupational Medicine and AEA Technology plc. February 2003).

The starting point for the valuation of health end-points is the identification of the components that comprise changes in welfare. These components should be summed to give the total welfare change, assuming no overlap between categories. The three components include:

- *Resource costs* i.e. medical costs paid by the health service in a given country or covered by insurance, and any other personal out-of-pocket expenses made by the individual (or family).
- *Opportunity costs* i.e. the cost in terms of lost productivity (work time loss (or performing at less than full capacity)) and the opportunity cost of leisure (leisure time loss) including non-paid work.
- *Dis-utility* i.e. other social and economic costs including any restrictions on or reduced enjoyment of desired leisure activities, discomfort or inconvenience (pain or suffering), anxiety about the future, and concern and inconvenience to family members and others.

The welfare changes represented by components (i) and (ii) can be proxied using market prices that exist for these items. This measure - in best practice - needs to be added to a measure of the affected individual's loss of utility, reflected in a valuation of the willingness-to-pay/accept (WTP/WTA), to avoid/compensate for the loss of welfare associated with the illness[89].

In reviewing the morbidity health end-points we use as our starting point the values derived in the EAHEAP working group report (EAHEAP, 1999). There has been one major new empirical study on the valuation of these end-points - CSERGE (1999) - and the results of this study have been used, alongside previous ExternE analysis (EC, 2001), together with hospital-based health care (Netten and Curtis (2000)), and the costs of absenteeism based on figures contained in Confederation of British Industry (CBI, 1998). A brief description by endpoint follows:

Cardiovascular-hospital admissions. To our knowledge, there have been no new studies undertaken to estimate the WTP for cardiac hospital admissions. As a consequence, we have used the generic hospital admission values derived by EAHEAP.

A&E visits for respiratory illness. The EAHEAP report did not derive a WTP valuation for this health end-point over and above the hospital costs. The CSERGE study does include such an end-point: it is described as a visit to a hospital casualty department, required for oxygen and medicines to assist breathing, followed by five days at home in bed. The mean unit value in the UK is £156 in 2002 prices.

GP visits: Asthma & Lower respiratory symptoms. The EAHEAP report did not directly derive a WTP valuation for this health end-point but instead used a proxy value of between £74 and £84. Netten and Curtis (2000) give unit values for the resource costs of the GP. These

[89] Note that there is the possibility of overlap between components since, for example, the individual will include both financial and non-financial concerns in his/her assessment of loss of welfare. Financial costs are often not borne fully by the individual but are shared through health insurance and public health care provision. Thus, we assume here that the financial costs are separable and measured in component (i). If this is not the case, then a part of the dis-utility measured in the WTP estimate will be incorporated in the private medical costs associated with treatment (or prevention) of the health end-point, and the total valuation should be reduced by an equivalent amount.

vary between £18 and £30 depending on whether the consultation period is 9.36 minutes or 12.6 minutes - the two unit periods suggested - and whether qualification costs are included. We assume that the longer period is more realistic for this condition. Added to this resource cost should be the WTP values for Asthma. CSERGE (1999) found a WTP to avoid a day of asthma attack of £50 and £104 per case for adult non-asthmatics, adult asthmatics respectively. These were the values for a sample of respondents that were asked to express their WTP to avoid one additional day of asthma attack (in addition to what they had experienced the last 12 months). The corresponding asthma daily values for a sample that were asked to value an additional day to 14 days were £10 and £11 respectively. For our purposes here, we think that a range of the two should be used, i.e. £10 - £104. The author of this element of the CSERGE study suggests using the marginal day value of £10 as a central unit value. For lower respiratory symptoms a value of £24 may be used. This value was derived for the symptom described as "a persistent phlegmy cough occurring every half-hour or so, and lasting one day".

Restricted activity days. The EAHEAP report did not directly derive a WTP valuation for this health end-point. A value is available from the CSERGE study. Here, the symptom is described as three days confined to bed, where there is shortness of breath on slight exertion. Adopting this as an appropriate description of a restricted activity day, the value to be used is £99[90].

Respiratory symptoms in people with asthma: Adults and Children. The asthma attack values given above for adult asthmatics - £104 per case and £11 per extra day - may be used. For asthma attacks among the respondents' own children the WTP per case was £210, and a WTP of £31 for each additional day of asthma symptoms. The value of £24 used for lower respiratory symptoms may be used instead but it is judged that the asthma value, whilst not the end-point being valued, allows us to consider the WTP values of people who suffer regularly from a similar condition. All these values are derived from the CSERGE (1999) study.

Summary of health end-point unit values.

Health end-point	**unit values (£)**
Cardiac hospital admissions 8 days hospitalisation: 14 days hospitalisation:	1,974 (central)
A&E visits for respiratory illness	222
GP visits: Asthma Lower respiratory symptoms	 40 54
Respiratory symptoms in asthmatics: Adults Children	 104 210
Restricted activity days	99
Chronic bronchitis	Not valued

The sensitivity analysis also considered the potential benefits of reductions of NO_2 (as recommended by COMEAP – see main text), CO and potential carcinogens.

[90] Note there may be an issue here with the application of this value in the context of the original study (consistency).

There is literature on the potential effects of carbon monoxide (CO) on health. COMEAP quoted studies that showed associations between CO and deaths brought forward and cardio-vascular admissions, but did not quantify because of the problems separating CO from other components of the air pollution mixture, and because of the lack of UK studies (though there are now London specific studies). The quantification report did however acknowledge that information on CO is accumulating, that assessment may be possible comparatively soon, and that this would be likely to include hospital admissions for cardio-vascular disease and deaths brought forward. ExternE (EC, 2001) recommended that the function for CO and acute hospital admissions for congestive heart failure was used. We have used this previous work to provide an indicative value for the road transport policies.

Benzene has been shown to be clearly genotoxic (able to damage DNA) in both in vivo and in vitro experiments. Benzene is classified by IARC as Category 1, a known human carcinogen. There is no convincing evidence of chronic non-cancer effects at ambient concentrations. There are many occupational studies investigating exposure to benzene and development of cancer, especially leukaemia, but at high concentrations. Risk quantification is complicated and many different assumptions can be used, i.e. for function shape, exposure pattern (see EC, 2000), but it is possible to use these studies and extrapolate and apply to low concentrations or doses (note this does assume extrapolation below the range of the original data). The US Environmental Protection Agency (EPA) risk assessment for benzene gives a unit risk factor of 8x10-6 per $\mu g/m^3$ (US EPA, 1990) which is the value used for ExternE, and the previous analysis has been used here to provide indicative values for road transport policies. It is stressed that the UK DoH does not recommend these risk factors for use.

1,3 butadiene is potentially carcinogenic to both the white and red cell systems. Animal studies have shown that it is carcinogenic both in rodents and in mice but there is no evidence available on cancer risks to the general population from ambient exposures. As a result, 1,3-butadiene is classified by IARC as Category 2a - Probable human carcinogen. Irritant effects also occur, but only at concentrations much higher than those relevant to road transport. The epidemiological evidence consists mostly of mortality studies that use qualitative estimates or exposure categories rather than estimates of actual lifetime exposures, and with limited consideration of other workplace exposures. The human studies cannot be used directly in quantified risk assessment because sufficiently reliable estimates of past exposures are not available. Thus, the US EPA (1990) unit risk factor (URF) of 3 (or 2.8) x 10-4 per $\mu g/m^3$ lifetime exposure is based on multi-stage modelling of animal (mice) experimental data. As with benzene, there are uncertainties with the use of these factors. This value was used in ExternE, and the previous analysis has been used here to provide indicative values for road transport policies. It is stressed that the UK DoH does not recommend these risk factors for use.

PAHs include a wide range of substances including **benzo[a]pyrene** (BaP). The relationship between BaP and other PAHs differs for various types of emission, but has been shown to be relatively similar in the ambient air of several towns and cities. There is strong evidence, including from epidemiological studies to suggest that certain components of PAHs, and specifically benzo[a]pyrene, are carcinogenic in humans; and that nitroaromatics as a group pose a hazard to health. In 1986 IARC and the US National Cancer Institute concluded that PAHs (PAH mixtures are Category 1 carcinogens) were a risk factor for lung cancer in humans. Benzo[a]pyrene specifically, rather than PAHs as a group, is labelled as a probable human carcinogen. As these compounds form complex mixtures and are also absorbed onto particulates, it is difficult to quantify levels of human exposure and so is difficult to estimate

risks reliably. Benzo[a]pyrene is the only PAH for which a suitable database is available, allowing quantitative risk assessment. The EPA unit risk factor of lung cancer for BaP is 1x10-4 per ng/m^3 (US EPA, 1990). Limitations in the use of benzo[a]pyrene as an indicator of PAH toxicity in air pollution are that some PAH is bound to particulates, and that some of the gaseous components are not included. WHO (1987) estimated a URF of 8.7 x 10-5 per ng/m^3; i.e. almost identical to that used by US EPA. ExternE quantified and valued potential effects, and the previous analysis has been used to provide indicative values for road transport policies. It is stressed that the UK DoH does not recommend these risk factors for use.

Additional Exposure-Response Functions recommended by ExternE, and used in the Sensitivity Analysis for Other Pollutants.

Receptor	Impact	Reference	Pollutant[1]	f_{er} [1]
ELDERLY 65+				
	Congestive heart failure	Schwartz and Morris, 1995	CO	5.55 E-07
ENTIRE POPULATION				
	Cancer risk estimates	Pilkington et al, 1997; based on US EPA	Benzene	1.14 E-07
			1,3-butadiene	4.29 E-06

The exposure response slope, f_{er} , has units of case events per year per person per µg/m^3 , except for mortality which is expressed as percentage increase per µg/m^3

[1] Sources: (EC, 1995: Hurley et al., 1997). Within ExternE, sulphates are treated as $PM_{2.5}$ and nitrates as PM_{10}.

Lead

There is no consistent evidence that lead is a carcinogen. Inorganic lead is currently classified by IARC as a possible human carcinogen (Category 2B). For organic lead there is as yet inadequate evidence for categorisation as a human carcinogen (Category 3). We have therefore not attempted to quantify a carcinogenic effect.

At high exposure, clinical lead poisoning occurs. At the ambient levels from road transport, the earlier literature (appraisals and ex post benefits studies) quantifies a number of health endpoints including hypertension in adults, coronary heart disease events, mortality (neonatal and adult), and IQ loss in children. Moreover, several non-cancer health effects have been proposed or examined. Most of the studies are US based.

Of these, the evidence is greatest for IQ loss in children and increased blood pressure in adults. Several studies have considered the possible relationship between blood lead levels and blood pressure. A meta-analysis of 19 studies by Staessen et al (1993) reported that a two-fold increase in blood lead was associated with 1 mmHg increase in systolic and a 0.7 mmHg increase in diastolic blood pressure. Communication from the Chief Medical Officer[91] also states that 'among adults, there is some evidence of a small increase in blood pressure - for example, an increase in systolic blood pressure of about 1 mmHg, with a smaller increase for diastolic pressure, for a doubling of blood lead from 0.8 to 1.6 µmol/1 (17 to 33 µg/dl^3)'. This suggests a significant weak positive association. However, recent reviews of quantification literature indicated there is still insufficient evidence to implicate low-level lead exposure as a cause of adult hypertension, as poor exposure data and inadequate consideration of confounding factors limit findings both in occupational and population based studies.

[91] *CMO'S Update* 18. Department of Health. May 1998.

ExternE (EC, 1995:1998: 2001) put forward functions for IQ effects. The main evidence with respect to lead in air and childhood IQ is in children. The pathway is in two parts, which need to be considered together:

a. the relationship between lead in blood and childhood IQ; and
b. the relationship between lead in air and lead in blood.

IQ as an endpoint is a very non-specific measure, and there is controversy about what it really represents. However, relationships with blood lead are better established with IQ, and are less variable across studies, than with more specific endpoints.

Lead in blood, and childhood IQ: For this part of the E-R function, consider an estimated reduction based on Schwartz (1994) of:

2.57 IQ points per 10 µg/dl blood lead; in school-age children.

Note, this compares with a communication from the Chief Medical Officer that 'blood concentrations greater than 0.5 µmol/ 1 in children are associated with small subclinical decrements in intelligence quotient (IQ) of about 1-3 points for an increase of 0.5 µmol/1[3]'.

We assume the relationship refers to children at age 10 (in practice, it is based on studies of school-children over a range of ages). Following the results of various meta-analyses, we assume no threshold.

The analysis has to adjust between lead in air and lead in blood. This is not straightforward. Especially in children, ingestion is the principal route of exposure. A UK review (EPAQS, 1998) uses a conversion factor of:

1 µg/m3 in air ~ 5 µg/dl in blood and so ~ 1.25 IQ points.

and this is used here.

There is also an issue of discounting/ time of occurrence. The adverse effects of exposure to lead will be greatest when the child's nervous system is developing most rapidly. We have assumed that the biologically relevant exposure occurs over three years, aged 0-2 inclusive. (There will be pre-natal exposure also; and presumably exposure after age three may have some effect. So clearly, this is a simplification). This implies a time-lag of about nine years, between exposure (at 18 months) and effect (at 10.5 years), which is relevant for discounting, but given the uncertainties in valuation is not accounted for in this study.

We have not modelled lead concentrations in this study. Lead concentrations have declined dramatically with the introduction of unleaded petrol, and there is less data available on concentrations (though some data are available from the ten background monitoring sites in the UK). The green accounting research project (GARP: Markandya et al, 1998) estimated the morbidity impacts from lead at UK concentrations in 1995, based on detailed air quality maps, and this has been used here in providing an indicative analysis. The function for IQ loss was applied to younger children, aged 0-2, using the conversion from lead air concentrations to lead blood levels. The logic for applying the function in this way is that the adverse effects of exposure to lead will be greatest when the child's nervous system is developing most rapidly, thus, the biologically relevant exposure is assumed to occur over

three years, aged 0-2 inclusive. The GARP study quantified the total loss of child IQ points age < 2 yrs in 1996 to be 69,800. Lead emissions were estimated at 1468 tonnes in this year, when compared to emissions of 2703 tonnes in 1990 and 8051 tonnes in 1980 (note, these numbers have since been updated within the NAEI. Extrapolating from the GARP report, the 69,800 IQ points was around 20% of the likely values that would have occurred when unleaded fuel was widely used, i.e. would be broadly equivalent to 350,000 IQ points in children under 2 years of age.

It is stressed that the valuation of IQ loss from lead is extremely controversial, and studies such as ExternE have backed away from recommending valuation. While some US literature does exist, much of it is old. The US strategic plan for elimination of childhood lead poisoning estimated, on average, that each loss of one IQ point resulted in lost lifetime earnings of $4,588 (1990 prices), whereas the URBAIR Guidance (1997) gave a value for the same endpoint of $1147 (1992 prices). Cannon (1990) reports a medical cost of a testing and treatment for children with high lead concentrations (>25µg/dL) as £1,070 (1988 prices) based on a US EPA RIA, with a compensatory education programme of £3,100 per child, and used a total costs of $4,165 (1988) for each case of elevated blood lead level avoided. This gave total annual health benefits in the US of $430 million from the policy. The US data also quantified reductions in cases of hypertension in adults (which derived a much higher annual benefit of $5.6 billion). Overall the programme was estimated to have annual benefits of $6 billion and total benefits (net present value) of $29 billion (1988 prices) between 1985 and 1992, compared with net present costs of only $3 billion. Maddison (1996, based on Rowe et al, 1995) gave a value of £340 per IQ point (1995 prices), though it is unclear if this is an annualised value or a lifetime cost, and is derived from a 1982 study. CDC (1991) used a value of $1,147/1 µg/dL increase (1989 prices) per child based on IQ and loss of lifetime earnings with no threshold – other studies show wage rates falling by 0.5% - 0.8% for one point decrease in IQ. The CDC value was used by ORNL/RFF (1994) as $1,147 per 0.25 IQ loss ($4588 per point), based on total present discounted lifetime earnings loss.

This study has used some of the above data to derive an illustrative value of the potential benefits of lead reductions from unleaded petrol. We stress that there are major problems with accounting for the exposure to lead over several years and its relation to IQ loss, and also in the valuation of IQ loss itself.

A simplified analysis has been undertaken, based on 115,000 IQ points lost per year (i.e. we adjust down the value of 350,000 points from all children age 0-2 by three to annualise exposure), with a lifetime present value loss per IQ point of £4100. This gives an annual benefit every year (with no unleaded petrol) of around £400 million. Over a 20 year period (1990 –2010, without discounting or inflating), this equates to £8 billion. It is stressed that our confidence in these numbers is extremely low, and they should not be quoted in specifically assessing the costs and benefits of unleaded fuel.

Health Benefits: PM_{10} and $PM_{2.5}$ Metrics and PM Measurement

Monitoring of PM_{10} levels in the UK has, to date, been largely based upon the use of TEOM analysers[92]. A principal concern with the TEOM instrument is that the filter is held at an

[92] The tapered element oscillating microbalance (TEOM) is used to continuously measure particulate concentrations at most sites. It automatically measures the mass collected on an exchangeable filter cartridge by monitoring the corresponding frequency changes of a tapered element. The sample flow passes through the filter,

elevated temperature (50ºC) in order to minimise errors associated with the evaporation and condensation of water vapour. This can lead to the loss of the more volatile species (some hydrocarbons, nitrates etc.) and has led to the identification of differences between TEOM and gravimetric measurements[93] at co-located sites.

Currently a factor of 1.3 is applied to all TEOM measured concentrations to estimate the gravimetric equivalent. Further studies have been commissioned by DEFRA to investigate these effects, and to provide a more robust relationship between the TEOM and the European transfer gravimetric reference method. The difference is important in interpreting the epidemiological results (functions) presented above. It means there is likely to be a difference between the measured concentrations for UK and US studies, and by implication, on the functions derived from the studies. The IOM study reviewed the PM_{10} functions to see which are likely to be based on TEOM measurements and which on gravimetric measurements. In general, acute UK functions assume TEOM measurements, whereas US functions (including chronic mortality) assume gravimetric measurement. This necessitates the use of a factor (of 1.3 upwards) for PM_{10} air quality mapping analysis when applying gravimetric functions (i.e. US functions).

Basis for concentration measurements for PM_{10} in exposure-response functions for different health impacts.

C-R Function	PM_{10} measurement Method
Acute mortality	TEOM
Respiratory hospital admissions	TEOM
Cardiac hospital admissions	TEOM
GP visits for asthma	TEOM
GP visits for lower respiratory symptoms	TEOM
Life years lost	Gravimetric
Respiratory symptoms in adults with asthma	Gravimetric
Respiratory symptoms in children with asthma	Gravimetric
Restricted activity days	Gravimetric
Chronic bronchitis (new cases)	Gravimetric
A&E visits for respiratory illness	TEOM

where particulate matter collects, and then continues through the hollow tapered element on its way to an electronic flow control system and vacuum pump.

[93] Gravimetric samplers collect particles on a filter using a pumped system. Mass concentration is determined by weighing of the filter and measurement of the air volume sampled during the monitoring period - normally 24 hours. Both gravimetric and TEOM samplers incorporate an inlet head which selectively samples only the PM_{10} fraction.