Air Quality damage cost update 2019

Report for Defra

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Executive summary

Air pollution can have damaging impacts on human health, productivity, amenity and the health of the environment. These detrimental impacts have associated economic and/or social costs (known as external costs or externalities) that are not captured in the market price of the goods or services consumed that produce the pollution. Cost-benefit analysis (CBA) is a tool commonly used to appraise the relative merits of different policy options. CBA attempts to value and contrast all costs and benefits associated with a given policy option, including any external costs that are not captured by market prices, to inform a comprehensive comparison across different options to achieve a policy objective.

The UK Department of Environment, Food and Rural Affairs (Defra) has produced guidance (Defra, 2013a) to steer the assessment of air quality impacts and the valuation of external costs such that these can be captured in policy appraisal. Defra's guidance details three approaches to assessing and valuing the impacts: one is the 'damage cost' approach, which is to be taken where impacts are valued to be less than £50m and when compliance with legally binding objectives is expected

Damage costs are a set of impact values defined per tonne of emission. These values estimate the external costs associated with a marginal change in pollutant emissions. They can be combined with forecasts of emission changes to provide an approximate valuation of the aggregate external impacts of a policy.

Ricardo Energy & Environment was commissioned by Defra to update the damage costs of air pollution. The objective of the project was to provide a focussed revision of specific elements of the damage cost methodology. These revisions identified developments in the underpinning evidence base that were considered most likely to have the most important impacts on the cost estimates. The review included:

- Update emissions-to-concentration modelling to 2014[1]
- Produce new NOx damage costs split by sectors
- Explore and produce a more disaggregated split of the industry PM and NOx damage costs for 'Part A' installations
- Update estimation of mortality effects of chronic exposure to NO_2 to reflect updated COMEAP recommendations (COMEAP, 2018a)
- Include new impact pathways: chronic exposure to PM_{10} on chronic bronchitis, impacts of exposure to ozone, impact pathways included in PHE's Estimation of costs to the NHS and social care due to the health impacts of air pollution (PHE, 2018) and impacts on productivity
- Reproduce chronic health impacts using life-table calculations
- Update baseline data for health impacts and population
- Include estimation of impacts on air pollution on ecosystems (including undertaking dispersion modelling to produce required concentration metrics).

The updated set of damage costs is presented in Table E1 below, alongside the low and high estimated sensitivities around the central values. Note that for these revised damage costs the change in $PM_{2.5}$ emission is the preferred metric for PM emissions. This table shows the national average damage costs. Sector specific damage costs have also been updated and are also presented in this report. A positive damage cost represents a cost associated with an increase in pollutant emissions or a benefit associated with a decrease in pollutants emissions.

Splitting the results by their contributing pathways, the effects of long-term exposure on mortality rates continues to be the dominant impact captured in the damage costs. This effect is captured alongside the effects of air pollution on mortality and hospital admissions (associated with acute exposure), chronic

[1] 2014 emissions-to-concentration modelling was used given this was the most recent modelling available at the time the update to the damage costs commenced.

heart disease, stroke, lung cancer, asthma in children, productivity, ecosystems, material damage and building soiling in the revised costs. The high damage cost includes a range of further health impacts (including chronic bronchitis) around which there is deemed greater uncertainty.

For the NOx damage cost, chronic exposure to PM on mortality is still an important effect (in this case PM is a 'secondary' pollutant), but the mortality effect of chronic exposure to NO_2 is the most significant pathway. This is the case even though the adjustment to account for the overlap between the two chronic effects has been applied to the NO_2 impacts, rather than the PM effects.

Another key impact pathway (for all damage costs) is the new PHE effect estimating impacts on asthma in children. Most other pathways make relatively small contributions.

Under the high damage cost, chronic mortality effects and asthma in children are important, but asthma in adults and chronic bronchitis pathways added under this sensitivity are also key contributors.

Each of the updated damage costs shows some variation relative to the set of damage costs published in 2015 (Defra, 2015). For NOx, the sector-specific damage costs have reduced relative to the latest published set of costs. For PM, damage costs have increased significantly relative to the 2015 set. The changes in each damage cost will reflect the different updates to the methodology underpinning the estimation set out in this report. It is not possible to fully disentangle which changes in the underlying damage cost methodology have contributed to the change in the different damage costs and to what extent. However, key factors in the changes will be:

- For the NOx damage costs:
    - improvements and updates to the underlying pollution modelling relative to the 2015 data costs, specifically: the use of specific dispersion modelling for NOx, accounting for NOx to NO_2 chemistry and using updated relationships between NOx emissions and PM concentrations.
    - Updates to method for estimating chronic mortality effects following updated guidance from COMEAP, in particular applying the adjustment to the co-efficient to account for overlaps with PM
- For all damage costs: the addition of new pathways, in particular
    - Productivity impacts, namely impacts on work-days-lost (WDL) have increased the PM damage cost
    - Ammonia's impact on CO_2 sequestration (captured under the ecosystem impacts) has reduced the ammonia damage cost significantly
    - The addition of asthma in children has significantly increased all damage costs, and under the high damage cost, asthma in adults and chronic bronchitis have had an even greater inflationary effect on the NOx and PM damage costs respectively.

Although the damage costs have been revised to reflect specific improvements in the underlying evidence base, the guidance regarding their use is still appropriate. The damage costs should only be used in appraisal where the cumulative monetised impacts sum to less than £50m or where the impacts are ancillary. This is to reflect the implicit assumptions made when applying the damage costs: in particular, that patterns of pollutant emission and exposure and baseline population and rates of health incidence could change over time and inherently represent an averaging of effects across the country as a whole or specific sector defined by the damage cost applied.

Further, users of the damage costs should note the wider caveats around their use, in particular regarding the uncertainty associated with their estimation and the coverage of impacts included and are encouraged to refer to the wider Defra guidance and original damage cost documentation (AEA Technology, 2006) for further information.

Although the scope of the impacts has been expanded as part of this update, it is important to note that the damage costs still only capture a sample of the range of impacts associated with air pollution, and some remain unaccounted for in the damage costs, including:

- The geographic scope of the analysis has not been revised for the updated damage costs. The updated damage costs only account for impacts of UK emissions on the UK and not on other countries.
- Some of the impact pathways included in PHE's 'Estimation of costs to the NHS and social care due to the health impacts of air pollution' have been included. However, some impact pathways identified in this work have not been included due to lower confidence around the supporting evidence base
- Some ecosystem impact pathways have been included based on the work of Jones et al. (2014) – those ranked as 'robust' and 'acceptable'. However, pathways assessed as 'improvements desirable' have not been included.
- The damage costs for VOC include impacts via the O_3 pathways only.

Table E1. Revised national average damage cost estimates and sensitivity bounds (2017 prices, impacts discounted to 2017). $PM_{2.5}$ is the preferred metric for the change in PM emissions

Pollutant Emitted	Central Damage Cost (£/t)	Low – High damage cost sensitivity range (£/t)	
		Low sensitivity damage cost	High sensitivity damage cost
NOx*	6,199	634	23,153
SO_2	6,273	1,491	17,861
NH_3	6,046	1,133	18,867
VOC	102	55	205
$PM_{2.5}$	105,836	22,588	327,928

*Note: NOx damage cost uses an adjusted coefficient for the impacts of NO_2 to account for overlap of effects with other pollutants, as advised by COMEAP and recommended by IGCB.

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Appendices

1 Introduction

1.1 Air quality and impact valuation

The quality of the air around us has a strong influence on both natural and man-made environments. Air pollution can have damaging impacts on human health, productivity, amenity and the health of the environment. These detrimental impacts have an associated economic or social cost (known as external costs or externalities) that are not captured in the market price of the goods or services consumed that produce the air pollution.

Cost-benefit analysis (CBA) is a tool commonly used to appraise options in Impact Assessment (IA) to support policy development. CBA attempts to value all the costs and benefits associated with a given policy option, including any external costs that are not captured by market prices. The UK Department of Environment, Food and Rural Affairs (Defra) has produced guidance (Defra, 2013a) to steer the assessment of air quality impacts and the valuation of associated external economic and social costs, based on the work of the Defra-led Interdepartmental Group on Costs and Benefits (IGCB). This guidance supplements the Green Book (HMT, 2018) which provides wider guidance for IA and valuation. Defra are currently in the process of updating their Green Book supplementary guidance. These processes are designed to support evidence gathering to inform policy development and evaluation.

Defra's air quality appraisal guidance details three approaches to assessing and valuing the impacts of policy on air quality. It recommends analysts follow the 'damage cost' approach where impacts are valued to be less than £50m and the more rigorous 'impact-pathway' approach (IPA) where impacts are more significant. Where changes in emissions could impact on compliance with legally binding obligations, an 'abatement cost' approach is advised to reflect potential additional policy costs required to ensure obligations are met.

1.2 Damage costs of air pollution

Where possible, IGCB recommend that the Impact Pathway Approach (IPA) should be used to appraise the external impacts of policies, projects or programmes on air pollution. The IPA charts a logical progression from a change in pollutant emissions, through to monetised impact. This is a more detailed modelling approach, which utilises specific information regarding the policy and its impacts on air pollution to produce a more rigorous estimate of the likely effects. The approach was advanced through a series of EC DG Research projects known as ExternE (2005) and was also extensively used previously in analysis of impacts at the UK and EU level.

However, the IPA is relatively resource intensive and may not be a proportionate approach in all policy appraisals. This is particularly the case where air pollutant impacts are ancillary to the central effects of the policy. As such, Defra commissioned AEA-Technology (2006) to develop a set of 'air pollution damage costs'.

Damage costs are representative estimates of the external costs associated with a marginal change in pollutant emissions. The costs are expressed per tonne of pollutant emission. They can be readily combined with forecasted changes in emissions to provide an approximation of the aggregate external costs. Damage costs represent the impacts of an average unit of emission in the UK. As such they necessarily imply a simplified approach relative to undertaking an assessment using the full IPA. A more rigorous assessment using the IPA would take into account all specific information regarding the nature and location of the specific change in pollutant emission. Hence it is recommended that the damage costs are only used for narrowing down a long-list of policy options (before undertaking more detailed assessment) or for policy appraisal where either the air pollution impacts are secondary, or the total level of impacts is valued to be less than £50m.

The initial set of damage costs for appraisal in the UK were estimated in 2006 by following the IPA for a range of impact pathways to capture the effects of an average emission in the UK. Since this initial

set was produced, several updates have been made to the damage costs. For example, slight amendments to the methodology underpinning the estimation of the damage costs were subsequently noted in Defra's Air Quality Strategy (or AQS; Defra, 2007), and a further updated set were published in 2011 (Defra, 2011a).

The most recent update to the damage costs was published by Defra in 2015 (Defra, 2015a). The key element of this update was to reflect recent developments in the underlying evidence base concerning the effects of chronic exposure to concentrations of nitrogen dioxide (NO_2) on mortality.

The present update to the damage costs refines the calculation of these effects based on the recently published 'Associations of long-term average concentrations of nitrogen dioxide with mortality' (COMEAP, 2018a). This report includes refined recommendations for quantifying mortality effects on the basis of long-term average concentrations of nitrogen dioxide (NO_2) from the UK Committee on the Medical Effects of Air Pollutants (COMEAP).

Several versions of the damage costs are referred to in this report. For clarity:

- The first set of damage costs produced in 2006 are referred to as 'original damage costs'
- The most recent set of damage costs published by Defra are referred to as the '2015 damage costs'
- The updated damage costs produced as part of the present study are referred to as 'updated damage costs'.

1.3 Project objectives and approach

Ricardo-Energy & Environment were commissioned by Defra to update the damage costs of air pollution. The key objective of the study was to update the published damage costs to reflect improvements to air pollution modelling and developments in the underlying evidence base.

The project did not intend to fully review every aspect of the damage cost methodology and provide a comprehensive re-assessment of the costs. The focus of this contract was instead to revise specific areas of the methodology where developments in the underpinning evidence base were considered to potentially have the most important impacts on the cost estimates. Activities included:

- Update emissions-to-concentration modelling to 2014[2]
- Produce new NOx damage costs split by sector
- Explore and produce a more disaggregated split of the industry PM and NOx damage costs for 'Part A' installations
- Update estimation of mortality effects of chronic exposure to NO_2 to reflect updated COMEAP guidance
- Include new impact pathways: chronic exposure to PM_{10} on chronic bronchitis, impacts of exposure to ozone, impact pathways included in PHE's Estimation of costs to the NHS and social care due to the health impacts of air pollution (PHE, 2018) and impacts on productivity
- Re-produce chronic health impacts using life-table calculations
- Update baseline data for health impacts and population
- Include estimation of impacts on air pollution on ecosystems (including undertaking dispersion work to produce required concentration metrics).

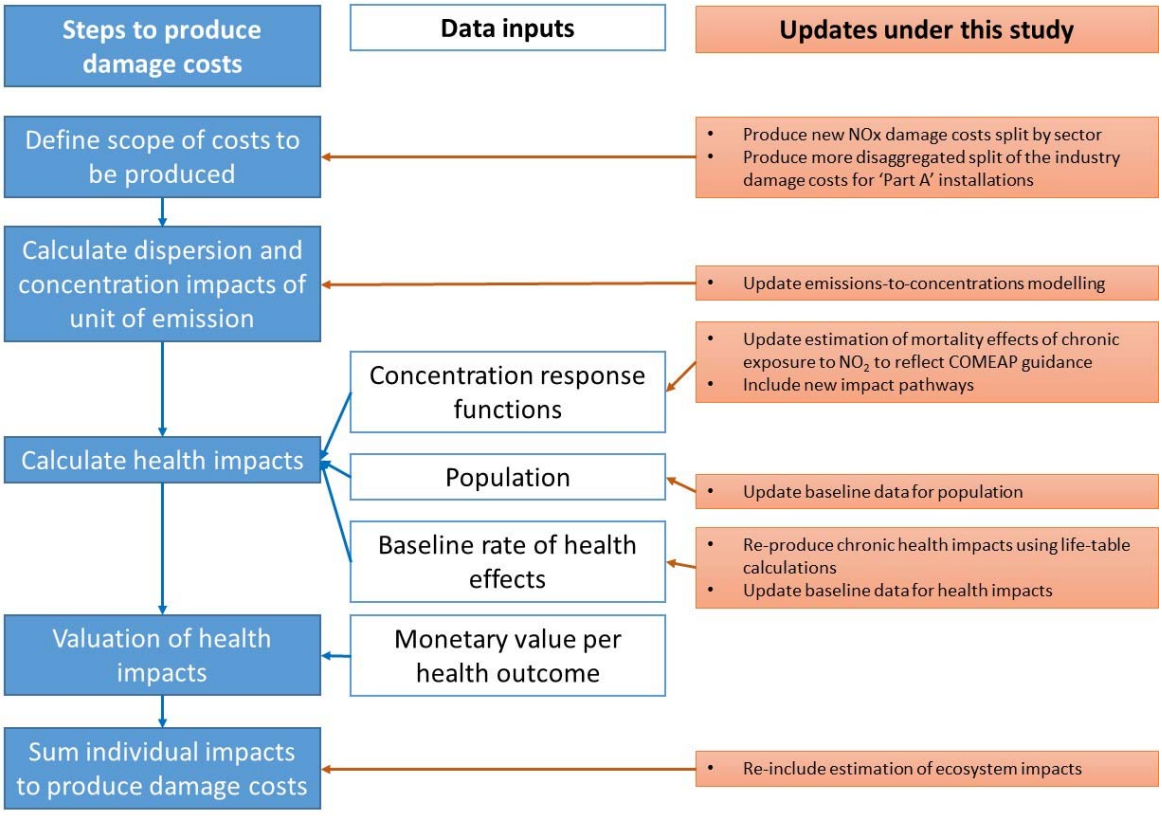
[2] 2014 emissions-to-concentration modelling was used given this was the most recent modelling available at the time the update to the damage costs commenced.

Although the scope of the impacts has been expanded as part of this update, it is important to note that the damage costs still only capture a sample of the range of impacts associated with air pollution, and some remain unaccounted for in the damage costs, including:

- The geographic scope of the analysis has not been revised for the updated damage costs. The updated damage costs only account for impacts of UK emissions on the UK and not on other countries.
- Some of the impact pathways included in PHE's 'Estimation of costs to the NHS and social care due to the health impacts of air pollution' have been included. However, some impact pathways identified in this work have not been included due to lower confidence around the supporting evidence base
- Some ecosystem impact pathways have been included based on the work of Jones et al. (2014) – those ranked as 'robust' and 'acceptable'. However, pathways assessed as 'improvements desirable' have not been included.
- The damage costs for VOC include impacts via the O_3 pathways only.

A simple flow diagram illustrating the process for developing the damage costs (i.e. following the IPA for an average unit of UK emission) and the changes implemented in this update are illustrated in Figure 1.

Figure 1 – Simple flow diagram illustrating development of damage costs and updates



This report is structured as follows:

- Section 2 sets out in greater detail the changes made to the air pollutant modelling underpinning the damage costs
- Section 3 sets out wider changes and updates to the damage cost methodology
- Section 4 outlines the changes made to damage cost sensitivities
- Section 5 presents the final set of updated damage costs.

2 Updates to air pollutant dispersion modelling

2.1 Introduction

The emissions to concentrations air quality modelling has been fully updated for the revised damage cost calculations. The following models have been used and these models are discussed below, including references to full descriptions.

- Relationship between changes in primary $PM_{2.5}$ emissions and $PM_{2.5}$ ambient concentrations for total emissions and for individual emission sectors (Pollution Climate Mapping (PCM) model)
- Relationship between changes in NO_x emissions and NO_2 ambient concentrations for total emissions and for individual emission sectors (PCM model)
- Relationship between changes in SO_2 emissions and SO_2 ambient concentrations for total emissions (PCM model)
- Relationship between changes in SO_2, NO_x and NH_3 concentrations and ambient concentrations of secondary inorganic aerosol (a component of ambient PM_{10} and $PM_{2.5}$) (PCM model emission sensitivity coefficients method)
- Relationship between changes in NO_x emissions and ambient O_3 concentrations (Ozone Source Receptor Model (OSRM) model)
- Relationship between changes in VOC emissions and ambient O_3 concentrations (OSRM model)

2.2 PCM model for the contribution of primary emissions to ambient concentrations

2.2.1 National damage costs

The PCM model has been used to calculate annual mean concentrations of PM_{10}, $PM_{2.5}$, NO_2 and SO_2 for 2014 on a 1 x 1 km grid. This model has been described in detail by Brookes et al (2015). Emissions-to-concentration modelling for 2014 was used given this was the most recent modelling available at the time the update to the damage costs commenced.

The PCM model results for each pollutant include contributions from a range of different sources. The calculation of damage costs requires the relationship between UK ambient concentrations and UK emissions (expressed as μgm^{-3} per tonne). Thus, only the sources within the PCM model that are related to UK emissions are relevant to the calculation of damage costs. These are the following contributions:

- Large point sources, modelled explicitly using the dispersion model ADMS
- Small point sources, modelled using a dispersion kernel approach (The model is run once for a unit emission rate from a single source and this is used to generate a dispersion kernel, which can be used to calculate concentrations from all sources considered).
- Area sources, modelled using the small points dispersion kernels for industrial emissions and dispersion kernels for other area sources, including kernels incorporating time varying emissions for domestic and road traffic sources.
- Regional concentrations of primary PM, modelled using the chemistry transport model TRACK.

The total concentrations of primary PM_{10}, $PM_{2.5}$, NO_2 and SO_2 associated with UK emissions inventory sources were calculated by summing these contributions and the population-weighted mean annual mean concentrations for 2014 were calculated for each pollutant using 1 x 1km population data from the 2011 census. The μgm^{-3} per tonne for each pollutant was then calculated by dividing this population-weighted mean by the 2014 UK total emissions for each pollutant that were used to calculate the ambient concentrations within the model. The emissions for 2014 were calculated by scaling data from the NAEI for 2013 forwards by one year using emission projections provided by the NAEI as described by Brookes et al (2015).

The impact of primary emissions of NOx on concentrations of NO_2 is expressed as ugm^{-3} of NO_2 per tonne of NO_x emitted. This has been calculated by multiplying the ugm^{-3} of NO_x per tonne of NO_x emitted by the total UK population-weighted mean of NO_2 from all sources divided by the by the total UK population-weighted mean of NO_x from all sources.

2.2.2 Sector specific damage costs

The approach described above provides the average relationship between emissions and the exposure of the UK population to ambient concentrations. The impact of emissions on exposure to ambient concentrations varies for different sources and geographically, since it depends on the release characteristics of the emissions and the proximity of these emissions to centres of population. We have calculated emissions estimates for each sector and have run the concentration models on a sector by sector basis. We have used this to calculate the change in concentration per unit emissions for each emissions sector.

The overall damage costs of air pollutants are dominated by the contribution from long term exposure to $PM_{2.5}$ and NO_2. Damage costs per tonne of primary $PM_{2.5}$ emitted via concentration of $PM_{2.5}$ have therefore been calculated for a range of specific emission sectors and geographical locations as detailed in Table 1. Sector specific damage costs per tonne of NO_x emitted via concentration of NO_2 have also been calculated. Sector specific damage costs have not been calculated for the contributions of emissions to secondary $PM_{2.5}$ or ozone because the release characteristics and location of emissions are less important for these pollutants. Sector specific damage costs have not been calculated for SO_2 because the direct SO_2 impact pathways typically only make a small contribution to the overall damage costs from emission releases, which are dominated by the contribution of SO_2 emissions to $PM_{2.5}$ pathways via the formation of secondary $PM_{2.5}$.

The road transport sources are area sources and have been separated by geographical location according to ‘area types’ defined by DfT (see Brookes et al, 2015). The concentrations for each sector also include the contribution from this sector to the regional primary PM concentration in addition to the local area sources.

The sector specific relationship between concentrations for NO_2 and emissions for NO_x have been calculated by multiplying the ugm^{-3} of NO_x per tonne of NO_x emitted for each sector by the total UK population-weighted mean of NO_2 from all sources divided by the by the total UK population-weighted mean of NO_x from all sources.

Table 1 - Sectors for primary $PM_{2.5}$ via $PM_{2.5}$ concentrations and NO_x via NO_2 concentrations

Sector
All Sectors (National)
Industry (area sources)
Commercial
Domestic
Solvents**
Road Transport Average
Aircraft
Off-road mobile machinery
Rail
Ships
Waste
Agriculture**
Other
Road Transport Central London
Road Transport Inner London
Road Transport Outer London
Road Transport Inner Conurbation
Road Transport Outer Conurbation
Road Transport Urban Big
Road Transport Urban Large
Road Transport Urban Medium
Road Transport Urban Small
Road Rural

** There are no NO_x emissions in the NAEI for Solvents or Agriculture, therefore dispersion modelling for these sectors has only been undertaken for $PM_{2.5}$

2.2.3 Damage costs for Part A processes

The release characteristics and location of releases in relation to centres of population are particularly variable for large industrial processes. These large industrial processes are known as Part A processes and the emissions are regulated by national regulators (The Environment Agency in England, Natural Resources Wales, The Scottish Environment Protection Agency and Department of Agriculture, Environment and Rural Affairs in Northern Ireland). We have therefore calculated damage costs for nine categories of Part A processes in order to account for differences in chimneystack heights and population density. The categories are summarised in Table 2.

Table 2 - Part A categories for primary $PM_{2.5}$ via $PM_{2.5}$ concentrations and NO_x via NO_2 concentrations

Average population density (persons per km^2)*	Stack Height <= 50 m and all small points	Stack Height > 50, <= 100 m	Stack Height > 100 m
<= 250	**Part A category 1**	**Part A category 4**	**Part A category 7**
> 250, <= 1000	**Part A category 2**	**Part A category 5**	**Part A category 8**
> 1000	**Part A category 3**	**Part A category 6**	**Part A category 9**

These damage costs have been derived in the same way as the rest of the sector specific damage costs (by dividing the total contribution to UK population-weighted concentrations from modelled sources within each category by the sum of emissions from the sources in each category). Note that the population density has been calculated for different areas for each stack height range. The areas are listed in Table 3.

Table 3 – Population density areas for Part A categories

Stack Height <= 50 m and all small points	Stack Height > 50, <= 100 m	Stack Height > 100 m
11 km x 11 km	21 km x 21 km	31 km x 31 km

2.3 PCM model emission sensitivity coefficients method for contribution to secondary $PM_{2.5}$

The PCM model has been used to calculate the impact of NO_x emissions on ambient NO_2 concentrations and of SO_2 emissions on ambient SO_2 concentrations. These μgm^{-3} per tonne have been used in the impact pathways for NO_2 and SO_2 concentrations. Emissions of NO_x, SO_2 and NH_3 also contribute to damage costs via the secondary inorganic aerosol (SIA) contribution to ambient PM concentrations and the long- and short-term exposure to PM concentration pathways. The PCM model emission sensitivity coefficients method has been used to calculate μgm^{-3} SIA changes per tonne of NO_x, SO_2 or NH_3 emitted.

SIA within the PCM model consists of SO_4, NO_3 and NH_4 and some additional counter ions and bound water. For compliance assessment modelling the concentrations of these components are derived within the model from ambient measurement data for SO_4, NO_3 and NH_4 by interpolation and application of appropriate scaling factors, as described by Brookes et al (2015).

Results from the EMEP model have been used to calculate emission sensitivity coefficients for the UK on a 50 x 50 km grid. The coefficients represent the proportional change in UK concentrations for the SIA species for changes in UK NO_x, SO_2 and NH_3 emissions. Coefficients have also been determined for the impact of changes in emissions in the rest of the EU, emissions from other countries and emissions from shipping but these are not required for the damage cost calculations. Emission sensitivity coefficients are required because the relationship between precursor emissions and SIA concentrations is complex and the change in concentrations is typically smaller than a 1 to 1 reduction in line with changes in emissions. There are also some complex effects such as changes in NO_x emissions potentially leading to small changes in SO_4 concentrations as a result of the complex atmospheric chemistry. The emission sensitivity coefficients provide a method of capturing these complexities in the results from chemistry transport models (the EMEP model in this instance) and parameterising them in such a way that they can be used in these damage cost calculations and other applications of the PCM model, such as projections for future years.

The emission sensitivity coefficients have been used to calculate the impact of 10% reductions of UK NO_x, SO_2 and NH_3 emissions in turn on population-weighted mean annual mean SIA concentrations in the UK. 10% reductions were chosen since changes in emissions of this magnitude should result in approximately linear responses within the EMEP model, which means that the emission sensitivity coefficients should be valid for this scale of reduction. The μgm^{-3} SIA (and thus PM) per tonne change in emissions was then calculated by dividing these changes in SIA concentrations by 10% of the UK total emission for these gases.

2.4 OSRM method for impact of changes in NO_x and VOC emissions on O_3

The Ozone Source Receptor Model (OSRM) has been used to calculate the impact of changes in NO_x emissions and VOC (non-methane VOC) emissions on ambient O_3 concentrations. The modelled change in μgm^{-3} O_3 per tonne of NO_x emissions or VOC emissions has then been used in the impact pathways for O_3 concentrations.

The OSRM was run to model the impact of a 10% reduction in UK NO_x emissions on O_3 concentrations on a 10 km x10 km UK grid. The impact of this scenario on various population-weighted mean ozone O_3 metrics (for the UK) was then calculated from the gridded results. The model was also run to assess the impact of a 10% reduction in UK VOC emissions on O_3

concentrations. The μgm^{-3} changes per tonne changes in emissions were then calculated by dividing the changes in the population-weighted mean ozone metrics by 10% of the UK total NO_x and VOC emissions.

The relationships between NO_x emissions and VOC emissions and O_3 concentrations are complex and non-linear. However, for the purposes of calculating per tonne damage costs, both relationships have been assumed to be linear. A reduction in VOC emissions results in a reduction in O_3 concentration. A reduction in NO_x emissions results in an increase in O_3 concentration.

Emissions of different VOC species have differing potentials to influence photochemical ozone creation. A consistent reduction in all VOC species has been assumed in the calculation of the impact of VOC emissions via the O_3 impact pathways. Consideration of damage costs for different VOC species is beyond the scope of this update to the damage costs.

Further information on the OSRM can be found in Cooke et al (2014).

2.5 Dispersion modelling to support estimation of ecosystem impacts

Specific O_3 concentration metrics were required to include the valuation of the three ecosystem impacts from O_3. These were POD_6wheat (mmol m^{-2}, the annual phytotoxic ozone dose for wheat with a threshold flux of 6 nmol m^{-2} s^{-1}) and 24-hour mean averaged over a seven-month growing season from 1st March to 30th September.

To produce the POD_6wheat metric, additional post-processing of OSRM results was carried out. This separate post-process was run on the OSRM model results from the impact of a 10% reduction in UK NO_x emissions on O_3 concentrations on a 10 km x 10 km UK grid. It was also run on the OSRM model results from the 10% reduction in UK VOC emissions scenario. The impact of these scenarios on area weighted mean POD_6wheat were then calculated from the results (following the method described in section 2.4).

Separate 7-month (rather than 12-month) OSRM runs were carried out to produce the 7-month 24-hour mean metric. The impact of the two scenarios (10% reduction in UK NO_x emissions & 10% reduction in UK VOC emissions) on area weighted 7-month 24-hour mean concentration were then calculated from the results (following the method described in section 2.4).

Further information on the OSRM can be found in Cooke et al (2014).

3 Updates to estimation and valuation of impacts

3.1 Concentration response functions (CRFs) for health outcomes

3.1.1 CRFs carried forward from previous damage costs

The estimation of the impacts of air pollution is carried out using Concentration Response Functions (CRFs). CRFs link a change in exposure to a pollutant to its consequent impacts by expressing a change in a health (or non-health) outcome for a given change in pollutant concentrations.

In its 2013 published guidance, the Interdepartmental Group on Costs and Benefits (IGCB) has recommended a set of CRFs describing the health impacts of air pollution that it suggests should be used for the appraisal of air quality impacts (Defra, 2013b). These CRFs were taken from an extensive underlying literature on the health effects of air pollution and follow the recommendations of COMEAP (see for example: COMEAP, 1998, 2009 and 2010). The health impact pathways included in the 2013 guidance are carried forward to the updated damage costs.

COMEAP have subsequently published a number of additional reports recommending health impact pathways for inclusion in the appraisal of air pollutant impacts (and the appropriate methodology for doing so). This includes:

- Impacts of ozone exposure on hospital admissions and deaths brought forward (COMEAP, 2015).
- Statement on quantifying mortality associated with long-term average concentrations of fine particulate matter ($PM_{2.5}$) (COMEAP, 2018b)

The CRFs for these pathways carried forward used for the estimation of the updated damage costs are set out in Table 4.

Table 4 – CRF's applied in updated damage costs (% per 10µgm^{-3} change in concentration for relevant averaging period)

			% change per 10ugm^{-3} change in pollutant		
Pollutant	Pathway	Air pollution metric	Low	Central	High
$PM_{2.5}$	Chronic mortality (1)	Annual average	4	6	8
PM_{10}	Respiratory hospital admission (2)	Annual average	0.8	0.8	0.8
PM_{10}	Cardiovascular hospital admission (2)	Annual average	0.8	0.8	0.8
SO_2	Deaths brought forward (2)	Annual average	0.6	0.6	0.6
SO_2	Respiratory hospital admission (2)	Annual average	0.5	0.5	0.5
O_3	Deaths brought forward (3)	Daily maximum of 8 hour mean	0.12	0.34	0.56
O_3	Respiratory hospital admission (3)	Daily maximum of 8 hour mean	0.3	0.75	1.2
O_3	Cardiovascular hospital admission (3)	Daily maximum of 8 hour mean	-0.06	0.11	0.27
NO_2	Respiratory hospital admission* (2)	Annual average	0.5	0.5	0.5

* Pathway only for inclusion in sensitivity analysis. Source: (1) COMEAP, 2018b; (2) Defra 2013b, (3) COMEAP, 2015

While there are CRFs for O_3, these are only relevant for the damage costs associated with NO_x and VOC emissions because O_3 is a secondary air pollutant, for which there are no emissions.

In its guidance, IGCB did not include a sensitivity range around the CRFs linking acute exposure to particulate matter, NO_2 and SO_2 to hospital admissions, nor SO_2 to acute mortality effects. Hence the CRFs used to assess these impacts are not flexed to derive the 'low' and 'high' damage cost sensitivities (see next section). In addition, the impact of acute exposure to NO_2 on hospital admissions is only included as a sensitivity in the 'high' damage cost.

In contrast to the calculations used to derive the original damage costs, this project has used a CRF for chronic mortality applied to concentrations of $PM_{2.5}$ rather than PM_{10} as the index of particulate air pollution (ambient measurement and model data for $PM_{2.5}$ in the UK were previously not sufficiently well advanced at the time for inclusion in the damage cost calculations). This is consistent with current IGCB guidance and with the recommendations of COMEAP (2009), which were published following the estimation of the original damage costs. In their review of evidence of the impact of long-term exposure to air pollution, COMEAP concluded that $PM_{2.5}$ was the most appropriate index of particulate air pollution for use in quantitative assessments.

Further, the uncertainty range around the CRF for mortality impacts associated with chronic exposure to particulates has also been narrowed relative to the range used to derive the original damage costs (see AEA-Technology, 2006). The range has reduced to a low and high value of 4% and 8% per $10\mu gm^{-3}$ respectively from 2% to 11% initially. This narrowing reflects latest COMEAP guidance (COMEAP, 2018b).

In the previous estimation of damage costs, impacts on health from ozone exposure were estimated using a range of thresholds, where a threshold represents a minimum level of concentration that must be reached before impacts on health start to occur. For this project, based on the most recent advice from COMEAP regarding the estimation of effects associated with ozone exposure (COMEAP, 2015b) we have not applied a threshold to the calculation of effects across all damage cost sensitivities.

3.1.2 Mortality effects of long-term exposure to NO_2

Since the 2013 guidance, COMEAP have also issued updated guidance around the assessment of long-term mortality effects associated with exposure to NO_2. This took the form of sequential publications:

- Nitrogen dioxide: interim view on long-term average concentrations and mortality (COMEAP, 2015b)
- Associations of long-term average concentrations of nitrogen dioxide with mortality (COMEAP, 2018a).

COMEAP's updated guidance (COMEAP, 2018a) suggests alternative approaches depending on the scope of the appraisal. In particular, this focuses on the scope of emissions assessed. The guidance suggests:

- For interventions that primarily target emissions of NOx: Use 25-55% of unadjusted coefficient (mid-point of range 40%) 1.023 (95% CI: 1.008, 1.037) per 10 µg/m3 annual average NO_2.

- For interventions that reduce all traffic related air pollutants: use the unadjusted NO_2 coefficient 1.023 (95% CI: 1.008, 1.037) per 10 µg/m3 annual average NO_2.

In order to maintain simplicity in the interpretation and implementation of the damage costs, IGCB agreed that it was preferable to have one method of estimating the effects that applies to all policy measures. Hence IGCB's guidance will be to apply, in all circumstances, the adjusted NOx coefficient (for interventions that primarily target emissions of NOx) to estimate damages associated with NOx emissions, alongside the unadjusted PM coefficient to estimate damages associated with PM emissions. The update to the damage costs has been undertaken in accordance with this direction.

The NOx damage costs include an assessment of the benefits of reductions in secondary nitrate (distant from source) due to reduced NOx emissions using the unadjusted $PM_{2.5}$ coefficient.

The CRFs used for the estimation of the updated damage costs are set out in Table 5.

Table 5 – CRF's applied in updated damage costs (% per 10µgm^{-3} change in concentration for relevant averaging period, CRFs with the adjustment for overlap with $PM_{2.5}$ applied are included in brackets) – chronic mortality and NO_2

			% change per 10ugm^{-3} change in pollutant		
Pollutant	Pathway	Air pollution metric	Low	Central	High
NO_2	Chronic mortality	Annual average	0.8 (0.2)	2.3 (0.92)	3.7 (2.035)

3.1.3 Chronic bronchitis

COMEAP have also subsequently published a report exploring the link between chronic exposure to particulate matter and chronic bronchitis: Impacts of long-term PM exposure on chronic bronchitis (COMEAP, 2016). In the report, COMEAP consider the number of cases attributable to current levels of pollution and the change in cases as a result of reducing pollution levels and provide comment intended to inform valuation of this health pathway. On the basis of the example presented in the paper, we have included valuation of this impact pathway in the updated damage costs.

The CRFs used for the estimation of the updated damage costs are set out in Table 5.

Table 6 – CRF's applied in updated damage costs (odds ratio per 10µgm^{-3} change in concentration for relevant averaging period) – chronic bronchitis and PM_{10}

			Odds ratio per 10ugm^{-3} change in pollutant		
Pollutant	Pathway	Air pollution metric	Low	Central	High
PM_{10}	Chronic Bronchitis	Annual average	1.02	1.32	1.71

For chronic bronchitis effects, the relationship between pollutant exposure and health outcome is expressed as an odds ratio. Hence relative to the other impact pathways, the calculation of impacts adopts a slight variation in approach. These effects have been captured by following the example demonstrated by COMEAP (2016) who calculated that a 1ugm^{-3} reduction in PM_{10} could reduce the number of people with symptoms by around 65,000.

In the report, COMEAP did not assign a monetary value to these effects, but did include further considerations and guidance as to how analysts could transform estimates of changes in the prevalence into a monetised effect. The methodology used in this updated set of damage costs closely follows COMEAP's guidance.

The change in prevalence is combined with a quality-adjusted life years (QALY) score for each case to calculate the total QALY loss. The selection of an appropriate QALY weight is highly uncertain. In particular, COMEAP highlight that the epidemiologic research from which the odds ratio is derived does not provide a precise definition of the effect, only referring to the chronic presence of phlegm. This does not elaborate on the duration and severity of effects.

To inform quantification, COMEAP refer to a study by Salomon et al (2012) which sought to assign QALY weights to a range of health effects. Figures drawn from this study show that respiratory diseases can imply a range of QALY weights depending on the severity of the disease: Salomon et al record QALY weights ranging from 0.009 to 0.383 between 'controlled asthma cases' to 'severe COPD cases' respectively. However, no QALY weight is specifically defined for 'chronic bronchitis'. COMEAP proceed to reference a further study to explore the severity of chronic bronchitis relative to other respiratory diseases. The evidence appears to suggest chronic bronchitis ranks somewhere between moderate and severe COPD in terms of its effects on quality of life.

For the updated damage costs, a QALY weight of 0.232 has been adopted. This represents a central point between the QALY weights for COPD moderate (0.192) and COPD severe (0.383). Rather than simply taking the central point, the value taken represents the position of chronic bronchitis relative to these two conditions on the scale of severity of effects on quality of life, hence capturing that chronic bronchitis is closer to COPD moderate than COPD severe.

The impact of exposure to PM_{10} on chronic bronchitis are included only as a sensitivity in the 'high' damage cost following the guidance of IGCB.

3.1.4 Pathways captured in PHE model

Alongside updating the damage costs for recent papers published by COMEAP, IGCB also directed that pathways for five diseases (asthma in adults, asthma in children, coronary heart disease, stroke, diabetes type 2 and lung cancer) explored by Public Health England (PHE, 2018) should also be included in the updated damage costs. This section sets out the method used to include the pathways in the updated damage costs.

Concentration Response Functions

Concentration Response Functions (CRFs) in relation to these new morbidity pathways for a NO_2 and $PM_{2.5}$ were extracted from the report provided by Public Health England (PHE), which in turn were obtained from scientific papers.

The NO_2 CRFs were adjusted by applying a factor of 40% to take account of overlaps between risks produced by $PM_{2.5}$, in order to be consistent with the approach adopted by PHE and the guidance provided by IGCB.

The health outcomes included and associated CRFs are:

- *Asthma in adults.* CRF for NO_2, with a value of Odds Ratio (OR) of 1.04 (0.996; 1.08) per 10 $\mu g/m^3$ of annual mean. Data sourced from Jacquemin et al., (2015).
- *Asthma in small children (≤ 6 years old)*. CRF for NO_2, with a value of Odds Ratio (OR) of 1.08 (1.01, 1.12) per 10 $\mu g/m^3$ of annual mean. Data sourced from Khreis et al., (2016).
- *Asthma in older children (7-15 years old).* CRF for NO_2, with a value of Odds Ratio (OR) of 1.03 (1.00, 1.06) per 10 $\mu g/m^3$ of annual mean; CRF for $PM_{2.5}$, with a value of Odds Ratio (OR) of 1.48 (1.22, 1.97) per 10 $\mu g/m^3$ of annual mean. Data sourced from Jacquemin et al., (2015).
- *Coronary heart disease (CHD).* CRF for $PM_{2.5}$, with a value of Hazard Ratio (HR) of 1.19 (1.01; 1.42) per 5 $\mu g/m^3$ of annual mean. Data sourced from Cesaroni et al., (2014).
- *Stroke.* CRF for $PM_{2.5}$, with a value of Hazard Ratio (HR) of 1.064 (1.021; 1.109) per 5 $\mu g/m^3$ of annual mean. Data sourced from Scheers et al., (2015).
- *Diabetes Type 2.* CRF for $PM_{2.5}$, with a value of Relative Risk (RR) of 1.10 (1.02; 1.18) per 10 $\mu g/m^3$ of annual mean. CRF for NO_2, with a value of Relative Risk (RR) of 1.05 (1.02; 1.07) per 10 $\mu g/m^3$ of annual mean. Data sourced from Eze et al., (2015).
- *Lung cancer.* CRF for $PM_{2.5}$, with a value of Relative Risk of 1.09 (1.04; 1.14) per 10 $\mu g/m^3$ of annual mean. CRF for NO_2 with a value of Relative Risk (RR) of 1.02 (1.00; 1.03) per 10 $\mu g/m^3$ of annual mean. Data sourced from Hamra et al., (2015).

All CRFs are assumed to represent a change in incidence, as suggested by most of the references that were used in the PHE report.

PHE applied these CRFs in a micro-simulation model to estimate the implied changes in disease incidence. The updated damage costs are not based on this type of micro-simulation model, and calculated values for the impact of change in concentrations on incidence from the PHE model have not been made available. Instead, we have assumed that the CRFs for all the diseases can be assumed to apply directly to incidence for the purpose of calculating damage costs. The CRFs are summarised

in Table 7. We have included only the pathways that are considered more certain in the central damage costs, see Table 10 below.

Table 7 – CRF's applied in updated damage costs (% per defined change in concentration for relevant averaging period) – PHE morbidity pathways

					% change (or change in Odds Ratio) per defined change in pollutant		
Pollutant	Pathway	Air pollution metric	CRF type	Reference change in concentration (μgm^{-3})	Low	Central	High
$PM_{2.5}$	CHD	Annual average	Hazard Ratio (HR)	5	1.00	19.00	42.00
$PM_{2.5}$	Stroke	Annual average	Hazard Ratio (HR)	5	2.10	6.40	10.90
$PM_{2.5}$	Diabetes	Annual average	Relative Risk (RR)	10	2.00	10.00	18.00
$PM_{2.5}$	Lung cancer	Annual average	Relative Risk (RR)	10	4.00	9.00	14.00
NO_2	Asthma (Adults)	Annual average	Odds Ratio (OR)	10	1.00	1.04	1.08
NO_2	Diabetes	Annual average	Relative Risk (RR)	10	2.00	5.00	7.00
NO_2	Lung cancer	Annual average	Relative Risk (RR)	10	0.00	2.00	3.00
$PM_{2.5}$	Asthma (Older Children)	Annual average	Odds Ratio (OR)	10	1.22	1.48	1.97
NO_2	Asthma (Small Children)	Annual average	Odds Ratio (OR)	10	1.01	1.08	1.12
NO_2	Asthma (Older Children)	Annual average	Odds Ratio (OR)	10	1.00	1.03	1.06

Baseline epidemiological data

Baseline epidemiological data for the diseases of interest was extracted from PHE (2018), which in turn have been collected from numerous sources:

- *Asthma in adults.* Incidence data for age groups older than 16, both genders. British Lung Foundation (BLF) statistics sourced from The Health Improvement Network (THIN) database[3].
- *Asthma in children.* Incidence data for small (≤ 6 years old) and older children (7-15 years old). British Lung Foundation (BLF) statistics sourced from The Health Improvement Network (THIN) database.
- *Coronary heart disease (CHD).* Incidence data for all age groups, male and female. Data sourced from the British Heart Foundation (BHF) cardiovascular disease statistics 2014.
- *Stroke.* Incidence data for all age groups, male and female. Data sourced from the British Heart Foundation (BHF) cardiovascular disease statistics 2014.
- *Diabetes Type 2.* Incidence data for age groups older than 20, male and female. Data sourced from the National Diabetes Audit 2015-2016.
- *Lung cancer.* Incidence data for all age groups, male and female. Data sourced from Cancer Research UK (2012-2014).

The data provided above was per 100,000 persons of each age group. To obtain an age- and gender-weighted incidence of a disease *i*, Equation 1 was applied:

$$I_i = \sum_j^J \sum_k^K \frac{N_{j,k}}{N} \cdot I_{i,j,k} \qquad (1)$$

[3] https://www.ucl.ac.uk/pcph/research-groups-themes/thin-pub/database

Where:

I_i is the age- and gender-weighted incidence of a disease *i*.

$N_{j,k}$ is the population of age group *j* and gender *k* in the United Kingdom.

N is the total population of the United Kingdom.

$I_{i,j,k}$ is the incidence of disease *i*, age group *j* and gender *k*.

Calculation of the change in incidence

The estimation of the change in incidence due to a decrease of 1 µg/m^3 of $PM_{2.5}$ or NO_2 is different depending on whether the CRF is based on the Relative Risk, Hazard Risk or Odds Ratio.

Relative and Hazard Risks

The change in incidence (ΔI_i) per 100,000 inhabitants when the CRF is based on either the Relative Risk or Hazard Risk is estimated as the product between the concentration of the pollutant, the baseline incidence, and the population as in Equation 2:

$$\Delta I_i = \frac{\Delta C_{Pol}}{C_{Inc}} \cdot \frac{RR}{100} \cdot \frac{N}{10^5} \cdot I_i \qquad (2)$$

Where:

ΔC_{Pol} is the concentration of a given pollutant ($PM_{2.5}$, NO_2).

C_{Inc} is the concentration increment on which the CRF is based (5 or 10 µg/m^3).

RR is the Relative Risk (or Hazard Risk, if applicable).

N is the total population of the United Kingdom.

I_i is the age- and gender-weighted incidence of a disease *i*.

Odds Ratio

The estimation of the change in incidence (ΔI_i) per 100,000 inhabitants when the CRF is based on the Odds Ratio (OR) is more complex, as it requires an estimate the odds of reporting the disease at the new concentration (κ_i) first, as in Equation 3:

$$\kappa_i = \exp\left(-\ln(OR) \cdot \frac{\Delta C_{Pol}}{C_{Inc}} + \ln\frac{I_i}{10^5 - I_i}\right) \qquad (3)$$

The change in incidence (ΔI_i) per 100,000 inhabitants can be then estimated as a function of the odds of reporting the disease at the new concentration (κ_i) as in Equation 4:

$$\Delta I_i = \frac{N(1+\kappa_i)}{\kappa_i(I_i - 1) + I_i} \qquad (4)$$

In the case where relative risk values were based on concentration increments of 5 µg/m^3 (C_{inc}), these were used in preference to those extrapolated in the PHE report to a 10 µg/m^3 concentration increment base. This was done in order to be consistent with the methodology explained above, since the extrapolation of relative risk values made in the PHE report was non-linear and the damage cost approach assumes a linear scaling.

Estimation of air pollution impacts and costs

The estimation of air pollution impacts is done using Quality-Adjusted Life Years (QALY) lost, which are then multiplied by the value of a life year (VOLY) to obtain the costs. Costs have been calculated for the change in disease incidence for the considered health outcomes. The calculation of QALY loss requires utility weights for the different diseases, which are then multiplied by the change in incidence as in Equation 5:

$$QALY\ Loss_i = (1 - w_i) \cdot \delta_i \cdot \Delta I_i \quad (5)$$

Where:

$QALY\ Loss_i$ are the quality-adjusted life years for disease *i*.

w_i is the utility weight for disease *i*.

δ_i is the discounted duration of disease *i*.

The utility weights used in this report are those originally published in Sullivan et al., (2011) Catalogue of EQ-5D scores for the United Kingdom (Table 8). Males and females were allocated the same EQ-5D score and the diseases were mapped onto conditions listed in the publication using matching, or closest matching ICD-9 Categories.

These weights represent the QALY loss associated with each condition whilst living with the condition.

Table 8 – List of EQ-5D values (QALY weights) allocated to males and females for each disease

Disease	w_i	Mapped ICD-9 Categories
Asthma	0.722	ICD-9 493 Asthma
CHD	0.61	ICD-9 410 Acute Myocardial Infarct
Stroke	0.63	ICD-9 433 Precerebral Occlusion
Diabetes	0.66	ICD-9 250 Diabetes Mellitus
Lung cancer	0.56	ICD-9 162 Malignant Neoplasm Trachea/Lung

The duration of the disease is reflected in the δ_i, which is calculated according to Equation 6:

$$\delta_i = 1 \quad \text{if } D = 1 \quad (6a)$$

$$\delta_i = 1 + \sum_{j=2}^{D}(1 + r)^{1-j} \quad \text{if } D > 1 \quad (6b)$$

Where:

D is the average years of duration of the disease.

r is the discount rate (*r*=0.035).

The average years of duration of the disease were provided by Defra and were calculated using the DISMOD II model (WHO 2018) and estimated based on the years of life with disability (YLD). The specific average years of duration for the diseases in this study are presented in Table 9.

As the duration of the disease has been taken into consideration, the QALY loss (which, by definition, looks at the impact of living with the condition for a single year) can provide an indication on the lasting effects that conditions have beyond the first year.

Table 9 – Average and discounted duration of disease

Disease	D [years]	δ [years]
CHD	9.50	8.93
Asthma in Adults	23.60	20.11
Asthma in Children	36.20	28.39
Stroke	14.80	13.41
Diabetes	9.10	8.58
Lung cancer	1.80	1.79

By combining the change in incidence, with the QALY weight of living one year with the disease, and the (discounted) duration of the disease, this then calculates the cumulative QALY weight over the expected duration of the diseases associated with all incidences of the disease in a given year.

Finally, the costs produced by increases in the concentration of either $PM_{2.5}$ or NO_2 is the product of the valuation of a QALY loss and the quality-adjusted life years for disease *i* as in Equation 7 (see Table 16):

$$Cost_i = QALY\ Value \cdot QALY\ Loss_i \quad (7)$$

Following discussion with PHE regarding the strength of the underpinning epidemiological evidence, IGCB recommended that these new pathways should be included in the damage costs (and the sensitivity around central values) as set out in Table 10. No pathways should be included in the low damage cost.

Table 10 – Inclusion of PHE pathways in damage cost sensitivities

	Long term exposure to $PM_{2.5}$	Long term exposure to NO_2
Low Damage cost	-*	-*
Central damage cost (Stronger evidence suggestive for a causal association)	Coronary heart disease Stroke Lung cancer Asthma (children)	Asthma (children)
High damage cost (Evidence less certain or emerging evidence of associations)	Chronic Obstructive Pulmonary Disease (as chronic bronchitis) Diabetes	Asthma (adults) Diabetes Lung cancer

* No pathways should be included in the low damage cost.

PHE's model also included impacts on low birth weight and dementia. However, following discussion between Defra and PHE there was some concern regarding the inclusion of these pathways and they were deprioritised relative to the inclusion of the other pathways, and not included in this round of updates.

3.1.5 Productivity impacts

Ricardo AEA (2014) explored the impacts of air pollution on productivity. The study developed a method to quantify these effects through five pathways. These focussed on the direct impacts of air pollution on human health via inhalation (and hence on labour as an input into production):

- Mortality (chronic and acute) in workforce
- Morbidity in the workforce (absenteeism)
- Morbidity in the workforce (presenteeism)
- Absence in the workforce due to morbidity in dependents

- Health impacts (mortality and morbidity) in non-market productive activities (e.g. volunteering and non-paid caring).

Eight other pathways were identified but not taken forward for quantification. These pathways included for example: impacts on visibility, animal health, and indirect impacts on human health via consumption of food or water.

The methodology to quantify the impacts under each pathway taken forward follows the widely recognised Impact Pathway Approach. The valuation of these health impacts uses the Human Capital Approach (HCA) to assess lost productivity: under the HCA, productivity loss is measured as the length of potential productive time that the person is unable to work multiplied by a value of marginal productivity revealed in the market.

The study estimated that the burden associated with 2012 levels of pollutants had a total cost of £2.7bn through its impact on productivity in that year. Some of the pathways captured in this analysis overlap with those pathways and impacts already captured in the existing damage costs and IGCB appraisal guidance. The study identified only £1.1bn of these costs are additional to those that would have been captured using the existing IGCB appraisal guidance.

The updated damage costs include an estimate of the impact of air pollution on productivity following the approach described in the report. Only those impact pathways that are deemed additional to those pathways already included in the existing damage costs are included to avoid double counting of effects (further discussion on the interaction and overlaps between these effects and those already captured by the IGCB guidance can be found in the underlying report (Ricardo-AEA, 2014)). The impact pathways included under the low, central and high damage costs are:

- absenteeism and work-days lost (WDL) for employees, volunteers and carers ($PM_{2.5}$)
- presenteeism and minor restricted activity days (mRADs) for employees ($PM_{2.5}$ and O_3).

In addition, the high damage cost also includes impacts on school days lost (SDL) (and consequent effect of absent workers to care for dependents) through exposure to PM_{10} and O_3.

A low, central and high estimate of the additional productivity impacts are included in the low, central and high damage costs respectively. Several parameters are varied to produce these different sensitivity estimates, alongside the impact pathways included as set out above. The parameters flexed under each sensitivity are set out in Table 11.

Table 11 - Parameters flexed to produce low, central and high productivity cost estimates

Productivity impact sensitivity	Low	Central	High
Impact pathways	WDL ($PM_{2.5}$), mRADS $PM_{2.5}$ and O_3)	WDL ($PM_{2.5}$), mRADS $PM_{2.5}$ and O_3)	WDL ($PM_{2.5}$), mRADS $PM_{2.5}$ and O_3), SDL (PM_{10} and O_3)
CRF applied from CRF confidence interval	Low	Central	High
Unit values	Average wage per worker	CBI value of average lost productivity per worker	Average GDP per day worked
Baseline rates of absence	Uses only air pollutant related health impacts (e.g. respiratory or cardio-vascular complaints) to set baseline absence rates	Uses total absence rate to set baseline (i.e. covering all causes, not just air quality related complaints)	Uses total absence rate to set baseline (i.e. covering all causes, not just air quality related complaints)

3.1.6 Health impact pathway summary

As described above, the health impact pathways included in the updated damage costs come from a number of sources. These are based on the original set of health impact pathways and CRFs in the initial damage costs, updated for subsequent COMEAP publications for:

- Impacts of ozone exposure on hospital admissions and deaths brought forward (COMEAP, 2015)
- Impacts of long-term PM exposure on chronic bronchitis (COMEAP, 2016).
- Statement on quantifying mortality associated with long-term average concentrations of fine particulate matter ($PM_{2.5}$) (COMEAP, 2018b)
- Associations of long-term average concentrations of nitrogen dioxide with mortality (COMEAP, 2018a).

In addition, a range of pathways have been included to reflect those captured in PHE's Estimation of costs to the NHS and social care due to the health impacts of air pollution (PHE, 2018).

The CRFs used for the estimation of the updated damage costs for the health pathways (apart from for the productivity pathways, which are summarised in Ricardo-AEA, 2014) are set out in Table 12.

Note: low, central and high in the table do not relate to the CRF values applied in the low, central and high damage costs produced. This simply presents the confidence interval bound around each CRF presented in the underlying literature. The CRF value from the confidence interval does vary between the low, central and high damage costs, however, the inclusion of the impact pathways themselves also varies between the sensitivities. Which impact pathways are included, and which CRF is selected from the underlying confidence interval in each damage cost is presented in Table 18.

Emissions of NO_x, SO_2 and NH_3 also contribute to damage costs via the secondary inorganic aerosol (SIA) contribution to ambient PM concentrations and the long and short-term exposure to PM concentration pathways. A full mapping of the different impact pathways included in each of the damage costs is presented in Table 13. Primary effects, such as the chronic mortality associated with $PM_{2.5}$ resulting from emissions of $PM_{2.5}$ are labelled 'P'. Secondary effects, such as the chronic mortality associated with $PM_{2.5}$ resulting from emissions of NO_x are labelled '2'.

Table 12 – CRF's applied in updated damage costs (% per 10μgm^{-3} change in concentration for relevant averaging period)

					% or Odds ratio change per 10ugm^{-3} change in pollutant		
Pollutant	Pathway	Air pollution metric	CRF type	Reference change in concentration (ugm^{-3})	Low	Central	High
$PM_{2.5}$	Chronic mortality	Annual average	Relative Risk (RR)	10	4	6	8
PM_{10}	Respiratory hospital admission	Annual average	Relative Risk (RR)	10	0.8	0.8	0.8
PM_{10}	Cardiovascular hospital admission	Annual average	Relative Risk (RR)	10	0.8	0.8	0.8
SO_2	Deaths brought forward	Annual average	Relative Risk (RR)	10	0.6	0.6	0.6
SO_2	Respiratory hospital admission	Annual average	Relative Risk (RR)	10	0.5	0.5	0.5
O_3	Deaths brought forward	Daily maximum of 8 hour mean	Relative Risk (RR)	10	0.12	0.34	0.56
O_3	Respiratory hospital admission	Daily maximum of 8 hour mean	Relative Risk (RR)	10	0.3	0.75	1.2
O_3	Cardiovascular hospital admission	Daily maximum of 8 hour mean	Relative Risk (RR)	10	-0.06	0.11	0.27
NO_2	Respiratory hospital admission	Annual average	Relative Risk (RR)	10	0.5	0.5	0.5
NO_2	Chronic mortality	Annual average	Relative Risk (RR)	10	0.8	2.3	3.7
PM_{10}	Chronic Bronchitis	Annual average	Relative Risk (RR)	10	1.02	1.32	1.71
$PM_{2.5}$	CHD	Annual average	Hazard Ratio (HR)	5	1.00	19.00	42.00
$PM_{2.5}$	Stroke	Annual average	Hazard Ratio (HR)	5	2.10	6.40	10.90
$PM_{2.5}$	Diabetes	Annual average	Relative Risk (RR)	10	2.00	10.00	18.00
$PM_{2.5}$	Lung cancer	Annual average	Relative Risk (RR)	10	4.00	9.00	14.00
NO_2	Asthma (Adults)	Annual average	Odds Ratio (OR)	10	1.00	1.04	1.08
NO_2	Diabetes	Annual average	Relative Risk (RR)	10	2.00	5.00	7.00
NO_2	Lung cancer	Annual average	Relative Risk (RR)	10	0.00	2.00	3.00
$PM_{2.5}$	Asthma (Older Children)	Annual average	Odds Ratio (OR)	10	1.22	1.48	1.97
NO_2	Asthma (Small Children)	Annual average	Odds Ratio (OR)	10	1.01	1.08	1.12
NO_2	Asthma (Older Children)	Annual average	Odds Ratio (OR)	10	1.00	1.03	1.06

Table 13 – Mapping of primary and secondary effects against each damage cost

Pollutant		$PM_{2.5}$	PM_{10}	PM_{10}	$PM_{2.5}$	PM_{10}	SO_2	SO_2	NO_2	NO_2	NO_2	O_3	O_3	O_3	O_3	O_3	PM_{10}	SO_2	SO_2	O_3	NO_2	NH_3	$PM_{2.5}$	NO_2	$PM_{2.5}$	$PM2.5$	NO_2	$PM_{2.5}$	NO_2	$PM_{2.5}$	NO_2	NO_2
Pathway		Chronic mortality	Respiratory hospital admission	Cardiovascular hospital admission	Productivity	Chronic Bronchitis	Deaths brought forward	Respiratory hospital admission	Respiratory hospital admission	Chronic mortality	Productivity	Deaths brought forward	Respiratory hospital admission	Cardiovascular hospital admission	Productivity	Material damage	Building soiling	Material damage	Ecosystems	Ecosystems	Ecosystems	Ecosystems	CHD	Asthma (Adults)	Stroke	Diabetes	Diabetes	Lung Cancer	Lung Cancer	Asthma (Children)	Asthma (Small Children)	Asthma (Older Children)
Damage cost	NO_x	2	2	2	2	2*			P*	P	P*	2	2	2	2*	2				2	P		2	P*	2	2*	P*	2	P*	2	P	P
	SO_2	2	2	2	2	2*	P	P										P	P				2		2	2*		2		2		
	NH_3	2	2	2	2	2*																P	2		2	2*		2		2		
	VOC											2	2	2	2*	2				2												
	$PM_{2.5}$	P	P	P	P	P*											P						P		P	P*		P		P		

P = primary effect; 2 = secondary effect; * = impact only included under 'high' damage cost

3.2 Chronic mortality and life-table calculations

3.2.1 Methodology for calculating long-term air pollution impacts

The methodology used to calculate the impacts of long-term (or 'chronic') exposure to air pollution on mortality is known as the 'life-tables technique' and is based on a report by IOM (2000) and a subsequent publication by Miller (2003). In the updated damage costs, life-tables are applied to calculate the mortality effects associated with chronic exposure to $PM_{2.5}$ and NO_2.

A life-table is a technique used to summarise patterns of survival in populations. Standard life-table calculations compute survival rates at different ages. It uses age-specific death rates, derived from numbers of deaths in each age group and mid-year population sizes for each age group. From these survival rates average life expectancy, from either birth or a specific achieved age, can be derived. Combining these values with numbers in the population affected allows prediction of the total numbers of life years lived at each age.

To derive health impacts associated with a change in pollutant concentrations, the basic approach for a given population is to:

- obtain information on current mortality rates
- predict future mortality using current mortality rates and assumptions about future demography using life-table calculations, in the absence of changes in air pollution
- create an alternative scenario by adjusting mortality rates according to evidence regarding the effect of pollution on mortality, leaving other baseline assumptions unchanged
- compare predicted life expectancy between the scenario without pollution changes and the alternative scenario to give estimates of the effect on the target population of the pollution change (in life-years).

Life-table calculations were undertaken by Brian Miller (Institute of Occupational Medicine, IOM) using the IOMLIFET[4] system. Calculations were based on mid-year population estimates and mortality rates for 2012[5]. These formed a baseline scenario in which it was assumed that mortality rates identified in 2012 remain constant over the assessment period and the impact of net migration does not alter population sizes or mortality rates.

Life-table calculations were undertaken for a one-year pulse reduction of $1\mu gm^{-3}$ in annual average $PM_{2.5}$ concentrations. When the damage costs were initially developed, chronic mortality impacts were calculated for an annual (1 year) and sustained (for 5, 20 and 100 year) pollution pulses. This update to the damage costs has only used a one-year pulse approach to be consistent with methodology underpinning the original damage costs. These costs used an annual pulse to provide flexibility in the damage cost approach: not all policies would be expected to last one year but a one-year reduction in emissions can readily be scaled up to provide an approximation for a variety of durations. As such, by using an annual pulse approach, this implicitly assumes that impacts of emissions changes are additive across different years of analysis (for example, where a policy has impacts on emissions for consecutive years, these can be added together) and in the short term the difference between assessing the impacts of a sustained change in concentrations and the sum of annual pulse changes over the same time period are negligible.

Calculations were undertaken for scenarios with different CRFs to reflect the low, central and high uncertainty ranges recommended by IGCB. The life-table outputs for the 'alternative' scenarios (i.e. including the impact of the marginal air pollutant change on mortality rates) were compared with those

[4] For further detail, see: http://www.iom-world.org/research/research-expertise/statistical-services/iomlifet/
[5] Data for population and mortality for single year age groups up to age 90 (aggregate population and mortality rates for ages 90+ were applied to all ages over 90) were sourced from the different UK statistics authorities (ONS, GRO Scotland and NISRA) for 2012.

for the baseline. This provided an estimate of the total life years gained for the population aged 30+ in the UK over a 100-year assessment period (an alternative scenario involving a one-year reduction is not predicted to have any impact on new birth cohorts). These results were subsequently scaled according the ratio of CRFs to derive life-table calculations for the mortality impacts of NO_2.

3.2.2 Cessation lag

The potential lag between a reduction in pollutant concentrations and a change in the risk rate of a chronic health outcome is unknown. When the damage costs were initially developed, a lag range between 0 and 40 years was assumed for all chronic mortality effects based on the then prevailing advice of COMEAP (DoH, 2001). It was noted that neither a lag time of 0 or 40 years would be likely for all affected persons, but evidence suggested that either could be feasible for a proportion of deaths depending on health condition. In summary, it was assumed that the average lag time for all-cause mortality was somewhere between the two extremes. The original damage costs varied the length of lag from 40 to 0 years between 'low' and 'high' damage cost sensitivities respectively.

Cessation lag is a term used to denote the time pattern of reductions in mortality hazards following a reduction in pollution. In their 2010 report, COMEAP note that there is little direct evidence regarding cessation lags but adopted the approach agreed by the US Environmental Protection Agency (EPA) in 2004 and re-affirmed in the EPA's analysis in 2010. This approach uses a distribution of impacts on mortality rates across different lag times. Specifically: 30% of the risk reduction occurs in the first year after pollution reduction, 50% occurs across years 2-5 with the remaining 20% distributed across years 6-20 with smoothed annual values.

COMEAP (2009) considered that while, in principle, it might take 40 years for all benefits to be achieved, in practice benefits are likely to occur earlier, with a significant proportion in the first five years. As such, the three components of the cessation lag approach were considered to represent the short-term, cardiovascular and lung cancer mortality effects respectively. The most recent version of IOMLIFET permits calculations with arbitrary lag patterns, and was used here to implement the EPA pattern of lags.

3.2.3 Results and interpretation

As described above, each alternative scenario, assuming a unit reduction in pollutant concentration combined with different CRF sensitivities, is compared to the baseline scenario to derive the impact on life years. A summary of the impacts across the scenarios is presented in Table 14 (results show cumulative life years gained across all age cohorts, sexes and calendar years from 2012 to 2112). A value for a 1μgm^{-3} one-year pulse reduction in NO_2 has been calculated from this value by linear scaling using the ratio of CRFs for $PM_{2.5}$ and NO_2 chronic mortality.

Table 14 – Life years gained by UK 2012 population aged 30+ from a 1μgm^{-3} one-year pulse reduction in $PM_{2.5}$

	IGCB CRF
Total life years gained	36,642
(Range from low to high CRF bounds)	(23,894 – 47,776)

3.3 Baseline population and health response rates

As part of the re-estimation of the damage costs, this project has updated the population and baseline health outcomes data used in the calculation.

Population data for the UK and each of the Devolved Administrations was taken from ONS's mid-year population estimates for 2015 (ONS, 2016a). This represents resident population, which is also used

when modelling exposure. Hence the calculations were based on a UK population of around 65.1m, of which 11.6m were over the age of 65.

Data for the number of deaths in the UK were aggregated from data for individual Devolved Administrations sourced from the ONS (2016b), NISRA (2016) and NRS Scotland (2016). These data were then combined with the population data to derive a baseline mortality risk rate against which the impacts of air pollution are assessed.

Information on the number of hospital admissions per annum split by cause was also aggregated from data for each Devolved Administration: from NHS Digital (2016) for England, DHSSPSNI (2016) for Northern Ireland and NHS Wales (2017). No consistent data were available for Scotland hence an average risk rate was calculated based on the numbers of hospital admissions in England, Northern Ireland and Wales and it is assumed that this is a reasonable approximation for the rate across the whole of the UK. The latest data available were for the year 2015/16.

Baseline rates for the prevalence of chronic phlegm in never-smokers (aged 16 and above) are taken from the same sources used by COMEAP (2016) in their calculation of chronic bronchitis effects in order to be consistent with their recommendations[6].

The risk rates used in the estimation of the damage costs are presented in Table 15 below.

Table 15 – Health outcome risk rates used for damage cost estimation (number of cases per 100,000 of population per annum)

Metric	Deaths	Cardio vascular hospital admission	Respiratory hospital admission	Chronic bronchitis
Risk rate all ages	886	1087	1814	N/A
Risk rate in ages 16+	N/A	N/A	N/A	4,966
Risk rate in ages 65+	N/A	4313	4926	N/A

The data underpinning the calculation of risk rates are for a given historical year for which the latest data are available. Hence where the updated damage costs are applied to changes in emissions in future years, this implicitly assumes that the population and risk rate data used to derive the damage costs are also appropriate for the year assessed.

3.4 Valuation of health outcomes

To value the impacts of exposure to air pollutants, the estimated quantity of health effects are combined with a monetary impact value of a single instance of each health impact. In this case, this is a value of life-year lost (or VOLY, used to monetise mortality impacts), a QALY value and a value per hospital admission. The impact values used for this updated set of damage costs are the same as those used in the calculation of the original damage costs.

The impact values used to monetise changes in life-years lost were originally estimated by Chilton et al (2004). This study estimated a VOLY associated with a life-year spent in good health of £27,630 and in poor health of £14,280 (2002 prices). This was based on a survey of participants undertaken in between November 2002 and January 2003. Uplifts have been applied to ensure these values are relevant to the assessment of impacts today.

[6] COMEAP in turn adopted baseline prevalence rates for chronic phlegm in England from the Health Survey for England (HSE, 2011) and in Scotland from the Scottish Health Survey (Scottish Government, 2011)

The air quality appraisal guidance recommends that all estimates of WTP to avoid detrimental health outcomes are uplifted annually by 2%. This advice reflected guidance published by the Department of Health (DoH, 2004) and represents the view that willingness to pay (WTP) to avoid detrimental health effects is influenced by (and hence can be expected to rise in line with) the income of the person or household. For the updated damage costs, the original values from Chilton et al therefore needed to be updated for both real income growth from 2002 and price base (the price base for the updated damage costs is 2017).

Real income growth has been relatively low over the period since 2002. Hence it was considered inappropriate to use a fixed 2% uplift each year to represent real income growth. Instead, data for real GDP per capita were sourced from the Webtag databook (DfT, 2018) to derive a trend for real income growth. Rather than growing at an assumed 2% per annum, these data suggested instead that real incomes on average have only increased at an average rate of around 0.9% per annum from 2002 to 2015. Hence using the assumed uplift could have led to substantial overestimation of the value of impacts. The annual rate of real GDP per capita growth sourced from Webtag were used to uplift WTP estimates between the 2002 and the assessment year 2015. The price base of the VOLY estimates was updated using the latest set of GDP deflators published by HMT (2017).

A similar approach was adopted to value the morbidity pathways. We have started with the original unit values used in the original estimation of damage costs, and applied uplifts using the latest data on real GDP per capita growth. These health values capture both the disutility and resource cost associated with a hospital admission of each type, with the range representing uncertainty in monetary estimates of disutility.

Following cross-departmental discussion, the IGCB group directed that the new morbidity pathways which express health impacts in terms of QALY loss (i.e. adopted from the PHE model and chronic bronchitis) should be valued using the Green Book recommended value of £60,000 for a QALY (Quality-Adjusted Life Year). This was decided since the method for valuing these impacts was considered analogous to the way QALYs are calculated. Furthermore, the IGCB agreed that the mortality estimates should continue to use the Chilton et al. value on the basis that a £60,000 ‘life year in perfect health’ would not be appropriate for the type of life years that are lost to air pollution-related deaths (e.g. in old age).

The unit impact values used in the analysis are set out in Table 16.

In variance to the previous derivation of the original damage cost estimates, this project has not used Monte Carlo analysis to derive central estimates of the damage costs within sensitivity bounds. Instead, an average of the ‘high’ and ‘low’ bounds for the value of hospital admissions is taken to provide a central estimate of the cost. This results in a central cost of £7,000 and £7,100 for respiratory and cardiovascular hospital admissions respectively.

Several impact pathways will have lasting effects after the first year of impact. This applies to chronic mortality effects, but also some of the morbidity impacts that are measured as changes in incidence. For analysis of future year impacts, the approach continues to assume a proxy for income growth is the long-run rate of economic growth of 2% per annum. Further, impacts in years after the year of emissions change are discounted using the Green Book discount rate of 3.5% (HMT, 2011).

Discounting is only applied to chronic mortality impacts and morbidity effects assessed through changes in incidence. Discounting is not applied to any other impact pathways given impacts occur in year in which change in emissions occur. Specifically:

1. Productivity impacts: are represented by a change in WDL and mRADs, which are acute events and happen fairly shortly after exposure to changes in pollution. These are assumed to occur in year
2. Material damage and building soiling: the value of the damage estimate has been annualised and can therefore be treated as if the impacts occur in year

3. Ecosystems: these values are taken from an underlying study which recommends damage costs for appraisal – hence impacts are either in year or discounted already in the recommended values

Table 16 – Health impact values used in analysis (£2017 prices)*

Health effect	Form of measurement valuations apply to	Health values used in analysis (Sensitivity range)	IGCB recommended values (Defra, 2013b)** (Sensitivity range)
Acute mortality	Number of years of life lost due to air pollution, assuming 2-6 months loss of life expectancy for every death brought forward. Life expectancy losses assumed to be in poor health	£22,100 (10-15% of LYL valued using 'good health' VOLY)	£21,000 (10-15% of LYL valued using 'good health' VOLY)
Chronic mortality	Number of years of life lost due to air pollution. Life expectancy losses assumed to be in normal health.	£42,800 (£32,000 – £53,300)	£40,800 (£30,700 - £51,100)
Respiratory hospital admissions	Case of a hospital admission, of average duration 8 days	£8,300 (£2,800 – £13,800)	£3,000 – £12,4500
Cardiovascular hospital admissions	Case of a hospital admission, of average duration 9 days	£8,500 (£3,000 – £14,000)	£3,500 – £11,500
QALY loss	Cumulative discounted QALYs over duration of disease	£62,800 (£31,400 - £83,700)	N/A

*Values rounded to nearest £100
**Values uplifted to 2017 using HMT deflators and adjustment made for income growth from 2012-17

3.5 Other non-health impact pathways

3.5.1 Material damage and building soiling

Three pathways have been included in the updated damage cost estimates. The pathways are limited to those where air pollution degrades or soils materials and buildings. Given the scope of the project, the cost estimates have been adapted from the original damage cost calculation rather than being re-estimated.

Concentrations of air pollutants in the atmosphere have been proven to have a detrimental impact on buildings in utilitarian applications (i.e. in houses, factories, etc.). The quantification of these impacts was assessed within various studies for the European Commission DG Research, in particular ExternE and associated projects. The pollutants most implicated in acid damage are SO_2 (most importantly), H+ and NO_2. The most significant impacts are on natural stone and zinc coated materials. The benefits of reducing material damage from SO_2 have been included in the updated damage cost estimates using the methodology used for the original damage costs (although with an update to the price base): this suggested an impact of around £237 per tonne of SO_2 emitted (2017 prices).

Damage to building materials covers limestone, sandstone, mortar and zinc used in galvanised steel. Quantification covers utilitarian buildings and infrastructure, but not cultural heritage. Response functions were taken from a major international research effort and are based on 8 years of exposure of material specimens across Europe. These demonstrate SO_2 to be the most harmful of the pollutants under conditions up to the mid-2000s, so analysis has focused on this pollutant. Valuation

is performed using repair cost data from the architecture and building sector, with repair assumed necessary once a critical loss of material (defined in relation to each material, taking account of how they are used) has occurred. Value is calculated via the change in frequency of repair operations. Full account of the methods used is provided in the reports of the European Commission funded ExternE Project[7] (ExternE 1995 p300,1998 p 381, 2005 p109).

Ozone can also have a damaging impact on materials, in particular on rubbers and paints exposed to ambient air. Holland et al (1998) undertook a large study into the impacts of ozone on a range of paints and rubber formulations representative of those in the UK market. The study found that impacts on paint were unlikely over the lifetime of their application, but did quantify a relationship between ozone and damage to rubber materials. The effect of a population weighted 1ppb change in ozone was estimated at £3.7m per annum (2005 prices). This relationship has been used in the new damage cost estimates with an update to the price base and conversion to be expressed in terms of population-weighted ozone concentration (1ppb to $2\mu gm^{-3}$) to gain the impact per tonne of NO_x or VOC emitted via this ozone pathway in 2017 prices.

Soiling of buildings by particles is one of the most obvious signs of pollution in urban areas. The degree of soiling of particles varies according to a number of factors specific to the particles themselves, the nature of emission, the surface affected and wider meteorological conditions: for example, blackness per unit mass of smoke, particle size distribution, and chemical nature of the particles. Although the relationship between particle emission and soiling is strong, quantification of impacts is not straightforward. The original damage cost estimates used an approach developed by Rabl et al (1998) which captured both the cleaning and amenity costs associated with building soiling. The same approach is adopted here which suggests that a 1 tonne change in PM_{10} has an associated cost of £565 (2017 prices).

In contrast to the other $PM_{2.5}$ pathways, the damage cost for building soiling does not take the location of emissions, dispersion conditions or the density of stock at risk into account. The contribution from this $PM_{2.5}$ pathway relative to other pathways is quite high for some part A sectors and for agriculture. The scope of this update to the damage cost did not included the development of sector specific damage costs for building soiling.

3.5.2 Ecosystem impacts

A key gap in the quantification of impacts associated with changes in air pollution are the effects on environmental health and the services ecosystems provide. The strength of evidence and methodologies to quantify these effects has lagged that of human health effects given the latter have been prioritised over the last couple of decades. That said, the initial set of damage costs did include impacts on crop yields.

In an attempt to start to fill this gap, Defra commissioned a tranche of projects to explore the impacts of air pollution on ecosystem service provision. One of the outputs of this work was a report by Jones et al (2014) titled ‘Assessment of the Impacts of Air Pollution on Ecosystem Services – Gap Filling and Research Recommendations’.

The aims of this study were to:

1. Review the evidence and data behind previous valuation studies of air pollution on ecosystem services.
2. Apply an improved spatially explicit methodology to value impact of selected ecosystem services.

[7] http://www.externe.info/externe_d7/sites/default/files/vol2.pdf, p.300, http://www.externe.info/externe_d7/sites/default/files/vol7.pdf, p. 381, http://www.externe.info/externe_d7/sites/default/files/methup05a.pdf, p 109.

3. Prioritise additional ecosystem services for valuation of air pollution impacts. Identify existing or planned projects and new research which might provide relevant information, and recommend appropriate research approaches to model them.
4. Collate damage costs from this and previous studies.

The study reviewed the evidence linking air pollution to a range of potential impacts on ecosystem services and collated damage costs associated with several pathways. Effects on ecosystem services have been included in this update to the damage costs based on the collated damage costs presented in this report.

Alongside collating the damage costs, Jones et al (2014) also provided direction on the rigour of the value estimate. To do so they scored each damage cost as either '## Robust', '# Acceptable' or '(#) Improvements desirable and not currently acceptable for policy appraisal'. IGCB have directed that all pathways scored either '## Robust' or '# Acceptable' should be included in the updated damage costs, and should be included in both the low and high sensitivity around central costs.

Jones et al (2014) also provided uncertainty ranges around the valuation of each damage. Following steer from IGCB, for those pathways included based on the rigour of the estimate, the low valuation sensitivity is included in the low damage cost, the central in the central damage cost and the high in the high damage cost.

The pathways included in the updated damage costs and the sensitivity range around the central valuation are presented in Table 17.

Table 17 – Ecosystem service impacts included in the updated damage costs based on Jones et al (2014)

			Provisioning services			Regulating services			Cultural services	
Pollutant	Unit	Sensitivity	Crop production	Timber production	Livestock production	CO_2 GHG Emissions	N_2O GHG Emissions	CH_4 GHG Emissions	Recreational fishing	Biodiversity
NO_2	£/tonne (2014 prices)	Central	-	-4.30	-8.80	-54.00	11.80	-	0.10	102.80
		Low	-	*-2.30*	*-5.60*	*-22.80*	*6.20*	-	*0.10*	*33.30*
		High	-	*-8.00*	*-11.80*	*-94.00*	*18.70*	-	*0.10*	*237.40*
NH_3	£/tonne (2014 prices)	Central	-	-93.10	-294.10	-1,267.10	338.40	-	2.20	413.80
		Low	-	*-49.70*	*-186.60*	*-535.40*	*179.10*	-	*2.20*	*139.10*
		High	-	*-170.70*	*-395.90*	*-2,204.00*	*537.40*	-	*2.20*	*1,021.50*
SO_2	£/tonne (2014 prices)	Central	-	-	-	-	-	-5.30	-	-
		Low	-	-	-	-	-	*-1.60*	-	-
		High	-	-	-	-	-	*-9.50*	-	-
O_3^*	£/ppb (7-month 24-hour mean) (2014 prices)	Central	-	-	1,051,000	5,740,000	-	-	-	-
		Low	-	-	*427,000*	*3,866,000*	-	-	-	-
		High	-	-	*1,705,000*	*7,939,000*	-	-	-	-
O_3^*	£/POD (2014 prices)	Central	100,555,000	-	-	-	-	-	-	-
		Low	*83,421,000*	-	-	-	-	-	-	-
		High	*118,970,000*	-	-	-	-	-	-	-

'-' denotes no relevant impact / no impact assessed, * Jones et al (2014) present costs as a negative integer and benefits as a positive integer for decreases in NO_2, NH_3 and SO_2 emissions and increases in O_3 metrics. This table presents costs as positive integers, associated with an additional unit of pollution (to be consistent with the way damage costs are presented in the rest of the report). As such we have reversed the sign of the values for O_3 impacts so that costs are shown as a positive integer associated with a unit increase for all pollutants (-ve numbers are benefits associated with an increase in emission).

4 Updates to damage cost sensitivities

4.1 Uncertainty in the estimation of damage costs

The estimation of the impacts of air pollution on both health and non-health pathways is inherently uncertain. The methodology for assessing the different impact pathways (which are subsequently aggregated to form the damage costs) is based on a number of assumptions around which there is a distribution of probable outcomes. The updated damage costs estimated under this project represent a best estimation of a 'central' damage cost estimate. However, there is uncertainty around: the emissions dispersion modelling, the interpretation of changes in air pollution concentrations into impacts and the valuation of those impacts.

The original set of damage costs attempted to illustrate the key uncertainties in the damage costs using sensitivity ranges around the central values. Two ranges were produced, a narrower 'low to high' damage cost range and a wider 'low sensitivity to high sensitivity' damage cost range. Given the importance of the impacts of long-term exposure to particulates in the overall damage cost calculation, these ranges explored the uncertainty around three key parameters in the appraisal of these impacts:

- CRF linking concentrations of particulates to health impacts: this varied between 1% and 12% impact per $10\mu gm^{-3}$ change in $PM_{2.5}$
- Lag time before the chronic mortality effect of particulates is felt: the lag time varied between 40 year and 0 year lag[8]
- Value of a life year (VOLY) lost: a range of monetary values were placed around the central VOLY estimate which varied from £26,300 to £43,800.

Further, a central estimate of the damage cost associated with each pollutant was derived using Monte Carlo analysis[9]. A Monte Carlo analysis was not within the scope of this current update and a central estimate based on central CRFs and other assumptions was considered appropriate, particularly given the revised approach to cessation lag adopted.

4.2 Areas of uncertainty for quantification

Given the changes in approach to estimating the damage costs under this project as outlined above, it has also been necessary to change the approach to illustrating the possible uncertainty range around the central damage cost estimates. In particular, although the CRF and VOLY ranges have been maintained in the analysis, this project has used the cessation lag approach to estimate the damage cost, which removes the ability to test uncertainty in lag times in the sensitivity analysis. As such, lags are no longer varied in the sensitivity range around the central estimates.

This project has developed uncertainty bounds around the central damage cost estimates based on the remaining two uncertainties associated with chronic exposure. Furthermore, the updated sensitivity bounds also capture several other uncertainties.

In this update, only one uncertainty range has been developed to reduce the complexity of the use and interpretation of the damage costs.

[8] Note: a longer lag time would produce a smaller damage cost as the impacts occur a longer distance into the future and hence are more heavily discounted.
[9] Further detail on the sensitivity analysis can be found in Annex 7 of Defra (2007)

4.2.1 Concentration response functions and adjustments

As in the original estimation of air pollution damage costs, the CRFs are varied between the low and high damage cost estimates.

For those pathways included in the central damage cost using the central CRF value, these are captured in the low damage cost applying the lower bound and in the high damage cost using the high bound of the CRF range.

Some pathways are excluded altogether from the central damage cost, and are only recommended for inclusion in the high damage cost (e.g. chronic bronchitis). Where this is the case, the pathways are only included in the high damage cost based on the central value of the CRF range. In addition to the new pathways discussed above, in the initial damage costs COMEAP (and subsequently IGCB) recommended a relationship between NO_2 and respiratory hospital admissions for quantitative analysis but noted that any impact should only be included as a sensitivity. As such, this project does not include this impact pathway in the low and central damage costs but adds it in for the high sensitivity.

A mapping of the point on the CRF range for each impact pathway across each damage cost is presented in Table 18.

Table 18 – Mapping of CRF bound chosen to each damage cost

		Damage cost sensitivity		
Pollutant	Pathway	Low	Central	High
$PM_{2.5}$	Chronic mortality	L	C	H
$PM_{2.5}$	Respiratory hospital admission	L	C	H
$PM_{2.5}$	Cardiovascular hospital admission	L	C	H
PM_{10}	Respiratory hospital admission	L	C	H
PM_{10}	Cardiovascular hospital admission	L	C	H
SO_2	Deaths brought forward	L	C	H
SO_2	Respiratory hospital admission	L	C	H
O_3	Deaths brought forward	L	C	H
O_3	Respiratory hospital admission	L	C	H
O_3	Cardiovascular hospital admission	L	C	H
NO_2	Respiratory hospital admission			C
NO_2	Chronic mortality	L	C	H
PM_{10}	Chronic Bronchitis			C
$PM_{2.5}$	CHD		C	H
NO_2	Asthma (Adults)			C
$PM_{2.5}$	Stroke		C	H
$PM_{2.5}$	Diabetes			C
NO_2	Diabetes			C
$PM_{2.5}$	Lung Cancer		C	H
NO_2	Lung Cancer			C
$PM_{2.5}$	Asthma (Older Children)		C	H
NO_2	Asthma (Small Children)		C	H
NO_2	Asthma (Older Children)		C	H
All	Productivity	L	C	H
All	Ecosystems	L	C	H

Note: L = Low end of CRF bound; C = central point of CRF bound; H = high end of CRF bound

For the effects of NO_2 on mortality, the sensitivity range also varies the adjustment applied to the CRF. This adjustment is applied to account for the overlap between the mortality impacts of NO_2 and PM.

An adjustment of 25%, 40% and 55% is applied in the low, central and high damage cost cases respectively to the coefficient linking chronic exposure to NO_2 and mortality.

4.2.2 Value of health impacts

Consistent with their original estimation, uncertainty around the value of health impacts is captured in the updated damage costs through flexing the unit impact values applied to the health outcomes between different damage cost sensitivities. This has been completed using the uncertainty bounds around valuation as set out in Table 16 above using ranges consistent with the original damage costs.

4.2.3 Value a proportion of acute deaths using the 'good health VOLY'

No range is recommended by the IGCB around the value of deaths brought forward from short term exposure and hence this value does not vary between low and high sensitivities. However, there is uncertainty around the quality of the life lost through the short-term mortality impacts of air pollutants.

As discussed in Defra (2007), it might be expected that acute deaths from respiratory disease occur in persons that are already ill. However, evidence suggests that for cardiovascular disease, some deaths occur in apparently healthy people (i.e. with no symptoms of prior underlying illness).

To address this uncertainty, the original damage cost report proposes that between 10 and 15% of acute deaths could therefore be valued using the 'good health VOLY' (value of life year lost in good health) used to value the effects of chronic mortality as a sensitivity. This project has included 15% of acute deaths being valued using this higher valuation in the high damage cost estimate.

4.2.4 Life-years-lost per acute death

In order to convert the number of deaths brought forward as a consequence of acute exposure to air pollution it is necessary to make an assumption around the number of months or years of life lost by an affected individual. COMEAP's estimate of between 2 and 6 months per death is recommended by the IGCB as the best estimate to use. It is important to note that there is still uncertainty around the amount of life lost through acute effects and this range was mainly inferred by COMEAP from the underlying evidence base rather than being based on direct evidence (for comparison the EU CAFE approach to the estimation of impacts assumes one life-year lost per acute death).

For this project, we have followed published IGCB guidance and have assumed the lower (2 months) and higher (6 months) levels of life lost under the low and high damage cost estimate respectively. For the central estimate, the project has assumed a central value of 4 months of life lost per death.

5 Updated damage costs

5.1 Damage costs update 2019

The updated set of damage costs are presented in the following tables, alongside the low and high estimated sensitivities around the central values. These values represent the damage costs associated with pollutant emissions in 2017, presented in 2017 prices. All sustained impacts of pollutant emissions have been discounted back to 2017. A positive damage cost represents a cost associated with an increase in pollutant emissions or a benefit associated with a decrease in pollutants emissions.

Note that for these revised damage costs the change in $PM_{2.5}$ emission is the preferred metric for PM emissions. The IGCB CRF for chronic mortality is for the impact of changes in $PM_{2.5}$ concentrations. Likewise, all pathways extracted from the PHE model associated with particulate matter are also expressed as $PM_{2.5}$. The IGCB CRFs for hospital admission and chronic bronchitis associated with PM are for PM_{10}, rather than for $PM_{2.5}$. For ease of use and given the dominant contribution of the chronic mortality pathway to the total damage costs, it is recommended that all changes in PM emissions valued using these updated damage costs are expressed as changes in $PM_{2.5}$ emissions. An adjustment is made to the PM_{10} pathways included for the ratio of primary $PM_{2.5}$ to PM_{10} emissions such that the change in emissions is expressed correctly when combined with these pathways. Ratios have been calculated from the NAEI emissions for 2013. The value for UK total emissions ($PM_{2.5}/PM_{10}$) is 0.642. Sector specific ratios have been used for the individual emissions sectors and these vary from 0.218 for agriculture to 1.000 for aircraft.

These damage costs have been produced applying an adjusted coefficient for long term mortality effects associated with exposure to NO_2 following COMEAP's advice for assessing 'interventions primarily target NOx' reflecting IGCB's steer. It is important to note that strictly COMEAP's recommendation regarding the estimation of mortality effects and the overlap with PM focused only road traffic emissions. This reflects that the epidemiological evidence for the CRF comes from studies where the main driver for the spatial variation in air pollutant concentrations was emissions from road traffic). The mix of 'all pollutants' emitted for other sectors is likely to be different because for most sectors the source emitting are not engines. Thus, using the adjusted NOx coefficient applied here may be considered less applicable, increasing uncertainty of applying these damage costs.

National damage costs are listed in Table 19. The damage costs for VOC include impacts via the O_3 pathways only. Sector specific damage costs for $PM_{2.5}$ and NO_x are provided in Tables 20 and 21.

Table 22 disaggregates a selection of the damage costs by their contributing impact pathways, including the low and high sensitivity damage costs. It can be seen from this table that:

- the impacts of long-term exposure to pollutants on mortality continue to be the most dominant impact valued across all damage costs.
- For PM, NH_3 and SO_2 damage costs, chronic exposure to PM is the dominant impact, irrespective of whether this is a primary or secondary impact. In fact, this impact contributes over 60% of the national average PM damage cost.
- For the NOx damage cost, chronic exposure to PM on mortality is still an important effect (but in this case PM is a 'secondary' pollutant), but the mortality effect of chronic exposure to NO_2 is the most significant pathway. This is the case even though the adjustment to account for the overlap between the two chronic effects has been applied to the NO_2 impacts, rather than the PM effects.
- Another key impact pathway is the new PHE effect estimating impacts on asthma in children
- Most other pathways are relatively small.

The balance of impacts is similar under the low and high damage costs. However, under the low damage cost the chronic mortality effects of primary exposure become even more significant as the PHE morbidity pathways are removed. Under the high, chronic mortality effects and asthma in children

are important, but asthma in adults and chronic bronchitis pathways added under this sensitivity are also key contributors. In fact, for the national average PM damage cost, chronic bronchitis is as large an effect as chronic mortality. This impact is only included as part of the sensitivity given concerns raised by COMEAP around the causality of effects. However, if it is assumed that there is a causal effect, this would have a significant impact on the valuation of damages associated with exposure to PM.

Table 19 – Revised national damage cost estimates and sensitivity bounds (2017 prices, impacts discounted to 2017). $PM_{2.5}$ is the preferred metric for the change in PM emissions

Pollutant Emitted	Central Damage Cost (£/t)	Low – High damage cost sensitivity range (£/t)	
		Low sensitivity damage cost	High sensitivity damage cost
NOx	6,199	634	23,153
SO_2	6,273	1,491	17,861
NH_3	6,046	1,133	18,867
VOC	102	55	205
$PM_{2.5}$	105,836	22,588	327,928

Table 20 – Revised sector PM damage cost estimates and sensitivity bounds (2017 prices, impacts discounted to 2017). $PM_{2.5}$ is the preferred metric for the change in PM emissions

Pollutant Emitted	Central Damage Cost (£/t)	Low – High damage cost sensitivity range (£/t)	
		Low sensitivity damage cost	High sensitivity damage cost
$PM_{2.5}$ Part A Category 1	8,666	2,473	25,060
$PM_{2.5}$ Part A Category 2	37,087	8,350	113,161
$PM_{2.5}$ Part A Category 3	81,059	17,444	249,465
$PM_{2.5}$ Part A Category 4	2,989	1,299	7,462
$PM_{2.5}$ Part A Category 5	6,392	2,002	18,013
$PM_{2.5}$ Part A Category 6	9,708	2,688	28,293
$PM_{2.5}$ Part A Category 7	2,557	1,209	6,125
$PM_{2.5}$ Part A Category 8	3,355	1,374	8,598
$PM_{2.5}$ Part A Category 9	4,223	1,554	11,289
$PM_{2.5}$ Industry (area)	95,847	20,679	308,503
$PM_{2.5}$ Commercial	63,797	13,636	183,869
$PM_{2.5}$ Domestic	85,753	18,171	247,526
$PM_{2.5}$ Solvents	194,078	41,485	692,660
$PM_{2.5}$ Road Transport	203,331	42,713	625,927
$PM_{2.5}$ Aircraft	194,269	40,571	560,317
$PM_{2.5}$ Offroad	153,487	32,181	446,162
$PM_{2.5}$ Rail	163,413	34,240	476,129
$PM_{2.5}$ Ships	33,739	7,443	97,124
$PM_{2.5}$ Waste	162,082	34,067	484,553
$PM_{2.5}$ Agriculture	46,442	11,732	192,401
$PM_{2.5}$ Other	251,877	52,538	738,774
$PM_{2.5}$ Road Transport Central London	1,111,831	230,582	3,430,456
$PM_{2.5}$ Road Transport Inner London	1,132,776	234,913	3,495,112
$PM_{2.5}$ Road Transport Outer London	602,201	125,195	1,857,233
$PM_{2.5}$ Road Transport Inner Conurbation	420,523	87,626	1,296,397
$PM_{2.5}$ Road Transport Outer Conurbation	250,221	52,409	770,676
$PM_{2.5}$ Road Transport Urban Big	305,377	63,815	940,942
$PM_{2.5}$ Road Transport Urban Large	247,045	51,753	760,871
$PM_{2.5}$ Road Transport Urban Medium	203,359	42,719	626,014
$PM_{2.5}$ Road Transport Urban Small	152,694	32,242	469,611
$PM_{2.5}$ Road Transport Rural	69,745	15,089	213,548

Table 21 – Revised sector NOx national damage cost estimates and sensitivity bounds (2017 prices, impacts discounted to 2017).

Pollutant Emitted	Central Damage Cost (£/t)	Low – High damage cost sensitivity range (£/t)	
		Low sensitivity damage cost	High sensitivity damage cost
NOx Part A Category 1	1,690	287	5,375
NOx Part A Category 2	2,701	365	9,362
NOx Part A Category 3	4,829	529	17,753
NOx Part A Category 4	1,625	282	5,119
NOx Part A Category 5	1,903	304	6,215
NOx Part A Category 6	2,576	355	8,871
NOx Part A Category 7	1,599	280	5,017
NOx Part A Category 8	1,665	285	5,277
NOx Part A Category 9	1,749	292	5,609
NOx Industry (area)	5,671	593	21,070
NOx Commercial	13,307	1,180	51,177
NOx Domestic	13,950	1,229	53,711
NOx Road Transport	10,699	980	40,896
NOx Aircraft	11,672	1,054	44,732
NOx Offroad	8,656	823	32,841
NOx Rail	9,009	850	34,230
NOx Ships	2,506	350	8,592
NOx Waste	6,766	677	25,391
NOx Other	7,426	728	27,990
NOx Road Transport Central London	57,517	4,576	225,472
NOx Road Transport Inner London	58,967	4,688	231,189
NOx Road Transport Outer London	31,326	2,564	122,215
NOx Road Transport Inner Conurbation	22,005	1,848	85,468
NOx Road Transport Outer Conurbation	13,200	1,172	50,754
NOx Road Transport Urban Big	16,010	1,388	61,834
NOx Road Transport Urban Large	12,994	1,156	49,940
NOx Road Transport Urban Medium	10,844	991	41,465
NOx Road Transport Urban Small	8,343	798	31,605
NOx Road Transport Rural	4,191	480	15,237

Table 22a – Updated national damage costs for 2017 and contributing pathways (£2017 prices, impacts discounted to 2017) - Central

Pollutant Emitted	NOx	SO_2	NH_3	VOC	$PM_{2.5}$
Damage Cost (£/t)	**6,199**	**6,273**	**6,046**	**102**	**105,836**
$PM_{2.5}$ Chronic mortality	593	2,305	2,528	-	40,238
PM_{10} Respiratory hospital admission	5	16	19	-	393
PM_{10} Cardiovascular hospital admission	3	10	12	-	240
SO_2 Deaths brought forward	-	14	-	-	-
SO_2 Respiratory hospital admission	-	26	-	-	-
O_3 Deaths brought forward	-9	-	-	4	-
O_3 Respiratory hospital admission	-47	-	-	18	-
O_3 Cardiovascular hospital admission	-4	-	-	2	-
NO_2 Respiratory hospital admission	-	-	-	-	-
NO_2 Chronic mortality	2,223	-	-	-	-
$PM_{2.5}$ Productivity	52	201	221	-	3,515
PM_{10} Productivity	-	-	-	-	-
O_3 Productivity	-56	-	-	22	-
O_3 Productivity	-	-	-	-	-
NO_2 Productivity	-	-	-	-	-
O_3 Material damage	-18	-	-	5	-
PM_{10} Building soiling	-	-	-	-	881
SO_2 Material damage	-	237	-	-	-
SO_2 Ecosystems	-	-6	-	-	-
O_3 Ecosystems	-18	-	-	11	-
O_3 Ecosystems	-19	-	-	40	-
NO_2 Ecosystems	63	-	-	-	-
NH_3 Ecosystems	-	-	-539	-	-
PM_{10} Chronic Bronchitis	0	0	0	-	0
$PM_{2.5}$ CHD	417	1,620	1,777	-	28,282
NO_2 Asthma (Adults)	0	-	-	-	-
$PM_{2.5}$ Stroke	157	610	669	-	10,642
$PM_{2.5}$ Diabetes	-	-	-	-	-
NO_2 Diabetes	-	-	-	-	-
$PM_{2.5}$ Lung Cancer	10	39	43	-	687
NO_2 Lung Cancer	-	-	-	-	-
$PM_{2.5}$ Asthma (Children)	309	1,201	1,317	-	20,959
NO_2 Asthma (Small Children)	1,958	-	-	-	-
NO_2 Asthma (Older Children)	580	-	-	-	-

Notes: Resp. HA = Respiratory Hospital Admission; CV HA = Cardiovascular Hospital Admission

Table 22b Updated national damage costs for 2017 and contributing pathways (£2017 prices, impacts discounted to 2017) - Low

Pollutant Emitted	NOx	SO_2	NH_3	VOC	$PM_{2.5}$
Damage Cost (£/t)	**634**	**1,491**	**1,133**	**55**	**22,588**
$PM_{2.5}$ Chronic mortality	299	1,163	1,276	-	20,303
PM_{10} Respiratory hospital admission	2	5	6	-	133
PM_{10} Cardiovascular hospital admission	1	3	4	-	84
SO_2 Deaths brought forward	-	7	-	-	-
SO_2 Respiratory hospital admission	-	9	-	-	-
O_3 Deaths brought forward	-2	-	-	1	-
O_3 Respiratory hospital admission	-6	-	-	2	-
O_3 Cardiovascular hospital admission	1	-	-	-0	-
NO_2 Respiratory hospital admission	-	-	-	-	-
NO_2 Chronic mortality	366	-	-	-	-
$PM_{2.5}$ Productivity	18	68	75	-	1,187
PM_{10} Productivity	-	-	-	-	-
O_3 Productivity	-17	-	-	7	-
O_3 Productivity	-	-	-	-	-
NO_2 Productivity	-	-	-	-	-
O_3 Material damage	-18	-	-	5	-
PM_{10} Building soiling	-	-	-	-	881
SO_2 Material damage	-	237	-	-	-
SO_2 Ecosystems	-	-2	-	-	-
O_3 Ecosystems	-11	-	-	7	-
O_3 Ecosystems	-16	-	-	34	-
NO_2 Ecosystems	17	-	-	-	-
NH_3 Ecosystems	-	-	-227	-	-
PM_{10} Chronic Bronchitis	0	0	0	-	0
$PM_{2.5}$ CHD	-	-	-	-	-
NO_2 Asthma (Adults)	0	-	-	-	-
$PM_{2.5}$ Stroke	-	-	-	-	-
$PM_{2.5}$ Diabetes	-	-	-	-	-
NO_2 Diabetes	-	-	-	-	-
$PM_{2.5}$ Lung Cancer	-	-	-	-	-
NO_2 Lung Cancer	-	-	-	-	-
$PM_{2.5}$ Asthma (Children)	0	0	0	-	0
NO_2 Asthma (Small Children)	0	-	-	-	-
NO_2 Asthma (Older Children)	0	-	-	-	-

Notes: Resp. HA = Respiratory Hospital Admission; CV HA = Cardiovascular Hospital Admission

Table 22c Updated national damage costs for 2017 and contributing pathways (£2017 prices, impacts discounted to 2017) - High

Pollutant Emitted	NOx	SO_2	NH_3	VOC	$PM_{2.5}$
Damage Cost (£/t)	**23,153**	**17,861**	**18,867**	**205**	**327,928**
$PM_{2.5}$ Chronic mortality	976	3,793	4,159	-	66,207
PM_{10} Respiratory hospital admission	8	26	31	-	653
PM_{10} Cardiovascular hospital admission	5	16	19	-	396
SO_2 Deaths brought forward	-	23	-	-	-
SO_2 Respiratory hospital admission	-	44	-	-	-
O_3 Deaths brought forward	-26	-	-	10	-
O_3 Respiratory hospital admission	-124	-	-	48	-
O_3 Cardiovascular hospital admission	-17	-	-	7	-
NO_2 Respiratory hospital admission	94	-	-	-	-
NO_2 Chronic mortality	6,067	-	-	-	-
$PM_{2.5}$ Productivity	115	446	489	-	7,791
PM_{10} Productivity	7	22	26	-	545
O_3 Productivity	-179	-	-	70	-
O_3 Productivity	-4	-	-	1	-
NO_2 Productivity	-	-	-	-	-
O_3 Material damage	-18	-	-	5	-
PM_{10} Building soiling	-	-	-	-	881
SO_2 Material damage	-	237	-	-	-
SO_2 Ecosystems	-	-10	-	-	-
O_3 Ecosystems	-26	-	-	16	-
O_3 Ecosystems	-22	-	-	48	-
NO_2 Ecosystems	170	-	-	-	-
NH_3 Ecosystems	-	-	-675	-	-
PM_{10} Chronic Bronchitis	796	2,673	3,202	-	66,585
$PM_{2.5}$ CHD	1,229	4,775	5,237	-	83,356
NO_2 Asthma (Adults)	2,082	-	-	-	-
$PM_{2.5}$ Stroke	356	1,384	1,518	-	24,167
$PM_{2.5}$ Diabetes	417	1,619	1,776	-	28,266
NO_2 Diabetes	5,091	-	-	-	-
$PM_{2.5}$ Lung Cancer	21	82	89	-	1,424
NO_2 Lung Cancer	73	-	-	-	-
$PM_{2.5}$ Asthma (Children)	703	2,730	2,994	-	47,656
NO_2 Asthma (Small Children)	3,837	-	-	-	-
NO_2 Asthma (Older Children)	1,521	-	-	-	-

Notes: Resp. HA = Respiratory Hospital Admission; CV HA = Cardiovascular Hospital Admission

5.2 Updated damage costs and comparison to existing set

For comparison, the updated central damage costs are presented alongside the original set of costs and those published in 2015 in Table 23. The original central damage cost estimates have also been included in the table to provide a more direct comparison between the updated and original damage costs (both original and 2015 damage costs have been uplifted to 2017 prices to remove the impact of changing price base. Note – no adjustment has been made for wage growth between these damage cost sets).

Table 23 – Updated and original central national damage cost estimates

Pollutant	Original damage costs (£2005/t)	Original damage costs (£2017/t)	Damage costs 2015 (£2015/tonne)	Damage costs 2015 (£2017/tonne)	Updated damage costs 2019 (£2017/tonne)
NO_X	875	1,099	*	*	6,199
NOx Domestic	*	*	14,646**	15,251	13,950
NOx Industry***	*	*	13,131**	13,673	*
NOx Industry (area sources)***	*	*	*	*	5,671
NOx Transport Av	*	*	25,252**	26,295	10,699
SO_2	1,496	1,879	1,956	2,037	6,273
NH_3	1,884	2,367	2,363	2,461	6,046
VOC	*	*	*	*	102
$PM_{2.5}$	*	*	*	*	105,836
$PM_{2.5}$ Domestic	25,770	32,376	33,713	35,105	85,753
$PM_{2.5}$ Industry***	23,103	29,025	30,225	31,473	*
$PM_{2.5}$ Industry (area sources)***	*	*	*	*	95,847
$PM_{2.5}$ Transport Av	44,430	55,819	58,125	60,525	203,331
$PM_{2.5}$ Waste	19,105	24,002	24,994	26,026	162,082

* = no damage cost estimated
** NOx damage costs presented are those 'where PM not valued'
*** Between the 2015 and updated damage costs there was a slight adjustment to the coverage of the 'industry' damage cost. The 2015 costs aggregated point and area sources, whereas the updated damage cost only focuses on area sources as point sources are separated out in the 'Part A' damage costs.

How do the damage costs compare?

As can be seen from the table, the updated damage costs show variance from both the original and the latest published sets of damage costs. The variance differs by damage cost.

For NOx:

- the damage cost has increased significantly from the original damage costs due to the inclusion of the mortality effects associated with chronic exposure
- However, relative to the 2015 damage costs, all NO_x sector-specific damage costs have decreased significantly.

For PM:

- The change from original to 2015 damage costs was fairly small (this damage cost update focused on the addition of NOx chronic mortality affects)
- The damage costs increased significantly between the 2015 and this latest update to the damage costs

The changes in each damage cost will reflect the different updates to the methodology underpinning the estimation set out in this report. It is not possibly to fully disentangle which changes in the underlying damage cost methodology have contributed to the change in the different damage costs and to what extent. That said, the following section discusses some of the key changes to the methodology and how these have impacted the estimation.

Key changes impacting on the damage costs: concentration response coefficients

The original damage costs did not include a pathway for the impact of long-term exposure to NO_2 on mortality. The damage costs 2015 did include this pathway and used a coefficient recommended by COMEAP in their 2015 Interim statement on quantifying the association of long-term average concentrations of nitrogen dioxide and mortality (COMEAP, 2015b). This interim coefficient was 1.025 (95 percent confidence interval 1.01–1.04) per 10μgm^{-3} NO_2.

The NOx damage cost is observed to reduce between 2015 and 2019 sets. This is likely predominantly due to the way these chronic mortality effects associated with NOx are assessed. Although the coefficient applied in the 2015 set is similar to the updated coefficient recommended by COMEAP (2018a) of 1.023 (95% CI: 1.008, 1.037) per 10 μgm^{-3} NO_2, COMEAP also recommended that an adjustment of 25-55% be applied to this unadjusted coefficient (mid-point of range 40%) for interventions that primarily target emissions of NOx to account for overlaps with the effect of PM.

Key changes impacting on the damage costs: dispersion modelling

One of the key factors in the change in damage costs is likely to be the update to the pollutant emissions-to-concentrations modelling. The μgm^{-3} per tonne for the updated damage costs will incorporate changes to the emission inventory estimates of emissions, the spatial patterns of emissions and dispersion modelling between the original and updated damage cost calculations.

For NOx emissions, in contrast to the 2015 data costs, the updated damage costs (and split by sources type) are based on specific dispersion modelling for NO_x emissions and individual sources. This will have two effects that will have a substantial impact on the size of primary impact pathways associated with NO_2 and will result in significant reductions in the updated damage cost relative to the 2015 cost:

1. The updated costs will now reflect improved understanding of the dispersion of NOx emissions (i.e. the relationship between NOx emissions and NOx concentrations) in the damage cost

2. The updated costs will specifically take account of NOx to NO_2 chemistry and the fact that ambient concentrations of NO_2 are lower than ambient concentrations of NO_x (i.e. not all NOx is present in the atmosphere as NO_2)

In addition, changes to the modelling will also influence the size of the secondary impact pathways associated with NOx emissions: i.e. the contribution of NOx emissions to secondary PM. Again, two effects could reduce the updated damage cost relative to the 2015 costs:

1. The use of the PCM model emission sensitivity coefficients method has led to lower estimates of the impact of reductions in UK emissions of precursor gases on SIA concentrations, since this method takes better account of the complex, less than 1 to 1, response to changes in precursor emissions. In short, the updated modelling reflects an improved understanding of the non-linear chemistry and contribution of non-UK sources

2. The revised estimated impacts associated with PM pathways have been calculated using changes in ambient $PM_{2.5}$ concentrations in contrast to the original and 2015 damage costs, which used PM_{10}. In the updated modelling, the contribution of SIA to ambient $PM_{2.5}$ does not include part of the coarse mode of the nitrate SIA that contributes to ambient PM_{10} but not ambient $PM_{2.5}$ (Brookes et al 2015), which in turn will have an effect on the estimated size of secondary effects through impact pathways associated with PM.

For NOx, the use of specific dispersion modelling for NOx and accounting for NOx to NO_2 chemistry are the changes that account for part of the difference between the 2015 and updated damage costs (in addition to the updated concentration response coefficients for the NOx emissions primary target option).

For some sectors, the values have not changed very much, for others the larger changes reflect more significant changes, either in emissions, the spatial pattern of emissions or dispersion modelling. For example, the increase in the damage cost for PM from Waste is likely to be due to the inclusion of two emission sources in the revised damage costs for this sector that were not included in the version of the NAEI used for the previous damage costs. These two sources (small-scale waste burning and accidental fires-vehicles) are both present in urban areas, while many of the other sources included in this sector are in non-urban areas and thus contribute less per tonne emitted to ambient PM concentrations.

Key changes impacting on the damage costs: New impact pathways

Several new impact pathways have been included in the damage costs in this 2019 update. This includes impacts on productivity, ecosystems, and wider morbidity outcomes that have been quantified by PHE. These will have had a material impact on the values relative to previous sets of damage costs. In particular:

- Productivity impacts, namely impacts on work-days-lost (WDL) have increased the PM damage cost
- Ammonia's impact on CO_2 sequestration (captured under the ecosystem impacts) has reduced the ammonia damage cost significantly
- The addition of CHD, stroke and asthma in children has significantly increased all damage costs, and under the high damage cost, asthma in adults, diabetes and chronic bronchitis lead to a further increase.

Other changes impacting on the damage costs

Part of the difference between the damage costs is likely to be explained by the unit impact values applied to monetise health impacts. Although the values themselves have not changed substantially, the 2019 damage costs include an uplift to account for wage growth to 2017 (based on GDP per capita growth). Such an adjustment has not been made to the original and 2015 values for comparison (i.e. they do not account for wage growth between their publication and 2017).

For NOx, several 'negative' impacts (i.e. a cost or reduced benefit associated with emissions reductions) through ozone impact pathways have now been included in the damage cost. These impacts are negative as the modelling assumes an increase in NOx leads to a reduction in ozone and the associated health impacts. These impacts slightly reduce the size of the marginal valued impact: these impacts appeared not to be included in the original NOx damage cost.

Other changes that will have some (although a smaller) influence on the damage costs will include the update to the life-tables calculations, the move away from the use of cessation lag approach, and choice of a specific central CRF for chronic exposure impacts (rather than use of Monte Carlo approach) of particulate matter on mortality.

Impacts that may be expected to have smaller effects are the changes to the baseline population and health impact rates, which are likely to have shown less variation over time.

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