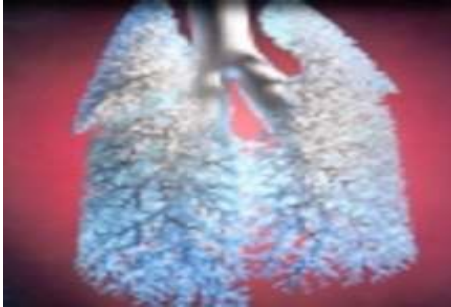

Damage Costs for Air Pollution



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Executive Summary

This document describes the approach and methodology used to derive the damage costs for air pollution for Defra as part of the Air Quality Strategy Review. It is stressed that the use of damage cost estimates is only recommended in certain circumstances e.g.:

- as part of a filtering mechanism to narrow down a wide range of policy options to a smaller number that are then taken forward for more comprehensive assessment;
- where air quality impacts are expected to be ancillary to the primary objectives.

Impacts and damages (£) per tonne for the pollutants PM₁₀, SO₂, NO_x, and VOCs have been derived for the UK (for UK damages) accounting for variation in the location and type of emission. The analysis covers the effects of air pollution on:

- Human health;
- Materials (excluding cultural heritage);
- Crops.

The approach used to estimate these damage costs is based on the impact pathway approach, and has used a detailed, highly disaggregated analysis to estimate the marginal pollution change, physical impacts and monetary damages, per tonne of pollutant emitted in the UK. It has used the national estimates of emissions in the National Atmospheric Emissions Inventory (NAEI), along with the national pollution-mapping tool, to assess a uniform relative reduction in emissions of each pollutant across the country, based on the existing emission profile.

For primary particulates (PM₁₀), the analysis has been more detailed. Damage costs are presented for different sectors. This reflects the importance of PM as a local pollutant and the influence of local stack height and population density in determining the population weighted exposure for PM (differences in population weighted exposure can lead to order of magnitude differences in PM damage costs between sources). For the secondary pollutants (ozone and secondary particulates), one uniform damage cost value has been derived for the UK. This reflects the fact that local stack height and population density are less important for these pollutants, as secondary pollutants form in the atmosphere over time and distance.

In interpreting the damage costs, it is essential to remember that a number of effects are excluded from quantification, including impacts on ecosystems and cultural heritage. Inclusion of these effects would further increase the values. A listing of those effects included and excluded from the analysis is given in the main text.

The following improvements can be made to the data presented in this report:

- To develop data-sets for other years (2015, 2020);
- To provide disaggregated ozone damage costs for VOC and NO_x that differentiate between sector and area, and recognise the strong non-linearities in ozone formation;
- To include uncertainty analysis in the values, to allow the application, for example, of probabilistic damage costs based on monte-carlo analysis;
- To extend the analysis to cover other air pollutants, notably ammonia, heavy metals, and organics (e.g. PAHs);
- To update the crop damage and building materials models.
- To make further investigation of excluded effects, particularly additional possible health effects, ecosystems, cultural heritage, VOC aerosols, to consider how these might change the damage cost estimates here (in sensitivity analysis).

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1 Introduction

1.1.1. Air pollution has a number of important impacts on human health, as well as on the natural and man-made environment. These include impacts of short-term and long-term exposure to air pollution on our health, damage to building materials, effects on crops (reduced yield) and impacts on natural and semi-natural ecosystems (both terrestrial and aquatic). These are described in the box below.

1.1.2. These impacts have a number of important economic costs – known as external costs or externalities, as they are not included in the price of goods or services that lead to air pollution¹. There has been significant progress in the quantification and valuation of these environmental costs over the past decade (see box).

1.1.3. There are a number of potential applications for the use of air pollution costs values across Government. These include:

- Project appraisal (project cost-benefit analysis);
- Regulatory Impact Assessment (policy cost-benefit analysis);
- Input to economic instrument design (setting of taxes, charges, or subsidies);

1.1.4. These applications can involve both:

- Specific air quality measures and policies;
- Wider policies that lead to air quality changes (e.g. in the areas of transport, waste, electricity, agriculture, households), where air quality improvement is not the primary policy objective².

1.1.5. There has been widespread use of environmental cost values for air pollution across Government, across a wide range of sectors. However, the exact approach and values have varied³. This has led to inconsistent policy analysis.

1.1.6. Ideally, each potential appraisal should undertake a rigorous analysis of the benefits (or dis-benefits) of policy on air quality, using a detailed ‘impact-pathway’ approach (see next section for details). This would involve modelling the change in emissions, use of dispersion modelling to assess the changes in air pollution, use of concentration response functions to assess health and non-health impacts, and monetisation of impacts. All of this should be undertaken using the latest approach and values recommended by the IGCB (Inter-Departmental Group on Costs and Benefits).

¹ In some policy areas, there have been moves to internalise these external costs through the use of economic instruments. For clarification, we refer to the quantification and valuation of ‘environmental’ costs and ‘environmental’ damage costs, rather than external costs. The use of these values in policy appraisal, or in the potential design of economic instruments, would have to consider potential issues of internalisation in existing policies.

² This could involve appraisal of policies that actually increased air emissions (though achieved other benefits).

³ For example, values used in different sectors have been taken from different sets of recommendations, some using the exposure response functions recommended in the 1998 COMEAP quantification report– some using the more comprehensive IGCB approach used in the review of the particle objectives including chronic effects. Other studies have used different approaches based on specific expert views, or relying on the EC’s BeTa values.

Box 1. Health and Non-Health Benefits

Studies of air pollution episodes (such as the London smog episodes of the 1950s) have shown that very high levels of ambient air pollution are associated with strong increases in **adverse health effects**. Recent studies also reveal smaller increases in adverse health effects at the current levels of ambient air pollution typically present in urban areas. The health effects associated with short-term (acute) exposure include premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, exacerbation of asthma and other respiratory symptoms. The evidence for these effects is strongest for particles (usually characterized as PM₁₀) and for ozone. For these pollutants the relationships are widely accepted as causal. Recent studies also strongly suggest that long-term (chronic) exposure to particles may also damage health and that these effects (measured through changes in life expectancy) may be substantially greater than the effects of acute exposure described above. These health impacts have major economic costs because of the additional burden they impose on the health service, the lost time at work, and the pain and suffering of affected individuals.

Air pollution also impacts on other receptors. The effects of atmospheric pollutants on **buildings and other materials** provide some of the clearest examples of air pollution damage. Air pollution is associated with a number of impacts including acid corrosion of stone, metals and paints in 'utilitarian' applications; acid impacts on materials of cultural merit (including stone, fine art, etc.); ozone damage to polymeric materials, particularly natural rubbers; and soiling of buildings. SO₂ is the primary pollutant of concern in building corrosion, primarily from dry deposition, but also from the secondary acidic species in the atmosphere. The approach for quantifying and valuing these impacts for 'utilitarian' buildings is based on impact pathway analysis, which links the 'stock at risk' of building materials to exposure-response functions. Impacts are monetised using repair and replacement costs, based on critical thickness loss. While a similar approach could, in theory, be applied to historic and cultural buildings, there is a lack of data on the stock at risk, and also the relevant valuation of building damage. The analysis of building soiling is concerned with the deposition of particles on external surfaces and the dis-colouration of stone and other materials. Although soiling damage has an obvious cause and effect, the quantification of soiling damage is not straightforward.

Ozone is recognised as the most serious regional air pollutant problem for the **agricultural sector** in Europe at the present time. Quantification of the direct impacts of ozone on agricultural yield uses exposure response relationships, with valuation of impacts based on world market prices. Some air pollutants other than ozone have been linked in the literature to crop damages (e.g. SO₂, NO₂, NH₃), but generally at higher levels than are currently experienced.

Air pollution also can impact on **natural and semi-natural ecosystems**. The effects of SO₂ and secondary pollutants on ecosystems ranging from forests to freshwaters are well known, and have been the prime concern until recently in international negotiations. Emissions of NO_x are also known to be responsible for a range of impacts on ecosystems particularly through their contribution to acidification, eutrophication and the generation of tropospheric ozone. However, despite the large, well-documented literature available on these effects, it is not currently possible to conduct an economic analysis of the effects of SO₂ and related secondary pollutants (sulphates and acidity), nor eutrophication or ozone effects on forests, other terrestrial ecosystems and freshwaters, with any confidence. A robust economic analysis would require knowledge of specific effects (change in species richness, productivity, etc.) over extended time scales and appropriate models are not available. Data for valuation of most impacts to ecosystems are also unavailable, or so specific that generalisation to the broader environment cannot be carried out with confidence. In consequence, predictive analysis in this field has almost solely followed the critical levels/critical loads concept.

1.1.7. However, this is extremely resource intensive. It is not practical or cost-effective to apply this methodology to all policy appraisals. To address this, one of the recommendations of the Air Quality Evaluation (Watkiss et al, 2005) was to generate a central set of air pollution benefit values (or 'damage costs') for appraisal⁴.

⁴ The EC's Clean Air for Europe (CAFE) programme has produced similar unit pollution costs (see Holland et al, 2005b), following from an initial set produced (the BeTa values (Holland and Watkiss, 2002) for the EC.

1.1.8. These would provide a consistent set of numbers for benefits analysis of air quality effects. The use of damage cost estimates is only recommended in certain circumstances e.g.:

- as part of a filtering mechanism to narrow down a wide range of policy options into a smaller number that are then taken forward for more comprehensive assessment;
- where air quality impact are expected to be ancillary to the primary objectives.

It is stressed that the values are not proposed for air quality appraisal, either of air quality policy (national policy) or of air quality measures (local schemes). For such schemes, the full impact pathway analysis is still recommended.

1.1.9. Following discussion at the IGCB, it was agreed that a set of damage cost values would be useful. The analysis of an up-to-date set of values has been generated, as part of the work supporting the current Air Quality Review. This has used the latest scientific information, and the latest guidance from the IGCB on valuation. This document sets out the methodology used for deriving this set of values.

2 Methodology

2.1 Introduction

2.1.1. There are a number of possible ways to derive estimates of air pollution cost values. These include ‘top-down’ and ‘bottom-up’ analysis. For nationally based estimates, the large data requirements have usually led to the use of top down assessments, working with nationally aggregated data sets. However, advances in spatial modelling, particularly through the use of Geographical Information Systems, has led to the potential for national estimates based on extremely disaggregated data sets. This allows the use of the impact pathway approach.

2.2 The Impact Pathway Approach

2.2.1. The usual approach taken for the detailed quantification of the benefits of air pollution emissions through to monetisation is often referred to as the ‘impact pathway approach’ a logical progression from emission, through dispersion and exposure to quantification of impacts and their valuation.

2.2.2. This approach was advanced through the series of EC DG Research projects under the ExternE banner (and its predecessor, the EC/US Fuel Cycles Study) through which it has been widely disseminated (ExternE, 1995a, 1999a).

2.2.3. It has also been used extensively in past work on UK and European air quality legislation and thus has been widely debated through the air quality steering group and the working parties that informed it. A similar approach has been used in the Clean Air for Europe (CAFE) programme⁵ by DG Environment.

2.2.4. The approach uses a logical and scientific approach, to build up the estimates of damages step by step. The approach is shown in the figure below. Following from the figure, impacts and damages under any scenario are calculated using the following general relationships:

$$\text{impact} = \text{pollution} \times \text{stock at risk} \times \text{response function}$$

$$\text{economic damage} = \text{impact} \times \text{unit value of impact}$$

2.2.5. Pollution may be expressed in terms of concentration or deposition. The term ‘stock at risk’ relates to the amount of sensitive material (people, ecosystems, materials, etc.) present in the modelled domain.

2.2.6. Although the underlying form of the above equation does not change, the precise form of the equation will vary for different types of impact. For example, the functions that describe materials damage from acidic deposition require consideration of climatic variables (such as relative humidity) and need to account for several pollutants simultaneously.

⁵ The Clean Air for Europe (CAFE) Programme led to the adoption of a Thematic Strategy on Air Pollution, fulfilling the objectives of the European Commission’s Sixth Environmental Action Programme. Relevant documentation relating to the CAFE cost benefit analysis can be found at <http://www.cafe-cba.org/>

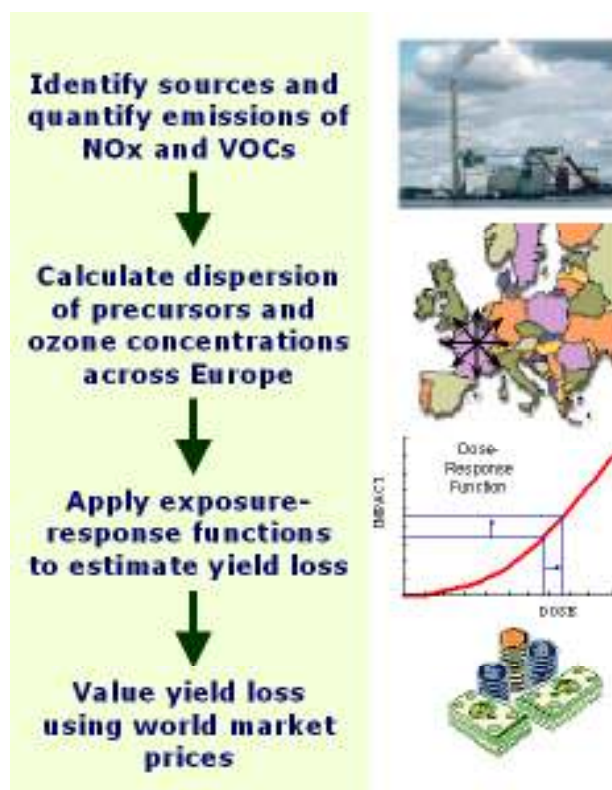


Figure 1. Illustration of the impact pathway approach taking the example of direct effects of ozone on crops

2.2.7. For any type of receptor it is necessary to implement a number of these impact pathways to generate overall benefits. So, for example, in the case of impacts of ozone on crop yield, it is necessary to consider, separately, impacts on a series of different crops, each of which differs in sensitivity. For health assessment it is necessary to quantify across a series of different effects to understand the overall impact of air pollution on the population.

2.2.8. The final stage, valuation, is generally done from the perspective of ‘willingness to pay’ (WTP). For some effects, such as damage to crops, or to buildings of little or no cultural merit this can be done using appropriate market data. Some elements of the valuation of health impacts can also be quantified from ‘market’ data (e.g. the cost of medicines and care), though other elements such as willingness to pay to avoid being ill in the first place are clearly not quantifiable from such sources. In such cases alternative methods are necessary for the quantification, such as the use of contingent valuation (for discussion of this and other valuation techniques see ExternE, 1999a; EAHEAP, 1999). Where impacts arise in the future it is necessary to discount monetised values.

2.2.9. All of this information is brought together for the calculations. The calculations are made for each cell within a grid system. For this analysis, we have automated this analysis within a Geographical Information System (GIS). This allows considerable flexibility in the grid resolution, and can at different scales from a 50 x 50 km grid down to 1km² grid resolution. The basic form of the analysis remains the same no matter what spatial resolution is adopted for the analysis.

2.3 National Estimates of Marginal Pollution Damages

2.3.1. The damage costs here – intended for policy appraisal – need to reflect the marginal damage costs of pollution, i.e. the additional marginal effect of one extra tonne of pollution (or the removal of one extra tonne of pollution). Previous studies have shown that the marginal damage costs of air pollution vary very significantly (per tonne of pollutant emitted) according to a range of parameters including:

- Location of emissions;
- Height of emission;
- Local and regional meteorology and other secondary pollutant precursors;
- Local and regional receptors (density of receptors and geographical spread).

It is stressed that the relationship between emissions and concentrations are not necessarily linear.

2.3.2. As the values are intended for use at a national level, i.e. for policies that potentially impact across entire sectors, with large geographical coverage, there is a conflict between trying to produce simple numbers intended to help policy analysts, versus the need to make sure that the values are actually representative and robust.

2.3.3. To try and address this, the analysis has varied the approach for different pollutants.

2.3.4. For primary particulates (PM₁₀), the analysis has produced separate values for each major sector. This reflects the importance of PM as a local pollutant, and takes into account the stack height and location of emissions (in relation to population density). This is extremely important, because previous analysis (see Watkiss, 2005) has shown that order of magnitude differences can occur in the damage costs of PM₁₀ between different locations and different sources. In summary, areas of higher population density/local population (urban areas) have higher damage costs, because emissions lead to higher population weighted exposure per tonne emitted.

2.3.5. For secondary pollutants (ozone and secondary particulates), one uniform value has been derived for the UK. This reflects the fact that local issues are less important for these pollutants. These secondary pollutants form in the atmosphere over time, and so the immediate local environment is less important in determining damage costs.

2.3.6. The use of a single value for secondary pollutants is more uncertain for the effects of NO_x on ozone. In practice, the formation of ozone from this precursor will vary significantly between urban and rural sites. This has not been taken fully into account in this analysis. Given that ozone damages are found to be small compared to PM effects, this should have little effect on the results. However, we recommend that further work to progress separate urban and rural values for NO_x in relation to ozone.

2.3.7. The main values derived are damage costs – the cost (£) per tonne of pollutant emitted (or the converse, i.e. benefits (£) per tonne of pollutant reduced). It is stressed that these relate only to the environmental effects, they do not include any mitigation costs (i.e. the costs of measures to reduce pollution). Separate values are presented for each pollutant (e.g. NO_x, SO₂, PM₁₀, VOC). The values are presented as a net present value (NPV) and an equivalent annualised benefit. These represent the damage costs in the UK from UK emissions. They do not include the impacts from UK emissions in the rest of Europe (i.e. trans-boundary

pollution). The values are also disaggregated by category (e.g. health, crops, buildings). The values include the health and non-health effects of both primary and secondary pollution from each pollutant – including secondary particulates and ozone. There is also a set of ‘impacts per tonne’ of pollutant emitted for the main health impacts, to allow estimation of respiratory hospital admissions per tonne, or years of life lost.

2.3.8. There are also separate values produced according to the time-scale of emissions reductions. Work for previous policy analysis has identified two types:

- Short-term pollution reductions, where the usual metric is to consider an annual change in air emissions.
- Long-term pollution reductions, associated with sustained pollution changes. The tradition in IGCB has been to consider these over a 100 year time-scale.

The analysis here has separated these into two damage costs – for an annual pulse, and a sustained pollution change over 100 years. Analysis has also been undertaken for a 5 and 20 year analysis reflecting sustained pollution changes over these time-scales.

2.4 Effects Included and Excluded

2.4.1. The analysis has considered a wide range of impacts, summarised in the table below.

Table 1. Effects included in the analysis

Burden	Effect
Human exposure to PM ₁₀ /PM _{2.5} (emitted directly or formed indirectly from NO ₂ or SO ₂)	Chronic effects on Mortality Acute effects on Morbidity (Respiratory and Cardiovascular hospital admissions)
Human exposure to ozone (formed indirectly from VOCs and NO ₂)	Acute effects on Mortality and Morbidity (Respiratory hospital admissions)
Human exposure to SO ₂ (emitted directly)	Acute effects on Mortality and Morbidity (Respiratory hospital admissions)
Exposure of crops to ozone	Yield loss for barley, cotton, fruit, grape, hops, millet, maize, oats, olive, potato, pulses, rapeseed, rice, rye, seed cotton, soybean, sugar beet, sunflower seed, tobacco, wheat
Damage to materials	Acidic deposition Ozone damage to polymeric materials Building soiling

Sensitivity analysis

Human exposure to PM ₁₀ /PM _{2.5} (emitted directly or formed indirectly from NO ₂ or SO ₂)	Acute effects on Mortality (deaths brought forward)
Human exposure to NO ₂	Acute effects on Morbidity (Respiratory hospital admissions)

2.4.2. However, it is also highlighted that not all potential benefits of air quality have been quantified / valued, because quantification is not possible or highly uncertain. Amongst those effects actually or potentially excluded are:

- The effects of VOC emissions on inorganic particle concentrations.
- Impacts on ecosystems through exceedance of critical loads and critical levels (including forests, freshwaters, etc.). This has long been regarded as a serious problem, with potentially significant consequences for ecological sustainability. With respect to acidification, which is linked to emissions of SO₂, NH₃ and NO_x, the problem is worst in areas of northern Europe where the bed rock is hard and weathers too slowly to counteract deposited acidity (e.g. Scandinavia) and much less severe in southern Europe (e.g. Spain, Greece). The most obvious impact of acidification is the loss of fish, particularly salmon and trout, though terrestrial ecosystems are also affected. Problems of eutrophication, caused by emissions of nitrogen-containing pollutants (NO_x, NH₃) are widespread in Europe, with particular hot-spots in a few countries, such as the Netherlands. The most visible effect is one of reducing the viability of rarer species of plant, allowing other species, particularly grasses, to invade land that was previously too nutrient deficient to support them, leading to a loss of species diversity. A modelling framework for describing exceedance of critical loads and levels is well developed across Europe. Valuation of these impacts is not yet possible because of limited research in this area that has specific relevance to reductions in air pollutant emissions.
- Additional health impacts from PM. A number of other major studies, including peer-reviewed work by CAFE, has identified a much larger number of morbidity effects from PM pollution than used in UK quantification⁶. These include serious effects such as infant mortality, chronic morbidity (e.g. bronchitis) and minor acute morbidity effects (lower respiratory symptoms, restricted activity days, etc). These additional effects add significantly to the overall damage costs. There are also additional effects suspected on morbidity from chronic (long-term) exposure.
- Additional health impacts from ozone. The same studies (as above) have identified a much larger number of morbidity effects from ozone. There may also be additional effects on morbidity and mortality from chronic (long-term) exposure to ozone.
- Damage to cultural heritage, such as cathedrals and other fine buildings, statues, etc has not been assessed (only damages to utilitarian buildings). Whilst the effects on historical buildings provided the earliest and clearest demonstration of air pollution effects, its importance has decreased substantially over time, as urban SO₂ levels have reduced. It is unknown whether this reduced rate of deterioration is still important. Analysis is not possible because of a lack of data on stock at risk (e.g. number of culturally important buildings, surface areas, number and size of statues) and repair and maintenance costs.
- Additional effects of ozone on crops in relation to visible damage (of marketed produce). Non-ozone effects on agriculture (e.g. through acid deposition, nutrient deposition, interactions between pollutants and pests and pathogens, etc.).
- Change in visibility (visual range) as a function of particle and NO₂ concentration. Research in the USA suggests that this results in a serious loss of amenity. However, following analysis carried out for EC DG Environment and the UNECE, and resulting debate, it was concluded that the issue is not regarded as being so serious in Europe (possibly because reduced visibility through poor air quality is now less of a problem than

⁶ In the UK, guidance from COMEAP has aimed to estimate effects that 'could be applied in the UK with reasonable confidence'. This increases the confidence in the estimates as quantified, but systematically underestimates benefits, because it does not seek to quantify the total impacts of air pollution. A more comprehensive approach of appraisal has been adopted for European policy appraisal (e.g. in CAFE) that aims to generate an unbiased set of estimates of the effects of air pollution on health, i.e. neither systematically *over-* or *under-*estimating effects, acknowledging that this increases the uncertainty.

it was a few years ago). It has been concluded that the US results are not transferable to Europe, though their inclusion would significantly increase the benefits values above.

- Macroeconomic effects of reduced crop yield and damage to building materials.
- Altruistic effects of health impacts⁷

2.5 Emissions and Air Quality Modelling

2.5.1. The starting point for the impact pathway analysis is the spatially disaggregated National Atmospheric Emissions Inventory (NAEI). The baseline methodology for the analysis can be found at the NAEI website (<http://www.naei.org.uk>) and is consistent with that reported in the UK Emissions of Air Pollutants. The detailed methodology is not repeated here but in essence the baseline takes account of all agreed future policies that might affect air quality, such as the Large Combustion Plant Directive (LCPD) for the electricity sector and agreed Euro standards for the road transport sector.

2.5.2. The baseline emissions profiles used were based on the recent work as part of the Air Quality Strategy Review. This includes a number of recent updates, including to the transport and ESI sectors. The work under the Review is being written up separately⁸.

2.5.3. The emissions are used as an input into pollution modelling. The analysis uses the UK Air Pollution Mapping model (Stedman et al, 2002). The modelling approach used is consistent with that used for other analyses presented in Air Quality Strategy Review. The analysis has been run for the baseline in 2010, i.e. the marginal change from the 2010 business as usual case.

2.5.4. Contributions to ground level PM₁₀ concentrations are influenced by emissions from point sources, area sources, secondary particles and coarse particles. These are all taken account of in the model to represent the chemical reactions in the atmosphere, enabling quantification of effects linked to emission, not simply to atmospheric concentration of the pollutant in the chemical state in which it was released. An important consequence of this is that effects caused by *secondary* particulates are not assigned to the PM₁₀ damage costs per tonne, but to damage costs per tonne of the primary pollutant from which they are formed (e.g. SO₂ for sulphate aerosol, NO_x for nitrate aerosol).

2.5.5. The UK Ozone Source-receptor Model (OSRM) was used to assess ozone concentrations in the UK. The OSRM model produces annual mean ground level ozone concentration (µg/m³) and other required impact metrics such as daily maximum of a running 8-hour mean threshold (100µg/m³), AOT40 exposure (ppm.hours), etc. Details of the model are provided in Hayman et al (2005). The model was run to assess the impact of VOC and NO_x in ozone formation⁹. The analysis has been run for the baseline in 2010, i.e. the marginal change from the 2010 business as usual case.

2.5.6. The pollution model outputs were combined with stock at risk data within a GIS (geographical information system) to provide relevant receptor weighted concentrations. This

⁷ Though these could involve double counting with other WTP estimates in the analysis.

⁸ See <http://www.defra.gov.uk/environment/airquality/index.htm#aqstrategy>

⁹ The model includes a surface-conversion algorithm, which is needed for ozone in urban areas to account for scavenging of ozone by NO, though it is possible that ozone is still slightly overestimated in urban areas.

was combined, in turn, with concentration-response functions to estimate impacts, and with monetary endpoints to calculate the economic values. Details are given by impact below.

2.6 Health Impacts

2.6.1. This section provides a summary of the general approach to health quantification. The main health outcomes quantified in the study are:

- Short-term (acute) pollution effects - deaths brought forward and respiratory and cardiovascular hospital admissions; and
- Long-term (chronic) effects - changes in life expectancy, known as chronic mortality.

These relate to the two types of evidence on health impacts, outlined in the box below.

Box 2. Health Effects

Two types of epidemiological study are relevant to the quantification of mortality impacts from health pollution:

- Time series studies, available for assessing the mortality and morbidity impacts of the short-term (**acute effects**) exposures to PM, SO₂, O₃ etc., which examine associations between daily pollution levels and daily numbers of deaths or respiratory or cardiovascular hospital admissions, i.e. the ways that air pollution on a given day or adjacent days can affect the health of people on the same day or on the days immediately following – typically within one week (though some analyses of acute exposure now include effects that occur up to 40 days from the relevant pollution days).
- Cohort studies which examine age-specific death rates (technically mortality hazards) in study groups of individuals followed up over prolonged periods. Having adjusted for other mortality risk factors measured for individuals (gender, race, smoking habit, educational status, etc.), differences in age-specific death rates between cities are assessed in relation to average pollution concentrations over periods of several years (chronic effects).

2.6.1 Chronic mortality from PM

2.6.2. Chronic effects on mortality have become the main focus for quantification of the health impacts of particulate exposure. The analysis here, consistent with similar studies, uses the risk estimates that are based on analyses of the American Cancer Society (ACS) cohort by Pope *et al* (1995,) and updated in 2002.

2.6.3. In terms of the physical impacts quantified, the analysis is consistent with the guidance from COMEAP and from WHO, and with an emerging consensus in HIA work, and expresses chronic mortality effects principally in terms of change in longevity. This also leads to estimates of the change in longevity aggregated across the population (otherwise referred to as ‘years of life lost’) as the most relevant metric. The analysis and implementation assume no threshold of effects.

2.6.4. While there is consistency on the use of the Pope study, and the use of years of life lost, there remain major differences on the exact implementation with respect to a number of areas. There are three major areas where differences occur.

2.6.5. Firstly, in relation to risk rates. The main results from the Pope *et al* (1995) study give a lower bound estimate of increase in death rates of 3% per 10 µgm⁻³ PM_{2.5}, a central estimate of 6% per 10 µgm⁻³ PM_{2.5}, and an upper bound estimate of 9% per 10 µgm⁻³ PM_{2.5}. An exhaustive reanalysis by the Health Effects Institute (HEI, 2000) confirmed these findings but

also found that adjustment for a possible direct effect of SO₂ reduced the estimate to 1% per 10 µgm⁻³ PM_{2.5},

2.6.6. COMEAP (2001) developed a series of estimates of the expected gains in life years for a sustained 1µg/m³ drop in PM_{2.5}. It considered a reduction in mortality rate of 1% (per 10 µgm⁻³ PM_{2.5}) to be ‘most likely’, a reduction in mortality rate of 3% to be ‘reasonably likely’ and a reduction in mortality rate of 6% to be ‘less likely’. COMEAP have recently issued an interim statement on mortality and long-term exposure to air pollutants, particularly relating to ambient particles.¹⁰ This is based on a detailed consideration of the more recent evidence and factors that can affect the best estimate of the size of the coefficient such as adjustment for sulphur dioxide, spatial autocorrelation and measurement error and the higher coefficients found in studies at smaller spatial scales. On balance, the Committee recommended using a coefficient of 6% per 10µg.m⁻³ PM_{2.5} from the largest most extensively analysed cohort study (Pope et al, 2002). The Committee quoted the 95% confidence intervals for this coefficient (2% to 11%) as an interim uncertainty range but noted that this only represented the statistical (sampling) uncertainty and not other factors contributing to uncertainty.

2.6.7. This is consistent with WHO/THF advice to the CAFE programme¹¹, and wider current practice, which also uses the recent extended follow-up by Pope *et al.* (2002) of the American Cancer Society (ACS) cohort. CAFE adopted the coefficient 6% change in mortality hazards (95% CI 2-11%) per 10 µg/m³ PM_{2.5} for the central estimate.

2.6.8. Secondly, in relation to the size metric. IGCB (2001) applied the risk estimates for PM_{2.5} from the Pope study directly to the change in marginal PM₁₀ concentrations. This assumes that most PM₁₀ from marginal pollution changes falls within the PM_{2.5} size fraction¹², but also that secondary particulates that form as a result of pollution, are also within this size fraction¹³. Other studies, e.g. CAFE, have worked directly with (anthropogenic) PM_{2.5} only.

2.6.9. Thirdly, there remains no consensus on the lag period for chronic effects, i.e. the period between exposure and impact. COMEAP 2001 gave a range assuming a lag of between 0 and 40 years. However, in its 2006 Interim Statement, COMEAP stated that, although the evidence was limited, its judgement tended towards a greater proportion of the effect occurring in the years soon after pollution reduction rather than later. CAFE assumes no lag between exposure and effect. This is consistent with recent guidance given by the WHO to the CAFE process, though it seems implausible that all health effects are effectively very short-term. The USEPA has recently considered a multi-step lag phase, which assumes 30% of the effect of reduced pollution on deaths rates occurs immediately (year1); 50% of the effect is distributed over years 2-5; and the remaining 20% is distributed over years 6-20.

¹⁰ ‘Interim Statement on the Quantification of the Effects of Air Pollutants on Health in the UK’, *Committee on the Medical Effects of Air Pollution*, Department of Health (2006b). Available at <http://www.advisorybodies.doh.gov.uk/comeap/pdfs/interimlongtermeffects2006.pdf>

¹¹ Advice given by WHO-TFH at its meetings of both 2003 (see <http://www.unece.org/env/documents/2003/eb/wg1/eb.air.wg1.2003.11.pdf>) and May 2004: (<http://www.unece.org/env/documents/2004/eb/wg1/eb.air.wg1.2004.11.e.pdf>).

¹² The validity of this assumption varies with sector. Recent work for the air quality review has shown that the modelled incremental change from air quality policies show a PM₁₀ fraction that is 30% larger than the PM_{2.5} fraction, though this may be due in part to model uncertainty.

¹³ Sulphates mostly fall within the PM_{2.5} fraction. Some, but not all nitrates fall within the PM_{2.5} fraction. Information on the possible size fractions of secondary particulates was provided in APEG.

2.6.10. For the analysis here, we have presented values for all three risk rates (1%, 3%, 6%), a zero and 40 year lag phase, applied to marginal changes in PM₁₀ (using equal risk rates for both primary and secondary PM₁₀). This leads to an extremely large range of estimates (a factor of twelve) between the low and high estimates of chronic health effects (expressed as years of life lost). Whilst the full range is presented, the recent advice from COMEAP suggests that damage costs at the upper end of the range (6%, zero lag) are more likely.

2.6.11. The analysis of chronic effects has been undertaken using a life-table approach, using the life-tables held by IOM. The analysis has assessed both the changes from a sustained pollution pulse (over 5, 20 and 100 years) and an annual pollution pulse. The discussion on these approaches is set out in the box below. The approach applies equally to primary PM, and secondary PM (formed from SO₂ and NO_x emissions as sulphates and nitrates).

2.6.12. Consistent with emerging practice, we do not separately quantify acute mortality impacts from PM₁₀ and add these to the chronic mortality estimates, as this would double count some impacts. However, the acute mortality effects are included as a sensitivity, using a risk estimate of 0.75% increase in daily mortality per 10 µg/m³ PM₁₀.

Box 3. Chronic Mortality

The approach for estimating the effects on mortality of long-term exposure to ambient air pollution uses life tables to estimate the effects on life expectancy, or more generally on life-years, of the changes in hazard associated with (changes in) annual average PM.

The previous approach in IGCB has been to use life tables to assess the change in mortality from long-term exposure to PM. This assumes that pollution reductions are sustained over time; i.e. that, once reduced, the mortality hazards remain at their lower levels. This work has been undertaken by IOM at Edinburgh (Brian Miller and Fintan Hurley). The main life table calculations are based on a 1% change (decrease) in mortality hazard rates, applied to the population of England and Wales and they draw directly on IOM's work for the Department of Health/ IGCB. The analysis is based on a 100 year sustained analysis. The approach applies changes in mortality hazards are applied only at ages 30 years or more, because the underlying ACS cohort study examined only adults at ages 30 years or more.

Some policies do not lead to sustained pollution pulses. The life tables have been used to investigate the impact of a 1 year change in hazard rates (after which time, hazard rates return to the previous levels). For this annual pulse approach, it is important to stress that the impacts of a 1 year pollution pulse are followed up over a 100 year period. To illustrate, with no lag, and a 1-yr pulse, the *mortality risks* change in Yr 1 only, and then they revert to previous levels. The reason for following up the population over a full lifetime is that the lower mortality risks in Year 1 under a pulse reduction imply: fewer deaths in year 1 (i.e. the number of deaths 'saved'), also and necessarily a slightly *increased* population in Year 2 and subsequently; and so slightly *more* deaths in Yrs 2 and onwards, because of the slightly larger population at risk. The analysis over a full lifetime 'tracks' how all this plays out. Another way of expressing it is that we analyse over 100 years to see when the deaths 'saved' in year 1 actually occur later, because necessarily they will occur. These 1-year pulse implementations were based *only* on the population alive at the time of the pulse – for analysis of that 1-year pulse, no account was taken of new birth cohorts born in later years. Note, that discounting is applied to YOLLS in future years during the valuation step.

This summarises a note, Annual 'Pulse' Or Sustained Changes In Hazard Rates – an interim analysis of time-related effects in life table analyses by Fintan Hurley and Brian Miller, IOM, Edinburgh

2.6.2 Acute mortality from ozone

2.6.13. The cohort studies used for the assessment of PM related mortality do not show a clear effect of ozone. For ozone it is therefore necessary to use data from time-series studies, which clearly link ozone to mortality.

2.6.14. The basic approach to estimating the effects of air pollution on ‘acute’ mortality is to use a concentration-response (C-R) function expressed as:

- i. % change in frequency of occurrence of an endpoint (relative risk (RR) of new or ‘extra’ cases) per $10 \mu\text{g}/\text{m}^3$ ozone; link this with:
- ii. the background rates of the endpoint (new cases per year per unit population – say, per 100,000 people) in the target population
- iii. the population size
- iv. the relevant pollution increment, expressed in $\mu\text{g}/\text{m}^3$ ozone

and express the result as estimated new or ‘extra’ cases per year.

2.6.15. The approach here has used the recommendations from the UK Department of Health’s *Committee on the Medical Effects of Air Pollutants* (COMEAP 1998). The implementation guidance from COMEAP was to quantify deaths brought forward and respiratory hospital admissions from ozone. The original quantification report quantified using risk estimates of a 0.6% increase in daily mortality per $10 \mu\text{g}/\text{m}^3$ O_3 and 0.7% increase in respiratory hospital admissions per $10 \mu\text{g}/\text{m}^3$ O_3 . The functions used are based on the APHEA studies, which are based on all year coefficients but COMEAP chose to do its calculation for the summer only.

2.6.16. The current study has used the 0.06% risk rates (per $\mu\text{g}/\text{m}^3$) with baseline rate estimates for the UK. The analysis has changed in that it considers annual rates (based on the annual average (mean) of the daily maximum running 8 hour mean) rather than just summer-time rates. Consistent with earlier COMEAP implementation, it has quantified without threshold, and with a threshold of 50 ppb. The analysis has also quantified at 35 ppb. This is the same as the cut-point recommended in CAFE of 35 ppb (for implementation, CAFE uses the metric SOMO35¹⁴). The analysis uses ozone as characterised by daily maximum 8-hr mean, in relation to daily all-cause mortality.

Impact	Function
Deaths brought forward from ozone	Risk estimate of 0.6% increase in daily mortality per $10 \mu\text{g}/\text{m}^3$ O_3 . Quantified at concentrations greater than 0 ppb, 35ppb and 50 ppb (maximum 8-hr mean). Baseline rate 990 per 100,000

2.6.17. The direct output from the analysis is the number of deaths brought forward. However, to quantify using the VOLY approach, it is necessary to convert the number of deaths into the number of life years lost. To do this, the period of life that is lost is needed. This is discussed in the valuation section.

¹⁴ In CAFE, the metric SOMO35 (sum of means over 35 parts per billion (ppb)) is used. Note there is not robust evidence for the presence of a threshold for ozone effects, though quantification is less reliable at low ozone concentrations. This measure represents accumulated exposure to concentrations greater than 35 ppb daily maximum 8-hr mean. This should not be taken as an indication that there are no effects under 35 ppb. WHO/TFH recognised using a cut-off point is a conservative approach to mortality effects of ozone.

2.6.18. The approach is used to quantify both NO_x and VOC contribution to ozone. For the results here, it is stressed that the two precursors lead to different changes in ozone: reductions in VOC reduce ozone (leading to health benefits), whilst at current concentrations, reductions in NO_x in urban areas increase ozone (leading to health impacts). This can be explained because reductions in either VOC or NO_x emissions will tend to decrease photochemical ozone concentration in rural areas. Reductions in NO_x emissions in urban areas, however, tend to lead to an increase in local ozone concentrations, because the scavenging of ozone by NO occurs to a lesser extent. In terms of population-weighted mean concentrations across the UK this local titration effect generally dominates. Large enough reductions in NO_x emissions will lead to reductions in ozone concentrations, particularly in the north and west of the UK. These issues of non-linearities do mean that a higher level of uncertainty is attached to the ozone analysis. Further work on damage cost estimates for ozone has therefore been recommended.

2.6.3 Acute Morbidity from PM and ozone

2.6.19. The basic approach to estimating the effects of air pollution on human morbidity is similar to 'acute' mortality above; i.e. using a concentration-response (C-R) function and background rate. The relevant functions as recommended by COMEAP, and used here, are shown below. Valuation is described later.

Table 3. Morbidity Concentration Response Functions.

Impact	Function
Respiratory Hospital Admissions PM ₁₀	Risk estimate of 0.8% increase in RHA per 10 µg/m ³ PM ₁₀ Baseline rate 980 per 100,000
Cardiovascular Hospital Admissions PM ₁₀	Risk estimate of 0.8% increase in CHA per 10 µg/m ³ PM ₁₀ Baseline rate 981 per 100,000
Respiratory Hospital Admissions ozone	Risk estimate of 0.7% increase in RHA per 10 µg/m ³ O ₃ . Quantified at concentrations greater than 0 ppb, 35ppb and 50 ppb (maximum 8-hr mean). Baseline rate 980 per 100,000

2.6.20. For PM, the approach applies equally to primary and secondary PM (the latter from SO₂ and NO_x emissions). Similarly, the approach is used for both NO_x and VOC contribution to ozone (though as above, reductions in VOC reduce ozone (health benefits), but reductions in NO_x increase ozone (health impacts)).

2.6.4 Mortality and Morbidity from SO₂ and NO₂

2.6.21. COMEAP also recommended quantification of deaths brought forward and respiratory hospital admissions from SO₂ as a gas (note SO₂ is also included via the formation of sulphates as PM above). The recommended concentration-response functions are shown below.

Impact	Function
Deaths brought forward from SO ₂	Risk estimate of 0.6% increase in daily mortality per 10 µg/m ³ SO ₂ . Baseline rate 990 per 100,000
Respiratory Hospital Admissions from SO ₂	Risk estimate of 0.5% increase in RHA per 10 µg/m ³ SO ₂ Baseline rate 980 per 100,000

2.6.22. COMEAP also recommended sensitivity for NO₂ as a gas (note NO_x is also included via the formation of nitrates as PM above, and in formation of ozone). The recommended concentration-response function is shown below.

Impact	Function
Respiratory Hospital Admissions from NO ₂	Risk estimate of 0.5% increase in RHA per 10 µg/m ³ NO ₂ Baseline rate 980 per 100,000

2.6.23. The analysis has not considered direct effects of VOCs (though their effect on ozone is captured as set out above).

2.7 Health Valuation

2.7.1. In previous appraisals of air quality policy proposals, it has not always been possible to value health mortality impacts due to a lack of empirical evidence as to the appropriate values.

2.7.2. Values for a range of health endpoints, including mortality, have now been agreed, following recommendations by the IGC. These recommendations have drawn upon recent research in the area, particularly the Defra-led study by Chilton et al (2004) which aimed to identify the willingness to pay to reduce the health impacts associated with air pollution, using a survey-style contingent valuation approach. This updates earlier recommendations from EAHEAP.

2.7.3. These agreed values have been used to monetise the health impacts. The values have been converted to 2005 prices (assuming an inflation rate of 2.5%). In subsequent years, the values have been uplifted by 2%. This reflects the assumption that willingness to pay will rise in line with economic growth. Where long-term sustained changes are assessed, the impacts are then discounted using the declining discount rate scheme recommended in the Green Book. The total monetised benefits for each health effect have then been estimated. Note the use of the uplift leads to different equivalent annual values between the 1, 5, 20 and 100 year analysis (i.e. the length that the lower pollution levels are sustained at). The net effect is to increase the equivalent annual benefit of the sustained pollution pulse for longer time-scales¹⁵, so that for example, the annualised benefit of the 100 year sustained pollution pulse is around two times higher than the 1 year benefit (for the same emissions pulse).

2.7.4. The values are summarised in the table below. There are some methodological notes associated with these values.

2.7.5. To value chronic mortality, the analysis uses the concept of the value of a life year (VOLY)¹⁶.

¹⁵ Using the definition in Green Book, the annualised value is the constant annualised value that when discounted and summed, produces the NPV. Therefore the annualised value for the sustained pollution pulse is presented as the annualised uplifted value.

¹⁶ This approach is also used in CAFE but as some regard the estimates of the VOLY as more uncertain, (especially in relation to age specific VOLYs), CAFE also quantifies premature mortality benefits based on the cohort studies in terms of 'attributable deaths' and values these using a Value for a Statistical Life, though it acknowledges that the quantification of such deaths is strictly speaking wrong ('attributable deaths' can only relate to a specified time period, the net difference in deaths (when comparing two populations of the same size with higher and lower pollution) is zero since everyone in both populations will die at some point).

Table 5. Summary of IGCB Recommendations on Health Valuation.

Health Effect	Form of measurement to which the valuations will apply	Valuation – (2004 prices)	
		Central Value	Sensitivity
Acute Mortality (ozone)	Number of years of life lost due to air pollution (life years) – assuming 2-6 months loss of life expectancy for every death brought forward. Life-expectancy losses assumed to be in poor health.	£15,000	10% and 15% of life years valued at 29,000 instead of £15,000 (to account for avoidance of sudden cardiac deaths in those in apparently good health)
Chronic Mortality	Number of years of life lost due to air pollution (life years) - Life-expectancy losses assumed to be in normal health.	£29,000	£21,700 - £36,200 (sensitivity around the 95% confidence intervals)
Respiratory Hospital Admissions	Case of a hospital admission - of average duration 8 days.	£1,900 – £9,100	£1,900– £9,600
Cardiovascular Hospital Admissions	Case of a hospital admission - of average duration 9 days.	£2,000 – £9,200	£2,000 – £9,800

2.7.6. For acute mortality from ozone, the direct output is deaths brought forward. However, to quantify using the VOLY approach, it is necessary to convert the number of deaths into the number of life years lost. This is difficult, as there is a lack of direct evidence for this part of the quantification. Previous work by the study team (ExternE 1999a, Hurley et al. 2000) has involved ‘conversion’ of attributable deaths from time series studies to changes in life years using an estimate of 6 months per life¹⁷. The guidance from IGCB has been to assume that between 2 and 6 months of life is lost, on average.

2.7.7. For morbidity, the estimates include resource costs (e.g. NHS costs) and dis-utility (opportunity costs, i.e. lost productivity, are considered in the sensitivity). COMEAP, in the quantification report, presents the functions for respiratory hospital admissions as ‘brought forward and additional’, recognising that some or all of these cases would have occurred in the absence of the additional pollution. As is usual in most HIA work, we have assumed that hospital admissions attributable to air pollution are additional to those that would have occurred anyway, and not simply the bringing forward of admissions that would otherwise still have occurred, but only later. In practice, there is likely to be a mixture of both, but the underlying time series studies are strictly uninformative about the balance between them. We highlight that this assumption does not have a significant impact on the overall economic benefits (because the effects of RHAs are so low compared to the overall values).

2.7.8. It is stressed that uncertainties remain regarding valuation, in particular:

- The amount of life expectancy lost due to the acute effects of air pollution;

¹⁷ When originally made, this estimate was considered by many to be an overestimate, though the peer review of the CAFE-CBA methodology thought that a larger value, on average, was warranted. A US evaluation of ozone and mortality has used an estimate of 12 months, and this is used in CAFE-CBA also. While there is no direct evidence to justify this figure, it seems an appropriate balance between ‘harvesting’ and the death of individuals who would have recovered and lived for significant periods.

- The quality of the life expectancy lost due to the acute effects of air pollution;
- The ability of respondents within the contingent valuation study to accurately value losses of life expectancy in poor health;
- The accuracy with which study respondents valued morbidity effects.

2.8 Materials

2.8.1. Damage to materials has historically been one of the major air pollution issues. A number of impacts exist, the most important of which are:

- Acid corrosion of stone, metals and paints in ‘utilitarian’ applications;
- Ozone damage to polymeric materials, particularly natural rubbers;
- Soiling of buildings and materials used in other applications.
- Acid impacts on materials of cultural merit (including stone, fine art, and medieval stained glass, etc.);

2.8.2. The method for quantification of damage to materials follows work carried out by the Europe-wide ICP Materials and quantification under various studies for DG Research, particularly ExternE and associated projects such as GARP (Green Accounting Research Project). The most significant impacts are those on natural stone and zinc coated materials. The ‘impact pathway’ approach works well for those applications that are used in every day life. This could in theory be applied to cultural and historic buildings. However, in practice there is a lack of data at several points in the impact pathway with respect to the stock at risk and valuation. As a result, effects of air pollution on cultural heritage cannot be quantified and thus need to be addressed qualitatively through the extended CBA framework.

2.8.1 Corrosion in utilitarian buildings (SO₂ and NO_x emissions)

2.8.3. The analysis of damages to materials in utilitarian applications, i.e. in modern houses, factories, etc. has been well advanced through work by the Europe-wide ICP Materials¹⁸ and quantification under various studies for the European Commission DG Research, particularly ExternE and associated projects. These studies have shown that the pollutants most implicated in acid damage are SO₂ (most importantly), H⁺ and then NO₂. The most significant impacts are on natural stone and zinc coated materials. A methodological approach exists for the quantification and valuation of material damage, based around the ‘impact pathway’ approach linking exposure-response relationships, the stock at risk, and building repair values. Note no uplift is applied to non-health values in future years¹⁹.

2.8.4. Previous analysis has shown low levels of benefits for current air quality policies, due to the progress in reducing SO₂ concentrations (the main pollutant of concern). A number of policies, notably targeting the industrial, domestic and marine sectors, can however lead to reductions in SO₂. The benefits in reducing material damages have been quantified using previous analysis as part of the Air Quality Evaluation.

¹⁸ ICP Materials (2003) Dose-response functions. http://www.corr-institute.se/ICP-Materials/html/dose_response.html

¹⁹ This would mean that the equivalent annual cost is the same for all time-scales, i.e. for the 1, 5, 20 and 100 year analysis, however, the 100 year analysis does give a different equivalent annual cost, because of the use of time-declining discount rates.

2.8.2 Materials Damage - Ozone

2.8.5. Although ozone is a major determinant of the lifetime of many rubber materials exposed to the ambient air, only two UK studies have investigated the problem from an environmental perspective. Lee et al (1996) estimated annual damages to the UK of £170 to £345 million for impacts on surface coatings (paints) and elastomers and the cost of anti-ozonant protection used in rubber goods. These estimates were based on US data from the late 1960s, demonstrating the dearth of information in this area. Lee's work served as a scoping study for a larger project (Holland et al, 1998) that undertook experimental assessments of a range of paints, representative of those in use in the UK market, and rubber formulations.

2.8.6. The analysis on paint found it unlikely that there would be significant ozone-induced damage during the expected service lifetime of the paint, though the possible effects of interactions of ozone with other environmental stresses in damaging paints were not addressed. In contrast, damage to rubber goods from ozone exposure in the UK was estimated at between £35 to 189 million, with a best estimate of £85 million/year. The effect of a population weighted 1 ppb change in ozone was estimated at £3.7 million/year. This estimate has been used to make an approximate estimate of ozone damage to rubber product, using the ozone model output and this relationship directly. Note no uplift is applied to non-health values in future years.

2.8.3 Building Soiling

2.8.7. Soiling of buildings by particles is one of the most obvious signs of pollution in urban areas. The soiling of buildings includes both "utilitarian" and historic buildings and causes economic damages through cleaning and amenity costs. Soiling is an optical effect (a darkening of reflectance) and results primarily from the deposition of airborne particulate matter to external building surfaces. The factors which can affect the degree of soiling are well known and include: the blackness per unit mass of smoke; the particle size distribution; the chemical nature of the particles; substrate-particle interfacial binding; surface orientation; and micro-meteorological conditions.

2.8.8. Different types of particulate emission have different soiling characteristics. For example, diesel emissions have a much higher soiling factor relative to petrol or domestic coal emissions factors due to their particulate elemental carbon (PEC) content (QUARG, 1993). Diesel emissions are the main source of atmospheric PEC in Western Europe. Secondary particulates are not considered to be involved in soiling – the effect is in relation to primary particulate emissions only.

2.8.9. Although soiling damage has an obvious cause and effect, the quantification of soiling damage is not straightforward. For the analysis, a number of different approaches and functions have been considered. A simplified approach is often used that quantifies soiling damage based on cleaning costs (in the absence of WTP data). Rabl et al (1998) extended this to quantify total soiling costs (i.e. the sum of cleaning cost and amenity loss), and Rabl's work has been used as the basis for quantification of soiling damage. Note no uplift is applied to non-health values in future years.

2.8.4 Acid damage to cultural heritage

2.8.10. The same approach that is used for modern buildings could, in theory, be applied to cultural and historic buildings. However, in practice there is a lack of data at several points in the impact pathway with respect to the stock at risk and valuation. As a result, effects of air pollution on cultural heritage cannot be quantified and thus need to be considered qualitatively.

2.8.11. Nevertheless, valuation studies of cultural heritage that show that people place a significant economic value on cultural heritage (see the review by Navrud and Ready, 2002). These data could be used in an extended framework to illustrate the potential significance of damage to cultural heritage, but it is not possible to include in the simple damage cost values.

2.9 Crop Damages

2.9.1. Ozone is recognised as the most serious regional air pollution problem for the agricultural and horticulture sectors. The analysis in the air quality review has directly quantified the changes in crop yields in the UK and valued these using international crop prices. The quantification needs the following data:

1. Information on stock at risk, in terms of the distribution of crop production, by species, across Europe.
2. Exposure-response functions for different crops, recognising the variability in response between species.
3. Valuation data.

2.9.2. The approach adopted for the analysis of methods in this area has been informed particularly by the Integrated Cooperative Programme (ICP) on Vegetation, and ICP/MM (Mapping and Modelling)²⁰. The approach has linked changes in ozone concentrations with data on the stock at risk²¹, and exposure-response functions for assessment of crop impacts from ozone²².

2.9.3. The valuation of impacts on agricultural production is reasonably straightforward, with estimated yield loss being multiplied by world market prices as published by the UN's Food and Agriculture Organization. World market prices are used as a proxy for shadow price on the grounds that they are less influenced by subsidies than local European prices (in other words, they are closer to the 'real' price of production).

2.9.4. The analysis of crop damages included here is based on the use of AOT40 relationships, combined with OSRM estimates of change in AOT40. Functions and other data

²⁰ ICP/MM (2004) Mapping Manual Revision. United Nations Economic Commission for Europe, ICP Mapping and Modelling. <http://www.oekodata.com/icpmapping/html/manual.html>

²¹ The stock at risk database has been developed by the Stockholm Environment Institute (SEI) in York and used in past analysis for ICP Vegetation.

²² Exposure-response functions for assessment of crop impacts from ozone take two forms. The first, sometimes called a Level I approach, relates yield change to ozone concentration, typically expressed as AOT40, the accumulated exposure to ozone in excess of 40 ppb during the growing season, measured in units of ppb.days. The second type of relationship, sometimes referred to as a Level II approach, seeks to equate yield change not simply to concentration, but to pollutant uptake, by accounting for crop development and climatic conditions. Quantification based on a Level II approach will be possible only later in 2005, drawing outputs of DEFRA's ICP Vegetation Contract held by CEH Bangor.

are shown below. Valuation data are based on world market prices as reported by FAO. Note no uplift is applied to non-health values in future years.

Table 6. Functions and associated factors for quantification of ozone damage to crop production.

The height factor accounts for variation in ozone concentration with height and is based on default estimates in the ICP Mapping and Modelling Manual (2004). The function shows proportional change in yield per ppm.hour.

Crop	Value (€)	Function	Height (m)	Height factor
Barley	120	0	1	0.88
Fruit	680	0.001	2	0.93
Grapes	360	0.003	1	0.88
Hops	4100	0.009	4	0.96
Maize	100	0.004	2	0.93
Millet	90	0.004	1	0.88
Oats	110	0	1	0.88
Olives	530	0	2	0.93
Potatoes	250	0.006	1	0.88
Pulses	320	0.017	1	0.88
Rapeseed	240	0.006	1	0.88
Rice	280	0.004	1	0.88
Rye	80	0	1	0.88
Seed cotton	1350	0.016	1	0.88
Soybeans	230	0.012	1	0.88
Sugar beets	60	0.006	0.5	0.81
Sunflower seed	240	0.012	2	0.93
Tobacco leaves	4000	0.005	0.5	0.81
Wheat	120	0.017	1	0.88

2.9.5. Some air pollutants other than ozone have been linked in the literature to crop damage (e.g. SO₂, NO₂, NH₃), but generally at higher levels than are currently experienced in the UK—and we have assumed that direct impacts of these other pollutants on agriculture are likely to be small. Note however that these pollutant may have indirect effects, for example by stimulating the performance of insects and other agricultural pests, enabling them to impact more severely on crop yield than in the absence of air pollution.

2.10 Uncertainty

2.10.1. There are a number of major uncertainties. These can be considered through

- Bias analysis: the purpose of the bias analysis is to highlight those issues that are considered likely to cause a systematic bias in the results.
- Statistical analysis: statistical analysis is used to provide a benchmark for uncertainty assessment.
- Sensitivity analysis: it is important to recognise that a variety of uncertainties cannot be described using standard statistical techniques.

2.10.2. The omission of impacts (see earlier section), particularly for ecosystems, is a major uncertainty but has to be seen in the context of the full range of uncertainties in the assessment. Whilst it does clearly bias to underestimation, the full set of uncertainties, including also model assumptions and statistical uncertainties, may push the results either way, up or down.

2.10.3. A number of key uncertainties are highlighted.

- Omission of ecosystem effects (acidification, eutrophication and ozone exposure).
- Omission of non-UK benefits (i.e. trans-boundary pollution to Europe);
- The health impacts of PM₁₀ – in relation to the different fractions of the pollutant mix (see box 4).
- Chronic mortality quantification (risk rate, lag phase and PM metric).
- Chronic mortality effects of SO₂ (*does it have direct effects??*)
- The valuation of mortality.
- The potential range of impacts considered for morbidity.
- Quantification of ozone effects on health with and without a ‘cut-point’ (effectively, the assumption of a threshold at 0, 35 or 50 ppb) and the assumption on the period of life lost.
- Whether NO₂ is acting as a marker for particles or whether it has its own direct effects.
- Interactions between NO and ozone in urban areas.

Box 4. Particles

Ambient PM₁₀ is a mixture of primary and secondary particulates. Emissions lead to both primary and secondary PM₁₀. As part of the analysis here, we have separated out the health benefits of primary and secondary particulates. It is stressed that there is different confidence in different fractions of the PM₁₀ mixture.

The evidence for primary PM₁₀ emissions is highest. Toxicological studies have highlighted that primary, combustion-derived particles have a high toxic potency

The evidence for the health benefits associated with reductions in sulphates is fairly strong for two reasons. Firstly, sulphates are within the PM₁₀ size fraction and, although it is widely believed that different constituents of PM₁₀ are associated with different toxicities to human health, it is rare that quantification seeks to take this into account. Thus, COMEAP does quantify secondary particulates in the same way as primary PM₁₀ (because it applies the PM₁₀ health concentration-response functions to all of the PM₁₀ size fraction). Secondly, several epidemiological studies in the USA and Canada have shown direct relationships between sulphates and various acute and chronic health endpoints, including mortality (reduced life expectancy) from long-term exposures (chronic mortality). However, it does not necessarily follow that there is a causal relationship – it may be that sulphates are a marker (surrogate) for other aspects of the pollution mixture (and that these other parts of the pollutant mixture also change at the same time as reductions in sulphates).

The evidence for secondary particulates as nitrates is less robust. The issues with quantification are similar to sulphates, though there are also some significant differences. Nitrates are part of the PM₁₀ size fraction, and it would be reasonable (applying a precautionary approach) to treat them as associated with similar PM₁₀ related effects as for sulphates above. As noted earlier, COMEAP in its quantification work does not differentiate between the various components of PM₁₀ (nor does the US EPA, other than distinguishing PM_{2.5}). However, unlike sulphates, there is almost no direct evidence linking nitrates to health effects, partly because it is difficult to measure nitrates reliably on the scale necessary for modern epidemiology, and most analysts remain cautious about the causality of nitrates as part of the PM₁₀ size fraction.

Related to this, any change in the health impacts attributed to different parts of the PM₁₀ mixture will have a major influence on the pattern of benefits from existing policies (and also future policy). For example, a sensitivity analysis in the Air Quality Evaluation showed that if greater emphasis is given to primary PM_{2.5}, and less to nitrates, this could dramatically affect overall benefits of different policies.

3 Application of the Analysis

3.1 Example of the application of the methodology

3.1.1. The damage costs are derived from comprehensive modelling analysis, using the impact-pathway approach. They are derived from runs that aim to estimate the marginal benefits of emission changes.

3.1.2. The starting point for the analysis has been the assessment of the baseline conditions, in 2010, consistent with the Air Quality Review. This uses the baseline NAEI emissions, and baseline air quality maps. The impacts of the baseline are quantified and valued, using the approach set out above.

3.1.3. The analysis then looks at marginal emissions reductions, by reducing the emissions individually by 10% in each sector, or by a suitable marginal quantity (e.g. 50,000 tonnes). The impact pathway analysis is re-estimated (changes in emissions, changes in air pollution concentrations, changes in impacts, changes in values). The marginal change in values is then normalised against the change in emissions (in tonnes) to produce a damage cost. At present we assume that the model response to different marginal changes will be linear (i.e. for smaller or larger changes than 10%). This approximation is appropriate for primary PM and the secondary PM analysis (where the model does effectively behave linearly²³). It is not as good for ozone.

3.1.4. An example of the process is shown below for one health endpoint for one pollutant – respiratory hospital admissions from secondary PM₁₀ formed from SO₂ emissions.

The marginal change in tonnes of emissions (50,000 tonnes) of SO₂ is taken, and then run in the pollution-mapping model to estimate the marginal changes in PM₁₀ concentrations across the UK. This is combined with population data to estimate the population weighted mean concentration change (which leads to a population weighted mean concentration change of 0.112 µg/m³ PM₁₀ relative to the baseline map).

This is combined with the concentration-response function (e.g. for respiratory hospital admissions 0.8 % per 10 µg m⁻³ change) and the baseline rate (980 per 100,000) to estimate the additional number of cases (51 cases per year).

The next step is to value this effect. For a 2005 emission, in 2005 prices, the valuation for a RHA is applied (at £1900 to £9100 per case) to give a total (£97,000 to £466,000). This is then divided by the initial tonnes of emissions (50000 tonnes) to give the cost per tonne (£2 to £9 per tonne SO₂). However, different values must be applied for different time-scales and different years, to take the 2% uplift in health values, and the 3.5% discount rate.

For the damage cost values here, the numbers are presented for a 2010 emission expressed in 2005 prices (i.e. with the value of a RHA increased at 2% to give a 2010 value, then discounted back to 2005). The values are presented both as a net present

²³ though note that the approach assumes that secondary particulates reduce across the UK in the same proportions – in practice, there will be different reductions patterns according to the exact spatial pattern of emission changes.

value and an annualised value (the latter assuming the equivalent annual cost starting in 2005). The same type of analysis has been repeated for the 5, 20 and 100 year analysis – in each case we assume that the same level of emissions leads to the same level of population weighted exposure and the same number of impacts in each year (for example, in the five year analysis, we assume that there are 51 RHA each year from 2010 to 2015). Note for the 100 year analysis, a declining discount rate is used, consistent with Green Book guidance.

This process is repeated for all health and non-health impacts, and aggregated to give an overall cost per tonne, though the 2% uplift is only applied to health effects (not to non-health effects).

A more complex approach has been used for the damage costs for chronic mortality (as years of life lost). This has been based on an underlying life table analysis, which has looked at the effects of a 1, 5, 20 and 100 year analysis, with and without lag phases, starting in 2010 for a 1% risk rate. The important difference here is that the analysis includes the pattern of impacts in future years over the 100 year time period – even for the annual pulse (1 year run). This reflects the fact that a change in the mortality rate in year 1 feeds through to the risk rates in subsequent years – and the health impacts in later years must be uplifted and discounted.

3.1.5. The table below shows the underlying data for this analysis, along with the effects of Cardio-vascular hospital admissions. The endpoints are added together to give a total damage cost.

Table 7. Example of the damage cost calculation.

Emissions (t)		SO₂
Population weighted concentration (TEOM)		50000
		0.112076923
Respiratory Hospital Admission (PM)		
Baseline rate per 100,000	979.7	
Risk rate % per µg	0.08	
Population	58279137.63	
Impacts		51.2
Impacts per tonne		0.0010
Valuation (low)	1900	97,267
Valuation (high)	9100	465,858
Valuation per tonne low (£)		1.9
Valuation per tonne high (£)		9.3
Cardio-Vascular Hospital Admission (PM)		
Baseline rate per 100,000	981.4	
Risk rate % per µg	0.08	
Population	58279137.63	
Impacts		51.3
Impacts per tonne		0.0010
Valuation (low)	1900	97,436
Valuation (high)	9100	466,667
Valuation per tonne low (£)		1.9
Valuation per tonne high (£)		9.3
Plus		
Years of Life Lost (PM) (GRAV)		
SO₂ and DBF as a gas		
SO₂ and RHA as a gas		

Impacts are calculated using baseline rate (/100000) multiplied by risk rate (%) by population

3.1.6. Of course, each pollutant has a different combination of primary / secondary pollutants and endpoints that need to be added together. These are summarised below.

3.1.7. The impacts are summed for each impact. Note that because there are three risk rates for chronic mortality, each with a zero and 40 year lag, there are six potential damage costs.

3.1.8. The damage costs for SO₂ as sulphates (PM), NO_x as nitrate, and VOC and NO_x as ozone have been presented a single values irrespective of sector or location. As these are secondary (regional) pollutants, this approximation is acceptable – though in practice there are strong non-linearities for ozone.

Table 8. Summary of effects included in the damage cost calculation for each pollutant

Impact	Primary PM	SO ₂	NO _x	VOCs
Chronic mortality (YOLL) from PM	✓	✓	✓	
Respiratory Hospital Admissions from PM	✓	✓	✓	
Cardio-vascular Hospital Admissions from PM	✓	✓	✓	
Deaths brought forward from SO ₂		✓		
Respiratory Hospital Admissions from SO ₂		✓		
Deaths brought forward from ozone				
0 ppb cut point (no threshold)			✓	✓
35 ppb cut point			✓	✓
50 ppb cut point			✓	✓
Respiratory Hospital Admissions from ozone				
0 ppb cut point (no threshold)			✓	✓
35 ppb cut point			✓	✓
50 ppb cut point			✓	✓
Building soiling	✓			
Building corrosion		✓		
Material damage (ozone)			✓	✓
Crop damage			✓	✓

3.1.9. For primary PM, there are much greater differences between sectoral emissions, because of the population-weighted increases from different emission sources. The analysis has therefore used the following sector split, i.e. specific damage costs are produced for:

- ESI;
- Domestic;
- Agriculture;
- Waste;
- Road Transport (average UK, plus a more disaggregated split by National Transport Model area);
 - Central London
 - Inner London
 - Outer London
 - Inner Connurbation
 - Outer Connurbation
 - Urban Big
 - Urban Large
 - Urban Medium
 - Urban Small
 - Rural

3.1.10. The table of damage costs for a selection of pollutants and impacts is presented in Appendix 1. Full results will be presented as part of a guidance note on the application of damage costs, to be prepared by the IGCB.

3.2 Caveats with the Numbers

3.2.1. The damage costs are based on a number of assumptions. These should be noted, along with a number of caveats, in any application of the values. These are:

- External costs of air pollution vary according to a variety of environmental factors, including overall levels of pollution, geographic location of emission sources, height of emission source, local and regional population density, meteorology and so on. These numbers take these issues into account to a certain degree only.
- Related to this, the numbers produced are based on national level analysis (and national averages). They are therefore potentially more relevant for national policies than specific local analysis²⁴.
- Values for NO_x and SO₂ include secondary particulate (PM₁₀) formation (nitrates and sulphates);
- Values for VOC and NO_x include ozone formation and effects;
- With the exception of ozone, all impacts are assumed to have no threshold of effects, and have been implemented using linear concentration-response functions;
- The relationship between emissions and concentrations is linear. This is an extremely simplistic assumption with respect to ozone formation. The analysis does not separate between urban and rural area in relation to ozone precursor emissions, nor fully take account of interactions between NO and ozone in urban areas.
- Future life years lost have been uplifted (at 2%) and discounted using a 3.5% discount rate, or for the 100 year analysis the recommended Green Book declining discount rates.
- The year of life lost analysis uses PM_{2.5} functions and applies these to changes in PM₁₀ pollution;
- PM mortality from acute (sensitivity) and chronic exposure to PM are not added.

3.2.2. It is also stressed that the values exclude a number of important effects.

- The numbers only include costs that occur in the UK - all trans-boundary pollution and impacts are excluded;
- The numbers exclude effects on ecosystems (acidification, eutrophication, etc);
- The numbers exclude effects on cultural or historic buildings from air pollution;
- The potential effects of VOC emissions on inorganic particle concentrations;
- Potential additional morbidity from acute exposure to PM and ozone;
- Potential mortality effects in children from acute exposure to PM;
- Potential morbidity effects from chronic (long-term) exposure to PM;
- Potential mortality and morbidity effects from chronic (long-term) exposure to ozone;
- Visibility (visual range);
- Non-ozone effects on agriculture.

²⁴ The most important variable in the applicability of the numbers is population density. Large local reductions in primary emissions in areas with little population will lead to smaller reductions in population-weighted means (and health benefits). Large local reductions in primary emissions in areas with very high population will lead to larger reductions in population-weighted mean (benefits).

3.3 Research Recommendations

3.3.1. A number of areas are identified for further improvement and research. These include:

- To develop data-sets for other years (2015, 2020);
- To provide dis-aggregated ozone damage costs for VOC and NO_x that differentiate between sector and area, and recognise the strong non-linearities in ozone formation;
- To include uncertainty analysis in the values, to allow the application, for example, of probabilistic damage costs based on monte-carlo analysis;
- To extend the analysis to cover other air pollutants, notably ammonia, heavy metals, and organics (e.g. PAHs);
- To update the crop damage and building materials models.
- To make further investigation of excluded effects, particularly additional possible health effects, ecosystems, cultural heritage, VOC aerosols, to consider how these might change the damage cost estimates here (in sensitivity analysis).

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Appendix 1: Extended Example of Damage Cost Calculations (2010 emissions, discounted back to 2005, presented in 2005 prices).

Pollutants	Primary PM (transport)	SO ₂	NO _x	VOCs
Emissions (tonnes)				
For PM calculation	26000	50000	50000	
For ozone calculation			50000	70000
Pollution (population weighted)				
Population weighted PM (GRAV)	1.4646	0.1457	0.0983	
Population weighted PM (TEOM)	1.1266	0.1121	0.07562	
Population weighted ozone (0 ppb)			-0.5664	0.0233
Population weighted ozone (35 ppb)			-0.2533	0.0165
Population weighted ozone (50 ppb)			-0.0734	0.0065
Chronic mortality (YOLL) from PM				
Impacts (YOLL) annual pulse (6% RR no lag)	60941	6062	4090	
Impacts (YOLL) per tonne annual pulse (6% RR no lag)	2.1	0.12	0.08	
Values annual pulse (6% RR no lag) £	1470 million	143 million	99 million	
Values per tonne annual pulse (6% RR no lag) £	49686	2926	1974	
Respiratory Hospital Admissions from PM				
Impacts	514	51.2	34.5	
Impacts per tonne	0.017	0.0010	0.0007	
Values (high - £9100) £	4462087	443891	299482	
Value per tonne (high - £9100) £	151	8.9	6.0	
Cardio-vascular Hospital Admissions from PM				
Impacts	515	51.2	34.6	
Impacts per tonne	0.017	0.0010	0.0007	
Values (high - £9200) £	4518949	449548	303298	
Value per tonne (high - £9200) £	153	9.0	6.1	
Deaths brought forward from ozone (0 ppb)				
Impacts			-196.0	8.05
Impacts per tonne			-0.00392	0.00012
Values (high - £7500) (annual pulse 2010) £			-341073	57548
Value per tonne (high - £7500) (annual pulse 2010) £			-28	0.8
Respiratory Hospital Admissions from ozone (0 ppb)				
Impacts			-226.4	9.300
Impacts per tonne			-0.00453	0.00013
Values (high - £9100) (annual pulse 2010) £			-1962888	80639
Value per tonne (high - £9100) (annual pulse 2010) £			-39	1.2
TOTAL HEALTH DAMAGE COSTS (HIGH) £				
Total health effects per tonne (£) High	49990	2944	1919	2

note excludes SO₂ as a gas (health) and non-health effects