## Appendix I.

### Abbreviations and Terminology

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AOT40</td>
<td>Accumulated concentration of ozone over a threshold of 40 ppb</td>
</tr>
<tr>
<td>CBA</td>
<td>Cost-benefit analysis</td>
</tr>
<tr>
<td>CLE</td>
<td>Current legislation scenario</td>
</tr>
<tr>
<td>CO</td>
<td>Carbon monoxide</td>
</tr>
<tr>
<td>CO₂</td>
<td>Carbon dioxide</td>
</tr>
<tr>
<td>CRP</td>
<td>Current reduction plan scenario</td>
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<tr>
<td>CV</td>
<td>Contingent valuation</td>
</tr>
<tr>
<td>DETR</td>
<td>UK Department of the Environment, Transport and the Regions</td>
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<td>DNMI</td>
<td>Norwegian Meteorological Institute</td>
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<tr>
<td>DTI</td>
<td>UK Department of Trade and Industry</td>
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<tr>
<td>EC</td>
<td>European Commission</td>
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<tr>
<td>EU</td>
<td>European Union</td>
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<tr>
<td>IIASA</td>
<td>International Institute for Applied Systems Analysis</td>
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<tr>
<td>IOM</td>
<td>Institute of Occupational Medicine</td>
</tr>
<tr>
<td>ITE</td>
<td>Institute of Terrestrial Ecology</td>
</tr>
<tr>
<td>LRTAP</td>
<td>Convention on Long Range Transboundary Air Pollution</td>
</tr>
<tr>
<td>MFR</td>
<td>Maximum feasible reduction scenario</td>
</tr>
<tr>
<td>NH₃</td>
<td>Ammonia</td>
</tr>
<tr>
<td>NO</td>
<td>Nitrogen monoxide</td>
</tr>
<tr>
<td>NO₂</td>
<td>Nitrogen dioxide</td>
</tr>
<tr>
<td>NOₓ</td>
<td>Oxides of nitrogen</td>
</tr>
<tr>
<td>O₃</td>
<td>Ozone</td>
</tr>
<tr>
<td>PM₁₀</td>
<td>Fine particles less than 10 µm in diameter</td>
</tr>
<tr>
<td>PM₂.₅</td>
<td>Fine particles less than 2.5 µm in diameter</td>
</tr>
<tr>
<td>REF</td>
<td>Reference scenario</td>
</tr>
<tr>
<td>SO₂</td>
<td>Sulphur dioxide</td>
</tr>
<tr>
<td>TFÉAAS</td>
<td>Task Force on Economic Aspects of Abatement Strategies</td>
</tr>
<tr>
<td>UNECE</td>
<td>United Nations Economic Commission for Europe</td>
</tr>
<tr>
<td>VOCs</td>
<td>Volatile organic compounds</td>
</tr>
<tr>
<td>VOLY</td>
<td>Value of life year</td>
</tr>
<tr>
<td>VOSL</td>
<td>Value of statistical life</td>
</tr>
<tr>
<td>WTA</td>
<td>Willingness to accept</td>
</tr>
<tr>
<td>WTP</td>
<td>Willingness to pay</td>
</tr>
<tr>
<td>YOLL</td>
<td>Years of life lost</td>
</tr>
</tbody>
</table>
MATHEMATICAL NOTATION

The following prefixes and suffixes are used in this work;

Ex, $E^{-x}$ as a suffix to a number, denotes that the number in question should be multiplied by 10 to the power $x$ or $-x$. Hence $6.4E^{-3}$ is equal to 0.0064.

The following prefixes to units are also used:
- $n = \text{nano} = 10^{-9}$
- $\mu$ or $u = \text{micro} = 10^{-6}$
- $m = \text{milli} = 10^{-3}$
- $k = \text{kilo} = 10^{3} = \text{thousands}$
- $M = \text{mega} = 10^{6} = \text{millions}$
- $G = \text{giga} = 10^{9} = \text{billions}$

This system is standard notation in the sciences. Note that m and M are not equivalent (by a factor of $10^{3}$) and hence should not be interchanged.
Appendix II

Review of Unpublished Papers

INTRODUCTION

So far as the published literature is concerned the ExternE Project Methodology report (European Commission, 1995) provides probably the most comprehensive summary of external costs assessment methodologies available at the present time. This will be superseded by another report in 1998, reporting on the current phase of ExternE (European Commission, 1998).

This section provides a review of material produced since 1995, little of which has been openly published to date.

The first group of reports considered concern the treatment of individual parameters;

- Valuation of air pollution related mortality (Markandya, 1997; NERA, 1997)
- Studies on the valuation of morbidity (Markandya, 1997; Navrud 1997)
- Studies on damages linked to crop loss (Tonneijck et al, 1997; NIAR/Mortensen/SSB, 1997)
- Assessment of materials damage (Glomsrod et al, 1996)
- Assessment of air pollution damage to historic buildings (Soguel, 1996)

The second group concerns studies that have sought to quantify damages at the European level;

- CBA of the EU Acidification Strategy (Holland and Krewitt, 1996)
- Assessment of ozone damages for the ExternE Project (Rabl and Eyre, 1997)
- EFTEC assessment of the marginal damages of NOx emissions in Europe (EFTEC, 1996)

These studies are reviewed in the following sections.

VALUATION OF AIR POLLUTION RELATED MORTALITY

Mortality related to air pollution has previously been found to be the most important element of damage estimates linked to the emission of acidifying pollutants and ozone precursors. Consequently the approach used for valuation of mortality is extremely important to our analysis. It is thus worth spending some time considering the issue, which has attracted persistent and considerable debate for several years now.
Some people will of course argue that it is wrong to put a value on life. This view ignores the fact that policy makers routinely have to allocate limited resources between healthcare, education, ecological protection, defence and so on. In doing so they intrinsically seek to balance expenditure against the real or perceived ‘value’ of different goods. Approaches alternative to monetary valuation are of course available. However, the use of monetary valuation as a weighting system has significant advantages - in particular it is a system that is universally understood. Following from this it has the potential to improve the transparency of policy making.

Three approaches for valuation of premature mortality have been used or suggested;
1. value of statistical life (VOSL)
2. value of a life year (VOLY)
3. gross output calculations

The first two approaches are based on ‘willingness to pay’. The third approach is based mainly on consideration of the future output, or gross-of-tax income, of victims. Allowance can be made for the contribution of those whose services are not marketed (e.g. those in the voluntary sectors and people undertaking work in the home). Allowance is also made for medical expenses. However, this approach is not considered further here as it has been widely discredited (see NERA, 1997), because people value safety principally, if not exclusively, because of an aversion to death or injury, rather than through concern for preserving national output and income.

The following sections deal first with the question of what, precisely, we must seek to value, based on the results of epidemiological studies. Next brief reviews are given of the advantages and disadvantages of the VOSL approach recommended by NERA (1997) and the VOLY approach recommended by Markandya (1997, to be published as part of European Commission, 1998) under the present phase of the ExternE project. In the face of genuine dispute as to which approach is preferable, suggestions are then made for alternative methods for conducting the valuation within the present study.

What are we trying to value?

Both short-term (acute) and long-term (chronic) exposures to urban air pollution have been linked to mortality in the epidemiological literature (Hurley, 1995). Although there is no consensus on the mechanisms involved, available evidence suggests that effects may be both non-carcinogenic, acting on the cardio-vascular and respiratory systems, or carcinogenic, depending on the pollutant in question. In this study we have restricted the analysis to non-carcinogenic effects. Reduction of VOC emissions may give benefits in terms of reduced cancers, but would require analysis at a finer level of detail than is possible within the constraints of this study.

Short term effects have been characterised using time series studies that compare daily mortality with air pollution either on the same day or for a few days earlier. Exposure-response functions relate ‘excess’ deaths to the concentration of specific pollutants. A problem arises because data from major pollution episodes have shown that many people
‘killed’ by air pollution have only an extremely limited life expectancy regardless of the quality of the air that they breathe, in the region of a few days rather than years. On this basis short term exposure to air pollution at current ambient levels typical of most if not all of Europe can only be regarded as a contributory factor in death. It is extremely unlikely that it is the only factor leading to death, or for the majority of cases even the most important factor. There are no direct estimates of the number of life years lost on average to acute effects of air pollution on mortality.

Chronic effects on the other hand are characterised from cohort studies, comparing the lifespan of known groups of individuals against long run pollution levels (accepting problems with the quantification of ‘lifetime’ exposure, because of a lack of data from the past). Exposure-response functions from these studies essentially quantify the loss of life years, in contrast to the acute studies which quantify the number of deaths. It seems that these functions implicitly account for those dying from short term exposures, so care is needed to prevent double counting. It seems reasonable to conclude that air pollution plays a more fundamental role in bringing life to a premature end for those affected by long term exposure than for those affected only by short term exposure (assuming that there are actually two distinct groups of people). Also that those affected by long term effects would lose a longer period of life to the effects of air pollution, on average, than those affected only through exposure in the short term.

One of the problems for this analysis is that the acute and chronic exposure studies provide different indicators of the loss of life, acute effects being expressed in terms of the number of people affected and chronic effects in terms of the overall shortening of life. Can we express both on a similar basis? For chronic effects it is possible to make some assumption about the average length of life lost to derive the number of deaths. However, such assumptions are based on indirect sources (such as premature mortality among smokers), and as such are subject to a high degree of uncertainty. Alternatively, some work is now being based on life table analysis (Brunekreef, 1997; Hurley and others, IOM, personal communication), though the extent to which it might answer the question of how many premature deaths are linked to chronic exposure to air pollution is not yet clear. Likewise for acute effects an average length of life lost could be assumed, but again there is a problem of adding further uncertainty to the analysis. A range for the average loss of life to acute effects of 2 months to one year has been suggested in work for the UK Department of Health (Hurley, personal communication). This is not based on epidemiological data, but inferred from numerous discussions regarding who it is that might be affected. Most deaths associated with acute effects of air pollution seem likely to be clustered at the low end of the frequency distribution. The average may be inflated well above the median by a small number of extreme cases. Confidence in any estimate is reflected in the language used here - ‘has been suggested’, ‘seem likely to be’, ‘may be inflated’. However, in the absence of definitive advice, and in the belief that a reduction in life span per se is relevant to the economic debate, it is important to make some estimate.

The Value of Statistical Life (NERA, 1997)

The paper “Valuation of Deaths from Air Pollution” by NERA forms a useful addition to the valuation literature. The paper uses the VOSL of road traffic deaths derived for the
UK Government’s Department of Transport (UK £800,000, or 1.1 M ECU) as a basis and attempts to classify and evaluate the changes which might be required to estimate the WTP for air pollution mortality reduction. It addresses most of the issues which might be considered to make air pollution mortality different from road traffic mortality, i.e.

- context - voluntariness, responsibility, controllability, understanding of risk,
- income effects, including utility correction,
- age,
- state of prior health,
- baseline level of risk, and
- non-WTP costs, e.g. health service costs

Effects of different factors that influence the transferability of the ‘roads’ VOSL to the context of air pollution are summarised in Table A1, taken from the NERA report.

The paper concludes that the context would lead to higher values for air pollution than for road accidents by a factor of two. This would raise the figure from analysis of road traffic accidents to a value (2.2 M ECU[1990]) much closer to that used in ExternE (2.6 M ECU[1990]) and most other externality studies.

However, other adjustments suggested in the NERA report would lead to a significant reduction in the estimate. The effect of age on VOSL is considered using published studies and it is concluded that multiples of 0.75 and 0.5 should be used for people over 70 and 80 years respectively. Most importantly, the effect of prior state of health is recognised as having the potential to reduce the value close to zero (at least for acute effects). Overall, therefore, the multiplier is less than 1 and ‘probably’ much less than 1. Combining the various multipliers leads to a range of 0 to 2 times the roads figure for acute effects (0 ECU to 2.2 M ECU), and a range of 1 to 2 times the roads figure for chronic effects (1.1 M ECU to 2.2 M ECU). Note that here, for chronic effects, we have not corrected for ‘health state’, as preceding morbidity may already be accounted for elsewhere in the analysis.
**Table A1.** Relative values of mortality risks from air pollution and from road accidents (from NERA, 1997).

<table>
<thead>
<tr>
<th>Effects</th>
<th>Context</th>
<th>Income</th>
<th>Age</th>
<th>Health state</th>
<th>Level of risk</th>
<th>Non-WTP costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Much higher WTP than for roads</td>
<td>Lower</td>
<td>Similar to roads except</td>
<td>Low WTP for those in substantial ill health, especially if so ill as to be close to death</td>
<td>Risk may be very high for some vulnerable groups, but their WTP may be very low because of ill health</td>
<td>Lower non-WTP costs (or higher non-WTP benefits) relative to roads. Figures will vary very widely with age and health state</td>
</tr>
<tr>
<td></td>
<td>WTP than roads but ethical</td>
<td></td>
<td>roads for the elderly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>considerations likely to over-</td>
<td></td>
<td>1 x roads value for</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ride this</td>
<td></td>
<td>people &lt;61</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.75 x roads value for</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>people &lt;71</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5 x roads value for</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>people &lt;81</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chronic</td>
<td>Similar to acute effects</td>
<td>Similar age of death to</td>
<td>Higher WTP because of preceding mortality</td>
<td>Low risk unless some highly vulnerable groups are identified</td>
<td>Similar to acute effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>acute effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>A adjustments for elderly as for acute effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2 x roads value</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>1 x roads value</td>
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</tbody>
</table>

Note: 1) The inclusion of higher WTP for chronic effects on account of morbidity preceding death will lead to double counting of damages if these morbidity effects are characterised separately.

The paper considers VOLS (value of life year) approaches to mortality valuation. It acknowledges the potential benefits of addressing lifespan reduction directly, but rejects the approach largely on the theoretical grounds that it is not strongly related to WTP. However, it might be noted that the use of a multiplier very much lower than 1 for people in poor health will have the same practical effect on damage values. The difference between the numerical recommendations of the study and advocacy of VOLS may therefore not be large. The range for the multiplier needs to be limited further if it is to be of any practical relevance, particularly in the context of this study. A multiplier of 1 could be used to justify the implementation of extensive additional abatement measures, whereas a value of 0 for acute effects on mortality might suggest that little more need be done if available information on chronic effects of air pollution on mortality were not considered sufficiently robust at the present time.

There are two issues where the paper raises interesting questions from the perspective of those involved in the practical cost benefit analysis of air pollution health effects: the meaning and relevance of VOLS for public policy decisions and the attribution of death to air pollution.

Relevance of VOLS

The VOLS measured by an individual’s own WTP is generally considered as the correct approach to measuring the value of small changes in mortality risk in welfare economics. There is a preference over VOLS on the basis that the latter appears to be far from constant as a function of age (as noted above). The explanation provided for this is not
that the utility of a life year varies a great deal as a function of age, but through the
inclusion of a ‘fear of dying’ in the VOSL.

The NERA paper considers this as a justification for preference of VOSL. It certainly
explains why WTP studies cannot readily be converted to VOLYs. But the implications
for public policy are not so straightforward. VOSL studies often claim to measure the
value of a risk in mortality reduction. Of course, this means the risk of mortality over a
given period (usually substantially less than the natural human lifespan), as the lifetime
risk of mortality is precisely unity for every individual. Public policy can affect risks to
reduce lifespan but cannot reasonably be expected to influence the ‘fear of dying’. To the
extent that VOSL is a measure of (a complex interaction of) the two, it values aspects of
dying which cannot be affected by public policy as well as those which can. Despite
VOSL’s validity as a utility measure, it may not therefore be the best indicator for policy,
or indeed the one the public would prefer to be used in public policy decisions.

Attribution of death
The NERA paper considers the problem of causality in air pollution mortality valuation as
part of an increased level of uncertainty (compared to road traffic accidents) in the overall
context. It is concluded that uncertainty per se will not affect valuation too much. But
causality issues may also have wider implications not addressed in the paper.

In acute (time series) studies, what is measured is the number of additional deaths in
episodes of high air pollution. With confounding variables adjusted for, it may be
concluded that air pollution is the cause of the precise timing of these deaths. However it
is not reasonable to infer that air pollution is the sole, or even the main, cause of death.
Certainly it will not be so recorded on death certificates. In this sense, air pollution deaths
are very different from traffic accidents, where the cause of death is also the cause of the
timing of death. For acute effects of exposure to air pollution, as was noted above, it is
generally believed that other factors are the principal cause of death.

Cohort studies of chronic mortality are somewhat different. The correlation of mortality
rate with long term pollution levels, with confounders allowed for, makes it reasonable to
infer that chronic pollution causes higher population mortality rates. In a constant
population, this rate is the inverse of life expectancy, so it is a correct conclusion that some
people (and probably quite a lot) have their lifespans reduced by air pollution. However,
air pollution will not usually be the cause of death in the normal sense, just one amongst
several stresses which cause death.

It is clear that extrapolation from the road traffic accident case to air pollution is
problematic. Traffic accidents are one of the few examples in which there is a single well
defined cause of death. In most cases is seems likely that death occurs earlier than it
otherwise would have done if any one of a number of environmental factors had been
optimised - temperature, diet and exercise regimes are obvious examples to add to air
pollution. It is clearly nonsensical to attribute the VOSL to each factor. Whilst each may
make death earlier than it otherwise would have been, they cannot all be the cause of
premature death in the normal sense.
Rational allocation of resources to risk reduction requires that the cause of death is attributed in some way between contributory stresses. For example, even if chronic air pollution makes the whole population of the UK die 3 months earlier than in the absence of air pollution (a result consistent with some current evidence), it does not imply that all mortality risk expenditure should be put into reducing air pollution.

The Value of a Life Year (Markandya, 1997 - see European Commission, 1998)

Within the ExternE Project it was first thought that the value of statistical life (VOSL) could reasonably be applied in the valuation of air pollution related mortality. A figure of 2.6 M ECU(1990) was obtained as an average of estimates of the VOSL based on wage risk, consumer market and contingent valuation studies (Markandya, 1995). Discussion within the project team soon raised concern over the application of this number, given the questions identified above regarding what it is that we are trying to value. It was concluded that an approach that took into account the length of life lost through ‘premature’ mortality was preferable. However, in spite of general interest in the concept of valuation of a life year, or considerations of quality of life, there were no data directly available. Recently Markandya (1997) has estimated the value of a life year based on a discounted stream of values covering remaining life expectancy. In view of the lack of specific valuation studies that have assessed valuation in terms of life years it was necessary to base calculations on published estimates of the VOSL (in this case a value of 3.1 M ECU(1995), updated from the earlier figure of 2.6 M ECU to account for inflation). It is acknowledged that this is not ideal, but it does offer a means of taking into account preference regarding the timing of death, until such time as there may be original research conducted to quantify the VOLY.

NERA (1997) criticise this approach on two grounds;
1. Available data suggest that the decline of VOSL with age is far slower than would be implied by the approach taken by Markandya. As such, valuation on the basis of ‘years of life lost’ (VOLY) appears to lack an empirical basis.
2. The conversion of a pre-determined VOSL into a time stream of individual values of life years (VOLYs) is essentially arbitrary, and unlikely to reflect the way that people think when quantifying their aversion to risk.

Regarding [1], it was noted in the previous section that some of the factors that contribute to the VOSL, related to the ‘fear of death’, cannot be influenced by policy. As such they may be irrelevant in this context. Once these factors are stripped out of the VOSL it is reasonable to suspect that the remainder would be more sensitive to assumptions about remaining longevity.

Regarding [2], it would clearly be better to estimate VOLY directly, but as no estimates are currently available (and that such work is beyond the remit of the JOULE Programme under which the ExternE Project is funded) it is necessary to base estimates on the VOSL, as Markandya has done, in order to get at least a first estimate. Readers should note that some experimental research in this area is likely to start in the USA next year (A. Krupnick, personal communication).
At a seminar held to discuss the interim findings of the NERA study, Newbery (1997) suggested that analysis be based on the VOLY concept. However, unlike Markandya he suggested deriving the value of a life year from health care expenditure. The advantage of this approach was that it would allow a common basis for assessment of expenditures on health improvement in different fields. An estimate of around 35,000 ECU per year of life saved was made. One problem with this approach is that it assumes that the action of politicians accurately reflects the preferences of the people that they represent. Another is that expenditure on health care is not based on a straightforward economic assessment of the benefits of that expenditure. A third problem is that health workers rarely characterise the rationale for their decisions in economic terms. When they do (in the UK at least) there is typically a great deal of public concern. For the purposes of this analysis the Newbery figure has therefore been rejected.

Position Proposed for the Present Study

It is clear that a number of different opinions regarding mortality valuation exist, even after a number have been eliminated (e.g. the gross output approach);

- Use of VOSL from the literature - either from an individual study or an average from a number of studies
- Use of an adjusted version of the VOSL, which seeks to better account for the nature of the risk faced from air pollution than the existing VOSL estimates
- Analysis based on the VOLY concept, deriving the value of a life year from estimated VOSL (at least until such time as direct valuations of life years are available).

Whilst apportioning the VOSL over time in VOLYs has been said to be ‘theoretically incorrect’ (from the perspective of the manner in which the VOSL is calculated), it does at least go some way towards ensuring that individual stresses are not accounted as the sole cause of premature death, when the reality is that each stress is only a minor contributor. On the other hand, if the VOSL concept is to be adhered to, it will be necessary to find some other method of apportionment over environmental factors to ensure that the aggregate risk of premature death over all factors remains strictly less than unity. On a purely practical level there is some consistency between the VOLY approach and the VOSL approach as recommended by NERA. The NERA range lies between 0 and 2 times the ‘roads’ VOSL, with a comment that the average may be much closer to zero for acute effects on mortality. However, no figure is specified. Depending on the discount rate adopted the VOLY approach essentially introduces a factor of around 0.1 per year of life lost, providing a value ‘much closer to zero’.

It was clear from the seminar held to discuss the NERA study that each approach had its advocates, and none is universally regarded as acceptable. The specific values represent a broad range:

- Literature VOSL: 2.6 M ECU/death
- Adjusted VOSL (NERA): 0 to 1.1 M ECU/death for acute effects on mortality
- 1.1 to 2.2 M ECU/death for chronic effects on mortality
- VOLY: 110 kECU/life year lost for acute effects on mortality
- 67 kECU/life year lost for chronic effects on mortality
Given the lack of consensus we have used the NERA figures and the VOLY approach in this study. The upper end of the NERA range is so close to the value of the VOSL derived from the literature that there is little additional benefit from using the ‘Literature VOSL’. These values have been adjusted as necessary in this study to reflect varying incomes in the UN ECE region. A single value is taken for all EU Member States, and PPP adjusted values for all other countries. Further details are provided in the main text of this report.

**VALUATION OF MORBIDITY (MARKANDYA, 1997; NAVRUD, 1997)**

The work carried out by Navrud identified problems in transferring valuation estimates for morbidity from the USA to Norway. The difference between the US and Norwegian estimates was explained in terms of improved CV survey and sample design, different preferences in Norway compared to the US, and different public health care systems. It was concluded that the results should provide better estimates for European morbidity than data transferred from the US. Although symptoms associated with air pollution were described, air pollution was not mentioned in the study, making it ‘non-contextual’. This was cited as one reason for the results being better transferable from one type of project to another, and possibly between countries. Further to this, the study compared results for different periods of suffering, comparing for example, WTP to avoid cough on 1 day only per year, and for 14 days. Results were higher for the second set of results, but only by a factor of about 3, rather than 14. Only ‘light symptoms’ were assessed (coughing, sinus congestion, throat congestion, eye irritation, headache, shortness of breath and acute bronchitis).

One of the problems in the application of Navrud’s work, as identified by Markandya (1997), is that the exposure-response functions available for analysis of air pollution health effects typically require a value of a typical ‘symptom day’ - and that the weight to be given to the different symptoms identified in unknown. However, comparing data between studies, Markandya concluded that Navrud’s results were within the range of those originally recommended under ExternE (Markandya, 1995). Markandya also identified new data from the US, providing values specific to chronic bronchitis in adults (105,000 ECU[1995] per case), and for acute bronchitis in children (225 ECU[1995] per case).

**VALUATION OF AIR POLLUTION EFFECTS ON CROPS (TONNEIJCK ET AL, 1997; NIAR/MORTENSEN/SSB, 1997)**

Only a brief summary of the work of Tonneijck et al was available to the study team. This work concentrated on effects of ozone, hydrogen fluoride, and the combination of SO₂ and NOₓ. Only direct effects on yield were considered; indirect effects mediated through frosts, pests and pathogens were ignored because of a lack of data. Valuation took account of changes in supply and price and welfare effects. This is more sophisticated than the analysis carried out under ExternE and a number of other studies, though these have tended to look at marginal change, where it is assumed that effects are
too small to affect supply and price to any significant degree. The most important pollutant was found to be ozone, and the least important, hydrogen fluoride.

The NIAR/Mortensen/SSB (1997) study provides an extensive overview of the effects of pollution on the agricultural sector in Norway. Compared to other work it is particularly noteworthy because of its consideration of effects on livestock production and of macroeconomic effects. Macroeconomic effects were equivalent to about 50% of the damage attributable directly to yield change. Unfortunately models and data are unavailable for an assessment of these indirect economic effects of changes on agricultural production for the EU. Results of the Norwegian study could be extrapolated to the EU situation, though their reliability would probably be very low, given the complexity introduced through the Common Agriculture Policy.

VALUATION OF AIR POLLUTION DAMAGE TO MATERIALS IN NORWAY
(GLOMSROD ET AL, 1996)

This is an extremely transparent study, providing details of databases, exposure-response functions, valuation and so on. In most ways the methodology is the same as that used in other work in this field (e.g. Cowell and Apsimon, 1994; European Commission, 1995). The coverage of materials goes further than in most studies, including wire fencing, transmission lines, infrastructure, etc. This is extremely important because the study found that damage to galvanised steel accounted for just under 50% of total materials damage (in accordance with the ExternE results for the UK). Like most other work on materials, no account was taken of damage to structures of cultural significance.

One particular area of innovation concerns quantification of the macroeconomic effects of damage to building materials. These were found to be very significant at 50% of the total increment in building maintenance cost. Extrapolation of this result to other countries is clearly problematic, but it would at least provide a first estimate of the macroeconomic consequences of materials damage.

There are questions as to whether brick and concrete should be considered in the analysis. They account for 12.3 and 11.3% of the stock at risk in Norway and 0.8 and 7.2% respectively of total damage costs. In total, use of concrete seems higher than in some other parts of Europe, and use of brick lower (comparison with Ecotec, 1994). The susceptibility of both materials to damage by the air pollutants of interest to this study has been much debated. Both are complex materials, porous, of variable composition, and of a strength partly determined by the quality of manufacture. Unfortunately the report by Iseke et al (1991) cited as the key reference on pollution damage to these materials is not included in the reference list.

Within the present study we have excluded brick on the grounds that there is very little evidence of damage to brickwork from air pollution in Europe, even in places where SO\textsubscript{2} levels were for many years grossly in excess of a concentration of 10 µg m\textsuperscript{-3}. Inspection of buildings in London shows that the assumption of a lifetime for brick of 65 years in an atmosphere where SO\textsubscript{2} > 10 µg m\textsuperscript{-3} is unreasonable. There are of course many different
types of brick, and some are likely to be more sensitive than others. However, the average brick would appear to be more resilient than Iseke’s prediction. From the Norwegian results it looks unlikely that damage to brickwork would represent more than 5% of the total damage to materials in any area of Europe, so the possible error involved here is small.

Exclusion of concrete is more contentious because it is known to deteriorate more quickly in urban situations than elsewhere. The over-riding mechanism for deterioration, particularly for re-inforced concrete, is carbonation - reaction of CO$_2$ from the atmosphere with calcium hydroxide in the concrete to form calcium carbonate. At current levels of pollution the carbonation profile proceeds well ahead of sulphation (the mechanism by which SO$_2$ causes damage). Given the comparatively greater success so far enjoyed in reducing sulphur emissions it is likely that the role of CO$_2$ will further increase in importance. We have thus excluded damage to concrete from this study.

**VALUATION OF AIR POLLUTION DAMAGE TO HISTORIC BUILDINGS (SOGUEL, 1996; MOURATO, 1997; AND OTHERS)**

Soguel conducted a contingent valuation survey in the Swiss town of Neuchatel. Sixteen buildings were identified as being of significant historical interest, being made of a sensitive material (a yellow limestone typical of buildings in the town) and being clearly exposed to road pollution. Eight interviewers questioned 200 people from the town, with the sample structured to reflect the demographic structure of the town. Of the 200, 114 were willing to contribute to protection of the buildings. Of the remaining 86, 22 were indifferent to the damage, 48 were regarded as ‘free riders’ and 16 could not afford to pay for protection of the buildings. On average the respondents thought that 6 of the 16 buildings were worthy of protection, presumably either because they were the most interesting buildings, or were clearly suffering more from pollution damage than the others. The average WTP per household (excluding the 11% of ‘indifferents’) was found to be 121 SFr (around 72 ECU at current exchange rates).

This is a substantial result, particularly considering that the buildings for protection were only those in Neuchatel, rather than all historic buildings in Switzerland or Europe. The extent to which this result might differ with a similar study quantifying WTP per household for protection of historic buildings across Europe is unclear. It is possible that it might not change much if people generally perceive their responsibilities to lie mainly with buildings in their home town or region.

Unfortunately the details given in the paper do not allow the result to be extrapolated more widely, because the pollution load is not described. It is also unclear exactly what sort of damage people were responding to - soiling of building surfaces or the loss of material. We will shortly contact Soguel to see whether further data are available that might allow us to use the results in the present study.

Mourato (1997) has recently reviewed economic valuation studies in the context of damage to cultural heritage. She concluded that the available literature was insufficient to
quantify the costs of damage to historic monuments and other cultural artefacts, particularly as they relate to air pollution. However, available data firmly suggest that total damages are substantial, and their omission provides a strong downward bias in the overall estimates made here of damage to materials. One study of particular note was the work by Mourato and Danchev (1997) on Bulgarian monasteries.

It has been suggested that the costs of pollution damage to cultural assets could be obtained directly from conservation bodies (in the UK this would include English Heritage, the National Trust, the Church of England, etc.). Unfortunately the records that do exist do not allow differentiation between the repair costs related to air pollution and the costs related to the inevitable and natural deterioration of buildings; in the interests of reducing costs maintenance programmes tend to address all problems affecting a given area of a building at the same time. Another problem arises because records of past pollution exposures in buildings that may be several hundred years old or more are not available.

**BENEFITS OF AN ACIDIFICATION STRATEGY FOR THE EUROPEAN UNION, HOLLAND AND KREWITT (1996)**

This paper by Holland and Krewitt is a synthesis of the state of the art of the methodology of the EC DGXI ExternE Project (in 1996, though noting that the ExternE project methodology has developed further since that time) so far as it applies to acidification issues in the EU.

The work was undertaken to look at the costs and benefits of closing the exceedence gap for critical loads for acidification by 45%, 50%, 55% and the maximum feasible reduction (MFR). Oxidised sulphur, oxidised nitrogen and reduced nitrogen emissions are considered and the impacts of primary and secondary pollutants included. ExternE regional scale atmospheric modelling is used except for ozone where European average results from Rabl and Eyre based on 1992 EMEP modelling are used, with rather crude apportionment between countries.

Abatement costs were taken from the work of Amann et al (1996) for EC DGXI, and therefore it is only damage results which are considered in any detail. The study by Holland and Krewitt is highly relevant to the present study, as it draws on the most thorough review of dose-response functions and impact valuations undertaken for air pollution in Europe. The methodology is the now well-established, ‘bottom-up’, impact pathway approach. Health, crops and building damages are included, but impacts on forests, fisheries and other ecosystems are not valued.

The dominant damages are human health mortality changes resulting from increases in PM$_{10}$ due to acid aerosols. Additional deaths are valued on a VOSL basis at 2.6 M ECU. The aggregate EU damages results are presented both with and without chronic mortality effects. Damages reductions are 16 GECU (59 GECU with chronic mortality) at 45% gap closure rising to 89 GECU (329 GECU) for the MFR. The cost-benefit comparison indicates that high levels of acid reduction have net benefits even if the more uncertain
chronic mortality effects are neglected (though note that the valuation of mortality was based on the VOSL, rather than the VOLY concept). The distribution of net benefits between EU countries was reported.

The results depend critically on the assumption that the health effects of nitrate aerosol are similar to those of other PM$_{10}$ species - a hypothesis which is not well-established. To our knowledge nitrate aerosol has not been specifically linked to health effects in epidemiological studies, though this could be simply a consequence of a lack of measurement data for this pollutant.

In summary, Holland and Krewitt provide a good starting point for much of the damage assessment needed. Nevertheless, its value to this project is limited for several reasons:

- emission reductions for the intermediate scenarios are confined to the EU rather than covering the whole UNECE region,
- it uses its own atmospheric modelling rather than EMEP transfer matrices, and so introduce some inconsistency with the cost-effectiveness analysis provided by Amann et al (1996, 1997).
- the policies examined are acidification reduction not nitrogen reduction,
- the ozone damages use European average estimates only,
- it does not include all the potential receptors, and
- the valuation of mortality does not reflect the latest thinking in this field.

Further assessment work is needed in these fields.

**ASSESSMENT OF OZONE DAMAGES, RABL AND EYRE (1997)**

This paper was written as a contribution to the current phase of the ExternE Project. It considers ozone effects on health (mortality and morbidity) and crops, but not materials, forests or other ecosystems. Air quality data were taken from two sources, the results of the EMEP Photochemical Oxidants Model (Simpson, 1992; 1993) which covered Europe, and the Harwell Global Ozone Model (Hough, 1989; 1991). The two models are very different. EMEP has a better geographical resolution, but just for Europe, and models only the boundary layer. The Harwell model concentrates on long distance transport and pollutant contributions from the free troposphere. The emission scenarios were also different, with EMEP following average European emissions of NO$_x$ and VOC, and the Harwell model looking at emissions of 1 Tg[N as NO$_x$]/yr at a latitude of 52°N. The lack of resolution with longitude clearly places a severe limitation on the accuracy of the global model. Specific account was not taken of very local effects of emissions on ozone levels. The extent to which these are reflected in the EMEP model is unknown.

The EMEP model was used for some simplified scenarios looking at the effects on ozone levels in Europe of variation in NO$_x$ and VOC emission (independently and together). From these data Rabl and Eyre calculated incremental ozone concentrations per unit of NO$_x$ and VOC release. The analysis provides an average estimate of effects per unit pollutant emission for the whole of Europe. Although fine for providing first estimates,
the usefulness of results is compromised, as the authors admit, and as is demonstrated elsewhere in the present report, by the potential for extreme non-linearities in ozone formation.

Exposure-response functions for health and crops damage were taken from the published reports of the ExternE Project (European Commission, 1995). Valuation of mortality was carried out in terms of years of life lost, calculated from a VOSL of 2.6 M ECU (1990). Assuming a discount rate of 3% this gave a figure of about 110,000 ECU per year of life lost. Each case of premature mortality was reckoned on average to involve the loss of 9 months of life.

It is clear that significant uncertainties affect the analysis carried out by Rabl and Eyre, but these are both well identified, and quantified in terms of the geometric standard deviation. This study will follow a similar approach, though a number of improvements can be made. The most important of these relate to the EM EP model, for which input data specific to the scenarios of concern will be used. Also, the analysis will be conducted at a suitable level of disaggregation such that results are country-specific, rather than European average. These changes will enable the study to deal with the non-linearities of ozone chemistry. Refinement of the global modelling will not be possible within this study. However, it is useful for suggesting that long range effects are important.

A further refinement that is necessary is for the study to use exposure-response functions derived from the European APHEA study, rather than US data.

**DAMAGE VALUATION ESTIMATES FOR NITROGEN BASED POLLUTANTS, EFTEC (1996)**

The EFTEC report ‘Research into Damage Valuation Estimates for Nitrogen based Pollutants, Heavy Metals and Persistent Organic Compounds’ represents an advance on earlier studies because of its attempts to estimate geographical dependence issues (by both source and receptor). These variations are important in Europe. This is well known for ozone (e.g. Simpson and Malik, 1996), but there is evidence in ExternE results that it is also significant for the nitrate aerosol components of small particulates.

The report emphasises the problems in using the EM EP ozone results to compute health effects. The following comments are relevant.

- EM EP model results relate only to a 6-month summer period and therefore the report points out that they may need to be corrected to be used with exposure response functions relating to the whole year. This is a valid point and needs further consideration in our work.

- European scale atmospheric model results reproduce rural ozone levels with reasonable accuracy, but not urban values, as the resolution is insufficiently fine to model local scale effects. This generally results in an over prediction of urban ozone concentrations, because ozone titration by NO, although included in the model, is not captured to the full extent at the surface in urban areas, where NO concentrations significantly exceed the grid square boundary layer average. EFTEC recommend
excluding the urban population from the ozone health impacts to account for this on the grounds that the urban population is ‘shielded from advected ozone’. This is a slight mis-representation of the problem. If implemented it would exclude impacts of VOC for which the comparable problem is not present. The urban ozone concentration is affected by regional scale ozone processes modelled, but it is also affected by local scale processes (incomplete mixing) which are not modelled. The EFT EC approach is therefore not ideal, but there is a real problem to be addressed in the treatment of low level surface emissions in urban areas in general (Eyre et al, 1997) and for ozone in particular.

- The report does not make clear whether the corrections necessary to adjust EMEP 6 hour average ozone metrics to the one hour metric more usually used in exposure-response functions have been made.

The report implies that the EMEP model is a linear approximation which is not correct. The marginal values taken from the model and used here are of course such an approximation, but the full model is not. The potential non-linearities, which may well be significant in VOC limited countries, are not fully considered by EFT EC.

Ozone marginal damages in European countries (shown in Table 7-2 of the EFT EC report) for UK emissions are taken from EMEP, but are not exactly the same as reported in Simpson and Malik (1996). It is assumed that they predate the published values. They are based on country to country matrices.

Aerosol modelling is reported in less detail, but appears to be based on EMEP country-grid matrices. It is not clear why a different modelling approach has been used from that for ozone. The long range of fine particulates would appear to make resolution less of a problem than for ozone, so it is not clear why a higher resolution is used.

Model results for aerosols report only the fraction of total nitrate aerosol in each country which is dependent on UK NO$_x$ emissions. It is not clear how concentrations of NH$_3$ emissions are allocated between NO$_x$ and NH$_3$ emissions. Linearity is assumed, although there is evidence from ExternE that the non-uniform distribution of NH$_3$ emissions makes this a problematic assumption. Results are reported for the marginal damages of UK emissions on the UK itself and on the whole UNECE region.

The presentation of results is not good. The summary of results (Table 10-4) reports total marginal damages from each European country, although neither the ozone nor aerosol damages are reported in this way. The values reported are highly counterintuitive with marginal damages (in ECU/t) from the Benelux countries more than an order of magnitude lower than those from Germany. The ordering indicates some correlation with size of country, implying some miscalculation.

Mortality is valued using the VOSL approach. Rather than differentiate VOSL by income in each country, an average value was taken, representative of the whole UNECE region. From a methodological perspective this accounts for the fact that the UNECE process deals with transboundary pollution. On a practical point one must also consider whether legislation that gave greatest weight to individuals in the richest countries would be
acceptable to poorer countries. This approach to benefits transfer seems appropriate to the present study.

The need to include macroeconomic effects of damage to buildings is raised in the report, but quantification of such effects was beyond the scope of the study.

Overall the EFTEC report raises a number of useful issues that need to be taken into account in the present study. However, as in the case of the other studies reviewed here, the EFTEC results cannot simply be extrapolated to the emission reductions under the various scenarios to be considered by UNECE.
Appendix III.

Review of Non-Quantifiable Effects

INTRODUCTION

Previous analyses of the external costs of air pollution emissions have concentrated on effects on health, materials and crops. However, it is well known that such analysis is far from comprehensive. A number of other types of damage have been quantified in the present, including timber loss from exposure to ozone, reduced visibility from particles and other air pollutants, and willingness to pay for the protection of natural ecosystems from air pollution damage. These areas are covered in the main text of this report, where it is noted that associated results are subject to significant uncertainty.

Even for those types of receptor that are included in the analysis, it is often the case that only a subgroup of impacts are assessed. For ease of reading Table 1, which dealt with the scope of the study in Section 1 of the main body of this report, was somewhat simplified. For example, ‘effects on crops’ in reality covers a range of effects, not just direct effects on crop yield. A detailed list for NO\textsubscript{x} emissions could include:

• fertilisation through addition of nitrogen to the system
• acidification, leading to a need for additional liming (though this is of little significance compared to the acidifying effect of harvest)
• stimulation of insect pests
• change in performance of pathogens
• direct inhibition of growth from exposure to O\textsubscript{3}
• foliar injury
• change in the interaction between plants and climate (e.g. drought, frost)
• indirect economic effects

It is clear that a complete analysis of crop damage (and other types of damage) requires more than the simple application of a single exposure-response function. Unfortunately a truly complete analysis for a given type of receptor (‘health’, ‘materials’, ‘crops’, ‘forests’, etc.) is seldom if ever possible because of a lack of data at some point in the analytical chain.

The following section reviews effects which cannot be quantified with confidence through a lack of data at the present time.
CARCINOGENIC IMPACTS OF VOCS

Some of the VOCs that would be controlled under the protocol are known to be carcinogenic (e.g. benzene). Analysis of the effects of these pollutants is possible and is being done in other studies, but would require a higher level of disaggregation of VOCs than is being attempted within the UNECE process, and a much finer level of spatial resolution than is possible within the remit of the present study.

MATERIALS DAMAGE

Effects on cultural buildings have not been assessed though the work of Soguel, Mourato and others certainly suggests that damage to these buildings may be significant (see Appendix II).

Effects of ozone on rubber, paint and textiles is also not quantified because of a lack of exposure-response data. DETR is, however, currently funding a study on these effects, though this is not due for completion until next year, after the completion of the present analysis.

Indirect economic effects of materials damage are also excluded from this analysis (though see the reference above in Appendix 2 to the study by Glomsrod et al, 1996).

CROPS

The introduction to this section listed a variety of pollution effects on crops. Of these the following are unquantifiable because of a lack of data;

- stimulation of insect pests
- change in performance of pathogens
- change in the interaction between plants and climate (e.g. drought, frost)
- effects of interaction between some pollutants
- indirect economic effects

It is possible that these effects are to some extent implicitly included in the exposure-response functions adopted. However, the artificial nature of experimental conditions makes it unlikely that interaction with these stresses is fully accounted for.

DIRECT EFFECTS OF AIR POLLUTION ON LIVESTOCK

There are few reports of direct effects of air pollution on livestock. Those that exist relate to extreme events (‘show’ cattle held in London at the time of the Great London Smog being a notable example). These events are not relevant to the current discussion.

Changes in the yield of pasture grass might be expected to feed through to a change in the rate at which livestock grow, or in the production of other products, such as milk. In another scenario farmers might be expected to respond to changes in animal production, so that meat and milk yields remain unchanged through the provision of additional
resources in the form of concentrates or extra pasture land. The NIAR study (see Appendix II) sought to assess this effect in Norway. One of the problems here would seem to concern the accuracy with which farmers can predict livestock yield. If pollution effects cause only small changes in grass production it would seem that farmers would need to be operating at, or very near to, optimal efficiency, before there would be any effect.

However, livestock production is extremely important, with production of cattle, sheep, goats and milk making up about 40% of total agricultural output in the European Union (EUROSTAT, 1990). Accordingly, effects on livestock productivity may well be too important to be ignored, so in spite of the uncertainties involved they have been brought into the analysis.

Effects on pigs and chickens need not be considered specifically as these species do not graze grass. To attempt to quantify impacts to these species would probably lead to double counting of damages quantified elsewhere in the study.

HEALTH

The epidemiology of air pollution effects at concentrations typical of those in Europe is all fairly recent. It is possible that some effects have not yet been detected. Coverage of chronic impacts appears particularly patchy. However, any assumptions regarding additional effects would obviously be little more than speculation. Note, however, that a number of reported effects have been excluded from the present analysis because of a lack of evidence in their favour (see Table 8 of the main text).

Valuation of altruistic effects of air pollution health impacts (i.e. damage relating to concern that someone else is unwell, or has died) is prone to much speculation and has thus been excluded from this study.

CLIMATE CHANGE EFFECTS

Some of the primary and secondary pollutants of interest here affect the radiative balance of the planet and hence affect the climate. However, these effects are extremely uncertain, and a satisfactory consideration of them is well beyond the scope of this study.

SECOND ORDER EFFECTS

Increased abatement of pollution can have a number of additional effects. These may individually be positive or negative, depending on the exact effect concerned. A well known example concerns the use of catalytic converters in cars - in addition to reducing tailpipe emissions of many pollutants (CO, benzene, NO, and others), ‘cats’ reduce vehicle efficiency, increasing CO₂ emissions. The use of ammonia in power station NO, scrubbers also involves second order costs as a result of emissions linked to ammonia manufacture. A full cost-benefit analysis would include these effects. However, this is beyond the scope
of the present study. In any case, analysis conducted in the present phase of the ExternE Project (unpublished at the time of writing) suggests that these effects typically cause small damages in comparison to the damages avoided.
Appendix IV

Sensitivity of Crops to Ozone Damage

This Appendix contains a summary of a draft paper written for the ExternE Project by Jones et al (ITE Merlewood) to be published next year (European Commission, 1998). The results identified in this paper have been used to attribute exposure-response functions to each of the crops considered in this paper.

It should be noted from this review that several of the authors of the papers referred to would prefer to exercise extreme caution in the application of their data in assessment of crop loss.

INTRODUCTION

It is possible to make some preliminary rankings of differential sensitivities of different types of vegetation to ozone. However, there are a number of factors that need to be taken into account in making these broad generalisations:

- Related species may show very different responses, and there may be large differences in sensitivity between different cultivars or clones of the same species
- Different stages in the life cycle may exhibit different sensitivities
- There are problems in transferring results from open top chambers to field conditions owing to effects on microclimate
- Interactions between ozone and other air pollutants may influence response
- Atmospheric humidity will play a vital role because of its effect on stomatal conductivity and therefore the diffusion of ozone into the plant
- Some plants can become rapidly adapted to high ambient ozone conditions
- The developmental stage at which exposure is commenced may have a large effect on the response
- Contradictory results from different experiments may be explained by different experimental conditions, such as hours of exposure, concentration levels, nutrient supply, and whether exposure is continuous or episodic

AGRICULTURAL CEREAL CROPS

Karenlampi and Skarby (1996) state that at Level I mapping the exceedence of the critical level of 3 ppm.h should not be converted into a yield loss estimate, but only used as an indication of the degree of risk. The AOT 40 for crops is calculated for a three month period during daylight hours, defined as those hours with a clear sky, global radiation of
50 W m\(^{-2}\) or more, and calculated as the highest running three month sum during the period when crops are grown.

**Wheat**
Analysis of data from open top chamber experiments demonstrated a linear relationship between relative yield of spring and winter wheat and AOT 40 (Fuhrer in Karenlampi and Skarby, 1996). This is the most complete data compilation to date and is based on results from 10 seasons, 6 countries and 10 cultivars. This work provides an AOT 40 value corresponding to 10% yield loss as 5.7 ppm.h and the following equation:

\[ y = 99.7 - 1.7 \times O_3 \text{ (ppm.h); } r^2 = 0.89 \]

Statistical analysis showed that the least significant deviation from a 100% yield which can be estimated with 99% confidence is 4-5% and the critical level for a 5% yield loss is approximately 2.8 ppm.h.

Numerous other papers are reviewed by Jones et al for wheat (Fuhrer and Ackermann, 1994; Heagle, 1989; Olszyk et al, 1988; Brewer et al, 1988; Brown et al, 1995; Soja and Soja, 1995). The final conclusion was to use the relationship developed by Karenlampi and Skarby (1996), acknowledging the concern that a Level II approach is essential for an accurate assessment of yield loss from exceedence of critical levels, depending on the availability of data on soil moisture deficit, phenological age, soil nutrient status and the presence of other air pollutants.

**Durum wheat**
Badiani et al (1996) investigated 2 cultivars of durum what, one of which was ozone sensitive, the other was not. According to criteria developed by UNECE a critical level of 5.4 ppm.h was calculated.

**Maize**
Data from Heagle (1989) indicate that maize appears to be considerably more ozone tolerant than wheat.

**Barley and oats**
Results from Olszyk et al (1988), Ashmore (1984) and Karenlampi and Skarby (1996) are contradictory, but in general barley appears to be relatively insensitive to ozone. Based on the review by Skarby et al (1993) oats also seem to be insensitive.

**Sorghum**
Heagle (1989) found only a small loss in yield, hence sorghum seems also to be fairly tolerant of ozone.
PASTURE GRASSES AND CLOVER

Ashenden et al (1996) showed a substantial reduction of yield of Lolium perenne of 37% in a 40 ppb ozone treatment with 2 peaks of 3 hours at 80 ppb followed by 1 hour at 100 ppb after 18 weeks exposure. The same treatment had no effect on Agrostis capillaris.

Heck et al (1986) reviewed the US NCLAN studies and found numerous effects on forage growth. Numerous authors have looked at effects of ozone on Trifolium repens, which appears to be the most ozone sensitive of the clovers (Becker et al, 1989; Heagle et al, 1996; Mortensen and Bastrup-Birk, 1996, Fuhrer, in Karenlampi and Skarby, 1996). Large differences in response between cultivars were reported but some appear to be at least as sensitive as wheat. Jones et al thus recommend setting a critical level at 4.7 ppm.h.

Effects on mixed pasture species are complicated because of competition between species. Based on studies by Ashmore and Ainsworth (1995) and Pleijel et al (1996), Jones et al concluded that ambient ozone levels are sufficient to affect the species composition of semi-natural grasslands, and cutting or grazing may influence community responses. Cumulative exposure above AOT 40 may be used to express critical levels and, although the effects are small they are sufficient to cause yield loss. Sensitivity is considerably less than for wheat.

VEGETABLE AND FRUIT CROPS

Potato
Data from Heagle (1989) and Heck et al (1986) indicate that sensitivity of potato is similar to that of wheat.

Beans
Results from Heagle (1989) and others suggest that soybean is slightly less sensitive than wheat. Similar results have been found for other species of bean (e.g. Phaseolus vulgaris and Vicia faba, Colls et al, 1993). It is thus recommended to use a similar critical level as for wheat.

Tomato
Tomato can be a very sensitive species, although there are wide differences in response between varieties. Heagle’s (1989) dose-response function shows a similar yield reduction to that of wheat, i.e. at 60 ppb loss of yield in wheat was 17% and in tomato was 18%. Available results are all from NCLAN. European data are lacking. Again, the critical level is assumed to be 5.7 ppm.h.

Grapes, fruit trees
Olszyk et al (1988) considered effects on grapes, lemons and oranges. Again sensitivity was similar to that of wheat. Work by Badiani et al (1996) suggested that peaches may be more sensitive when looking at different parameters such as fruit firmness which affect crop value significantly.
Other crops
Olszyk et al (1988) found no effect on lettuce, strawberries or sugar beet. Spinach was found to be slightly sensitive. Pepper, carrot and parsley were all found to be sensitive in work by Heck et al (1988).

SUMMARY

Effects of ozone on crops have been studied extensively, and exposure-response functions are available for a variety of species. The use of these functions at a European level is prone to uncertainty because of the numerous factors that will influence response.
Appendix V

Survey on Uncertainty

INTRODUCTION

Formal statistical methods can be used in benefits analysis, but fail to account for some important aspects of uncertainty. One proposal for integrating uncertainty to the analysis is based on ranking impact types by order of perceived uncertainty (note the use of the word perceived - the ranking is subjective, and the exercise is inevitably biased by the viewpoint of the person developing the questionnaire).

The purpose of this approach is to focus attention on the impact categories that are regarded as most certain, to some extent taking attention away from extremely uncertain impacts that may have little effect on the outcome of the cost-benefit analysis.

This is particularly important in the present study because we have sought to push the boundaries of quantification, trying to bring in effects that had previously been excluded from externalities analysis. This inevitably brings in some very speculative estimates of damage. We believe that it is better to quantify these effects than to ignore them altogether, but that they should not be allowed to dominate the analysis.

Once impacts have been ranked, calculated damages can be added sequentially, starting with those with perceived lowest uncertainty, until they exceed the abatement costs (should they reach this point at all). This can be repeated using alternative estimates based on additional uncertainty analysis.

Possible outcomes are as follows;

1. Only a few benefit categories are needed to exceed costs, excluding some that are perceived to be relatively well characterised. In this case the conclusion that benefits will exceed costs looks robust.

2. All relatively well characterised impacts (however identified) are needed for benefits to exceed costs. Confidence in the conclusions is reduced, but the conclusion that benefits exceed costs is still likely to look justified because several types of impact remain to be added in, counteracting (albeit to an extent unknown) possible over-estimates amongst the effects that have been included.

3. Many impact categories are needed, including some that are thought to be quite poorly characterised. Further loss of confidence in the robustness of the conclusion that benefits exceed costs.
4. The total of all quantified benefits added together is not sufficient to exceed costs. This outcome would suggest that the scenario under consideration is too extreme. However, consideration would still need to be given to the question of whether overall damages are underestimated (e.g. from the exclusion of some impact types such as damage to cultural assets, or from any downward biases present in the modelling framework). Consideration would also have to be given to the question of whether estimated costs were excessive, for example through exclusion of options such as fuel switching.

Of course, it is not enough to simply demonstrate that benefits will exceed costs to justify a given course of action. Ideally one should consider the ratio of benefit to cost for a variety of actions, selecting those where the ratio is highest to gain the most benefit from scarce resources. This is extremely difficult to do, particularly when dealing with issues of contrasting uncertainty. The methods that we are suggesting for uncertainty and sensitivity analysis (in this appendix and in the main text of the report) head in the right direction, but certainly leave scope for further improvement.

The full questionnaire on sensitivity was distributed to some UK analysts, and policy makers in several UK government departments. Full responses were obtained from the authors of the earlier report (Holland et al, 1998) and four environmental policy makers. Additional responses were obtained from policy makers in health and transport, who felt unable to complete the ranking exercise because of a lack of familiarity with some areas of the analysis. The full questionnaire is reproduced on the next page.
SENSITIVITY ANALYSIS QUESTIONNAIRE

Given that a full and reliable assessment of uncertainty is not possible at the present time it is necessary for individuals to use their experience to generate a ranking. Because this is judgmental it is useful for rankings to be made by people from different areas of expertise, and this has been done under the present study. Steering group members were asked to produce their own rankings for the impacts listed in the following table. The table is followed by guidance notes for its completion.

<table>
<thead>
<tr>
<th>Paint damage from acidic deposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crops - N fertilisation effects</td>
</tr>
<tr>
<td>Visibility</td>
</tr>
<tr>
<td>Macroeconomic effects of crop yield change on agriculture</td>
</tr>
<tr>
<td>Acute morbidity (excluding restricted activity days)</td>
</tr>
<tr>
<td>Restricted activity days</td>
</tr>
<tr>
<td>Crops - ozone damage (low estimate taken as 50% of total calculated to account for ozone x drought interaction)</td>
</tr>
<tr>
<td>Crops - ozone damage (high estimate - no account taken of possible interaction with water stress)</td>
</tr>
<tr>
<td>Damage to natural ecosystems</td>
</tr>
<tr>
<td>Acute mortality (low valuation based on value of life years)</td>
</tr>
<tr>
<td>Acute mortality (high valuation based on value of statistical life)</td>
</tr>
<tr>
<td>Crops - need for additional liming to counteract acidification</td>
</tr>
<tr>
<td>Materials damage (excluding paint)</td>
</tr>
<tr>
<td>Forest damage</td>
</tr>
<tr>
<td>Materials damage (macroeconomic effects)</td>
</tr>
<tr>
<td>Chronic effects on mortality (low estimate - VOLY approach)</td>
</tr>
<tr>
<td>Chronic effects on mortality (high estimate - VOSL approach)</td>
</tr>
<tr>
<td>Chronic morbidity (excluding bronchitis)</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
</tr>
</tbody>
</table>

1.1.1 Rules
1. In the left hand column write down the order in which you want to rank effects, 1 being given to the damage estimate that you have most confidence in, through to 19 for the estimate that you regard as being most uncertain.
2. ‘Low’ estimates for ozone effects on crops and acute mortality have to come in before ‘high’ estimates. Note, this does not necessarily imply that, for example, the VOLY approach is preferable to the VOSL approach for mortality valuation: if you prefer the VOSL approach you would probably agree that damages would have to be at least those generated using VOLYs.
3. It may be helpful to start by grouping effects according to the broad level of confidence that you have in them.
4. Pay no attention to the listings presented elsewhere in this report. We need an independent ranking from each contributor.
5. Don’t assume that anyone knows the ‘right’ answer - this is not a test of what people know, it is supposed to be an honest survey of perceptions about the uncertainty of different types of impact. Conventional wisdom can be very wide of the mark!

6. Tied rankings, even multiple tied rankings (within reason - a 19 way tie will be assumed to indicate that people did not try hard enough) are preferred where you cannot distinguish between impacts.

[end of questionnaire]
RESULTS

The results from all respondents who completed the questionnaire are summarised in Table A2 against an aggregated ranking. The Table is followed by a ranking of health impacts (not including valuation) by a health policy maker and comments by a transport policy maker. Based on the responses made effects have been put into 5 sets, I to V, of reducing confidence.

Table A2. Responses to sensitivity questionnaire. Note comment for respondent G in the key:

Column 1: Overall rank based on mean score
Columns 3 to 8: Ranking from individuals:
a - External cost analyst (trained in ecology)
b - External cost analyst (trained in physics)
c - Economic adviser to environmental decision makers
d - Environmental decision maker
e - Environmental decision maker
f - Environmental decision maker
g - Integrated Assessment Modeller (results received too late to be integrated with others)

Column 10: Mean score
Column 11: Grouping of effects

<table>
<thead>
<tr>
<th>Rank</th>
<th>Effect</th>
<th>a</th>
<th>b</th>
<th>c</th>
<th>d</th>
<th>e</th>
<th>f</th>
<th>g</th>
<th>Mean</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Materials damage (excluding paint)</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>10</td>
<td>1</td>
<td>5</td>
<td>4.0</td>
<td>I</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Crops - N fertilisation effects</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>14</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td>4.5</td>
<td>I</td>
</tr>
<tr>
<td>3</td>
<td>Acute mortality (low valuation based on value of life years)</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>2</td>
<td>5</td>
<td>5</td>
<td>4.7</td>
<td>I</td>
</tr>
<tr>
<td>4</td>
<td>Acute morbidity (excluding restricted activity days)</td>
<td>3</td>
<td>5</td>
<td>7</td>
<td>4</td>
<td>7</td>
<td>6</td>
<td>5</td>
<td>5.3</td>
<td>I</td>
</tr>
<tr>
<td>5</td>
<td>Crops - ozone damage (low estimate)</td>
<td>4</td>
<td>3</td>
<td>8</td>
<td>2</td>
<td>13</td>
<td>3</td>
<td>3</td>
<td>5.5</td>
<td>I</td>
</tr>
<tr>
<td>6</td>
<td>Crops - need for additional liming to counteract acidification</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>12</td>
<td>6</td>
<td>1</td>
<td>6.2</td>
<td>II</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Restricted activity days</td>
<td>11</td>
<td>8</td>
<td>12</td>
<td>4</td>
<td>1</td>
<td>12</td>
<td>5</td>
<td>8.0</td>
<td>II</td>
</tr>
<tr>
<td>8</td>
<td>Paint damage from acidic deposition</td>
<td>15</td>
<td>11</td>
<td>4</td>
<td>2</td>
<td>15</td>
<td>3</td>
<td>3</td>
<td>8.3</td>
<td>II</td>
</tr>
<tr>
<td>9</td>
<td>Crops - ozone damage (high estimate)</td>
<td>8</td>
<td>12</td>
<td>9</td>
<td>2</td>
<td>14</td>
<td>6</td>
<td>5</td>
<td>8.5</td>
<td>II</td>
</tr>
<tr>
<td>10</td>
<td>Chronic morbidity (excluding bronchitis)</td>
<td>7</td>
<td>7</td>
<td>14</td>
<td>4</td>
<td>5</td>
<td>17</td>
<td>11</td>
<td>9.0</td>
<td>II</td>
</tr>
<tr>
<td>11</td>
<td>Acute mortality (high valuation based on value of statistical life)</td>
<td>16</td>
<td>19</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td>11</td>
<td>9.7</td>
<td>III</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Macroeconomic effects of crop yield change on agriculture</td>
<td>10</td>
<td>9</td>
<td>11</td>
<td>14</td>
<td>9</td>
<td>12</td>
<td>11</td>
<td>10.8</td>
<td>III</td>
</tr>
<tr>
<td>13</td>
<td>Materials damage (macroeconomic effects)</td>
<td>9</td>
<td>10</td>
<td>10</td>
<td>14</td>
<td>11</td>
<td>12</td>
<td>?</td>
<td>11.0</td>
<td>III</td>
</tr>
<tr>
<td>14</td>
<td>Chronic bronchitis</td>
<td>12</td>
<td>15</td>
<td>16</td>
<td>4</td>
<td>4</td>
<td>17</td>
<td>11</td>
<td>11.3</td>
<td>III</td>
</tr>
<tr>
<td>15</td>
<td>Ozone damage to forests</td>
<td>17</td>
<td>13</td>
<td>17</td>
<td>11</td>
<td>6</td>
<td>10</td>
<td>5</td>
<td>12.3</td>
<td>IV</td>
</tr>
<tr>
<td>16</td>
<td>Chronic effects on mortality (low estimate - VOLY approach)</td>
<td>13</td>
<td>14</td>
<td>13</td>
<td>11</td>
<td>17</td>
<td>6</td>
<td>11</td>
<td>12.3</td>
<td>IV</td>
</tr>
<tr>
<td>17</td>
<td>Chronic effects on mortality (high estimate - VOSL approach)</td>
<td>19</td>
<td>18</td>
<td>15</td>
<td>11</td>
<td>18</td>
<td>12</td>
<td>11</td>
<td>15.5</td>
<td>V</td>
</tr>
<tr>
<td>18</td>
<td>Damage to natural ecosystems</td>
<td>14</td>
<td>17</td>
<td>19</td>
<td>19</td>
<td>16</td>
<td>16</td>
<td>11</td>
<td>16.8</td>
<td>V</td>
</tr>
<tr>
<td>19</td>
<td>Visibility</td>
<td>18</td>
<td>16</td>
<td>18</td>
<td>14</td>
<td>19</td>
<td>19</td>
<td>11</td>
<td>17.3</td>
<td>V</td>
</tr>
</tbody>
</table>
Response from health policy maker

Ranking of health impacts (excluding the valuation stage):
- acute effects on mortality
- acute effects on morbidity
- chronic effects on mortality
- chronic effects on morbidity

Comments from transport policy maker

The transport policy maker was unable to complete the exercise due to unfamiliarity with some effects. However, the following comments were made:

- the identification of non-human impacts, e.g. materials, involve more certainty than identifying health effects

- for estimates involving the valuation of life there is a need to take on board the findings of the NERA report. In terms of certainty, given the lack of theoretical validity for the VOLEY approach, this should be ranked lower in terms of certainty than the VOSL. On the VOSL figures they should include the acute effects using the range identified by NERA (attaching greater certainty than the values closer to zero, given that the report suggests that the average is ‘possibly much closer to zero’).

Authors comment: This raises a question about the practical implementation of the results of the NERA report, which were first raised during the NERA study. NERA were not explicit about how close to zero the average might be - the issue arising because of the likely health state of people whose deaths are linked to the acute effects of air pollution. Any multiplier from 0.0000001 to 0.25 and possibly higher could reasonably be said to be ‘much nearer to zero than 1; NERA themselves would only refine the range to 0 to 0.5, if asked (note that the present authors have sympathy with this view, given the lack of data in the area). For practical purposes the lower end of the NERA range is zero. There is likely to be no argument with the statement that the absolute lowest figure possible for acute air pollution effects on mortality is zero. In the context of this ranking exercise it would be given a ranking of 1 by default, but not influence the outcome of the assessment. Most people argue that the correct figure is higher than zero, though by how much? By stipulating that the VOLEY result be included above the VOSL (see the rules for the ranking), we place a greater confidence in a figure that is much closer to zero. We thus regard our approach as a pragmatic response to the difficulties faced.

- Visibility should be placed last on the list.

The ranking used in the sensitivity conducted in this report is based largely on the views of two of the authors of the Holland et al (1998) report (respondents a and b), rather than the full set of data (Table A3). In cases where average rankings were tied, the order reflects the likely magnitude of damages on the basis that uncertainties in small numbers will have a smaller effect on the overall cost benefit analysis than uncertainties in larger numbers. Overall, there is little real difference in the rankings - the groupings in the final column remain almost intact. The most major difference relates to the position of the high valuation of acute effects on mortality, based on VOSL - reflecting different opinions on the validity of the VOSL approach. To take account of the difference in views this effect
has been moved up, above visibility effects, damage to forests and damage to natural ecosystems.

**Table A3.** Ranking based on respondents i and ii only, and final ranking.

<table>
<thead>
<tr>
<th>a, b</th>
<th>‘Total’ Diffference</th>
<th>Effect</th>
<th>Final ranking</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>1 Crops - N fertilisation effects</td>
<td>1</td>
<td>I</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>5 Crops - need for additional liming to counteract acidification</td>
<td>2</td>
<td>II</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>2 Crops - ozone damage (low estimate)</td>
<td>3</td>
<td>I</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>0 Acute morbidity (excluding restricted activity days)</td>
<td>4</td>
<td>I</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>-4 Materials damage (excluding paint)</td>
<td>5</td>
<td>I</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>-2 Acute mortality (low valuation based on value of life years)</td>
<td>6</td>
<td>I</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>3 Chronic morbidity (excluding bronchitis)</td>
<td>7</td>
<td>II</td>
</tr>
<tr>
<td>8</td>
<td>12</td>
<td>4 Macroeconomic effects of crop yield change on agriculture</td>
<td>8</td>
<td>III</td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>1 Restricted activity days</td>
<td>9</td>
<td>III</td>
</tr>
<tr>
<td>11</td>
<td>9</td>
<td>-2 Crops - ozone damage (high estimate)</td>
<td>10</td>
<td>II</td>
</tr>
<tr>
<td>12</td>
<td>8</td>
<td>-4 Paint damage from acidic deposition</td>
<td>11</td>
<td>II</td>
</tr>
<tr>
<td>13</td>
<td>14</td>
<td>1 Chronic effects on mortality (low estimate - V O L Y approach)</td>
<td>12</td>
<td>II</td>
</tr>
<tr>
<td>13</td>
<td>16</td>
<td>3 Chronic bronchitis</td>
<td>13</td>
<td>IV</td>
</tr>
<tr>
<td>15</td>
<td>15</td>
<td>0 O zone damage to forests</td>
<td>14</td>
<td>III</td>
</tr>
<tr>
<td>16</td>
<td>18</td>
<td>2 Damage to natural ecosystems</td>
<td>15</td>
<td>IV</td>
</tr>
<tr>
<td>17</td>
<td>19</td>
<td>2 Visibility</td>
<td>16</td>
<td>V</td>
</tr>
<tr>
<td>18</td>
<td>11</td>
<td>-7 Acute mortality (high valuation based on value of statistical life)</td>
<td>17</td>
<td>V</td>
</tr>
<tr>
<td>19</td>
<td>17</td>
<td>-2 Chronic effects on mortality (high estimate - V O S L approach)</td>
<td>18</td>
<td>V</td>
</tr>
</tbody>
</table>

**SUMMARY**

The ranking derived in this appendix seeks to take a pragmatic approach to the problem of uncertainty in the assessment of benefits of reducing pollution. Use of the ranking does not magically ‘solve’ the problem, but it should help by making more explicit which parts of the analysis people have most confidence in, and which parts people have least confidence in. Applied to the sensitivity analysis it provides some guidance on how robust the conclusions of the cost-benefit analysis are likely to be. Importantly there is much consistency in the responses obtained.
Appendix VI

Illustrative Calculation

The analysis follows a similar pattern for almost all of the impacts assessed in this study, as illustrated in the following figure taken from the main text of this report:

- **Pollution load** → Concentration, deposition
- **Stock at risk** → Distribution of people, etc.
- **Sensitivity of stock at risk** → Baseline mortality, etc.
- **Exposure-response functions** → Rate of damage/unit pollutant
- **Impact assessment** → Change in crop yield, mortality, etc.
- **Valuation** → Willingness to pay
The ALPHA model developed by AEA Technology contains all of the databases of stock at risk, exposure-response functions and valuation data required for the analysis (as described in the main text of this report).

The first stage of the analysis is to quantify the effect of pollutant emissions in each country on pollutant concentration and deposition in each cell of the EM EP grid. The ALPHA model does this by extrapolation from EM EP results (kindly supplied by the Norwegian Meteorological Institute, DNMI) for all pollutants except ozone. The complexity of ozone chemistry means that extrapolation of ozone data would lead to serious error, and hence ozone data need to be modelled explicitly by EM EP for each scenario. The air quality and deposition module of the ALPHA model is derived from EM EP transfer matrices based on real emissions data and real meteorology for a series of six years from 1989 to 1994. These provide estimates of the contribution to concentration and deposition of different pollutant species (e.g. S deposition, SO$_2$ concentrations, sulphate concentrations) in each grid cell as a consequence of known emissions from each country in the following format:

<table>
<thead>
<tr>
<th>Coordinates</th>
<th>Countries</th>
</tr>
</thead>
<tbody>
<tr>
<td>X Y</td>
<td>Albania</td>
</tr>
<tr>
<td>1 1</td>
<td>Conc$_{1,1}$ - ALB</td>
</tr>
<tr>
<td>1 2</td>
<td>Conc$_{1,2}$ - ALB</td>
</tr>
<tr>
<td>1 3</td>
<td>Conc$_{1,3}$ - ALB</td>
</tr>
<tr>
<td>1 4</td>
<td>Conc$_{1,4}$ - ALB</td>
</tr>
<tr>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>n n</td>
<td>Conc$_{n,n}$ - ALB</td>
</tr>
</tbody>
</table>

A table of transfer factors was generated by dividing the data in each cell by annual emission from the country concerned. This was repeated for each of the nine pollutant species considered (deposition of sulphur and reduced and oxidised nitrogen, concentrations of NO, SO$_2$, NH$_3$, NO$_3^-$, SO$_4^{2-}$ and NH$_4^+$). For each species factors were then averaged across the six years to account for variation in meteorology. To calculate concentrations and depositions in each scenario the transfer factors are simply multiplied by national emissions.

Ozone data are taken directly from the results of the EM EP model, applied specifically to each scenario.

Following this the calculations are straightforward, multiplying together a series of variables. Take the quantification of effects of SO$_2$ emissions on some morbidity impact such as coughing in people over 65 years old, via the formation of sulphate aerosol. The first stage is to apply the table of transfer matrices showing the effects of SO$_2$ emissions on sulphate concentrations, as just described. The rest of the analysis is illustrated using dummy data.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration of sulphate in cell 1,1</td>
<td>2 µg/m²</td>
</tr>
<tr>
<td>Number of people living in cell 1,1</td>
<td>2,000,000</td>
</tr>
<tr>
<td>Fraction of population aged over 65</td>
<td>0.14</td>
</tr>
<tr>
<td>Exposure-response function</td>
<td>10 cases/year/1000 population</td>
</tr>
<tr>
<td>Valuation data</td>
<td>12 ECU per episode of coughing</td>
</tr>
</tbody>
</table>

Multiplying these variables gives 5,600 episodes of in cell 1,1 in a year, valued at 67,200 ECU.

Results are then summed to give national totals. To enable this each grid cell has been allocated to one or more countries depending on the proportion of area of the cell in each country. If relevant, additional factors may then be introduced. An example might be national incidence of disease, or factors to adjust willingness to pay for variation in national income.

Results are summed across impacts to give the total damage for the scenario under investigation. The final stage is subtract damages under the scenario in question from those of the REF scenario, giving the incremental annual benefits of the scenario under investigation.
Appendix VII

Comments Received on the Interim Report, and Responses

This appendix reproduces written comments received following issue of our interim report for this study. The original text of the authors is presented in italics, the authors’ response is given in normal type.

Commentator: Suzie Baverstock, CONCAWE, 1/7/98

Questions/Points of Concern on the Ozone Benefits Evaluation

Q1. Has there been any progress in resolving/agreeing which benefits should be evaluated, i.e. the AEA list of benefits versus those contained in the position paper?
R. There are differences between the list of benefits in the position paper and the list of benefits in our report, reflecting the different purposes and approaches of the two documents. The position paper identifies the group of effects that most strongly suggest that ozone causes damage to a variety of receptors, from people to materials. It was not intended to define the remit for the benefits assessment (there would be a problem if there was clear disagreement between the two papers, but this is not the case). In our study we take a more holistic approach, whilst placing much emphasis on exploration of uncertainties, in seeking to build an overall image of the benefits attributable to ozone reduction. This is consistent with the precautionary principle. The uncertainty and sensitivity analysis conducted in this study guard against the precautionary principle driving the conclusions that may be drawn from being overly conservative.

Q2. How can mortality (including ozone mortality) be ranked as the third most certain monetary value when WHO have advised that there is no strong evidence of an ozone induced mortality association. (As a minimum PM and O₃ mortality values should have different places in the ranking).
R. As noted, ozone related mortality is only one component of the ‘acute mortality’ impact class. The latest version (8.1) of the position paper from the ozone working group is somewhat ambivalent on ozone-related mortality. Mortality is not mentioned until a study by experts in the UK is summarised, quantifying impacts with and without thresholds. There is a difference of course in saying that ‘there is no evidence of an effect’ and that ‘there is no strong evidence of an effect’. In view of the uncertainty in this area our report assesses the inclusion/exclusion of the ozone mortality function.

The ranking was derived in an earlier study funded by the UK Department of the Environment, Transport and the Regions to inform the UN ECE Task Force on Economic
Aspects of Abatement Strategies (TFEAAS). It was collated from responses made by analysts, academics and civil servants in the UK. At meetings convened since then (by the UN ECE, European Commission and UNICE) the authors of this report have repeatedly requested that others submit their own versions of the ranking, following the rules specified. So far no further returns have been submitted. To ensure traceability a revised ranking will not be made until we receive further (complete) submissions. Note that for the present study this is now too late.

It should also be remembered that acute effects on mortality are ranked third only for assessment based on the VOLY approach. As noted in Section 5 of the report results show that this valuation of acute effects on mortality makes little difference to the overall cost:benefit ratio. With valuation based on VOSL these effects do influence the ratio noticeably, but the VOSL-based valuation is already ranked lower.

Q3. Related to [2] - we submitted a written comment on O₃ and mortality and attached a paper from HEA. Was this passed to AEA by Martin Lutz?
R. See the letter from D. Cloquet of UNICE reproduced below, and the response that follows it.

Q4. Have separate cumulative curves been produced which do not double count VOSL and VOLY valuations of the same chronic/acute mortality cases?
R. The curves presented in the interim report did not double count VOSL and VOLY valuations, though this was not clear from the figure captions. It was subsequently agreed that it was logical to separate them more clearly, and this has now been done.

Q5. Direct and indirect benefits should be clearly differentiated in summary materials and diagrams so we can see what benefits arise from addressing O₃ and what results from reducing NOₓ.
R. Ozone and non-ozone effects are now reported separately in some of the summary material presented in the report. The terms ‘direct and indirect effects’ should be avoided as they could cause confusion with, for example, indirect economic effects of changes in agricultural production.

Q6. Crop yield evaluations are unreliable if they do not consider the other factors affecting crop yield and the impact of ozone (Level II effects). Factors should include increased sunlight, drought, disease and be seen in the context of CAP payments to reduce yield.
R. Our reports have always recognised these issues. However, at the present time it is not possible to integrate the effects mentioned. The potential for these factors to influence the results was discussed in the interim report, and here, in Sections 3 and 4, with particular consideration given to drought. It was shown that substantial areas of southern Europe are irrigated to counteract drought problems. Irrigation is likely to concentrate on the most sensitive crops, and those of highest economic value. These two factors should go some way to mitigating error in the results from a failure to account for water stress in detail. The direction of interaction with ozone of some of the other stresses not explicitly accounted for (e.g. pest damage) could also run counter to the direction of the effect of water stress.
The alternative to the approach used here would be not to quantify crop yield effects at all in this study. Implicitly this could be taken to give a zero value to ozone effects on agriculture. We are not aware of anyone who would say that this is true. It is certainly possible to dispute the estimates of damage made in this study, as our repeated reference to associated uncertainties shows. However, without quantification it would not even be possible to say whether available information on exposure-response suggest that effects are trivial or significant.

Any account taken at the present time of the effect of the CAP on the value of yield changes is subject to arbitrary assumptions on the form of the CAP in 2010: for transparency we have not attempted to speculate on the future of the CAP. Note of course that throughout this study changes in agricultural production are valued at World Market prices rather than the higher CAP prices.

Commentator: Daniel Cloquet, UNICE, 30/4/98

Re: Health Effects Institute Study concerning Ozone

As before, the original comments are given in italics, responses in normal text.

I am writing to follow up an intervention made by Mike Hawkins at the recent AQFD Steering Group Meeting. Please find attached the title page and an abstract from a study conducted by the Health Effects Institute (HEI) concerning ozone mortality and daily exposure to which he referred [Reference: HEI, 1996 Research Report Number 75: Ozone exposure and daily mortality in Mexico City: A time-series analysis. Loomis, D.P., Borja-Aburto, V.H., Bangdiwala, S.I. and Shy, C.M.]. The study lends support to the view taken by WHO which was included in the Position Paper, i.e. that there is no strong evidence that ozone is causally related with mortality.

So far DGXI has relied heavily on the advice of WHO in choosing air quality targets for ozone, yet its advice seems to be downplayed when it comes to the consideration of ozone and mortality. In particular, UNICE notes that the;

• AEA Technology ozone cost/benefit study presents benefit figures for ozone-related mortality, and
• Draft Position Paper chapter on Risk Assessment makes reference to the UK Committee on the Medical Effects of Air Pollutants (COMEAP) report, which includes estimates of mortality based on statistical associations with ozone concentrations.

Although neither study claims that ozone causes mortality, they include figures as if this were the case, and use them in assessing potential benefits.

UNICE is very concerned that these two studies (and their promulgation and apparent endorsement by DGXI) lend undue credence to the hypothesis that exposure to ozone at current ambient levels is causally related to mortality - a matter which remains highly speculative. UNICE urges DGXI and its consultants to ensure that it is made absolutely
clear in all relevant communications that there is no strong support to suggest that ozone is causally related with mortality. DGXI is also requested to instruct AEA Technology to exclude the corresponding benefit figures from their cost/benefit analysis.

**Response to the letter from Daniel Cloquet**

As noted elsewhere in our report, the ozone-mortality association is subject to uncertainty. This is clearly reported and its effect within the cost-benefit assessment is analysed. We do not regard the existence of uncertainty as reason to ignore reported effects. In fact, it would appear equally valid to turn the WHO statement from the position paper (‘that there is no strong evidence that ozone is causally related with mortality’) around to say: ‘The limited number and inconsistent outcomes of studies on all-cause mortality do not provide strong evidence that ozone is not causally related with mortality.’

HEI funded the Loomis et al study in Mexico City because the location offered a number of advantages over many locations in the USA and Europe. Firstly, ambient ozone levels in Mexico City are high. Secondly it was felt that these levels were a more reliable metric of personal exposure than was available in the USA because indoor heating and air conditioning are rare in Mexico. Thirdly it was felt that weather related variables would be less likely to affect the results.

There are of course also reasons for supposing that Mexico City may not provide a good model for the USA and Europe. A review of relevant demographic data (Philip’s Geographical Digest, 1995-96) shows for example that the population of Mexico is generally younger than those of the EU Member States. In all countries except Germany, the % of the population aged over 60 years is between a factor 2 and 4 higher than in Mexico. Even then, data for Germany show a much higher % in the 25 to 59 age bracket than in Mexico (65% against 35%). This is reflected in the lower death rate for Mexico overall (roughly a factor 2 lower per unit population than in the EU). Data on GDP per capita show that Mexican incomes are lower than those throughout the EU, on average being roughly half those in EU Member States. This affects access to health services.

There are also major contrasts in living conditions between what might be considered typical of the EU. Much of the housing in Mexico City is temporary, many areas are lacking in basic amenities.

In view of the major differences between the study population and the population of the EU we conclude that whilst there were indeed good reasons for studying effects of ozone in Mexico City, extrapolation of the results of the HEI study elsewhere may well be prone to the same level of error as extrapolation of the results of any single epidemiological study.

Considering total suspended particulates (TSP), SO\textsubscript{2} and ozone individually, the HEI study found statistically significant associations with mortality, particularly for individuals aged over 65 years. However, when all three pollutants were brought together in the model only the association with TSP remained significant. Loomis et al concluded that;

“Although the investigators found an increase in mortality associated with periods during which ambient levels of ozone were elevated, this increase cannot necessarily be attributed
to ozone because it was not statistically significant when other air pollutants were included in the analysis. Overall the results of this study conducted in Mexico City appear largely consistent with what has been reported in other locations world wide, namely that short term increases in indices of particulate air pollution are associated with increased daily mortality. However, further research is needed to disentangle the effects of the various pollutants and to gain insights into the association of individual pollutants with morbidity and mortality.”

The paper therefore does not state that ozone is unrelated to mortality - the issue is identified as an uncertainty.

Consider also work such as the recent study on the relationship between ozone and mortality in the hot summer of 1994 in Belgium by Sartor et al (1997). In that study a significant relationship with ozone was found after account was made of the effects of SO₂, NO₂ and fine particles.

Overall, our study follows the rationale of COM EAP (1997), who reported after a review of the available data including the work of Loomis et al that; “Taking all the existing evidence into account, it is concluded that an association exists [between ozone and premature mortality] and is reasonably consistent across Europe and with large cities in the US. This justifies an estimate of risk...”

Inclusion of a quantified estimate of the effect of short-term ozone exposure on mortality is consistent with the broader rationale of this study; to quantify impacts as fully as possible whilst clearly identifying the uncertainties. It is then the responsibility of policy makers to act as they see fit, reflecting their own position with respect to acceptance of risk on both sides of the cost-benefit equation.

Commentator: Peter Rombout, RIVM, May 1998


As before, the original comments are given in italics, responses in normal text. The comments made below largely reflect the time pressure between receipt of data on emissions and ozone concentrations, and the deadline for production of the interim report dictated by a meeting of the steering group (a matter of only 6 days). In this time it was not possible to undertake the level of sensitivity analysis regarded as appropriate by the study team. However, production of the report at that time enabled members of the steering group the opportunity to inspect the methods, functions, valuations, etc. and provide appropriate feedback which has been useful in the second half of the study.

Comment: The main comment concerning benefit assessment is not the principle or the methodology of benefit assessment as such, but concerns the fact that the currently available input data lead to benefit assessments in the interim report which may be grossly exaggerated. A factor of 100 or more would not surprise me. In my opinion the
methodology needs other input data than those that were used. Such data are currently not available and cannot be easily generated, because either the basic science is lacking or the development and testing of necessary models and concepts is not adequate yet.

Response: The factor 100 error mentioned appears to be based largely on concern over the valuation of mortality following the value of statistical life (VOSL) approach, and the general validity of mortality assessments. The interim report assessed both the VOSL approach and the value of life years (VOLY) approach to mortality valuation. The authors' preference is for the VOLY approach, though we accept that the current approach to estimating the value of a life year is not ideal.

On reflection the earlier presentation of results did not draw sufficient distinction between this mutually exclusive choice. Combined with assumptions on lost life years this makes a factor 40 difference in the estimated benefits from reduced acute effects of exposure on mortality: uncertainty in mortality assessment of this order was thus clearly reported in the interim report. With respect to chronic effects of exposure on mortality the difference is less largely because of the conservative assumptions that are applied to the VOSL based assessment.

It should be noted that the authors' preference for the VOLY approach is not shared by many environmental economists. The UNECE Task Force on Economic Aspects of Abatement Strategies recommends the use of the VOSL, and so opt for the higher estimate of benefits. In the face of diverse opinion here, as elsewhere, we have represented the positions regarded as valid by different respected experts in Europe, and investigated the effects of these positions on the outcome of the CBA.

In terms of scientific purity it would indeed be better to wait until such time as more robust input data were available in some key areas. However, results are needed now to inform those responsible for the development of policy on ozone. Our opinion is that it is appropriate to use available data provided that account is taken of the magnitude of potential errors. Sufficient time has been available for production of the final report for us to do this more clearly than in the interim report (noting that the interim report itself contained substantial discussion of the errors in the analysis).

Comment: The implicitly underlying question of benefit assessment is, if a government or political entity should do something about an environmental problem even if the economic benefits are smaller than the cost of abatement? Should the intrinsic need for a government to do something about environmental problems be, that it wants to shield its population from the harmful effects of pollution?

If so, of course there still is a need to evaluate the amount of resources that should be spent on control measures, to evaluate the levels of pollution reached after such measures have been taken and the then resulting health risk for the population. But it does not necessarily imply that pollution abatement measures should only be taken as long as the economic benefits are (or seem) bigger than the cost. Try to imagine the reverse position when, with higher costs than benefits, abatement measures might not be taken.

Response: The question of the meaning of the results of the CBA for policy is outside the remit of this study as the authors are scientists, not the politicians charged with deciding what level of action against air pollution is appropriate! We have simply brought together
a body of data that we regard as being a reasonable representation of the information available, or where different opinion exists that others regard as so, and then conducted error and sensitivity analysis around the results generated from those data.

Comment: Two important constraints in these comments on the interim report have to be stated. First is that the comments are made from the perspective of the Netherlands, which might implicate that a number of the facts presented in the interim report on which critical comment is given, might still be right for another European country. The second constraint is that the comment is from the perspective of human health only, which implies that NO$_x$ aerosols and ozone are the important features that have been taken into account. These comments concern the items presented with the numbers 3, 4, 5, 8, 11, 13 and 14 (from 16) in Figures 1 and 2 of the interim report.

For the sake of clarity the chain used in the interim report (pollution load [a], stock at risk [b], sensitivity of stock at risk [c], exposure-response functions [d], impact assessment [e] and valuation [f]) will be followed. On each of the stages, as far as necessary, comments will be presented.

Comment on [a]. Pollution load was not included in the report as such. RAINS and other models were used to estimate this factor. It is a pity these basic calculations have not been presented in the interim report because the reference scenario already indicates the NO$_x$ emissions in the Netherlands to be approximately half of what they currently are. It is not clear whether the expected benefits might even be bigger as the reference scenario might be slightly optimistic in its pollution loads.

Response: Emissions data were given in Tables 2 and 3 of the interim report. Data on concentrations and deposition were left out of the report because of constraints on space and time. Since production of the interim report IIASA’s REFERENCE scenario has been revised, increasing annual emissions from the 270 kt per year given in our interim report to 292 kt per year.

Comment: A more important point is the fact that the models estimate concentrations in a 150 x 150 km$^2$ area. Because of this grid size the interim report only estimates the ozone concentrations for the rural areas of the Netherlands and not for the urban areas. The Netherlands is a highly urbanised country in which a considerable amount of the population lives in cities. For ozone in summer in the Netherlands concentrations in cities are a certain fraction of those in non-urban areas, which implicates that population health risks within a country of 200 x 200 km$^2$ area may vary considerably, especially if also the fact is taken into account that ozone health risks do not seem to be linear in the lower concentration ranges. Probably for reasons of simplicity the interim report overlooks such a complicating factor.

Response: The interim report like the final report did not overlook this issue (see Section 3.3.3 of both reports), though it was only possible to deal with it qualitatively. Given that analysis has not been conducted for ozone levels at the localised scale that would be necessary for more accurate assessment a more refined analysis based on modelled outputs is not possible. Within the UNECE forum we raised the question of whether an arbitrary correction (reduction of urban ozone x health damages by 50% and 100%) should be carried out for urban areas given the problem of the NO$_x$ sink. It was decided there that the issue was best raised in the text and that no (arbitrary) correction be carried out. No further suggestions on dealing with this problem have been received.
Comment on [b and c]. There are no comments on the stock at risk (distribution of people) or the sensitivity of the stock at risk (baseline mortality) which would lead to a very different picture from the one that was presented.

Comment on [d]. There are though, serious comments on the exposure-response functions (rate of damage / unit of pollutant) as presented in Table 6 of the interim report. The main concern is the omission of a threshold for the ozone related health effects in the interim report. The figure of 0.059% per µg/m$^3$ ozone for acute mortality in the interim report ties in nicely with the 3% per 50 µg/m$^3$ of ozone presented in Table 6.5 of a recent UK report: “Quantification of the effects of air pollution on health in the United Kingdom, 1997”. In this last report a clear indication is presented for the existence of a threshold between peak 8 hour ozone concentrations and hospital admissions (Figure 6.1). The authors of this last report indicate that a threshold of 80-120 µg/m$^3$ ozone only seemed to be valid in a city and not in rural areas, and on the basis of theoretical arguments they assumed no threshold to exist for ozone. In the last report (“Quantification...etc.) a calculation has been made of the ozone related deaths in the summer in the UK. Fortunately they included estimates with and without a threshold for the numbers of deaths during summer in the UK (Table 8.2). With a threshold 700 deaths were calculated related to ozone and without a threshold this was 12,500, which is a difference of a factor of nearly 20. In my opinion this indicates that inclusion or omission of a threshold is a very important decision which dictates the outcome from the exercises.

Hoek et al., (1997) have recently presented an analysis of daily mortality and air pollution in the Netherlands (1986-1994) which showed that the mortality of ozone was non-existent in the winter and significant in the summer. In my opinion this result also suggests some kind of threshold for ozone associated mortality, certainly if also the fact is taken into account that the ozone levels indoors are a fraction of the ambient levels outdoors. Therefore a linear estimation with a zero threshold for the ozone related mortality as has been done in the interim report does not seem justified. In my opinion the ozone results of the interim report are not valid and should be recalculated with a non-zero threshold (in the case of linear modelling) or some kind of a sigmoid curve which encompasses the epidemiological concentration response relationships in the low dose range. The least that could be done is to present two estimates, just as has been done in the UK report on quantification. A second remark concerning ozone is that the results in the interim report do not indicate if the right basis for the calculations has been used, as it is necessary to use the peak 8 hour levels during a day to calculate the health effects with the dose response curves that have been presented in Table 6 of the interim report.

Response: The problem here for us was not whether a threshold should be used, (we agree that with the current weight of evidence the result both with and without a threshold is important) but at what level to set that threshold. The second chapter of the draft ozone position paper almost completely ignores both mortality and thresholds. All that the position paper said about thresholds was a reference to some epidemiological studies that suggest no threshold for morbidity effects. The value of comparing summer and winter data in Northern European countries seems very limited given variation in personal habits (people spending less time exposed to ambient air). Also, the lower day to day variability in ozone concentrations in winter would make it more difficult for epidemiological analysis to detect relationships. Overall I don’t think any firm conclusion on thresholds is
possible from the Hoek paper, though the results are obviously useful to set alongside other data.

Despite a lack of clear evidence on possible threshold levels the study team has now investigated the possible influence of a 50 ppb threshold on the overall CBA by extrapolation from the UK analysis (dividing estimated ozone-mortality damages by a factor 18).

Comment: For the PM related health effects also a serious objection needs to be made against the calculated results in the interim report. The models calculate the levels of nitrate, whilst all the exposure response slopes are for PM\textsubscript{10} in general and not for nitrate in particular. Nowhere in the interim report, or to my knowledge in the recent literature, has there been a justification that the exposure response slopes of both substances are exactly the same, even assuming that association is the same as causality. In the Netherlands approximately 10\% of the PM\textsubscript{10} is nitrate on a yearly average basis. Recently reported results of epidemiological research in the Netherlands (Hoek et al., 1997) indicate no relationship between nitrate concentrations and mortality, a similar result from a study concerning hospital admissions in the Netherlands will be published this summer.

Response: Without clear guidance on the role of nitrate we have treated it as PM\textsubscript{10}. We regard the question of likely variability of health effects between different fractions of PM as a subject requiring urgent further research. The results of the Hoek paper are certainly of much interest as for some time in the ExternE Project we have questioned the role of nitrate, though without evidence either way. However, Hoek et al is only a single study (and one that is yet to be published in the peer-reviewed literature as we understand). The problems of assuming that the findings of individual epidemiological studies are correct are clear from Dr. Rombout’s own concerns about ozone, above.

Comment on [e]. These comments above on [d] of course also refer to the impact assessment (change in mortality etc.), so there is no need to dwell any longer on this part of the chain.

Comment on [f]. The last point the valuation is again one with serious differences of opinion with the interim report. Skipping the very difficult point of the value of a human life and agreeing with the figures presented in the interim report, the main objection to the valuation is that it does not take into account the currently assumed biological basis for the mortality and morbidity affects of low levels of air pollution: it most probably are not the people in the prime of their lives who are at risk, but possibly people which are already seriously compromised with cardiovascular or respiratory problems. Possibly these people are currently undergoing treatment in a hospital already, so economically speaking it could be argued that it might even save money that these people die (a short period) prematurely. With our current assumptions of the mechanisms involved and the groups deemed at risk a totally different valuation (saving money instead of costing money) for the acute risks would seem at least as economically (but of course not morally) justifiable.

Response: The model adopted for valuation deals with personal preference as well as other factors such as cost of illness. This paradigm is well accepted in Europe and the USA. The valuation issue is dealt with explicitly in this report and the interim report through use of both the VOSL and VOLY approaches, though there remains sensitivity to
the assumption regarding the period of lost life expectancy. It is unfortunate that Dr. Rombout does not distinguish between the two approaches, though this could be a result of the way that some results were presented in the Interim Report.

Given the much lower levels of ozone indoors than out, it seems unlikely that a strong signal would be detected in the epidemiological studies if a substantial fraction of the people at risk were in hospital undergoing treatment (partly because the numbers affected would be small, and partly because of the capacity of epidemiological analysis to pick up trends in day to day mortality against a limited background of variability in ozone concentrations). This is even more the case for people whose health is seriously compromised, because of the greater control that is typically exercised over their environment (e.g. for those in intensive care units, or those breathing through a face mask).

Comment: For the chronic risks a mortality rate per µg/m$^3$ per year of 0.00036 is used (Table 6.) in the interim report. This number implies for the Dutch conditions with an average PM$_{10}$ level of 40 µg/m$^3$ a chronic PM mortality of 0.00144 per year per person. This figure is unrealistically high for Dutch conditions as it would lead to a life expectancy of 69 years solely due to PM mortality. The life expectancy in the Netherlands is higher than 69 years and also other causes of death (caused by some of our bad habits as smoking, overeating, drinking alcohol, not exercising regularly etc.) surely play some part in our health status and mortality rates. Therefore such high figures for chronic PM mortality as presented in table 6 are quite unrealistic for our country (there is a strong tendency among experts not to use figures from for instance the USA for Europe). Other remarks concerning the chronic mortality figures could also be made, for sake of space and time this is omitted here.

Response: I believe that there is some misunderstanding here of the way in which the function has been applied. The factor used does not relate to mortality rates (as does the acute function) but to life years lost. [In any case, simple inversion of the figure 0.0144 (which would give an average age at death of 69 if the function did reference mortality rate) would only work in a population with a uniform age distribution: in EU countries there is a well known shift towards a more elderly population.]

Taking up the point that other remarks could be made about the chronic mortality figures, reference to the report demonstrates that we ourselves have low confidence in the estimates of chronic effects on mortality. Indeed, some (downward) adjustment has already been made to the risk factor reflecting broader comparisons between European and US epidemiological data, and concern over the effect of exposure to higher concentrations historically. There is a strong tendency amongst the health experts of the ExternE Project team not to recommend US data in Europe where alternative European sources are available. In the case of chronic effects of particles on mortality no European study currently exists, and so we are faced with the choice of using the US data or not quantifying the potential risk. The latter option would go against the rationale followed in this study, based on the precautionary principle, which is to quantify where quantification is possible and then to conduct extensive uncertainty and sensitivity analysis (apologies for the repetition). The limited literature available on assessment of these chronic effects has also drawn criticism, but set against this should be considered the
scale of the Pope et al. study, which followed 500,000 people in 150 US cities over an 8 year period. Note also that preliminary calculations suggest that our analysis is consistent with Brunekreef’s study in the Netherlands using the Pope et al. data.

As a final comment, there is an interesting difference in reported PM$_{10}$ exposure between the Netherlands and the UK. Based on the use of fine resolution maps (1 x 1 km) for the whole of the UK we have found that average exposures are just under 20 ug/m$^3$, half the level reported for the Dutch population.

Comment: When the various objections are taken together the economic benefits for the Netherlands could easily be a factor of 100 less than those that have been presented in the interim report. Therefore the current numbers seem rather unrealistic. In the future with better input data the methodology which has been devised in the interim report could be used to assess the economic benefits, if a realistic biological scenario can be agreed upon. Response: This comment seems to apply only to the commentator’s views on the mortality results based on application of the VOSL. This represents only one strand of the analysis presented in the interim report (noting that the sensitivity analysis is presented much more explicitly in this final report).

It also presumes that component results have equal validity which ignores the development of the confidence ranking exercise and other methods used to describe and discuss uncertainty which are given in the report.

We believe that our work on describing and dealing with uncertainties is at the forefront of error analysis in environmental CBA. A problem does remain in dealing with very large quantities of information. Comments on approaches to presenting these data in a still more transparent manner are welcomed.

Comments by page

p. vi.

It is not clear what morbidity effects have been considered apart from restricted activity days. In my opinion the most important health effects of ozone are on morbidity effects and not on mortality. The associations of mortality from acute ozone exposure have been reported in a rather small number of studies. The outcome of these studies is rather variable.

Response: The functions used are clearly listed in our report (Table 6) and include four morbidity endpoints for ozone. With respect to the inclusion of ozone-mortality effects our study follows the rationale of COM EAP (1997), from which we quote: “Taking all the existing evidence into account, it is concluded that an association exists and is reasonably consistent across Europe and with large cities in the US. This justifies an estimate of risk...”. As noted above this assessment considered not just those studies that found an internally robust relationship, but also contrary evidence such as that of the HEI study in Mexico City by Loomis et al. Taking note of these comments, the sensitivity of results to different views on the validity of the mortality functions is now demonstrated more clearly in this report. Finally, we are not aware of any analysis that shows that
morbidity effects outweigh those on mortality. The idea that they are should not be ignored, but is a subjective interpretation of available evidence.

A most important feature of this report is that the health effects of chronic exposure have not been considered. Although quantitative exposure-response data are hardly available, this point must be stressed in the report since chronic irreversible health effects may likely have a rather large impact on health risk.

Response: In view of the lack of data there is nothing by way of chronic effects of ozone that can be quantified at the present time. Hence we do not agree that it is possible to conclude that chronic effects on morbidity have a large impact on health. If we accept this point, however, it may be expected also that ozone would influence life expectancy, and hence that the association with the timing of death is real and causal.

Ozone and PM do not solely act on a regional scale. In fact the anthropogenic Northern hemisphere background levels may rise in absolute and general terms in the future.

A major sensitivity is the fact that epi studies consider associations. In this report these associations are (almost) considered as causal.

Response: This issue is raised in the report (see bullet points a to d following Table 7 in Section 3.3.2).

A number of assumptions on stock at risk will have an impact on the confidence level of the outcome. They should be discussed in uncertainty terms.

Response: This appears to contradict the earlier comment that ‘There are no comments on the stock at risk (distribution of people) or the sensitivity of the stock at risk (baseline mortality) which would lead to a very different picture from the one that was presented.’

Some discussion of uncertainties relating to the stock at risk was given in Section 3.3.1. We concluded that the uncertainty associated with definition of the stock at risk was not significant in the context of the overall analysis. Given the very broad scope of this analysis it is not possible to take explicit account of every potential source of error, though we believe that the uncertainties likely to most affect the results have been considered.

Table 6. The number of studies providing input for the generation of exposure-response relationships is extremely limited. More thorough (meta)analyses are definitively needed here. The uncertainty rating is highly debatable. The number of health endpoints is limited and must be expanded.

Response: This is a misinterpretation of the work underlying our study. Many studies were assessed within the ExternE Project of the EC DGXII JOULE Programme, those cited were carefully selected as being reasonably representative of the published literature. From the comment regarding page vi of the interim report it is apparent that Dr. Rombout thought that only one morbidity endpoint was characterised for ozone, when in fact we addressed four, three of which, respiratory hospital admissions, minor restricted activity days and symptom days each cover a range of health states. Expansion of the number of health endpoints can only be achieved with more data, which are not currently available.
With respect to the number of health endpoints assessed, the listing given in this report corresponds closely to that provided to the US EPA for their benefit analyses of revised standards for ozone and particulate matter.

p.22
The main problem with “epi data relates to causality and covariation in parameters.” Last paragraph APHEA cannot establish causality.
Response: Distinctions between association and causality are made in Section 3.3.2 in both the interim and final reports.
The information on additive effects of ozone and PM is as yet not convincing.
Response: This issue is now accounted for in the more extensive sensitivity analysis presented in the final report.

p.25
Validation of the model predictions against ozone monitoring sometimes shows large discrepancies (certainly for Spain, Belgium and the Netherlands) instead of the reasonable agreement as mentioned.
Appendix VIII

References


HEI (see Loomis et al).


