

## Valuing the Impacts of Air Quality on Productivity

**Final report** 



Report for the Department for Environment, Food and Rural Affairs

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## **Executive summary**

Air pollution can have damaging impacts on human health, productivity, amenity and the health of the environment. These impacts have an associated economic cost. Recent estimates suggest that particulate air pollution reduces average life expectancy in the UK by around 6 months per person, an impact valued at £16bn per annum. Defra has produced guidance to steer the assessment of air quality impacts and the valuation of associated economic costs. This guidance facilitates the appraisal of a number of impacts but several are not captured, including impacts on amenity, ecosystems and productivity.

Ricardo-AEA, alongside its partners the Institute of Occupational Medicine (IOM) and Metroeconomica, were commissioned by Defra to undertake a project to identify the links between air quality and productivity and where possible, develop a methodology with which productivity impacts could be assessed, quantified and valued. The aim of the project was to enable a more comprehensive valuation of impacts and improve understanding and communication of the significance of air quality. This project was carried out in the context of air quality impact studies in the EU and US which included an assessment of productivity effects in cost-benefit analysis (CBA) alongside other impacts.

Productivity typically represents the efficiency with which an input is used in the production process. The present analysis took a wider definition of productivity effects as impact pathways which affect the relationship between the output of an economic unit and its factor inputs (e.g. labour). The overall impact is measured by the consequent change in final output. Air pollution can therefore influence output through either the quantity of the factor input used (e.g. the amount of time people are at work) or the efficiency with which that factor is used in the production process (e.g. what someone produces while at work).

Based on this definition, an initial long-list of hypothetical impact pathways was defined through which air pollution can potentially influence productivity. These pathways are spread across three different factors of production: capital; labour and natural capital. The long-list was then refined based on the strength of evidence supporting whether each pathway exists and is significant for the UK and also whether necessary quantitative information was available with which impacts could be appraised.

Five pathways were identified as being suitable and feasible for quantitative assessment. 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. For each pathway, an appropriate concentration response function (CRF) was identified which defines a given impact per unit change in pollutant. This CRF is combined with the concentration of the pollutant to be assessed and applied to an appropriate population and background rate of health response. Combining these four parameters provides the estimated health impact burden associated with current levels of (or the impact of change in) air pollutants. The present project focuses

on the effect of current concentrations (the air pollution burden) but the proposed methodology can also be applied to assess the impacts of changes in pollutants.

The analysis of mortality effects mainly focused on the effect of long-term ("chronic") exposure to particulate matter (PM) on mortality in adults. This is generally the dominant health effect of outdoor air pollution in an all-ages analysis but it is unclear if this is the case for people of working age (where death rates are lower than in older people) and for employed people (where death rates may be lower still in comparison to unemployed people). The project also developed an assessment of the impacts of acute mortality (from  $O_3$  and  $NO_2$ ) on productivity.

The analysis of morbidity effects focussed principally on working days lost (WDL) as a result of exposure to PM which is the single, most direct measure of productivity available from the literature. However, the CRF by which PM impacts on WDL are assessed is highly uncertain as it is based on a study conducted in the US over 25 years ago. To test this uncertainty, we considered estimating morbidity effects indirectly through a "bottom-up" alternative approach. This looked at different health outcomes and attempted to estimate their combined effect on productivity. Although the methodology proposed uses the existing relationship to WDL, the alternative methodology proved a useful sense-check of the results and provides additional confidence in the results derived using the proposed approach.

The WDL CRF focuses on the effects of acute rather than long-term exposure. However, the present analysis considers that this CRF will capture impacts in both those with and without underlying chronic ill health, provided that they are in employment. A robust assessment of the impact on productivity of chronic morbidity could not be developed given concerns of double-counting with the WDL CRF and data limitations: it is difficult to estimate impacts indirectly given that many individuals with chronic illness could be fit enough to work on most days.

For all of the health endpoints considered (both short term and long term), it is important to note that air pollution is one of many causal factors. Individual cases will result from a combination of risk factors rather than solely as a result of air pollution. It is not possible to identify individual deaths or other events that solely result from air pollution. The quantification of effects is based on the well-known approach in health Impact Assessment of calculating the "attributable fraction": the proportional impact of the risk factor (in this case air pollution) on the total number of deaths or other specific health endpoints. In practice, a much greater number of people are likely to be affected to some degree by air pollution than implied by the number of attributable cases.

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. We have taken values of marginal productivity from the Confederation of British Industry's (CBI) 'Workplace Health and Absence' survey to capture the wage and non-wage, direct and indirect costs associated with absence. Some of the pathways taken forward were excluded from those recommended to be included in CBA given the potential overlap of valuation with existing appraisal guidance.

Combining the assessment of health impacts and valuation methodology, a proposed approach was developed with which the productivity impacts of air pollution can be assessed. The methodology assesses the burden associated with levels of, or impacts of a change in, pollutant concentrations for  $PM_{2.5}$ ,  $PM_{10}$ ,  $NO_2$  and  $O_3$  for a given year on an average basis across the UK as a whole. This methodology can be used to develop CBA estimates or impacts on the UK's GDP.

To demonstrate the use of the proposed methodology and assessment tool, the burden associated with levels of air pollutants for the UK in 2012 were assessed. This analysis shows that the burden associated with current levels of pollutants had a total cost of £2.7bn through its impact on productivity. Further, £1.1bn of these costs are additional to those that

would have been captured using the current appraisal guidance<sup>1</sup>. Not all of the impacts assessed would feed through to the measurement of GDP: considering only those impacts captured by GDP, the burden associated with current levels of pollutants could have reduced GDP in 2012 by around £1.7bn or by 0.11% of total GDP. The ability to capture these additional productivity impacts in policy or pollution appraisal going forward will increase the comprehensiveness of analysis and reduce the likelihood that the benefits of reducing pollution are under-estimated (these impacts are presented in the table below).

Analysis type	Coverage	£m (PV, 2012 prices)	% of GDP (2012)
For inclusion in Cost-benefit analysis	Total cost	1120	0.07%
Assessment of GDP Impact	Total lifetime cost	2308	
	First-year cost <sup>2</sup>	1730	0.11%
All impacts assessed	Total cost	2710	

#### Monetised burden associated with pollutant levels in 2012

Any methodology for estimating impacts is necessarily limited by the weakness of the underlying evidence base. Within these constraints, the methodology developed under this project is a practicable way forward that provides reasonable indicative estimates of impacts, avoiding major overlaps with the impacts captured under the current air quality appraisal guidance.

Further, it is important to note that the estimation of productivity impacts using the tool is inherently uncertain (the estimates above represent a central view of impacts) and the methodology is based on a number of assumptions. These factors should be taken into consideration when producing analysis using the assessment tool as they could imply the figures produced are either an over or under-estimate of the true impacts of air pollution.

In the tool we have included functionality to explore the potential impact of five key sources of uncertainty. The two parameters which drive greatest variation in impacts assessed are the estimation of health impacts (varying the CRFs between low and high values produces estimates of all impacts of £2.0bn and £3.4bn respectively) and the unit values of productivity (using a 'top-down' measure of a unit of productivity would increase the value of all impacts to £4.9bn).

In addition, through the development of the tool the project team have identified possible further actions which could improve the methodology. Specifically, it is recommended that Defra take into account any future discussions and conclusions of COMEAP related to the evidence base on which this methodology has been developed. They should also look to further discussion regarding the consistency of the approach to valuing health impacts in CBA across UK Government departments.

<sup>&</sup>lt;sup>1</sup> This (smaller) estimate is the value that is consistent with (and hence can be considered additional to) the previous estimated impact per annum of £16bn (although this was derived for an earlier year) <sup>2</sup> This is the cost in the year for which pollutants are assessed. This excludes monetised impacts from chronic effects in future years which will not

<sup>&</sup>lt;sup>2</sup> This is the cost in the year for which pollutants are assessed. This excludes monetised impacts from chronic effects in future years which will not impact on GDP in the first year of assessment

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## **1** Introduction

### 1.1 Air quality and impact valuation

The quality of the air around us has a strong influence on natural and man-made environments. Air pollution can have damaging impacts on human health, productivity, amenity and the health of the environment. These impacts have an associated economic cost. Recent estimates suggest that particulate air pollution reduces average life expectancy in the UK by around 6 months per person, an impact valued at £16bn per annum (Defra, 2010).

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 economic costs. This guidance supplements the Green Book (HMT, 2011) which provides wider guidance for impact assessment and valuation. These processes are designed to support evidence gathering to inform policy development or evaluation.

The existing air quality appraisal guidance facilitates the assessment of a number of impacts. Although this approach enables some key impacts to be monetised, a number of other impacts are not captured, including the impact on productivity. For social cost-benefit analysis (CBA) to be an effective tool with which to assess, compare and communicate policy impacts, all significant impacts need to be captured and monetised as far as possible.

### 1.2 Project aims and this report

Ricardo-AEA, alongside its partners the Institute of Occupational Medicine (IOM) and Metroeconomica, were commissioned by Defra to undertake a project to identify the links between air quality and productivity and where possible, develop a methodology with which productivity impacts could be assessed, quantified and valued.

This document is the final report of this project. It summarises: the methodology developed and the detail on which it is based, an illustrative assessment of the productivity burden associated with current levels of air pollutants, and instructions for using the tool as part of project or policy appraisal. The report is structured in the following way:

- Section 2 sets out in greater detail the objectives of this study and the approach taken
- Section 3 outlines the definition of productivity adopted and potential impact pathways
- Section 4 describes the approach taken to assess the health impacts of each pathway taken forward for assessment
- Section 5 describes the approach taken to value the economic cost of these impacts
- Section 6 assesses the productivity burden associated with current levels of pollutants and considers uncertainties around estimation
- Section 7 concludes with a discussion of recommendations for further evidence gathering.

## **2** Background and approach

## 2.1 Air quality appraisal guidance

The impacts of air pollution on human health, the health of the environment, amenity and productivity have an associated economic cost. These are known as external costs or externalities are not captured in the market price of the goods or services consumed that generate air pollution.

Cost-benefit analysis (CBA) is a tool commonly used to appraise policy options in Impact Assessment (IA) to support policy development. CBA attempts to value all costs and benefits associated with a policy including those not captured by market prices. The balance of costs and benefits in a comprehensive analysis subsequently informs whether such a policy could imply a net cost or benefit to society.

Specific guidance to steer the appraisal of air quality policy has been developed by Defra to supplement the wider Green Book guidance on IA. This guidance details three approaches to assessing and valuing the impacts of policy on air quality. It recommends analysts consider 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.

Many of the key impacts of air pollution are captured within this existing appraisal guidance. This includes the impacts of air pollution on: human health (chronic and acute mortality and hospital admissions), crop yields and building materials and soiling. However, not all impacts can be assessed using this guidance, including: amenity value of buildings, ecosystems and productivity effects. Given these impacts are not captured any estimation using this guidance is likely to under-estimate the true net cost of pollution.

Recent impact studies of air quality policy in the EU and US have sought to value productivity impacts in CBA alongside other impacts. These studies were reviewed in detail under this project and are referenced throughout this report. A brief summary of the approaches taken and impacts assessed are included in the information boxes below.

#### Information box: ExternE, CAFE, EC4MACS and air quality assessment at an EU level

The valuation of the impacts of air pollution at an EU level is based on the Atmospheric Long-range Pollution Health and environment Assessment model (or ALPHA) (EC4MACS, 2013). The original version of this model was developed by AEA-Technology in the 1990's, drawing extensively on the ExternE research programme. This model was then used, among other things, to assess the impacts of the National Emissions Ceiling Directive and the Gothenburg Protocol to the UN/ECE Convention on Long Range Transboundary Air Pollution<sup>3</sup>.

An updated version of the model was subsequently developed under the EU's Clean Air For Europe (CAFE) programme, and later reviewed and adopted as part of the European Commission's LIFE+ Programme's EC4MACS (European Consortium for Modelling of Air Pollution and Climate Strategies) Project.

<sup>&</sup>lt;sup>3</sup> The latest update to this analysis can be found at: AEA (2011)

A number of studies assessing the impacts of air pollution (or scenarios of pollution reduction) at the EU level have been completed using this framework<sup>4</sup>. This includes two relatively recent pieces of work: a technical briefing note supporting the Climate Cost study which assessed the ancillary air quality benefits of greenhouse gas (GHG) mitigation (Holland et al, 2011a and Holland et al, 2011b) and a CBA of emissions control scenarios after 2020 (Holland, 2012) as part of the Thematic Strategy on Air Pollution (TSAP)<sup>5</sup>. The latter study included updates to the methods used to account for new information from the Review of Evidence on the Health Aspects of Air Pollution (REVIHAAP; see WHO, 2013a) and the Health Risks of Air Pollution in Europe (HRAPIE; see WHO, 2013b) studies being led by the World Health Organisation (WHO). The HRAPIE project developed concentration response functions (CRF) for application in impact assessment and provide the new standard basis for analysis undertaken for the European Commission. This evidence was developed through an intensive review process involving a wide range of experts in air pollution and health.

This EU assessment framework (and the IAs based on this framework) captures a wide range of impacts associated with air pollution, including impacts on human health, crop yields and building amenity. Further, several of the morbidity health endpoints assessed explicitly capture the impact of lost productivity.

To value these impacts, a cost per day of work absence is derived from a survey by the Confederation of British Industry (CBI; see CBI, 1998). This is used to estimate the cost of: Restricted Activity Days or RADs (valued at £130 per day in addition to a value of the Willingness To Pay (WTP) of a person to avoid a RAD) and hospital admissions (valued using this unit cost of absence alongside a WTP to avoid admission and the resource cost of hospitalisation).

Other health endpoints are also assessed without productivity being explicitly valued as part of the assessment. In particular, the impact of air pollution on chronic and acute mortality is valued using both the Value of a Statistical Life (VSL) and Value of Life Year (VOLY) given ongoing debate regarding the most appropriate valuation methodology. Further, minor RADs (mRADs) are valued using the WTP of a person to avoid an mRAD.

The Climate Cost study concludes that there are substantial air quality benefits associated with the GHG mitigation scenario assessed. In 2050, the mitigation scenario results in 480,000 life years gained, 43m fewer RADs and 4.9m fewer mRADs annually, leading to a total co-benefit of mitigation of  $\in$ 43bn: the valuation of fewer RADs alone is estimated to be  $\notin$ 4.1bn (around 10% of the total estimated benefit).

#### Information box: US EPA air quality impact assessment

The Environmental Protection Agency (EPA) in the US has undertaken a number of air quality policy appraisals where costs and benefits are estimated<sup>6</sup>. This includes the relatively recent third update to the assessment of the Clean Air Act Amendments of 1990 (CAAA; see US EPA, 2011a). The analysis estimated the costs and benefits of reducing emissions of air pollutants by comparing a 'without-CAAA' against a 'with-CAAA' scenario. A critical tool in the US EPA assessment is the Environmental Benefits Mapping and Analysis Programme (BenMAP; see US EPA, 2012a), which captures and monetises a range of impacts associated with changes in air pollution.

A number of health outcomes explicitly capture the impact of air pollution on productivity. The Programme assesses the number of: working days lost (WDL) which are valued using the average wage rate of \$149 per day (2006 prices); school days lost (SDL) which are

<sup>&</sup>lt;sup>4</sup> These IAs are referred to collectively in this paper as either the 'EU' studies or 'EU CAFE' approach.

<sup>&</sup>lt;sup>5</sup> Further studies include: EU IA of the CAFE (EC, 2013) and report by HEAL (2010)

<sup>&</sup>lt;sup>6</sup> The IAs based on this approach are referred to collectively in this report as the 'US' or 'US EPA' studies or approach

converted to WDL (and valued in the same way as direct WDL) by adjusting for the likelihood that a SDL will lead to a WDL in a parent; hospital admissions which are valued using lost earnings plus direct medical costs; and non-fatal myocardial infarction which also include a valuation of lost earnings alongside other costs. Other health impacts are also captured where productivity impacts are not explicitly valued. This includes the impact on chronic and acute mortality which are valued using VSL and mRADs which are valued using a WTP of a person to avoid that health outcome.

The results of the third update suggest the CAAA has a significantly positive net present value (NPV) of \$2,000bn by 2020. The majority (85%) of benefits are the result of reductions in premature mortality but the reduction of WDL (17m by 2020 valued at \$2.7bn) and mRADs (110m by 2020 valued at \$6.7bn) are also significant impacts.

## 2.2 Project objectives and approach

The aim of this project was to identify the links between air quality and productivity and where possible, develop a methodology with which productivity impacts could be assessed, quantified and valued. This would subsequently enable a more comprehensive valuation of impacts and improve understanding and communication of the significance of air quality.

The specific objectives of the project were to:

- 1. Identify the pathways by which air quality affects productivity by conducting a review of existing literature
- 2. Assess the availability of information, strength of the evidence and ability to value impacts for each of these pathways and prioritise the order in which to consider them in greater depth
- 3. Propose possible valuation approaches for each pathway and recommend which would be most appropriate. Agree and develop this approach, including relevant sensitivities and consider the robustness of the approach
- 4. Produce an appraisal tool that applies the agreed methodology to estimate the monetary impact of a change in air quality on productivity
- 5. Produce a report detailing the work undertaken and including discussion of how the methodologies could be developed further and suggestions for future research.

Our project team comprised of experts from Ricardo-AEA, IOM and Metroeconomica. The expertise within the team has been instrumental in the development of research and methodology to quantify and value air quality impacts over the years.

The project was carried out in five stages. The initial scoping stage of the project defined productivity and developed an initial set of impact pathways. Second, a wide-ranging critical review of published research on the assessment and valuation of air pollution impacts from UK, EU and other international sources was completed. Third, based on the findings of this review, the long-list of potential pathways were 'prioritised' such that those where the evidence base was considered stronger were taken forward for quantification. Fourth, a methodology was developed to assess productivity impacts and estimate the associated economic cost. Finally, this methodology was then captured within a modelling tool for use in policy appraisal in the future. The rest of this document sets out the detail of the methodology developed and proposed under this project.

## **3 Impact pathways and productivity**

## 3.1 Defining productivity and air pollution impacts

Productivity typically represents the efficiency with which an input is used in the production process. The productivity of an input is commonly presented as the quantity (or value) of the total output per the quantity (or value) of the given input used to produce that output.

The objective of this project was to value the economic cost of air quality through its impact on productivity. The methodology developed to value these impacts can then be applied in CBA to support policy appraisal or evaluation. Given this objective, an impact on productivity was defined in this project as an impact pathway which affects the relationship between the output of an economic unit and the factor inputs which have gone into producing that output (Pass, et. al., 1988). The overall impact is measured by the consequent change in final output. This definition is consistent with that taken by air quality IAs in the US and EU.

The relationship between output and factor inputs is defined in the economic production function:

Production = Productivity of capital x amount of capital + Productivity of Labour x amount of Labour + Productivity of natural capital inputs x amount of natural capital inputs +  $...^7$ 

It is evident from this function that air pollution influences output through its impact on either the quantity of the factor input available or the efficiency with which that factor is used in the production process.

### 3.2 Identification of pathways and prioritisation

Based on this definition of productivity, an initial long-list of hypothetical impact pathways through which air pollution can potentially influence productivity was defined. These potential impact pathways are spread across three different factors of production: capital; labour and natural capital and are set out in Table 3.1 below. At this initial stage in the project this represented a list of hypotheses which could be tested through further evidence gathering.

This initial long-list of pathways was developed by considering how air pollution could influence each factor of production (or the efficiency with which this factor is used in the production process) in turn. This exercise was informed by: pathways known to be included in existing air quality IA (both in the UK and otherwise); pathways mentioned in wider air quality literature; and the expertise of the project team.

In this study, we have widened the typical interpretation of the definition of labour to include non-paid work such as caring for loved ones and voluntary work. Use of a broader interpretation was judged to be important since a number of health impacts resulting from poor air quality particularly afflict the retired population. This group in turn may be more likely to be responsible for care provision (e.g. to spouses, grand-children, etc.) or to undertake other forms of voluntary work in the community, rather than being paid monetarily for employment in the formal labour market.

<sup>&</sup>lt;sup>7</sup> Note: the inputs depicted in a production function will depend on the specific good or service being produced. The factor inputs specified above have been included as these were considered the inputs more likely to be influenced by air pollution.

Factor of production	Impact pathway	Potential productivity impact			
Pathways taken forw	Pathways taken forward for quantification				
Labour	Mortality (chronic and acute) in workforce	Persons are removed from employment prematurely through death			
Labour	Morbidity in the workforce (absenteeism)	Persons withdraw temporarily (or permanently) from employment through illness (e.g. missed days of work)			
Labour	Morbidity in the workforce (presenteeism)	Productivity of persons in employment is reduced through illness (where a person is not absent from work)			
Labour	Absence in the workforce due to morbidity in dependents	Persons withdraw temporarily from employment to care for dependents who are ill			
Labour	Health impacts (mortality and morbidity) in non-market productive activities	Persons are unable to undertake productive activities outside formal employment (e.g. caring or volunteering) due to illness or premature death			
Pathways not taken forward for quantification					
Capital	Air pollution curtails operation of sensitive capital assets	Productivity (or stock) of machinery that relies on clean air as an input is reduced			
Capital	Buildings and other fixed productive assets are corroded through acid rain	The stock of useable buildings or machinery is reduced through corrosive effects of acid rain			
Capital	Decreasing returns to capital curtails additional investment	In long-term, reduction in labour supply through mortality and morbidity reduces returns to capital, reducing investment in (and accumulation of) capital			
Labour	Impact of absenteeism on longer term productivity growth	Absence from work or school due to poor health reduces productivity of workers in the long-term due to reduced learning			
Labour	Air pollution and Visibility	Poor visibility causes lost work time (or lost productivity) either through delays in travel to work or interruption of outside occupations			
Labour	Indirect impacts on human health	Human health is affected through contamination of food and/or water supply by air pollution			
Natural capital	Impact on animal health	Air pollution reduces output of livestock agriculture			
Natural capital	Impact on outputs of commercial crops	Air pollution reduces crop yields of commercial crops			

#### Table 3.1 – Initial long-list of hypothetical impact pathways

Once the initial long-list was defined, the evidence supporting each pathway was then assessed against a simple set of decision criteria. This assessment informed whether each pathway could be prioritised: i.e. taken forward under the project to develop a methodology with which the impact could be quantified. The criteria used were:

- What is the strength of evidence that this impact exists? Is there evidence to suggest this pathway exists in the UK? Has this impact been included in other air quality impact assessments?
- What is the likely significance of the impact pathway? Is there evidence to suggest this pathway is significant in the UK?
- Does the necessary quantitative evidence exist with which an impact can be quantified?

Where pathways were not considered or quantified in existing air quality impact assessments, a wider evidence gathering exercise was completed to support the prioritisation process.

Using these criteria, five pathways were identified as being suitable and feasible for quantitative assessment. Eight pathways were de-prioritised and not taken forward for quantification. The pathways and recommendations for prioritisation were discussed and agreed with the Project Steering Group before the project progressed to developing a quantification methodology.

The pathways and a summary of the rationale underpinning the outcome of the prioritisation process are set out in Table 3.2. This presents a RAG (red-amber-green) rating for each pathway against each criterion to assess whether the evidence under each criterion prevented the pathway from being taken forward. A green rating suggests the criterion does not present a problem for robust quantification; red signifies that the evidence under that criterion implies quantification would be problematic and amber suggests that some evidence exists to support quantification, but the strength of evidence in this initial screening would lead to concerns regarding the robustness of any estimate.

It is important to note that not all pathways taken forward for assessment were included in the final assessment methodology. Throughout the development of the pathways taken forward, further consideration was given to the strength of evidence and appropriateness in the UK context which subsequently informed the final recommendation around pathways to be included in CBA.

The pathways taken forward for quantification focussed on the direct impacts of air pollution on human health via inhalation. These pathways subsequently impact on productivity through lost time participating in employment or non-market productive activities. In addition to having sufficiently robust evidence and data on which an estimate could be derived, it was considered appropriate to carry these pathways forward either because the pathway is captured by EU or US air quality IA approaches (i.e. absenteeism, presenteeism, and absenteeism due to dependents) or the health outcome is already captured in the existing UK appraisal guidance but the productivity impacts (both market and non-market) have not been directly explored (i.e. chronic and acute mortality). The methodology taken to quantify and value these impact pathways is set out in the following sections.

The remaining pathways were excluded from being taken forward as either: the impacts are already captured in the existing appraisal guidance (e.g. impacts on buildings and crop yields); there is no information to suggest that these potential pathways are significant in the UK (e.g. impacts on visibility, animal health, or indirect impacts on human health via consumption of food or water); or there is no information to suggest that this impact exists, either in the UK or more widely (e.g. longer-term impacts on learning or capital accumulation).

None of the pathways which are excluded from further analysis but deemed significant for the UK have been included in the published assessments of air quality under the EU CAFE programme or by the US EPA (except for impacts on crop yields and materials damage already captured by existing Defra appraisal guidance). Further description of the excluded pathways and evidence gathering against the criteria can be found in Appendix 2.

#### Table 3.2 – Summary RAG rating of long-list of pathways for quantification

Impact Pathway	Does evidence exist to support this impact pathway?	Is this pathway likely to be significant?	Does information exist with which a quantitative impact can be estimated?	Pathway carried forward for quantification?
Pathways taken forward for quantification	·			
Mortality (chronic and acute) in workforce	Yes – loss of life (but not productivity directly) is assessed in existing Defra appraisal guidance	Yes – loss of life (but not productivity directly) is assessed in existing Defra appraisal guidance	Yes – loss of life (but not productivity directly) is assessed in existing Defra appraisal guidance	~
Morbidity in the workforce (absenteeism)	Yes – included in EU / US IAs	Yes – included in EU / US IAs	Yes – included in EU / US IAs	✓
Morbidity in the workforce (presenteeism)	Yes – included in EU / US IAs	Yes – included in EU / US IAs	Yes – included in EU / US IAs	✓
Absence in the workforce due to morbidity in dependents	Yes – included in US IAs	Yes – included in US IAs	Yes – included in US IAs	4
Health impacts (mortality and morbidity) in non-market productive activities	Yes – health outcomes included in existing UK / EU / US IAs but non-market impacts not explored directly	Yes – health outcomes included in existing UK / EU / US IAs but non-market impacts not explored directly	Yes – health outcomes included in existing UK / EU / US IAs but non-market impacts not explored directly	4
Pathways not taken forward for quantification	on			
Air pollution curtails operation of sensitive capital assets	No evidence of direct impact but some machinery has preventative measures	No evidence to suggest impacts are significant in UK	No evidence linking changes in air pollution to impacts on capital assets	×
Buildings and other fixed productive assets are corroded through acid rain	Yes – included in existing Defra appraisal guidance	Yes – included in existing Defra appraisal guidance	Yes – included in existing Defra appraisal guidance	×
Decreasing returns to capital curtails additional investment	Impact theoretical - no evidence of direct impact of air pollution	No evidence of direct impact of air pollution	No evidence of direct impact of air pollution	×
Impact of absenteeism on longer term productivity growth	No evidence of direct impact but link from lost learning to reduced productivity acknowledged more widely	No evidence of direct impact of air pollution	No evidence of direct impact of air pollution	×
Air pollution and Visibility	Yes – included in US IA and noted in UK historically	No evidence to suggest impact is significant in UK currently	No – literature review suggests US concentration response functions may not be applicable	×
Indirect impacts on human health	Yes – potential impacts are documented qualitatively in air quality IAs	No evidence to suggest impacts are significant in UK	No evidence linking changes in air pollution to indirect health impacts	×
Impact on animal health	Yes – potential impacts are documented qualitatively in air quality IAs	No evidence impact in UK is significant	No evidence of direct link from air pollution	×
Impact on outputs of commercial crops	Yes – included in existing Defra appraisal guidance	Yes – included in existing Defra appraisal guidance	Yes – included in existing Defra appraisal guidance	×

## **4 Impact pathways for analysis**

## 4.1 Introduction

The prioritisation process narrowed down the long-list of potential impact pathways to identify five to be taken forward for quantification. The pathways included were those where the initial screening of evidence suggested that it could be significant in the UK and relevant information could be available to produce a quantitative assessment.

The pathways taken forward focussed on human health related impacts of air pollution and the subsequent impact on productivity. These pathways were:

- Mortality (chronic and acute) in working age population
- Acute morbidity in working age population (will also include acute effects arising from chronic morbidity)
- Morbidity in dependents of working population
- Reduced efficiency in workplace due to ill health (presenteeism)
- Mortality and morbidity in (non-working) carers and voluntary sector.

The analysis of mortality effects mainly focused on the effect of long-term ("chronic") exposure as this impact in adults is widely understood to be the dominant health effect of outdoor air pollution. Certainly this is true in an all-ages analysis but as discussed below, it is unclear if this is the case for people of working age and more specifically, the working population. The project also developed an assessment of the impacts of acute mortality on productivity.

Our analysis of morbidity effects focussed principally on an acute measure of morbidity: working days lost (WDL). This was the single most direct measure of productivity available from the literature. The main available study of the effect of air pollution on WDL focused on absences among employed people, regardless of whether or not they have any chronic underlying health conditions. It would be difficult to estimate the WDL that might arise from long-term health effects (such as chronic bronchitis) for which a concentration response function (CRF) is available, given that many affected individuals with chronic health conditions will be fit enough to work on most days. Even if it is possible to estimate WDL associated with chronic bronchitis, the inclusion of both acute and chronic effects could lead to some double-counting as most of the WDL associated with chronic ill health would arise from acute exacerbation of the individual's condition. However, limiting the quantification of morbidity to acute effects may lead to an under-estimate of overall productivity impact because a proportion of individuals who develop chronic illness may take early retirement as a result of ill health.

For all of the health endpoints considered (both short-term and long-term), it is important to note that air pollution is one of many causal factors. Individual cases will result from a combination of risk factors rather than solely as a result of air pollution. It is not possible to identify individual deaths or other events that solely result from air pollution. The quantification of effects is based on the well-known approach in health Impact Assessment of calculating the "attributable fraction": the proportional impact of air pollution on the total number of deaths or cases of other specific health endpoints. In the case of mortality, this distinction between "attributable cases" (i.e. an estimate of the total population effect of air pollution) and "etiological cases" (i.e. an estimate of the number of individuals whose life may have been shortened to some extent by air pollution) was recognised and discussed by COMEAP (2010).

To quantify the impacts under each pathway, the methodology follows an approach consistent with the widely recognised Impact Pathway Approach (IPA). For each pathway, an appropriate CRF is identified which defines a given impact per change in pollutant. This CRF is applied to an appropriate population in combination with the change in or current levels of pollutant to be assessed and the background rates of occurrence of the health response. Combining these four parameters provides an estimated health burden associated with levels of or impacts of changes in air pollutants which can then be valued.

The set of CRFs that are proposed under this project have been taken from a number of different sources, as set out in the remainder of this section alongside the rationale underpinning their selection. In particular this project has drawn heavily on the recent HRAPIE project (WHO, 2013b). For certain health endpoints, the methodology uses CRFs which have not been included in HRAPIE and the CRFs selected originate from different sources than those used in existing Defra guidance (Defra, 2013b). These include the US EPA Cost Benefit analysis of the Clean Air Act. Some further information on health endpoints that were not included in the earlier reviews and on the potential impact of predicted health outcomes on productivity was sought by searching PubMed (free to access online database of the medical literature maintained by the US National Institutes of Health Library) as described in Appendix 3.

The three different sets of CRFs (those used in existing Defra guidance, HRAPIE and recommended by this project) have been included in Appendix 4 for comparison and in the assessment tool developed to allow the user to compare the impacts of using the different CRF sets. This report only presents the results derived using the project recommended CRFs.

### 4.2 Chronic exposure and mortality (PM)

#### 4.2.1 Background and productivity impact

The effect of long-term exposure to particulate matter (PM) on adult mortality is generally regarded as the most important health impact associated with air pollution. In the current air quality appraisal guidance, a methodology exists to estimate the impacts of air pollution on chronic mortality (i.e. on mortality from long-term exposure).

Chronic (and acute) mortality can impact on productivity and economic output where the person affected was in employment at the time of death or where the person retired from employment on grounds of ill health attributable to air pollution<sup>8</sup> prior to death. These outcomes remove that person from the workforce with an associated loss of days or years that would have otherwise been worked had that person lived longer. Most attributable deaths are in those aged 65 or more and consequently the impacts of PM on mortality at younger ages may have a smaller impact on productivity than the impacts of morbidity. One purpose of the present project was to establish if this is so.

Of the various ways of presenting the mortality effects of air pollution, COMEAP (2010) suggest that population total survival time is the most appropriate approach. This can be estimated using attributable deaths and associated life-years-lost (LYL) per death. This calculation would provide an estimate of the aggregate LYL across the whole population. To calculate productivity impacts, subsequent additional steps are required to illustrate what proportion of this time could have been spent in productive activities which are lost through the effects of air pollution.

COMEAP (2010) distinguishes calculations and indices relevant to the two different kinds of question, which it calls (a) *the burden question*, i.e. what is the current annual mortality effect

<sup>&</sup>lt;sup>8</sup> As discussed in Section 4.5, the present analysis focuses only on the impacts on persons employed at the time of death and does not capture the impact where persons withdraw from the labour force early due to chronic health conditions.

of long-term exposure to air pollution at levels similar to those occurring now; and (b) *the impact question*, i.e. what is the effect on mortality, over a time-period to be specified, of a *change* in air pollution levels. The impact question is generally more relevant to policy analysis but, as described by COMEAP (ibid), when assessing impacts careful analysis requires that consideration be paid to dynamic population effects (i.e. how over time changes in risks affect the size of the population and so the numbers of attributable deaths per year). These effects are usually captured in the calculation known as the 'life-tables approach'. For this study we favoured simpler calculations based on mortality burden overlooking these time-related difficulties and looked on the effect of a change as the difference between burdens from two scenarios each of which has reached long-term stability. This involves assumptions about the time-relatedness of exposure and effect which for some pathways, in particular mortality risks of long-term exposure to PM<sub>2.5</sub>, do not account for these important time dependencies (COMEAP, ibid). However this approach was a necessary simplification which allowed the project to focus on interpretation of results (what metric to use and what it means for productivity) rather than the calculations themselves.

#### 4.2.2 Concentration response functions

#### 4.2.2.1 Particulate matter

In this project the analysis has focussed on using the CRFs developed on behalf of the WHO for application within the EU by HRAPIE. These CRFs will provide the new standard basis for analysis for the European Commission and were developed through an intensive review process involving a wide range of experts in air pollution and health.

There may be differences in the UK population age structure and health relative to the populations included in the HRAPIE analysis that affect the applicability of the CRFs. However, HRAPIE gives a credible, up-to-date framework and the focus of the present project is on how estimated effects of air pollution on health translate into effects on productivity. It is not intended to re-examine all existing issues on estimating air pollution health effects UK-wide. These CRFs have not yet been considered by COMEAP who may in due course recommend some differences for standard applications in the UK.

For chronic mortality, HRAPIE recommend the CRFs for  $PM_{2.5}$  derived in the Hoek et al (2013) meta-analysis of 13 different studies undertaken in Europe and North America. The authors derived a Pooled Effects Estimate of excess risk per 10 µgm<sup>-3</sup> increase in annual average  $PM_{2.5}$  of 6% (95% Confidence Interval (CI) of 4% to 8%) for "all-cause" mortality based on 11 studies and 11% (95% CI 5-16%) for cardiovascular (CV) causes based on 10 studies. The all-cause coefficient is consistent with those recommended by COMEAP (2009 and 2010) and used in the UK in recent years. The current COMEAP recommendations are based on results from the American Cancer Society (ACS) study only (Pope, 2002). The new HRAPIE CRFs, although practically identical, are better in that they are based on much wider evidence. Hence as well as drawing on a wider evidence base, they have smaller CIs than those from the ACS study used currently in the UK.

Hoek et al noted that there was wide variability in  $PM_{2.5}$  effects reported in different studies and suggested that this was probably due to differences of particle composition, indoor air quality, population differences and differences in exposure assessment and confounder control. Although Hoek et al suggested that differences in the populations studied were a possible cause of the variability of the  $PM_{2.5}$  effects on mortality they were unable to draw conclusions on which factors were most important as other confounding factors may have led to the differing outcomes. Some of the studies reviewed by Hoek et al found limited evidence of higher percentage increase in mortality due to  $PM_{2.5}$  in subjects of lower educational status and in obese subjects, which may reflect a wider range of factors such as lower dietary intake of fruit and antioxidants.

#### 'Age-specific' or 'working population specific' effects

Typically quantification studies have investigated the mortality impacts of air pollution in adults at all ages above 30 years, rather than focussing on age-specific impacts. However, given a person's productivity changes substantially with age and employment status, it is important to consider the implications of using all-age CRFs for this project.

In an all-ages analysis a substantial proportion of the number of deaths is in the 65+ age group because the death rate (all internal causes) increases markedly with age. This has an important bearing on mortality at working age below 65. HRAPIE indicate that the source epidemiological data from air pollution cohort studies do not provide a strong evidence base for deriving age variable coefficients for the CRF for mortality effects, partly because there is limited power to estimate effects in the younger age groups. We have not undertaken our own detailed assessment of all of the source studies underlying the HRAPIE recommendations for evidence of age-specific differences in relative risk (RR).

HRAPIE also describe how epidemiological studies of other risk factors for both ischaemic heart disease and stroke indicate that RRs decline with logarithm of age but it is not known whether this effect occurs with ambient air pollution. HRAPIE cite an analysis undertaken to support the WHO Global Burden of Disease Study by Lim et al (2012) that indicates that pooled epidemiological studies of CV risks show RR decreases with age in a roughly log linear factor with the RR reaching 1 at 100-120 years for all CV factors. It is difficult to translate this to the CRF linking  $PM_{2.5}$  to all-cause mortality but it seems likely that the CRF for the population under 65 is steeper, possibly much steeper, than for those over 65.

Considering the working population more specifically, one issue that is particularly difficult to address is that people in the workforce may on average be fitter and less susceptible to the adverse effects of air pollution than people of the same age who are not working. A proportion of those not working will have never worked because they have a congenital health issue or other long-term disability and therefore are potentially more susceptible. In addition, some are unable to work because of the long-term lack of employment opportunities and the adverse effects on health and life expectancy associated with deprivation may increase their susceptibility to air pollution.

In the absence of age-specific or working population specific CRFs, this study uses the allage CRF as recommended by HRAPIE. However, given that most deaths occur in older age groups, it is possible that CRFs based on whole population data (and therefore largely on deaths in older age groups) have limited predictive value specifically for younger age groups or for the working population and may over-estimate mortality impacts in the workforce. Given this lack of working-population specific or age-specific CRF, additional calculation steps are required to derive productivity impacts from the initial estimation of all-age chronic mortality impacts (as set out in Section 4.2.3 below).

#### All-cause and cause-specific CRFs

It is generally found that the major mortality impact of long-term exposure to PM is on cardiovascular rather than respiratory deaths; although coefficients are available variously for respiratory deaths and lung cancer as well as ischaemic heart disease and cerebrovascular disease. In contrast to the international experience reviewed by Hoek et al, recent UK studies have shown stronger relationships between PM and respiratory deaths than between PM and CV deaths (Yap et al, 2012; Beverland et al, 2012;Carey et al, 2013). Carey et al (2013) suggest that population differences, particularly more widespread use of statins, might have led to a smaller PM impact on CV deaths than observed elsewhere, but were unable to explain the size of the observed difference.

The present analysis uses the all-cause CRFs recommended by HRAPIE. However, the convention of using all-cause mortality rather than cause-specific mortality may become less tenable when assessing impacts in the working rather than overall population. This is due to the extent that the pattern of causes of death in the target population is different from the pattern of causes in the cohorts originally studied. The proportion of deaths due to CV or respiratory causes is lower in the working age population (around 27%) relative to the

population as a whole (43%)<sup>9</sup>. Hence using all-cause estimation could bias upward the number of "attributable" deaths.

These differences raise questions about whether the HRAPIE estimates need to be modified for use in the UK and it is likely that COMEAP will consider this in due course. For this project, we retain the use of all-cause CRFs in the first instance to maintain consistency with the considerations underpinning the assessment of mortality impacts in the existing appraisal guidance. It may be that the international values will remain the coefficients of choice. The numbers will change according to coefficient used; but this need not greatly affect the methodology of how to estimate the effect on productivity effects of how air pollution affects mortality.

Impact pathway	Concentration response function	Source
Chronic mortality in working age population	6% change in "all-cause" mortality (95% Confidence Interval (CI) of 4% to 8%) per 10 μgm <sup>-3</sup> increase in annual average PM <sub>2.5</sub>	Hoek et al (2013)

#### 4.2.2.2 Other pollutants

The recent WHO linked evaluations of REVIHAAP and HRAPIE summarise the available evidence linking mortality risks with long-term exposure to  $NO_2$  and to ozone. The evidence is less compelling than for long-term exposure to  $PM_{2.5}$ . For ozone, it is based on two different analyses of data from the American Cancer Society study. REVIHAAP and HRAPIE recommend inclusion in sensitivity analyses only.

For NO<sub>2</sub>, numerous studies give evidence of association and so, for quantification of longterm exposure to NO<sub>2</sub> and mortality in adults, HRAPIE recommend the CRF derived in the Hoek et al (2013) meta-analysis for all-cause mortality (based 11 studies) of a 5.5% (95% CI 3.1, 8%) increase per 10  $\mu$ gm<sup>-3</sup> increase in annual mean concentrations of NO<sub>2</sub>. HRAPIE indicate that impacts should only be calculated for NO<sub>2</sub> concentrations above 20  $\mu$ gm<sup>-3</sup> (effectively the annual mean concentration subtracting 20  $\mu$ gm<sup>-3</sup>).

There are however difficulties about the spatial scale on which NO<sub>2</sub> is assessed, about the independence or not of an NO<sub>2</sub> related effect from that of PM and about the extent of double-counting that would arise if mortality impacts are estimated for long-term exposure to both  $PM_{2.5}$  and NO<sub>2</sub> and then added. These difficulties are identified and addressed, though not completely resolved by REVIHAAP and HRAPIE, where it is re-stated that the most reliable relationship for quantification is that in long-term exposure to  $PM_{2.5}$ . Currently quantification of long-term exposure on mortality for policy making in the UK is based on  $PM_{2.5}$  only (COMEAP 2009 and 2010).

It was agreed between Ricardo-AEA, Defra and the Project Steering Group that the present project was not the place to try to resolve these difficulties with quantification of the mortality effects of long-term exposure to  $NO_2$  and whether or not these should be added to impacts via quantification in  $PM_{2.5}$ . Instead we would focus on mortality and long-term exposure to  $PM_{2.5}$ . This was considered sufficient for highlighting the methodological issues of how mortality effects from long-term exposure translate into reduced productivity. In addition it was agreed that the impacts of long-term exposure to  $NO_2$  would be noted as part of our exploration of uncertainties around the estimation of mortality impacts but quantifications of each pathway would not be added together to derive a total estimated impact.

We have included mortality from acute exposure (i.e. from daily variations, in time series studies) to both  $NO_2$  and ozone in our consideration of productivity impacts (see Section 4.3,

<sup>&</sup>lt;sup>9</sup> Figures based on mortality data for 2012 from ONS (see Section 4.2.3.3 below)

below). The likelihood of double-counting with these impacts is small as time series studies give better possibilities for separating the effects of particular pollutants<sup>10</sup>.

#### 4.2.3 Population at risk and metric

The productivity of a person (and hence the impact on productivity of the detrimental impacts of air pollution) differs significantly by age. This is because a person's engagement in productive activities, both inside and outside the formal labour market, varies with their stage in life.

As explained above, there is no CRF available to depict age-specific or working-population specific health impacts associated with air pollution. Hence to be able to derive an impact on productivity, the methodology first follows the conventional approach to develop an estimate for "total population survival time lost" (total life years lost or LYL) per year. This can be thought of as an aggregate of "deaths" and "life years lost per death". Additional calculation steps are then required to demonstrate what proportion of total life years lost (LYL) could have been productive.

#### 4.2.3.1 Estimating "attributable" deaths and total LYL

To estimate total LYL associated with a given concentration of air pollutants, the methodology first estimates the number of "attributable" deaths. To do this, it combines the all-age CRF with pollutant concentrations and underlying data on the numbers of deaths per annum in the UK.

Mortality data split into five-year age ranges (from age cohort 0-1 to 90+) was sourced individually for England and Wales (ONS, 2012a), Scotland (GRO-Scotland, 2012) and Northern Ireland (NISRA, 2012) and aggregated to the UK level. Data have been taken for 2012 as the latest year for which data are available<sup>11</sup>. The CRF is applied to all deaths excluding external causes and is applied consistently across all age cohorts over 30.

Each "attributable" death is then combined with an estimate of LYL per death to calculate total population survival time lost in units of LYL. In this case, the number of attributable deaths is multiplied by an average LYL per death. Total survival time lost can be most comprehensively estimated using age-specific estimates of LYL for each attributable death (where deaths are first distributed across age cohorts) and aggregating across all deaths. However, for this initial calculation, the use of age specific LYL would have no or negligible impact relative to using a population-wide average (in practice there may be some differences due to rounding). As such the approach adopted was considered suitable and pragmatic given the likelihood of negligible differences.

This analysis takes the average LYL per death calculated by COMEAP (2010) of 11.8 years and applies this to the number of "attributable" deaths to derive a central estimate of the total LYL. This average LYL was estimated for 2008: if this analysis were repeated for 2012, it is likely that the answer would be slightly different. However, given this would only imply a small difference we have used the available figure from COMEAP. This discussion of what LYL to attach to "attributable" deaths should be seen as part of a calculation of total population survival time in terms of LYL. As discussed in detail in COMEAP (2010) and summarised below, this does not reflect the number of individuals whose life is shortened by air pollution and the average life-shortening caused by air pollution rather than by other factors.

#### 4.2.3.2 Deriving an estimate of LYL falling within each age cohort

#### Depicting a number of deaths at each age cohort

COMEAP (2010) encouraged the use of total population survival time as the key metric with which to communicate mortality impacts. This is because total population survival time: (i)

<sup>&</sup>lt;sup>10</sup> Further, this project has used CRFs recommended by HRAPIE which were based on the results of 2-pollutant models where considered robust. <sup>11</sup> As such the numbers of deaths in 2012 are assumed to be representative of deaths in the assessment year for which the tool is used.

seemed to represent well the total impact of air pollution and mortality and (ii) was insensitive to the actual number of individuals affected (i.e. deaths), a number which strictly cannot be known. There are many combinations of "number of deaths" and "average LYL per death" that aggregate to the same total population survival time lost. In the same paper, COMEAP also note that it is not possible to know how LYL associated with chronic mortality may be distributed across individuals or across an age distribution.

However, a person's productivity (and hence the productivity impact of their death) varies substantially by age as represented by differences in employment rate (or involvement in non-market activities) and wage-rates across age cohorts. As such combining population-wide averages for employment rates (and wage rates) with total LYL could provide a misleading estimate of productivity impacts. These averages would not suitably represent the overlap between the likely distributions of air-pollution associated deaths and productive activities across age groups.

To be able to demonstrate the productivity impacts associated with chronic mortality effects, it was therefore necessary to: (i) define a number of deaths for use in the analysis and (ii) develop an illustration of how deaths and LYL could be distributed across ages. It is important to note that although the hypothetical illustration of how impacts are distributed across the population is useful in the calculation of impacts, it merely represents one of a number of possible ways of allocating total population survival time lost. Hence this approach should be regarded as a way of illustrating the overall effect and not as a literal description of what is actually happens to particular individuals in reality.

Once the total number of "attributable" deaths is estimated in the preceding step, it is then assumed that these deaths are distributed across age-cohorts in proportion to the spread of underlying all-cause deaths across age cohorts<sup>12</sup>. Of course, the distribution of "attributable" deaths across ages could be very different but in the absence of age-specific CRFs, this is a necessary but simplifying assumption. We use the distribution of all-causes of deaths rather than cause-specific data to remain consistent with the use of all-cause CRFs.

The methodology subsequently calculates the total number of LYL associated with deaths in each age cohort by combining the number of "attributable" deaths with the average value of LYL (11.8) used to calculate the overall number of LYL. This produces the same estimate of overall survival time lost as the first step of the calculation above (Section 4.2.3.1).

It is then necessary to depict the number of LYL which would have fallen between each age cohort (which implies a further step after deriving the total LYL associated with deaths at each age cohort as described above). This is required as although a death will fall in a given age cohort (determined by the age at death), some of the LYL associated with this death would fall in different age cohorts given the progression of a person through age cohorts over time. For example, a death in the 30-34 age range may have lived between 1 and 5 years in that range before moving up to the next age cohort of 35-39, leading some LYL to fall in this older category too. Given economic parameters vary across age bounds, this calculation is required to ensure the potential productive output of each potential LYL is approximated by the relevant economic parameters of the appropriate age group.

#### Developing a demonstration of how life-years lost could fall between age cohorts

To be able to depict the progression of LYL across age cohorts, we must again return to the assumption of average LYL per death and "attributable" deaths. To calculate total LYL and LYL allocated to deaths in each age cohort as described above, we have used the average value from COMEAP. However when depicting the counterfactual progression of an affected person across time, different considerations need to be taken into account.

The analysis produces an estimate of "attributable" deaths, a number which expresses the mortality impact of air pollution as if air pollution were the only cause of death. This is

<sup>&</sup>lt;sup>12</sup> Mortality data from the national statistics authorities are available in five-yearly cohorts. We have retained these cohorts in our analysis

convenient for these calculations because it provides a measure of the direct population impacts of air pollution rather than other factors on mortality. It may however not be the best means of deriving impacts on productivity.

As described by COMEAP (2010), "attributable" deaths are a measure of population effect and should not be interpreted in terms of individuals affected. The actual number of individuals affected is likely to be much greater as in most cases air pollution is a contributing factor in, but not the sole cause of, death. For example, COMEAP (ibid) estimated that there were about 29,000 deaths "attributable" to long-term exposure to air pollution per annum in the UK with an average LYL of 11.8 years. In reality a much larger number of individuals may experience some shortening of life due to air pollution. The number of deaths affected is likely to fall between two extremes: the number of "attributable" deaths representing a lower bound; and affecting all deaths at age over 30 to a much lesser extent.

COMEAP (2010) speculated that given that much of the impact of air pollution on mortality is linked with cardiovascular deaths, it would be more reasonable to consider that air pollution may have made some contribution to all CV deaths, about 200,000 per annum UK-wide, with an average LYL of less than 2 years per death (i.e. with the same overall population survival time lost).

If the issue were solely one of estimating total population survival time lost, then it would not matter which approach were used (as discussed above in Section 4.2.3.1). However, there are differences when deriving working LYL: working with "attributable" deaths and associated longer average LYL per death produces a lower estimate of total working LYL relative to the case where a larger number of individuals are assumed to be affected with a shorter average LYL per death. Under a longer average LYL per death, more persons are depicted to cross age-boundaries in the calculation and most importantly, more persons are depicted to cross the key age boundaries around retirement age where participation in the labour market significantly reduces, therefore reducing the number of productive LYL estimated.

Further, alongside influencing the number of working LYL, the choice of average LYL per death has two additional impacts on the estimated cost. Different average LYL will impact on the wage assigned to each working LYL and how many years into the future these impacts are reported to occur, impacting the extent to which these impacts are discounted. For the shorter average LYL, less discounting will be applied as these impacts only stretch over 2 rather than 11.8 years from the year in which the concentrations are assessed.

As under the calculation of total population survival time lost above, this project considered the use of age-specific estimates of LYL which differ by age cohort (i.e. using life-tables to derive age-specific LYL and attach these to age-specific attributable deaths). It is conceivable that using age-specific estimates of LYL here would depict a more representative illustration of how LYL fall across the age boundaries. However, it was not possible to use of age-specific estimates of LYL under this project given this data was unavailable. As such a population-wide average of LYL was used to depict the counterfactual progression of affected persons.

We have chosen to use a population-wide average LYL of 2 years with the associated larger value of deaths as our central estimate. Using "attributable" deaths and associated 11.8 LYL on average almost certainly focuses the effect on too few people: it was considered that there is a need not to base the analysis on "attributable" deaths which may be understood as impacting individuals as the main analysis. In contrast, a much more plausible alternative was to focus the effect on all CV deaths, even though this is still speculative. If all CV deaths are affected, then the relevant attributable LYL is 2.

#### 4.2.3.3 Deriving working LYL from LYL at each age

The total LYL at each age cohort are then combined with employment rates to generate the number of likely working LYL at each age cohort. This accounts for the fact that not all LYL will be productive: some people of working age are not available for employment, because of ill health, congenital disability, unpaid work (e.g. parenting or other care-giving), early

retirement or inherited income. Others may wish to work but are unable to find paid employment.

Data are available on the employment rate across different age cohorts from ONS's 'Pension Trends' (ONS, 2012b) and 'Labour Force Survey' (ONS, 2013a; LFS) publications. Although the data are available for more up-to-date years in the LFS<sup>13</sup>, the analysis for the present project uses data from the Pension Trends publication as it offers greater disaggregation of employment rates in older age cohorts: this is important as air pollution is likely to have greater impacts in older age cohorts. Comparing the two sets of data, there appears to be very little difference between the employment rates across age cohorts, which provides confidence that using the Pension Trends dataset is appropriate for 2012.

## 4.3 Acute exposure and mortality (NO<sub>2</sub> and O<sub>3</sub>)

#### 4.3.1 Background

Acute mortality (as with chronic mortality discussed in section 4.2 above) can impact productivity where employed persons are affected. The impacts of acute mortality are currently captured in existing Defra appraisal guidance and valued using willingness to pay (WTP) estimates. As with chronic mortality, we have developed an approach to directly assess the productivity impacts associated with acute mortality.

For acute mortality, the relevant RR's are derived from time series studies of daily pollution levels, where daily pollution may be characterised in a variety of ways (e.g. as 24-hour daily average or 8-hour daily max or 1-hour daily max). In these studies the time-period between exposure and effect is clear-cut: time series studies of air pollution and mortality focus on increased risk of death on the same day, or in the days immediately following, the day for which pollution is characterised and typically within one week. This is what is typically included in quantification (it is, for example, what has been used in the recent WHO project HRAPIE) and it is what we have assumed for the present project.

There is a much smaller evidence base of so-called distributed lag time series studies, which look at effects over longer time periods of up to about 6 weeks, and give higher RRs. To date, these have not been proposed for quantification by e.g. COMEAP, US EPA, WHO or the EU. Use of the distributed lag studies might give rise to some differences in interpretation of effects in relation to productivity; for now, we focus on the conventional approach.

As with chronic mortality, the effect is then expressed as "extra" or "attributable" deaths per annum, derived fundamentally as "extra" or "attributable" deaths per day aggregated over a year.

HRAPIE proposes CRFs for "acute mortality" based on  $PM_{2.5}$ ,  $NO_2$  and ozone. Following HRAPIE and indeed many previous Health Impact Assessment (HIA) projects, we will not include quantification in  $PM_{2.5}$  because of the strong likelihood that this would involve double-counting with the mortality effects of long-term exposure (see Section 4.3.3 below).

#### 4.3.2 Concentration response function

#### 4.3.2.1 Nitrogen dioxide

Air pollution is experienced as a mixture and as such it is difficult to identify the effects on mortality of different pollutants separately or even in combination; the roles of PM and nitrogen dioxide ( $NO_2$ ) are an important case in point. There has been considerable debate about the role of long-term exposure to  $NO_2$  in giving rise to adverse health effects and most quantification studies have focussed on PM in the belief that effects attributed to  $NO_2$  are largely due to concurrent exposure to some component of PM. Thus, for example, COMEAP

<sup>&</sup>lt;sup>13</sup> Last year of data available is for 2012 in LFS relative to 2011 in Pension Trends

(2009) concluded that quantification using  $PM_{2.5}$  was the best way of representing the mortality effects of long-term exposure to air pollution, and it did not recommend any additional quantification in long-term exposure to  $NO_2$ . This was consistent with other evaluations (e.g. WHO, EU, US EPA) at that time.

Anderson et al's (2007) meta-analysis of time series studies however provides strong evidence using single and multi-pollutant models that the NO<sub>2</sub> metric is independent of PM metric in acute mortality effects (although this finding does not eliminate the possibility that NO<sub>2</sub> is a marker of another pollutant). It is also biologically plausible that NO<sub>2</sub> acts independently of PM and there is small amount of experimental data showing adverse respiratory effects at concentrations experienced on high pollution days. This led to a recommendation for quantification by HRAPIE of various relationships linking NO<sub>2</sub> with health. The relationship with short-term exposure was considered among the most reliable for quantification (HRAPIE Group A); the RR per 10  $\mu$ gm<sup>-3</sup> NO<sub>2</sub> daily maximum 1-hour mean was given as 1.0027 (95% CI 1.0016 to 1.0038). This relationship is included in our quantification of acute mortality impacts. The RRs are based on the APHEA-2 analysis of data from 30 European cities and the RR is adjusted for PM<sub>10</sub>.

Impact pathway	Concentration response function	Source
Acute mortality in working age population (NO <sub>2</sub> )	0.27% change in "all-cause" mortality (95% Confidence Interval (CI) of 0.16% to 0.38%) per 10 $\mu$ gm <sup>-3</sup> NO <sub>2</sub> daily maximum 1-hour mean	Air Pollution and Health: a European Approach (APHEA)-2 project

#### 4.3.2.2 Ozone

HRAPIE indicate that the impacts of  $O_3$  for concentrations above 35 ppb (70 µgm<sup>-3</sup>) maximum daily 8-hour means (using the sum of means over 35 ppb: SOMO35) should be calculated using a linear function with RR coefficients of 1.0049 (95% CI = 1.0013, 1.0085) per 10 µgm<sup>-3</sup> for cardiovascular and 1.0029 (95% CI = 0.9989, 1.0070) per 10 µgm<sup>-3</sup> for respiratory mortality. Further, the RR coefficient for all-cause mortality recommended by HRAPIE was 1.0029 (95% CI 1.0014, 1.0043) per 10 µgm<sup>-3</sup> increase in daily maximum 8-hour mean. This relationship is included in our quantification of acute mortality impacts. The source of these coefficients is the APHENA study (Katsouyanni et al., 2009).

Impact pathway	Concentration response function	Source
Acute mortality in working age population (O <sub>3</sub> )	0.29% change in "all-cause" mortality (95% Confidence Interval (CI) of 0.14% to 0.43%) per 10 $\mu$ gm <sup>-3</sup> O <sub>3</sub> daily maximum 8-hour mean (using a threshold of 35 ppb (70 $\mu$ gm <sup>-3</sup> )	Katsouyanni et al. (2009)

HRAPIE also calculated the coefficients for all-cause and cardiovascular mortality for all ages in relation to daily maximum 1-hour mean. To do so they used a weighted average of the APHENA results for ages 75+ years and below 75 years, based on the proportion of subjects in the European population aged 75+ years (6.4% calculated as the mean of the city-specific proportions) in the APHENA study. Further, HRAPIE used a conversion factor of 0.72 between daily maximum 1-hour mean concentrations and daily maximum 8-hour mean concentrations that was derived in the APHENA study.

The acute mortality effects of ozone were considered to be part of the core set of CRFs for impact analysis (Group A). HRAPIE indicate that additional analysis for  $O_3$  concentrations above 10 ppb (20 µgm<sup>-3</sup>) using the sum of means over 10 ppb (SOMO10) should also be performed.

The APHENA study investigated age and employment status as possible effects modifiers but did not find a relationship. It did however find steeper CRFs for people aged under 75 than for the over 75 age group (see Table 4.1 based on summarised CRFs listed by REVIHAAP, note that the CRFs presented in this table are presented here for information and have not been used in the methodology developed). The difference in apparent response in the under and over 75s is possibly due to differences in the proportion of time spent outdoors and potential exposure to ozone rather than necessarily an indication of decreased sensitivity with age.

Health endpoint	Percentage increase in effect per 10 $\mu$ gm <sup>-3</sup> increase in daily maximum 1 hour O <sub>3</sub>		
	Single pollutant model	Adjusted for PM <sub>10</sub>	
All-cause mortality	0.18 (0.07-0.30)	0.21 (0.10-0.31)	
CV mortality 75+ years	0.22 (0.00-0.45)	0.21 (-0.01-0.43)	
CV mortality <75 years	0.35 (0.12-0.58)	0.36 (0.10-0.62)	
Respiratory mortality	0.19 (-0.06-0.45)	0.21 (-0.08-0.50)	

Given that concentrations of ozone in urban areas, where the majority of the working population live, are typically lower and elevated concentrations of ozone mostly (but not always) arise in rural areas with relatively low population densities, the impact of ozone on mortality is anticipated to be relatively small compared with that of PM.

#### 4.3.3 Population at risk and metric

To calculate working LYL associated with acute mortality, a similar methodology is used to that taken to estimate working LYL under chronic mortality impacts above. First, the CRFs for acute mortality are applied to the same underlying data for numbers of deaths per year to calculate number of "attributable" deaths. However, the average LYL for acute mortality will differ to that for impacts of chronic exposure, because of likely large differences in susceptibility of the deaths involved.

Interpretation of these deaths in terms of LYL and effect on productivity is not simple, because time series studies give no direct information on LYL associated with these "attributable" deaths. It is however widely accepted that the relationships with mortality identified in time series studies reflect effects in people with pre-existing serious cardio-respiratory disease: i.e. that even in conjunction with other factors, higher pollution on one day is insufficient to cause death in otherwise healthy people. The implication is that the "extra" or "attributable" deaths in time series studies are typically deaths in people with serious pre-existing cardio-respiratory disease, possibly undiagnosed, and whose life expectancy at that time, i.e. irrespective of air pollution, was in some sense small relative to the general population of the same age. For that reason COMEAP's first Quantification Report (1998) used the phrase "deaths brought forward", to indicate that in at least some people the LYL is short.

The CAFE Methodology Report (Hurley et al, 2005) considered the issue further and recommended that, in the face of uncertainties (but with informed indirect reasoning as above), the LYL per death be estimated as *one year, on average*, and this was done for both short-term effects of both PM and ozone (CAFE did not quantify a mortality effect of NO<sub>2</sub>). The Interdepartmental Group on Costs and Benefits' (IGCB) analysis for the 2007 Air Quality Strategy (Defra, 2007) is reported as using 2-6 months of life-expectancy (assumed in poor health, except for 10-15% of deaths). The issue was not re-visited explicitly by the recent WHO led projects of REVIHAAP and HRAPIE but insofar as the issue was discussed, no

alternative approach was proposed, and we understand that current calculations for EU policy assessments continue to use it.

Additionally, to avoid double-counting, CAFE did not add effects in PM from time series studies (short-term impacts) to those estimated in PM from cohort studies (long-term impacts); but it did add time series effects in ozone to cohort effects in PM. HRAPIE uses a similar convention, e.g. it proposes, among its most reliably quantified pollutant-health outcomes, to add cohort effects in  $PM_{2.5}$  to time series effects in  $O_3$  and  $NO_2$ . If  $NO_2$  effects from cohort studies are included, then  $NO_2$  effects from time series studies should not be included also.

We use the same convention in the present project: the number of "attributable" deaths is combined with one average LYL per death to calculate total LYL. This in turn sets an upper limit on productivity time lost. As under the calculation of chronic mortality impacts above, it is assumed that these deaths are spread across age cohorts in proportion to the rates of overall deaths. As under that calculation, this demonstration of the possible spread of deaths is a simplification and is made as it is necessary to develop an illustration of productivity impacts.

Taking the number of LYL in each age cohort, these are then combined with the same rates of employment used in the calculation of chronic mortality affects above to generate an estimate for working LYL. Indeed, given that these are deaths in people with serious preexisting cardio-respiratory disease, it may be that only a small proportion of these LYL are available for paid work. Hence as for chronic mortality above, this approach could lead to an upward bias in our estimates of the number of working LYL as impacts are likely to be more prevalent among persons with existing health issues, who are hence more likely to have already withdrawn from the labour force.

## 4.4 Acute morbidity and absenteeism (PM)

#### 4.4.1 Background

Alongside its impact on mortality, air pollution also has a number of non-fatal health impacts, causing acute and/or chronic health conditions. These health effects in turn have a direct impact on a person's ability to function and undertake different activities. Where acute periods of illness (or acute worsening of symptoms associated with chronic conditions) affect employed persons, this may cause the affected person to be absent from work with a consequent impact on productivity.

Some impacts of air pollution on morbidity are captured in the current Defra air quality appraisal guidance. This guidance focuses solely on the incidence of hospital admissions and values these outcomes according to the associated resource costs. The primary measure of acute morbidity impacts used for this pathway in the present project will be WDL. In principle WDL covers all work absence due to ill health including that associated with hospital admissions (although most emergency hospital admissions will arise in people who are not working and hence any overlap is considered as negligible for this analysis).

As before, we have as far as practicable based quantification on existing well-established reviews and have concentrated on understanding: (i) the extent to which results are specific to particular age groups and (ii) the exact meaning of the health outcomes studied and what this may mean for productivity<sup>14</sup>.

As noted earlier, the companion projects REVIHAAP and HRAPIE together provide the most recent comprehensive evidence review for quantification of the health effects of air pollution in the EU. REVIHAAP does not consider WDL, presumably because it is a health outcome

<sup>&</sup>lt;sup>14</sup> Where appropriate, the description below leans on that in the CAFE Methodology Report (Hurley et al, 2008)

rarely used in epidemiology. It has however been used in quantifications such as CAFE for the EU and in various quantifications by the US EPA and HRAPIE also recommends a quantification of the relationship between  $PM_{2.5}$  and WDL, based on a paper by Ostro (1987).

Ostro (1987) examined the relationship between air pollution and various kinds of Restricted Activity Days (RADs). HRAPIE also recommends a CRF for  $PM_{2.5}$  and RADs, based on the same Ostro paper and CRFs for ozone and Minor RADs (mRADs), based on a paper by Ostro and Rothschild (1989).

#### 4.4.2 Concentration response function

#### 4.4.2.1 The US Health Interview Study (HIS)

Both Ostro (1987) and Ostro and Rothschild (1989) are part of a series of papers produced between 1983 and 1990 examining air pollution and various kinds of RADs. These papers included: Portney and Mullahy (1986), Ostro (1987), Ostro and Rothschild (1988), Ostro (1990) and Mullahy and Portney (1990), all of which were examined for the present summary description. These papers all use data from the Health Interview Study (HIS), carried out annually since 1957 by the National Center for Health Statistics (NCHS; see Centers for Disease Control and Prevention) in the USA. In order to understand the implications of "attributable" WDL and RADs, it is necessary to understand something of the design of the HIS and the definition of the various health outcomes studied.

The HIS (or NHIS, where N stands for National) is a multi-stage probability sample of 50,000 households from metropolitan areas of all sizes and regions throughout the USA (Ostro and Rothschild, 1989). The design is of repeated annual cross-sections; the HIS is not a longitudinal study at the individual level. This leads to analyses year-by-year, with data for 1979 relating to approximately 110,000 individuals. Portney and Mullahy (1986) further describe that the data are based on interviews with: "each respondent or the family member responding for him or her" and that: "All acute illness experienced during the 2-week period prior to the date of interview was to be reported". Ostro (1990) says that: "Questionnaires are administered in the home by trained interviewers using standardized procedures".

Both Ostro (1987) and Ostro and Rothschild (1989) study adults of working age, from ages 18 to 64. Ostro has some analyses restricted to people in employment, and other analyses for all adults of working age. Ostro and Rothschild studied only people in employment because: their days are more structured, implying more consistent activities and so "restrictions in activity are easier to detect" (Ostro, 1990). Further, it was considered that there is a more consistent pattern of exposure to outdoor air pollutants and a more reliable recall of events. Also, workers' daily activity patterns and daily length of exposure to outdoor air pollutants are more similar than for the population generally.

Both these studies use Poisson regression analysis of the number of events (e.g. RADs, WDL and mRADs) per subject over a two-week period. "Respondents in the NHIS can report as many as 14 such days" in the relevant two-week period and so: "possible responses are measured as integers in {0,1,...,14}" (Mullahy and Portney, 1990).

The studies were based on 49 metropolitan areas throughout the USA. Air pollution was included as the relevant two week average of particulate matter, estimated from airport visibility data as fine particles ( $PM_{2.5}$ ). Adjustments were made for between-city differences (e.g. in factors such as time spent out of doors, building construction, and health practices) by using a fixed effects model, which focused the analysis on how individual observations differed from their city means. These studies also made adjustment for other possible confounding factors such as sex, race, education, income, and average (daily) minimum temperature in the two week period of recall for each individual.

#### 4.4.2.2 Definitions of health outcomes

A restricted activity day (RAD) is a day when a study subject: "was forced to alter his or her normal activity" (Ostro, 1990). Within the HIS, RADs are classified in three mutually exclusive categories according to degrees of severity (Portney and Mullahy, 1986):

- Bed disability days
- Work or school loss days: only WDL are relevant to the age-groups studied by Ostro (1987) and Ostro and Rothschild (1989)
- Minor restricted activity days (mRADs): these do not involve work loss or bed disability but are where the subject: "did suffer an acute impairment sufficient to cause restriction of activity in some noticeable way".

Presumably the forced change in normal activity implied by a RAD was for reasons of the individual's own health, but confirmation or otherwise of this has not been found in the papers examined<sup>15</sup>. It seems reasonable to conclude that WDL relate to loss of work for reasons of the individual's own health and not as a care-giver. WDL were not specific to causes such respiratory illness that might be expected to be linked to air pollution. In addition, days of restricted activity were also split between those attributed to respiratory conditions or not. Ostro (1987) says that Respiratory RADs (RRADs) were "determined from diagnoses reported in the HIS".

The CRFs for WDL and RADs based on these studies are proposed by HRAPIE. HRAPIE describe these as Group B CRFs, that is, pollutant–outcome pairs for which there is more uncertainty about the precision of the data used for quantification of effects than Group A CRFs recommended as core for quantification. In this analysis, we have taken the CRF for WDL to calculate the impacts of air pollution on acute morbidity and absenteeism in the workforce.

Impact pathway	Concentration response function	Source
Acute morbidity in working age population (PM)	0.46% change in WDL (95% CI of 0.39% to 0.53%) per 10 $\mu$ gm <sup>-3</sup> change in PM <sub>2.5</sub> annual average	Ostro (1987)

There is no strong evidence that air pollution has different health effects in North America than in Europe: estimates of air pollution impacts on mortality and on emergency hospital admission are similar. There is also no expectation that the health status of the general population in the US would be very different from that in much of the EU although it is difficult to assess how changes in health and in attitudes to health might affect the incidence of RADs and WDL in the UK now as opposed to the US in the 1980s.

One issue that may lead to an over-estimate of impacts is if the CRF is applied to current "allcause" sickness absence rates in the UK: a substantial proportion of sickness absence is due to stress related and musculoskeletal conditions where a link to air pollution seems highly unlikely. It is not known how comparable the causes of sickness absence in the UK in 2014 are to those in the US in the 1980s. In addition, economic insecurity and the changing nature of work (proportionately fewer employed in manual work) are likely to have influenced individual's attitudes towards taking sick leave affecting both baseline absence rates and likelihood that mild illness will lead to WDL.

<sup>&</sup>lt;sup>15</sup> However, the NHIS website includes a lot of historical reports giving methods and results. One such report is: "Current Estimates From the National Health Interview Survey, United States, 1979" (Series 10, Number 136).Jack (1981) (1979 was chosen because it is near the middle of the years 1976-81 whose HIS data was used by Ostro (1987) and Ostro and Rothschild (1989)). This includes several Tables in a section entitled "Disability Associated With Acute Conditions" including Tables for "Days of Restricted Activity Associated with Acute Conditions".

#### 4.4.3 Population at risk and metric

The CRF is specified as a change in all WDL across all employed persons. Data for the number of WDL were taken from the ONS' Labour Force Survey (LFS; ONS, 2014a). This dataset contains information on the total number of sickness absence days in the UK with data up to the year 2013: data for 2012 are used to be consistent with population data used for other pathways.

According to the ONS survey, there were 134m sickness absence days across the UK in 2012: equivalent to 4.5 days per each member of the workforce. The dataset also breaks the total number of absence down according to reason for absence: around 38m sickness absence days were due to causes potentially associated with air pollution (i.e. cardiovascular, respiratory or minor - including coughs and colds - illnesses). Given only a small proportion of absences are due to conditions that may be associated with air pollution, there is a possibility that using all-cause sickness absence as a baseline could over-estimate the amount of WDL caused by air pollution. However, it is important to note that the ONS data record only the primary reason for absence and not all contributing factors hence typical air pollution related illnesses could have featured in a greater number of absence days than reported here.

Alternative sources of data are available which estimate total sickness absence in the UK from the Confederation of British Industry (CBI; CBI, 2013) and CIPD (CIPD, 2013) surveys. Both surveys report a higher number of total sickness days and average absence per person relative to the ONS dataset, hence using the ONS dataset will produce relatively conservative (i.e. lower) estimates of WDL relative to using these sources. These surveys do not split the number of WDL according to different causes but illnesses which are associated by air pollution (i.e. respiratory or cardio-vascular problems) score highly among the most common reasons for absence reported by employers.

In addition, using the rates of absence per employee from ONS implies a level of absence consistent with the average level of absence used in the EU CAFE approach. Therefore using the same CRFs as EU CAFE will produce a comparable level of absence attributed to air pollution. Some further support for our estimate is given in our estimate of WDL through our bottom-up estimate of WDL (see Appendix 3).

### 4.5 Chronic morbidity and early retirement (PM)

#### 4.5.1 Background

Exposure to air pollution can lead to the development of either acute or chronic morbidity effects which have a subsequent impact on productivity and economic output. Consider for example, chronic bronchitis, in so far as it may be caused by air pollution. Where the symptoms associated with this disease are sufficiently significant, affected persons employed in the workforce could be forced to retire early as a consequence. Hence chronic morbidity could have the impact of causing early withdrawal from employment resulting in lost working days, weeks or years that would otherwise have been completed.

#### 4.5.2 Concentration response function

It is difficult to assess the potential role of air pollution in leading to ill health retirement as there are few published studies of ill health retirement and no studies of the impact of air pollution on age of retirement. Much respiratory ill health retirement is attributable to smoking and/or workplaces exposures. It is likely that less than 1% of the workforce retires as a result of respiratory ill health and many of those retiring are likely to be 60+ in age (Solomon et al, 2006; Pattani et al, 2001).

HRAPIE recommend a RR for the change in baseline incidence of new cases of chronic bronchitis of 1.117 per 10  $\mu$ gm<sup>-3</sup> increase in annual mean PM<sub>10</sub> (CI 95% from 1.040 to 1.189).

Based on this CRF the" attributable" fraction of new cases of bronchitis arising from current levels of population mean exposure to  $PM_{10}$  in the UK (14.5 µgm<sup>-3</sup> in 2012) might be about 17%, so it may be reasonable to attribute around 17% of cases of respiratory ill health retirement to exposure to  $PM_{10}$ . There is considerable uncertainty in the CRF for chronic bronchitis and HRAPIE did not include it as one of their core CRFs for quantification.

Impact pathway	Concentration response function	Source
Chronic bronchitis in working age population (PM)	11.7% change in new cases of chronic bronchitis (95% CI of 4% to 18.9%) per 10 $\mu$ gm <sup>-3</sup> change in PM <sub>10</sub> annual average	HRAPIE: Combination of results from longitudinal studies Loma Linda University Adventist Health and Smog (AHSMOG) and SAPALDIA

An estimate of the impact of air pollution on chronic cardiovascular ill health (which is similarly multi-causal) has not been included in the methodology due to limitations of the existing evidence base. However, as an illustration of the potential size of impacts, it is possible that about 1% of the workforce may retire as a result of cardiovascular ill health and many of those retiring are likely to be 60+ in age. It is difficult to assess the role of air pollution. If the "attributable" impact was proportionately similar to that on mortality, it is possible that about 12% of early retirements due to cardiovascular ill health might be "attributable" to air pollution based on current levels of population mean exposure to PM<sub>2.5</sub> in the UK (about 10.6  $\mu$ gm<sup>-3</sup> in 2012) and the mortality CRF recommended by HRAPIE (an increase in 11% per 10  $\mu$ gm<sup>-3</sup> PM<sub>2.5</sub>). The use of the mortality CRF to predict morbidity effects is consistent with the approach taken in the Global Burden of Disease study which assumed that for most endpoints mortality and morbidity CRFs would be the same (Lim et al, 2012). Lim et al state, however, that particulate related CV mortality had a steeper CRF than that associated with morbidity so simple application of the mortality CRF might lead to an over-estimate of morbidity impacts.

#### 4.5.3 Population at risk and metric

The CRF is defined as an increase in new cases of chronic bronchitis for a given change in pollutants. Following an extensive search, no data could be found for the UK regarding the number of new cases of chronic bronchitis per year. Hence to gain an estimate, the analysis takes the recommended baseline of number of new cases of chronic bronchitis per year from HRAPIE (390 new cases per 100,000 people per year). This rate is then combined with population data for the UK (ONS, 2013b, GRO-Scotland, 2013; NISRA, 2014) regarding the total number of persons in 2012 to obtain an estimate of the number of new cases of chronic bronchitis in the UK in 2012. It should be noted that this rate of new cases may not be appropriate for application in the UK, given it was derived from two studies looking at chronic bronchitis in Switzerland and California.

The number of new cases is then combined with data for the UK around the prevalence of chronic bronchitis across age cohorts to spread the number of new cases across the potential ages at which they could occur<sup>16</sup>. These data are for England and hence the methodology assumes this prevalence is common across all countries in the UK.

Information on the number of retirements due to chronic bronchitis is sparse: to produce a quantified impact in this project, we have taken an estimate of the number of retirements from a survey by Education for Health (2011). In the survey, it is reported that around a

<sup>&</sup>lt;sup>16</sup> Prevalence data is from 2008/9 and hence could represent a relatively out-dated picture of the prevalence of the disease, but we have used this data here given this pathway is only included for illustration.

quarter of persons reported retiring early as a consequence of developing COPD (which is a slightly broader category of illnesses including chronic bronchitis). It is important to note that this survey was relatively small survey (around 2,500 respondents) and covered six countries (including the UK).

Finally, the number of retirements in each age cohort is combined with the average retirement age (ONS, 2012c) to identify the numbers of years left to retirement and hence the number of working years lost as a consequence of having to retire early due to the disease.

The quantitative estimate of the impact of chronic bronchitis on lost work years has been developed for information only to illustrate the likely size of impacts. We do not advise that the estimated impact is included in CBA given the uncertainty around the data used to develop the estimate (rate of new cases based on HRAPIE data rather than UK-specific information and a small survey estimate of rates of retirement based on COPD rather than chronic bronchitis). Further, the definition of chronic bronchitis has a tendency to differ between studies in terms of its severity: in the studies which developed the estimates of RR, several members of the study were shown to have recovered from the illness by the end of the study period. This clearly does not tally with assuming that persons who contract chronic bronchitis permanently withdraw from the labour force through early retirement. Finally, given our estimation of the impact on acute morbidity above and the all-encompassing nature of the WDL metric used, we cannot rule out that some of the productivity impacts of air pollution through chronic bronchitis are not already captured in our estimate of WDL: in particular where these are acute impacts.

## 4.6 Dependent morbidity and absenteeism (PM and O<sub>3</sub>)

#### 4.6.1 Background

Alongside its impacts on productivity through its direct influence on workers' health, air pollution can also impact on productivity through its effects on the non-working population: either children or adult dependents.

Under this pathway, air pollution impacts on the health of a child leading to school absence days: this subsequently leads to an impact on productivity through the loss of adult working days as a consequence of the need to care for the child. There would also be an economic cost in the case where a working day was not lost by the parent, but alternative care provision needed to be arranged. The US EPA includes an estimated economic impact of parents missing work to care for sick children in their air pollution IAs.

In addition to providing care for children absent from school, some individuals may need to take time off work to care for adult dependents. We have investigated the potential to estimate productivity impacts associated with this pathway however no methodology has been proposed with which these impacts have been quantified. Although CRFs and population estimates exist with which morbidity outcomes of individuals can be assessed for non-child dependents, there is a lack of information to support a judgement as to whether that person will consequently require the care of a family member. No useful or readily available information could be found to inform the number of WDL caused by RADs occurring in other (non-child) family members or dependents; or where these happen in persons already being cared for, whether this increases the number of days of care that these persons require.

#### 4.6.2 Concentration response function

CRFs for school absence days are available for  $PM_{10}$  and  $O_3$  from studies conducted two decades ago in the US (Ransom and Pope, 1992; Park et al, 2002; Chen et al, 2000). Only the  $O_3$  relationship was used in the US EPA quantification of effects and the PM CRFs are highly uncertain. A CRF for PM (Ransom and Pope) and for  $O_3$  (Chen et al) have been

included in our proposed methodology for assessing productivity impacts but only as part of the sensitivity analysis around the central estimation of impacts.

Impact pathway	Concentration response function	Source
School days lost (PM)	4% change in all-cause SDL (95% CI of 2.5% to 6.4%) per 10 $\mu$ gm <sup>-3</sup> change in PM <sub>10</sub> annual average	Ransom and Pope (1992)
School days lost (O <sub>3</sub> )	0.13% change all-cause SDL (95% CI of 0.06% to 0.25%) per 10 $\mu$ gm <sup>-3</sup> change in O <sub>3</sub> 1-hour daily maximum	Chen et al (2000)

In the Ransom and Pope and the Chen et al studies, the CRF is for all-cause school absence and takes account of weather, month of year and holidays. The Park et al study is specific to illness-related absenteeism.

The Ransom and Pope study was a small study in terms of geographical coverage but did include a reasonably large elementary school population of about 7000 to 8000 children and was conducted over 6 years. However, the community was living in area impacted by emissions from steel mills with high average levels of  $PM_{10}$  (about 50 µgm<sup>-3</sup> with 24-hour maximums reaching 365 µgm<sup>-3</sup>). The CRF is stated as about 40% per 100 µgm<sup>-3</sup> of  $PM_{10}$ : this level of pollution is consistent with the variability of  $PM_{10}$  concentrations in the study area during the period of the study but very different from current conditions in the UK. As such it is questionable whether the CRF derived is appropriate in a UK context and therefore we excluded the impact pathway of PM on school days lost (SDL) from our central impact estimation.

The Park et al study is extremely small being based on one school in Seoul in South Korea and 4 years of data. Societal and climatic differences combined with relatively high pollution levels give rise to a very substantial uncertainty in extrapolation to the UK. Both Chen et al (2000) and Gilliland et al (2001) failed to find a relationship between school absence and  $PM_{10}$ .

There is clearly also uncertainty in the extrapolation from the source studies regarding the effects of ozone to the UK in 2014. Concentrations of  $O_3$  in heavily populated areas of the UK are generally low and we would anticipate that PM and  $NO_2$  would have more important impacts on children's health and school absence days than  $O_3$ . There are no CRFs linking  $NO_2$  and school absence. Hence we also do not include the  $O_3$  CRF from Chen et al in our central impact estimation, but this CRF (alongside the CRF for PM from Ransom and Pope) is included in the sensitivity analysis. The exclusion of the SDL CRFs from our core analysis is consistent with HRAPIE which included no CRFs linking air pollution to SDL in its impact assessment recommendations.

#### 4.6.3 Population at risk and metric

Baseline rates of school absence due to respiratory causes are uncertain as schools are not required to record the detailed cause of absence. It is possible that baseline rates of respiratory absence in the studies that have found an association between absence and air quality were very different from those in the UK.

To estimate the number of "attributable" SDL, the CRF is combined with data regarding the number of illness-related school day absences for the UK (DoE, 2013; Scottish Government, 2013; Welsh Government, 2013; DENI, 2013) for 2012. Although the CRFs are specified for all-cause school absence, the present analysis applies the CRF to illness-related authorised absences in primary school children only to allow for a conservative estimate to be produced given concerns regarding the applicability of the CRF.

It is conceivable that sickness absence in children outside primary schools (e.g. pre-nursery children in day-care, children attending special schools with care needs, younger children in

secondary school) may cause parents to miss work days. However, these potential impacts have been excluded in this analysis as the CRFs focus on impacts in primary school aged children.

All SDL will not necessarily imply parents will incur a WDL as a result: some families have mixed or no employment hence parents may be 'available' (i.e. not working) and able to care for a sick child. In these circumstances, a SDL does not result in any WDL. To account for this, the analysis applies an estimate of the proportion of children who live in 'working' families (i.e. where all adults are in employment). Based on data from ONS (ONS, 2013c), around 52% of children live in working families (ratio applies to all UK Households in 2012).

However, this adjustment may under-account for the number families where an adult member may be available to care for a sick child: some working families may have an adult member who works part-time or has flexible working practices. Further, even in the case where both parents work, families may have other mechanisms through which child care can be provided in the case of illness: for example the child could be cared for by wider family members or friends or parents could take annual leave to care for the child. Given these factors, it was considered appropriate to make a further adjustment to the estimate of SDL to account for this. A ratio of 36% is applied to the estimate based on the Palmer et al study to depict the likelihood that a SDL will lead to a WDL in a working household<sup>17</sup>.

## 4.7 Morbidity and presenteeism (PM and O<sub>3</sub>)

#### 4.7.1 Background

Presenteeism is defined as attendance at work whilst ill, rather than taking time-off work to recover (absenteeism). Hence, presenteeism can reduce productivity as the affected person's activity is inherently reduced by their illness relative to days of good health. Air pollution can therefore have an impact on productivity through morbidity and presenteeism in addition to its impact through absenteeism considered above.

#### 4.7.2 Concentration response function

In the existing published evidence, there is no defined CRF which sets out a change in the number of working days where the person is ill, but where illness does not constitute WDL, for a given change in air pollution. However, as discussed above, CRFs do exist for RADs and WDL.

Impact pathway	Concentration response function	Source
Presenteeism in employed persons (RADs) (PM)	4.7% change in all-cause RADs (95% CI of 4.2% to 5.3%) per 10 $\mu gm^{-3}$ change in $PM_{2.5}$ annual average	Ostro (1987)
Presenteeism in employed persons (mRADs) (O <sub>3</sub> )	1.53% change in new cases of chronic bronchitis (95% CI of 0.6% to 2.49%) per 10 $\mu$ gm <sup>-3</sup> change in O <sub>3</sub> 8-hour daily maximum	Ostro and Rothschild (1989)

The definition of a RAD includes bed days and minor RADs alongside WDL, where a mRAD is a day of restricted activity that does not result in the person having to stay in bed or miss work. Hence, the number of days where a person's health is affected by air pollution but

<sup>&</sup>lt;sup>17</sup> This report estimates that for every family where all adults were employed and where a child reports an incidence of illness, 0.4 additional days of work were lost relative to other households. From the same study the average estimate of WDL per incidence of illness was 1.1 days. Assuming this estimate of sickness days per incidence applies equally to SDL, for every illness and absence of 1.1 days from school 0.4 days of work is assumed to be lost. Hence for every SDL, the number of WDL will be 36% (the ratio between 0.4 and 1.1)

does not result in a WDL could equate to some proportion of bed days and mRADS: i.e. total RADs minus WDL.

To derive an estimate for the number of presenteeism days, our approach uses the CRF from Ostro (1987): 4.7% change in the number of RADs for a 10  $\mu$ gm<sup>-3</sup> change in PM<sub>2.5</sub>. This CRF was included by HRAPIE in their B category of CRFs.

The analysis also includes a CRF for mRADs resulting from concentrations of  $O_3$  from Ostro and Rothschild (1989): again this CRF was also included in HRAPIE's B-rated CRFs and was also used as part of the EU CAFE air quality appraisal methodology.

#### 4.7.3 Population at risk and metric

A baseline for the number of presenteeism days was constructed using data from the ONS' General Lifestyle Survey (ONS, 2013d). This provides a baseline estimate for the average number of RADs per person in the UK in 2011<sup>18</sup>. These data include all causes of RADs (hence also including WDL). To develop a baseline of potential presenteeism days (i.e. RADs that are not WDL), we subtract the average number of WDL per person per annum (used above to calculate the number of WDL) from the average number of RADs per annum per employed person. Hence we assume all RADs that are not WDL are potential presenteeism days.

The total number of "attributable" presenteeism days is calculated by combining the CRFs, average number of RADs (less WDL) per employed person per annum, multiplied by the number of employed persons.

Given the general definition of the metrics used to represent presenteeism days, it was also considered prudent to adjust the total number of potential presenteeism days to account for those instances which may occur when the affected person is legitimately away from work: for example at weekends or during periods of leave. These instances would not incur a productivity loss as the person is not at work. To do so, the number of presenteeism days is combined with a proportion of all days in a year that are likely to be worked by an employed person (derived from ONS' LFS the average person works 221 days per annum).

Although a person attends work on a presenteeism day, the productivity loss occurs through the reduction in their potential activity relative to a usual healthy day at work. Hence the productivity impact will depend on the proportion of total productivity that is lost on a given presenteeism day. The definition of RADs and mRADs might be informative in terms of potential productivity impacts but we have not identified a reliable source of information about the impacts of RADs on productivity. It is likely that mRADs might have relatively smaller impact on productivity in comparison to RADs (if the latter are not severe enough to lead to absence from work). Given that there is no set percentage reduction in productivity that can be easily taken from the RAD-related literature, a wider literature search was undertaken.

There is a burgeoning evidence base of published literature which attempts to quantify the impacts on productivity of presenteeism. Many studies (e.g. Stewart et al, 2004; Goetzel et al, 2004; Cooper and Dewe, 2008; and Mitchell and Bates, 2011) have suggested that the costs of presenteeism could be larger than absenteeism. However, it is recognised that there is no agreed methodology with which presenteeism impacts can be assessed (Mattke et al (2007)) and there is significant variation in the quantification and subsequent valuation of productivity impacts across studies (Johns, 2010).

Most published investigations of presenteeism appear to focus on stress-related illness and much less information is available about the impacts of other types of illness. In a comparison of the effects on work performance of mental and physical disorders, de Graaf et

<sup>&</sup>lt;sup>18</sup> The tool calculates an estimate of the impacts in 2012: hence it is assumed that the average number of RADs per annum per person is the same in 2012 as it is in 2011.

al (2012) reported that physical disorders were associated with a reduction in efficiency of about 10% on the affected days.

A few studies have focussed specifically on presenteeism associated with respiratory symptoms mostly in the context of infection and/or allergic conditions. Palmer et al (2010) in a study of influenza-like illnesses estimated that employees with these conditions were on average less productive for 4.8 hours of each day that they worked while sick. In a Canadian study of productivity loss in people with asthma, Thanh et al (2009) equated sickness absence through RADs with a reduction of functional level of 20-30%.

Several studies have looked at the impacts on productivity of allergic rhinitis. In a review of studies of the impacts of rhinitis on productivity, Vandenplas et al (2008) concluded that rhinitis has a small effect on absenteeism but estimates of lost productivity attributable to reduced on-the-job effectiveness ranged from 11 to 40%. In an earlier study, Lamb et al (2006) reported that employees were unproductive for 2.3 hours per workday when experiencing symptoms. Hellgren et al (2010) found an average level of presenteeism of 22% during an episode of allergic rhinitis or common cold.

No single study provides a direct estimate of the impact of air pollution on reduced productivity during a presenteeism day. However, drawing together the findings of the studies regarding productivity loss associated with respiratory related illnesses, the analysis here has assumed a 20% level of reduced productivity during presenteeism days. This was chosen given it lay at the conservative end of estimates of reduced productivity. In practice the level of productivity loss will depend on a number of factors including the person, the illness type and the job type.

# 4.8 Impacts on non-market productive activities (mortality and absenteeism)

#### 4.8.1 Background

In the pathways considered thus far for quantification, the focus is on the productivity impacts through formal employment: i.e. air pollution causes a loss of productivity where a person misses a day at work or is permanently removed from the labour force. This analysis is sufficient if we were only considering what the impact of air pollution could be on GDP. However, in practice people undertake a number of activities outside the formal labour market which provide a real economic and/or social benefit.

In this project we have defined the productive activities undertaken by persons outside formal employment as 'non-market' productive activities. A number of activities fall under this definition: housework, childcare, volunteering, caring for other dependents, etc. All these activities have a value and if lost, imply a cost to society. Hence when undertaking a comprehensive CBA, it is necessary to consider what potential 'non-market' activities might be lost through the impacts of air pollution.

There are potentially a large number of people in these groups and the impacts of their mortality or morbidity on productivity are likely to be difficult to assess because of the diversity of their contributions to national productivity. Some of these individuals are providing unpaid care that would otherwise be provided by a paid carer whereas other individuals are providing services to the wider community that would not otherwise be provided (for example, many visitor attractions, sports clubs or initiatives aimed at children and young people are highly dependent on volunteers in order to be able to function). There are older people caring for one another, volunteering or looking after grandchildren, as well as people of working age who are not at work but providing unpaid care or working as volunteers and people in work who give time to the voluntary sector. This group does not include those in paid employment who are absent from work due to the need to care for others.

#### 4.8.2 Concentration response function

Under this pathway, we use the same health impact pathways as discussed in the preceding sections. In this case only mortality (chronic and acute) and WDL have been considered as these are likely to be the most significant impacts. SDL and presenteeism have not been estimated as these impacts are likely to be less significant for carers and volunteers and it is uncertain whether SDL will necessarily imply lost productive days of non-market activity given the nature of those activities.

#### 4.8.3 Population at risk and metric

As with the CRFs used, this pathway also uses much of the same information as is used to estimate the impacts of air pollution on formal market activities. However, where adjustments were made to focus on persons in employment, under this pathway data are instead used to adjust metrics to focus on either carers or volunteers.

We have chosen to focus on carers and volunteers in this analysis to illustrate the potential size of non-market impacts lost. In practice, each non-market productive activity will have an associated economic cost if lost through air pollution. However, to capture all non-market activities would significantly increase the size and scope of the modelling tool. We have chosen to focus only on carers and volunteers to rationalise the number of potential impacts assessed and given these activities were considered to be 'less deferrable' than others: for example, where a carer or volunteer incurs a RAD, they may not be able to carry out their care or volunteer responsibilities which need to be completed on a given day. Whereas (for example) for housework, it may be arguable that this activity is more deferrable to other days when the affected person recovers, with no consequent loss in overall productivity.

For mortality, the same underlying data are taken to calculate the total LYL. However, rather than applying an employment rate to these LYL, data are used to estimate the number of LYL for either carers or volunteers. This is then combined with the prevalence of non-market activities and average time spent in these activities to estimate hours of lost care or volunteering.

For carers, data on the number of carers and the average length of time spent caring over a given period are taken from HSCIC (2010a). The data used are for 2009/10 and for England only: for this analysis we assume these data are appropriate for the UK in 2012. Further, these data only focus on care provided in a non-professional capacity. The analysis uses data for sole carers only (where the carer is the only person providing care to the person cared for). This was considered more consistent with productivity loss as where a sole carer is ill, it is more likely that this care is not replaced. However, only considering sole carers may place a downward bias on the estimation of lost care hours given care may also be lost where there is more than one carer, but other carers cannot fill in for absent carers.

For volunteers, data are taken regarding the prevalence of volunteering and average time volunteered from the Cabinet Office's Community Life Survey (Cabinet Office, 2013). This provides data for England in 2012/13: it is assumed that the rates of volunteering are applicable to the UK for 2012. Data are only taken for those engaging in 'formal' volunteering on a regular basis: those undertaking informal volunteering or on an irregular basis are excluded as the impacts on these people will be less significant.

For morbidity, the approach is the same with one further step. The CRF for WDL is combined with underlying rates of sickness absence (although the CRF for WDL is specified for employed persons, we have assumed that persons undertaking non-market activities incur a similar level of sickness per annum – i.e. carers and volunteers will suffer the same number of days of sickness days that would otherwise have caused WDL had/if these people were employed in the formal labour market) to generate total 'WDL' among carers and volunteers.

The analysis then also adjusts for the frequency of care or volunteering: employed persons typically attend work more than carers or volunteers undertake these activities, hence a number of the 'WDL' among carers or volunteers would occur on days when they are not
undertaking these activities, implying no productivity loss. Conservative assumptions are made regarding frequency (i.e. persons tend to care or volunteer for longer hours less frequently) to produce a lower bound estimate of where a WDL and a care or volunteering day coincide. This is then combined with data for the time spent in caring or volunteering activities to determine potential hours of care or volunteering lost.

It should be noted that the WDL, RADs and mRADs CRFs were established in studies of working people and may not be representative for older people or others who are not in work and who may potentially have a lower baseline health status. Further, for activities such as formal volunteering, there is likely to be a lower incentive to attend the activity relative to paid employment. The uncertainties in extrapolation from studies conducted in the US in the 1980s to the UK in 2012 are even greater for the unpaid sector than for the working population.

# **5 Valuation of impact pathways**

# **5.1 Introduction**

#### 5.1.1 Social cost-benefit analysis and literature review

Under this project, we have identified a number of key pathways through which air pollution can impact productivity via human health. This section of the report discusses the proposed approach to valuing the impact taken forward for quantification.

The objective of the project was to develop a methodology to value productivity impacts of air pollution for use in appraisal. This analysis could then be included as part of a more comprehensive CBA of impacts. Given its intended use in social CBA, the methodology developed focuses on the measurement of the net social change in productivity (i.e. the productivity change from a society-wide perspective). This framing contrasts with productivity change attributable to a given person and/or producer. In this regard, it is consistent with the social welfare perspective which frames the UK Government's approach to CBA of public projects and policies, including air quality regulation.

To support the development of the valuation methodology, a detailed and wide-ranging literature review was completed to assess the approaches taken to valuing impacts on productivity via health in existing published studies. This review covered: other air quality impact assessments (for example EU CAFE and US EPA approaches), impact assessments produced by UK Government departments which value health impacts (but not specifically air quality related), approaches to valuing impacts on health from other organisations outside the UK and a wider review of relevant academic literature.

#### 5.1.2 A simple model of production

In a simple model, a fall in output for a given time-period, Qn, for the nth person has a price, Pn. If we assume that one unit of output is lost, then the lost value, Vn, is equal to Pn. Generalising this to a larger number of units:

Vn = Qn \* Pn = Value of lost output per person.

The total productivity loss, ( $\Sigma Vn$ ), will then depend on the duration of the illness and number of persons affected.

In certain situations the loss of value may be reduced if other people involved in the production process compensate for the fall in productivity of an individual by helping out with his or her tasks. This is more likely to occur in a production context where collective output is rewarded or where there is an incentive (such as extra pay, promotion, goodwill, etc.) in place to encourage others to fulfil the short-fall in output. This type of temporary compensation mechanism is also more likely in the short-term, and when there is the expectation that the person will fully recover and either:

- a) Return to the production process after being absent or
- Return to full productivity having incurred a period of lower productivity due to illness (presenteeism).

The loss in value per person will then be:

F\*ΣVn

where F is the fraction of lost output which is not compensated for, and  $0 \le F \le 1$ .

In both cases the direct cost of the loss in productivity will be determined by the length of time for which the person is not working or not working at full productivity.

In the longer term, there are two principal options:

- A new person may be recruited to replace the ill person and/or additional capital may be employed. Thus, formerly unemployed (or employed) resources are now employed (or reemployed) to prevent the short-fall in output from continuing longer
- b) The ill person is not replaced, and either the short-fall in output is borne permanently, or some form of compensation mechanism is retained.

### 5.2 Over-arching approach: Human capital versus frictioncost approach

There are two different schools of thought regarding the quantification of productivity impacts in the existing literature: the Human Capital Approach (HCA) and friction cost approach. The difference between these approaches is demonstrated in the discussion of the simple model of production above.

Under the HCA, productivity loss (associated with either mortality or morbidity) is measured as the total length of time that the person is unable to work combined with a value of marginal productivity (typically assumed to be the average wage rate). This approach values all potential productivity loss associated with the person's absence from work and hence (consistent with an assumption of 'full employment'<sup>19</sup>) implicitly assumes that the person is not replaced, either on a temporary or permanent basis. Under full employment, there is no spare capacity with which a compensation mechanism could be used (implying in our simple model that F = 1).

The friction cost approach, on the other hand assumes that the affected person would be replaced in the labour force (or at least a replacement would be sought) at some point in time and so is a more flexible measure of the value of productivity. The friction cost approach also includes other costs, such as hiring and training new workers, in the overall valuation. On balance, the HCA is likely to provide a higher estimate of the value of productivity change relative to the friction cost approach and the assumption of no replacement has led some to suggest that the HCA may over-estimate this cost component and hence overall productivity loss (Koopmanschap et al, 1995).

Applying the friction cost approach would require an empirical estimate of how likely it is that a person is replaced. The ability to replace any given worker would be dependent on levels of skill, age-related experience and location of available labour relative to the vacancy to be filled. This might be expected to differ significantly between different sectors, firms and locations. Relaxing the assumption of full employment under the friction cost approach also implies that the length of loss associated with each health impact (measured by (Qn)) will be reduced. Again, an empirical estimate of this reduction would be needed<sup>20</sup>.

When considering the potential impacts of air pollution in practice (in particular in the case of mortality), the friction cost approach could initially be considered more appealing. This is particularly the case when only considering either the specific job affected in isolation or the impacts in the short-term. If a person is removed from the work-force, it is likely that the firm will attempt to seek a replacement such that output is not lost.

<sup>&</sup>lt;sup>19</sup> Full employment is a term in economics used to refer to a situation where all available labour resource is being used in the most economically efficient way: and unemployment is frictional i.e. where workers are moving between jobs.
<sup>20</sup> Note that loosening the assumption of full employment does not imply that there is therefore an infinite supply of labour available to replace

<sup>&</sup>lt;sup>20</sup> Note that loosening the assumption of full employment does not imply that there is therefore an infinite supply of labour available to replace those incapacitated by air pollution-related health conditions. As well as relating to skill levels and sectoral experience, this constraint is likely to be affected by other factors in the case of non-marginal loss of labour, such as that resulting from serious smog, when the volume of replacement labour may not be easily mobilised immediately.

However, when assessing the impacts from the perspective of the whole economy or society, the person could feasibly be replaced by someone who is already employed elsewhere: hence the productivity loss would still accrue to the economy overall, just to a different firm than originally affected. Further, in standard economic theory in the long-term an economy is typically assumed to operate at 'full employment'. Where a person leaves the labour force through death under this assumption, there is a permanent reduction in productivity associated with the lost remaining potential productive life of that person.

In existing health impact appraisal, the HCA is the typical methodology used to estimate productivity impacts. Both the EU CAFE approach and US EPA studies of air pollution impacts use the HCA to estimate the impacts on productivity assuming workers are not replaced when removed from the workforce. The HCA has also been used in other studies of the impacts of air quality on productivity in Australia (DEC (NSW), 2005; and ATSE, 2009) and Canada (ENVIRON, 2009; and Stieb et al, 2002).

Further, outside the appraisal of air quality impacts, a number of IAs undertaken by the UK Government (HSE, 2013; McCrone et al, 2008; and Scottish Executive, 2006) and current UK Government IA guidance from DfT (see: DfT, 2012; and O'Reilly, 1993) do not assume replacement of workers where they are removed from the labour market through detrimental health impacts, implying some consistency in approach. In addition, the HCA is also widely used in an academic context to measure productivity impacts of health outcomes (see for example, Mitchell, 2011 and Filipovic et al, 2011).

The methodology developed to assess productivity impacts under this project uses the HCA to value losses in productivity through both mortality and morbidity affects. This approach is proposed as this maintains consistency with existing UK Government IAs and IA guidance and with standard economic assumptions in the long-run. This approach will also reduce the complexity of the approach and of updating the tool going forward.

## 5.3 Empirical measurement of productivity

In order to value productivity loss, a value for each unit of lost work (Vn) is needed. This unit value represents the productivity of workers at the margin and values the productivity lost to the firm (and, under our full employment assumption, the whole economy) associated with each unit of lost production.

The measurement of Vn can be undertaken in a number of ways. Measurement methods can be divided into "top-down" and "bottom-up" methods. Top-down measures take aggregate measures of output and factors of production (in this instance, labour), and divide the former by the latter: for example, GDP per capita or GDP per worker. By contrast, bottom-up measures use output and employment estimates at the micro-scale (such as at the firm or sector level) to construct measures of Vn.

At the margin in a competitive market, it is assumed that the marginal revenue of a unit of labour is equal to the marginal cost. Marginal revenue represents the value of the marginal production of each unit of labour. However marginal revenue is difficult to measure at the micro-level given lack of available data. As such bottom-up methods seek to construct a value of the marginal cost of labour with which to value productivity losses.

In practice, the average market wage rate is typically used as a proxy for the marginal cost of labour. This is the case for many of the air quality IAs (US EPA, DEC (NSW), 2005; and Stieb et al, 2002) and wider IAs (HSE, 2013; McCrone et al, 2008; Scottish Executive, 2006; and British Thoracic Society, 2006) assessed as part of this literature review.

However, it should be noted that the observed market wage rates may not equal marginal revenue of a unit of labour for a number of reasons: for example, if a job involves team production, or if there is time sensitivity to outputs, the actual productivity loss may be greater than that measured by the wage rate (Zhang et. al., 2011). Further, imperfect labour markets

may reflect inequalities such as race, gender, employer market power or risk aversion in workers which lead to workers receiving a wage less than their marginal productivity.

Further, using only the marginal wage overlooks a number of other costs which an employer incurs to employ a given person: for example, tax, National Insurance and pension contributions, IT and facilities costs, etc. These costs should also be factored into the estimate of the marginal cost to an employer of employing a worker. These wider costs were included the valuation of productivity impacts by DWP (2013) in their recent review of sickness absence.

The literature also distinguishes between direct and indirect costs. Direct costs include those costs associated with the loss of output resulting from the health impact in a given time period. For example, the value of the fall in number of car exhaust units produced resulting from a day of absenteeism would constitute a direct cost. Indirect costs include, for example, costs associated with lower consumer satisfaction and lower quality products (CBI, 1998) and are harder to measure accurately. In theory, given both are costs that accrue to the employer through absence, both should be included in any valuation of the marginal cost of a worker (and hence also of marginal revenue). Thus, we might imagine that:

Vn = Vnd + Vni,

where d = direct and i = indirect.

For this study, we propose using the bottom-up values of productivity per worker derived by the CBI in their regular survey of employers regarding the costs of absence (CBI, 2013). In doing so, it is assumed that in responding to the survey employer's factor in wage, nonwage, direct and indirect costs of an employee's absence. This approach is consistent with the unit values used for EU assessments by CAFE which also uses estimates from these surveys<sup>21</sup>.

Given uncertainty around the measurement of the marginal cost (and hence marginal revenue) of labour, the assessment tool also includes alternative unit values as part of the sensitivity analysis (discussed in further detail Section 6.4). This sensitivity analysis provides a sense check of the central estimates against impacts valued using average wage<sup>22</sup>; average wage plus 30% (uprated to represent the inclusion of indirect costs as advised by UK Standard Cost Model (BERR, 2005)); and a top-down estimate (GDP per worker per day worked<sup>23</sup>). Central estimates of impacts use CBI's values as these include non-wage and indirect costs (whereas average wage does not) and represent a more conservative (i.e. lower) value than the top-down measure.

### 5.4 Consistency with existing valuation guidance

Under this project, we have developed a methodology specifically to appraise the productivity impacts associated with air pollution through its impacts on human health. It is intended that this is then added to the existing Defra appraisal guidance to facilitate a more comprehensive assessment of impacts. It is therefore important to ensure that there is no potential overlap between the two sets of guidance. This relates most particularly to the measurement of productivity change proposed under this project and the valuation of other components of welfare change which are included in existing Defra guidance, specifically the disutility from pain and suffering associated with health impacts.

A measure of the WTP to avoid the risk of premature death (and hospital admissions) is currently included in the appraisal guidance<sup>24</sup>. This attempts to capture the value that a

<sup>&</sup>lt;sup>21</sup> Estimates of the cost of absence are also available from a survey by CIPD but the CBI values were selected to maintain consistency and hence greater level of comparability with estimates of productivity losses estimated by the EU CAFE approach. <sup>22</sup> Data for average wage was sourced from ONS' Annual survey of Household Earnings (ONS, 2012d)

<sup>&</sup>lt;sup>23</sup> This was derived using ONS data on GDP, number of employed persons and average numbers of days worked per annum. (ONS, 2013e) <sup>24</sup> A measure of WTP to avoid morbidity outcomes is not currently included, hence there is no potential overlap for these pathways

person places on the extension of his or her life. WTP is typically estimated by surveying the population about the strength of their preferences, as measured in monetary terms. It is conceivable that this WTP may capture an individuals' value of their potential future productive output and/or their (dis-) utility of working, alongside capturing the utility derived from other activities such as time spent with friends and family, lost consumption, etc.<sup>25</sup>. Therefore, including a separate measurement of productivity changes as developed under this project to existing guidance would constitute double-counting.

Indeed, the fact that such impacts may be captured in survey-based estimates of WTP appears to be the reason why many empirical mortality risk estimates in air quality related IAs not to consider productivity impacts related to mortality separately<sup>26</sup>. The EU CAFE work to monetise the potential impacts of air pollution did not include any additional valuation of the productivity impacts of mortality.

Further, a study by the DEC (NSW; 2005) valued health impacts using WTP values where possible as it was considered to include, alongside other factors, the productivity value of extending life. In addition, reports by the World Bank (2007) and ENVIRON (2009) use WTP values to assign an economic cost to both the mortality and morbidity impacts of air pollution as this was considered a better approximation of the overall social costs of health impacts relative to a 'cost of illness' approach under which productivity impacts would have been assessed directly. Further, the DfT impact assessment guidance only uses WTP to value mortality impacts with productivity impacts assumed to be implicitly included in the values. However, assessing wider existing IAs, examples can be found where a separate valuation of productivity impacts has been included alongside the use of WTP estimates to value changes in life expectancy. For example, in HSE (2013) impacts valued using WTP are added to the direct assessment of productivity impacts.

The extent of any potential overlap will be determined by the basis on which each individual taking part in the WTP survey derives their response; this will be based on the activities or services from which an individual derives utility but which will be lost through death (or other health outcome). This will inherently vary from person to person. The design of the survey could influence the potential overlap (where participants may explicitly be asked to include/exclude specific factors from their valuation) as could the context of the survey; Steib et al (2002) note that WTP values may not include the costs of medical treatment or lost earnings where the individual will not bear these costs.

Looking at the design and wording of the original study on which Defra's WTP estimates for air quality mortality risks are based (Chilton et al) there is no basis to suggest that the WTP estimates are either inclusive or exclusive of productivity effects. Given this is not explicit either way we have concluded that separate and additive treatment of the productivity impacts associated with mortality cannot be justified.

In this analysis we propose that the direct productivity impacts of mortality are <u>not</u> considered additional to the existing valuation of the benefit of individuals to extend life in Defra's appraisal guidance. Based on the WTP studies on which the guidance is based, we cannot be certain that there would be no overlap between individual's WTP values and our separate valuation of productivity. Hence all productivity impacts associated with mortality are not included in our recommended impacts for CBA to avoid the risk of overlaps. The analysis does include productivity impacts of morbidity given the low potential for overlap between the morbidity metrics captured in this assessment and those included in existing guidance.

<sup>&</sup>lt;sup>25</sup> The interaction of productivity impacts assessed as part of a cost-of-illness assessment and valuation of health outcomes using WTP values is summarised in Figure 2 of a report by the OECD (2008).
<sup>26</sup> A second, practical, reason for excluding productivity impacts of mortality is the fact that the majority of air pollution mortality impacts are likely

<sup>&</sup>lt;sup>20</sup> A second, practical, reason for excluding productivity impacts of mortality is the fact that the majority of air pollution mortality impacts are likely to be borne by the elderly retired population.

### 5.5 Treatment of consumption impacts

In addition to the supply-side effects of air pollution associated with productivity change, it is also possible to conceive of potential demand-side effects – at least in principle. Consumption of household goods may be expected to fall permanently, as a result of mortality, or temporarily, as a result of morbidity impacts<sup>27</sup>. This issue is likely to be more pertinent in the case of mortality than morbidity, in particular given the morbidity impact pathways assessed under this project focus on acute health outcomes where consumption could be conceived to be simply deferred rather than reduced.

Consumption (and hence changes in consumption) could be considered to represent a resource cost to society. For example, where a person lives an additional year, they will consume goods and services that would consequently be unavailable to the wider society, either in that or future years. In the case of improving air quality, any subsequent increase in consumption associated with improving health outcomes could therefore be included as an additional cost.

Outside air quality IA, the impacts of reducing consumption are sometimes included in CBA (e.g. reductions in energy consumption through improvements in energy efficiency (see for example: DECC, 2010)). Further, the value of consumption change associated with changes in life expectancy has been considered in previous assessment of health impacts: In DfT's appraisal guidance, the valuation of mortality due to road accidents includes the cost of the lost 'net output' of the person (i.e. the balance of total productivity and total consumption over a person's remaining lifetime).

Although consumption impacts are captured in the DfT guidance, these impacts were not typically included in air quality IA or CBA of health impacts. Consumption impacts are not captured in US or EU air pollution economic appraisals or in UK Government IA's of policies to improve health (HSE, 2013; McCrone et al, 2008; and Scottish Executive, 2006).

We propose not to include consumption impacts as part of the valuation of mortality or morbidity impacts in this analysis given the current lack of consistency in approach across IA more widely. Further, the focus of this project was to develop a methodology to assess the impacts on *supply-side* productivity change.

Should consumption impacts be considered for inclusion in the future, further contemplation is required as to the consistency of inclusion with the existing valuation of changes in mortality risk using WTP values and the risk of double-counting. In addition, more detailed data are likely to be required on which to base a reliable estimate of consumption per person than is currently available. Information on overall domestic and government expenditure is available at a national level (as used by DfT in their unit values of lost life; ONS, 2014b) but given air pollution tends to affect older persons, an average derived across the population may be less representative and lead to a less robust estimation of consumption impacts.

## 5.6 Value of non-market impacts

Using the HCA to value productivity impacts relies on the use of a price revealed in the market as a unit cost of lost productivity. This analysis includes an assessment of the impacts of air pollution on non-market labour activities: caring and volunteering. By definition, these activities do not have a price which is revealed in the market, as these activities are unpaid. Hence to be able to value these activities, alternative values revealed elsewhere are required.

A study by UNRISD (2008) proposes four potential methodologies to value care: (1) use average wage rate across the economy; (2) use average wage rate paid to unpaid carers

<sup>&</sup>lt;sup>27</sup> The latter effect may result from the individual being less active, and/or reducing consumption on the basis that she has lower wages, or expects lower wages, and so saves more for the future.

where those persons are also formally employed; (3) use average wage rate paid to persons undertaking similar activities (i.e. domestic workers) to carers in formal economy; and (4) use specific wage rates paid to persons undertaking care activities in the formal economy. This multi-country study used the first and third options to place a value on care given data limitations associated with other alternatives. However, a study of the value of carers in the UK (Carers UK, 2011) uses an estimate of the costs of professional care services (equivalent to the fourth option from UNRISD). This draws on estimates of health care services provided in the UK developed by PSSRU (2012).

In this project, we propose to use the values developed by PSSRU (equivalent to option (4) in the UNRISD study) as the basis for valuation of a unit of care lost. This provides a reliable and relatively accurate valuation given that this represents the replacement cost of providing care lost through professional alternatives.

It is more difficult to place a value on a unit of volunteering; volunteering includes a more diverse range of activities and with a lack of comparable occupations in the formal labour market from which a value can be derived. Several approaches are discussed in Ironmonger et al (2006) with which to value volunteering. This includes the Volunteer Investment and Value Audit (VIVA) approach which seeks to value volunteering based on the cost of resources used to support volunteers (e.g. training, HR, etc.). Volunteering could also be valued by focussing on the outputs of volunteering and using prices of comparable goods and services produced in the formal market.

In this analysis, we propose to use the average wage rate in the UK economy as a proxy for the value of volunteering. Using this approach is consistent with the majority of IA's undertaken in the existing literature (see: JRF, 1997; IFRC, 2011; Teasdale, 2008; and Mayer, 2003). Further, given the likely size of impacts assessed, this relatively simple approach to valuing volunteering time will provide a reasonable illustration of the value of impacts.

It should be noted that there is ongoing work within the UK Government to develop robust values with which to appraise volunteering time. In particular, this work is exploring a new approach based on deriving a value of volunteering activities to the volunteer (Fujiwara et al, 2013). This uses a 'well-being valuation' approach to assess the value of volunteering. The development of this approach could present an additional impact for inclusion in the valuation of changes in volunteering time; using average wage could under-estimate the true cost of lost volunteering time associated with air pollution given this additional utility for the volunteer is not captured. However, consideration would need to be given as to whether these values should be added to or used instead of the use of average wage given the well-being approach seems to focus on the value to the participant rather than the recipient.

# 6 Application of analysis and sensitivities

### 6.1 Summary of assessment methodology

#### 6.1.1 Using the tool to appraise air quality impacts

The preceding sections of this report set out in detail the proposed methodology with which the productivity impacts of air pollution can be assessed. A core set of pathways for which the evidence base was considered relatively robust and sufficient quantitative information was available to support assessment have been 'prioritised' from a longer list of potential pathways for quantification. A proposed approach has been set out for each 'prioritised' pathway to assess and value the health impacts.

The methodology has been developed to assess the burden associated with levels of, or impacts of changes, in pollutant concentrations in a single year for  $PM_{2.5}$  and  $PM_{10}$ ,  $NO_2$  and  $O_3$ . This includes any lasting impacts which occur in future years. Where this is the case these impacts are valued and discounted back to the base year.

When assessing the burden associated with levels of, or impacts of changes in, concentrations over multiple years each year must be assessed individually using the tool (and appropriate discounting applied to the results) but impacts assessed across different years are additive.

The levels (or change in levels) of pollutants are assessed on an average basis across the UK as a whole (i.e. one average UK-wide value is used for each pollutant with no further disaggregation of impacts between geographical region, sector, etc.). The methodology has been developed to assess impacts in 2012 prices.

The use of pollutant concentrations as an input in the assessment tool is different to the damage cost tool, in which emissions are used as an input. Using concentrations is however consistent with the valuation of air quality impacts via the IPA which also takes pollutant concentrations as input. If only emissions information is available then this will need to be converted to concentrations outside of the assessment tool using techniques such as air quality models. In future work, the productivity impacts could be incorporated into the damage cost tool so the productivity impacts could be an add-on to the damage costs and estimated directly from changes in emissions.

The tool developed has undergone a rigorous procedure of checks under standard Ricardo-AEA Quality Assurance processes to test the quality and accuracy of the tool. This process consisted of: peer review by an independent, qualified team member; production of traceable and internally consistent worksheets and use a colour-coding and annotation system<sup>28</sup> to inform the provenance of the data and the process/calculation used to derive the results; and all project staff being appropriately trained with key technical tools.

#### 6.1.2 Refining pathways for consistency and analysis-type

The methodology specifies those impacts that are appropriate for inclusion in CBA given this methodology is intended to be used alongside the existing Defra air quality appraisal guidance. As discussed in the previous section, given that we cannot be certain that the

<sup>&</sup>lt;sup>28</sup> Similar to that adopted by the National Atmospheric Emissions Inventory (NAEI).

WTP estimates which are used to value reductions in mortality exclude a valuation of productivity impacts, all productivity effects associated with mortality are excluded from CBA. Further, productivity impacts through SDL are excluded from the central estimation of impacts given uncertainty around the applicability of CRFs in the UK. Finally, the impact pathway through chronic bronchitis and early retirement is also excluded given the high level of uncertainty around valuation.

As part of this project, a methodology was also developed to quantify the productivity impacts of air pollution on the UK's GDP. Estimating the productivity impacts of air pollution on GDP has similarities to estimating impacts for inclusion in CBA, but also has important differences. Whereas CBA seeks to incorporate all costs and benefits into a net impact for society, assessing the impacts on GDP aims to capture only those impacts which accrue to the national measure of gross output.

Whilst GDP can be calculated in three different ways (income, value added and expenditures approach) our methodology for valuing each impact pathway is most consistent with the income approach: each health impact has been valued using a unit cost which is inclusive of wage and non-wage, direct and indirect costs. It is assumed that the full value of productivity lost using the HCA will accrue to GDP<sup>29</sup>.

The impact pathways through chronic bronchitis and SDL are also not included in the assessment of impacts on GDP for the same reasons as excluding these impacts in costbenefit analysis. Further, the non-market impacts of air pollution on carers and volunteers are also not be included in the GDP impacts given that these are non-market activities; hence the value of these activities is inherently not currently captured in estimates of GDP.

A summary of the proposed approach to valuing each impact pathway is included in Table 6.1. The table also identifies which pathway is appropriate for inclusion in which type of analysis: CBA, assessment of impacts on GDP, both, or neither.

#### 6.1.3 Using the tool to assess changes in concentrations

As discussed in Section 4.2, COMEAP (2010) draws attention to the dynamic population effects associated with chronic mortality impacts which occur as a consequence of changes in pollutant concentrations, i.e. how over time changes in risks affect the size of the population and so the numbers of attributable deaths per year. As discussed in COMEAP (ibid) and elsewhere, these effects are taken into account by using life table methods appropriately. Such life table calculations are however computationally intensive and were outside the scope of the present study. Instead, we focused on the burden of air pollution in a given year, as it simplified the calculations and allowed the project to focus on interpretation of results (what metric to use and what it means for productivity) rather than the calculations themselves.

Within this framework we have looked on the effect of a change in concentrations as the difference between burdens from two scenarios each of which has reached long-term stability. This ignores the population dynamic effects that life tables address. We have not tried to quantify the extent of the resulting approximations.

Given that the tool considers the burden of levels of pollution, chronic mortality impacts are assumed to occur with no lag. Where the tool is used in its existing state to assess the impacts of a change in concentrations this assumption is unrealistic but is of no consequence if, as recommended above, chronic mortality effects are omitted from those pathways included in CBA, given potential overlaps with the existing damage cost estimates.

<sup>&</sup>lt;sup>29</sup> When measuring impact on GDP, consumption impacts are no longer additive to productivity impacts. Although in theory these could comprise separate impacts for inclusion in CBA, these impacts become 'opposite sides of the equation' when measuring impacts on GDP: ie in theory, impacts on GDP could be measured using production (i.e. income) or consumption, not both. We have chosen to use the productivity values given the uncertainty around our estimation of the impacts on consumption given data limitations.

#### Table 6.1 – Summary of proposed approach to valuing productivity impact pathways

Impact	Pollutant	Population Affected	Metric to value	Valuation	Inclus	sion in	assessment	Notes
impact	Fonutant	r opulation Affected		Valuation	CBA	GDP	All productivity costs	NOLES
Chronic Mortality	PM <sub>2.5</sub>	Employed persons	Working years lost	CBI absence cost per day (uprated to year)		~	~	Impacts not additional to WTP values already in CBA guidance
Acute mortality	$NO_2$ and $O_3$	Employed persons	Working years lost	CBI absence cost per day (uprated to year)		~	✓	Impacts not additional to WTP values already in CBA guidance
Absenteeism	PM <sub>2.5</sub>	Employed persons	WDL	CBI absence cost per day	~	~	✓	
Chronic morbidity and early retirement	PM <sub>10</sub>	Employed persons	Working years lost	CBI absence cost per day (uprated to year)				Assessment of impacts very uncertain and could overlap with absenteeism
Absenteeism (via dependents)	$PM_{10}$ and $O_3$	Employed persons (via school children)	WDL (via SDL)	CBI absence cost per day				Excluded given uncertainty around application of CRF to UK
Presenteeism	$PM_{2.5}$ and $O_3$	Employed persons	WDL	CBI absence cost per day	~	~	✓	
Chronic Mortality	PM <sub>2.5</sub>	Carers	Care hours lost	Unit value of care (PSSRU)			*	Impacts not additional to WTP values already in CBA guidance; and non- market impacts not included in GDP
Acute mortality	$NO_2$ and $O_3$	Carers	Care hours lost	Unit value of care (PSSRU)			~	Impacts not additional to WTP values already in CBA guidance; and non- market impacts not included in GDP
Absenteeism	PM <sub>2.5</sub>	Carers	Care hours lost	Unit value of care (PSSRU)	~		✓	Non-market impacts not included in GDP
Chronic Mortality	PM <sub>2.5</sub>	Volunteers	Volunteer hours lost	Average wage (ONS)			~	Impacts not additional to WTP values already in CBA guidance; and non- market impacts not included in GDP
Acute mortality	$NO_2$ and $O_3$	Volunteers	Volunteer hours lost	Average wage (ONS)			*	Impacts not additional to WTP values already in CBA guidance; and non- market impacts not included in GDP
Absenteeism	PM <sub>2.5</sub>	Volunteers	Volunteer hours lost	Average wage (ONS)	~		~	Non-market impacts not included in GDP

Where the tool is used to assess changes in concentrations in isolation of damage costs or to assess impacts on GDP, it will be necessary to add lags to the chronic mortality impacts to ensure that these are consistent with the analysis to which the effects are being added. Adding lags to chronic impacts would provide a more consistent assessment of changes in concentrations but would still not capture the additional level of sophistication of dynamic population effects were life-tables also to be used in the analysis.

# 6.2 Example of application

To demonstrate the use of the proposed methodology and assessment tool, analysis has been produced to assess the burden associated with levels of air pollutants for the UK in 2012. The metrics used in the example application are as described in Table 6.2. These population-weighted mean values have been calculated from PCM<sup>30</sup> (PM and NO<sub>2</sub>) and OSRM<sup>31</sup> (O<sub>3</sub>) modelling results.

Pollutant	Values	Unit	Туре
PM <sub>2.5</sub>	10.6	μg/m³	Annual mean concentration
PM <sub>10</sub>	14.5	µg/m³	Annual mean concentration
O <sub>3</sub>	6.2	µg/m³	Annual mean of the daily maximum of the running 8-hour mean concentrations using a $70\mu g/m3$ (i.e. 35ppb cut-off)
0 <sub>3</sub>	88.4	μg/m³	Annual mean of the daily maximum 1-hour mean concentrations
NO <sub>2</sub>	34.6	μg/m³	Annual mean of the daily maximum 1-hour mean concentrations

 Table 6.2 – Levels of pollutants in 2012

It is important to note that the pollutant concentrations used as inputs in the assessment tool need to be for specific metrics. For some pollutants the metrics available from the modelling results are consistent with those needed for direct input into the tool. However, for others, the metrics available from the modelling need to be converted to other metrics before use as inputs. Details of the conversion factors used are given in Table 6.3 below: note that the daily maximum 1-hour mean values should be larger than the daily maximum 8-hour mean or annual mean values.

For the present analysis, the concentrations for assessment were developed for 2012 for the UK for the appropriate pollutant metrics and thresholds as required by the specific CRFs proposed under each health pathway.

The overall impacts for inclusion in CBA and GDP impacts, and the detailed results, are set out in Table 6.4 below.

The analysis shows that levels of pollutants in 2012 had an additional impact on productivity<sup>32</sup> with an associated economic cost of £1.1bn (or 0.07% expressed as a percentage of GDP). Hence any appraisal of the costs of the current levels of pollutants (or policy to reduce pollutants) using the current Defra guidance will under-estimate the costs (or benefits) associated. This estimate is consistent with (and hence can be considered additional to) the previous estimated burden associated with levels of air pollutants per annum of £16bn (although this was derived for an earlier year).

<sup>&</sup>lt;sup>30</sup> For further information, see: Brookes et al (2013)

<sup>&</sup>lt;sup>31</sup> For further information, see Cooke et al (2013)

<sup>&</sup>lt;sup>32</sup> Additional to the valuation of impacts using the existing Defra appraisal guidance

Pollutant	Units	Metric output by model	Conversion factor used (modelled concentrations multiplied by this factor)	Method used to calculate conversion factor	Metric needed in tool
PM <sub>2.5</sub>	µg/m³	Annual mean concentration	N/A	N/A	Annual mean concentration
PM <sub>10</sub>	µg/m³	Annual mean concentration	N/A	N/A	Annual mean concentration
O <sub>3</sub>	µg/m³	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70 μgm <sup>-3</sup> (i.e. 35 ppb) cut off	N/A	N/A	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70 μgm <sup>-3</sup> (i.e. 35 ppb) cut off
O <sub>3</sub>	µg/m³	Annual mean of the daily maximum 8-hour mean concentrations	1/0.72	From the APHENA study	Annual mean of the daily maximum 1-hour mean concentrations
NO <sub>2</sub>	μg/m <sup>3</sup>	Annual mean concentrations	1.96	Calculated from the average ratio of the measured 2012 annual mean NO <sub>2</sub> concentration and the 2012 annual mean of the measured daily maximum 1- hour mean NO <sub>2</sub> concentrations for all urban background, urban industrial, suburban background and rural background monitoring stations in the UK AURN (using a 75% data capture threshold).	Annual mean of the daily maximum 1-hour mean concentrations

#### Table 6.3 – Conversion of metrics between model outputs and productivity tool inputs

# Table 6.4 – Impacts for inclusion in cost-benefit analysis and GDP impacts of 2012 levels of pollutants

Analysis type	Coverage	£m (PV, 2012 prices)	% of GDP (2012)
Cost-benefit analysis	Total cost	1120	0.07%
GDP Impact	Total lifetime cost	2308	
	First-year cost <sup>33</sup>	1730	0.11%
All impacts assessed	Total cost	2710	

<sup>&</sup>lt;sup>33</sup> The difference between lifetime cost and first-year cost is explained in the text below

#### Table 6.5 – Detailed results of all direct productivity impacts of 2012 levels of pollutants

Impact	Pollutant	Population affected	Life years lost	Deaths brought forward	Working years lost	WDL (000's)	SDL (000's)	Care hours (000's)	Volunteering hours (000's)	Productivity Loss (£m, PV, 2012 prices)	Percentage of total productivity loss (%)
Chronic Mortality	PM <sub>2.5</sub>	All employed (productivity) and all persons (consumption)	420426	210213	47033					1209.4	44.6%
Acute Mortality	NO <sub>2</sub>	All employed (productivity) and all persons (consumption)	5123	5123	599					15.5	0.6%
Acute Mortality	O₃ (35 ppb threshold)	All employed (productivity) and all persons (consumption)	993	993	116					3.0	0.1%
Absenteeism	PM <sub>2.5</sub>	All employed (productivity)				6522				765.4	28.2%
Presenteeism	PM <sub>2.5</sub>	All employed (productivity)				2250				264.1	9.7%
Presenteeism	O <sub>3</sub> (35 ppb threshold)	All employed (productivity)				431				50.6	1.9%
Chronic Mortality	PM <sub>2.5</sub>	Carers						13431		237.8	8.8%
Acute Mortality	NO <sub>2</sub>	Carers						167		3.0	0.1%
Acute Mortality	$O_3$ (35 ppb threshold)	Carers						32		0.6	0.0%
Absenteeism	PM <sub>2.5</sub>	Carers						1760		31.7	1.2%
Chronic Mortality	PM <sub>2.5</sub>	Volunteers							10698	118.7	4.4%
Acute Mortality	NO <sub>2</sub>	Volunteers							133	1.5	0.1%
Acute Mortality	O <sub>3</sub> (35 ppb threshold)	Volunteers							26	0.3	0.0%
Absenteeism	PM <sub>2.5</sub>	Volunteers							735	8.3	0.3%
Total			426542	216329	47748	9203	0	15390	11593	2709.8	

The level of pollutants in this year is shown to have a total impact on GDP of around £2.3bn over the lifetime of these impacts. Some of the impacts associated with chronic mortality occur in future years: this will impact on GDP in future years but these impacts have been discounted back to 2012 to show a present value impact. A more tangible estimate is the first-year impact on GDP: this illustrates the potential impact on GDP in 2012 of the levels of pollutants in 2012. Concentrations in 2012 are estimated to have potentially reduced GDP in that year by £1.7bn (or 0.11% as expressed as a percentage of total GDP) through mortality and morbidity affects.

Across all the pathways assessed, the direct assessment of the productivity impacts of air pollution is valued to be greatest for chronic mortality and absenteeism: these pathways account for around 45% and 28% of all valued impacts respectively. Presenteeism is also a significant cost, accounting for around 12% of the total valued impacts. The impacts on carers and volunteers also contribute a not-insignificant impact to the total (in particular for chronic mortality and absenteeism impacts). The smallest impacts arise through acute mortality due to the low numbers of deaths attributed to the levels of pollution in 2012.

It is also apparent from the table above that the majority of productivity impacts are associated with PM. Relatively few impacts are associated with  $NO_2$  due to the low significance of acute mortality impacts, and the most significant impacts associated with ozone are through presenteeism. This result is consistent with the appraisal of impacts at the EU level: although the scenarios considered in the Climate Cost study depict greater reductions in  $SO_2$  and  $NO_x$  than PM, PM still accounts for the vast proportion of 'productivity-related' benefits estimated (i.e. just focusing on RADs and mRADs).

# 6.3 Uncertainty and discussion of results

The methodology developed to appraise the productivity impacts of air pollution is based on a number of assumptions. These should be taken into consideration when carrying out analysis using the assessment tool. These caveats could imply that the impacts estimated are either an over- or under-estimate of the true impacts of air pollution.

Those which could lead to impacts being over-estimated are:

- With the exception of ozone, all impacts are assumed to have no threshold<sup>34</sup>
- Use of all-cause rather than cause-specific CRFs, in particular in the case of chronic mortality as discussed in Section 4.2.2.1 above
- For mortality, a population-wide CRF is applied to all persons rather than a specific CRF applied for employed persons only. The death rate is likely to be higher in the unemployed, leading to lower deaths in employed persons than is estimated here. However, no data could be found depicting specifically the mortality rate of persons in employment hence this effect could not be accounted for in the present analysis
- Comparison of the number of WDL through absenteeism estimated using this model relative to the breakdown of underlying reasons for sickness absence in the baseline data suggest the estimate of WDL due to air pollution may be relatively high (however, our bottom-up sense check of the number of WDL (see Appendix 3) and wider reasoning provides support for our estimation of WDL)

Those that could lead to impacts being under-estimated are:

• Other impacts of air pollution on health have been excluded to avoid the potential for overlaps, for example acute mortality through PM, chronic mortality from non-PM

<sup>&</sup>lt;sup>34</sup> Meaning there is no non-zero concentration that is safe for the population as a whole and a significant impact is found where concentrations of the pollutant are above a certain concentration (the threshold).

pollutants (i.e. NO<sub>2</sub> and ozone) and other morbidity outcomes (e.g. hospital admissions, symptom days, etc.). In particular for the latter, the interpretation of the WDL metric in this study could be considered conservative in comparison to other air quality impact assessments which have included a quantification of WDL or RADs alongside other morbidity outcomes (US EPA and DEC NSW)

- Some pathways that have been quantified have been excluded from final valuation due to lack of appropriate data (e.g. chronic morbidity) or concerns regarding the applicability of CRF (e.g. SDL see section on sensitivity analysis below)
- Only two types of non-market activities are included here for assessment: in practice, those affected by air pollution are likely to carry out other productive activities with an associated economic benefit which may be lost through the mortality or morbidity impacts of air pollution.
- Use of a threshold of 35 ppb rather than 10ppb for ozone impacts
- Estimates exclude a number of relatively minor pathways with impacts on productivity that were de-prioritised as part of the pathway prioritisation exercise (see Section 3 above)
- Numbers only include costs that occur within the UK: all trans-boundary impacts of UK pollution on productivity have not been included, consistent with Green Book guidance.

Those where the direction of impact is uncertain (or more general caveats around the use of the tool) are:

- The external costs of air pollution vary according to a variety of specific environmental factors including: geographic location of emission sources, height of emission source, local and regional population density, meteorology, etc. The assessment tool appraises impacts on a UK-wide average basis only. In practice, impacts may be more or less severe depending on the specific nature of the concentrations of pollutants
- Given the tool assesses impacts on a UK-wide basis, the tool is therefore potentially more relevant for national rather than regional or local policy appraisal but could be adapted where necessary with appropriate considerations attached
- All pathways have been implemented using linear CRFs: although in practice this is relatively appropriate for PM, it may be less relevant for ozone
- No direct estimate of the impacts of air pollution on reductions in productivity through presenteeism has been made: our analysis uses a proxy for the potential productivity loss of a minor RAD from the literature for (related but) different health problems.

## 6.4 Sensitivity analysis around central estimates

The detail above sets out the proposed approach to estimating a central value for the impacts of air pollution on productivity. However, there are a number of uncertainties in this valuation which are derived from the underlying parameters used in the estimation of impacts. In the tool we have included functionality to explore the potential impact of five key sources of uncertainty:

- Concentration response function variation
- Average life-years lost and number of deaths associated with chronic mortality
- Inclusion of SDL
- Valuation of productivity impacts using bottom-up values other than CBI values

• Baseline rates of sickness absence used in WDL calculation.

Functionality has also been added to the model to vary assumptions around the discount rate and average annual productivity growth applied to the valuation of impacts. This section also explores a further sensitivity of the results to the thresholds used for the ozone concentration metric.

#### 6.4.1 Concentration response functions

CRFs as proposed in the literature and used here are typically reported with a 95% confidence-interval around the central estimate to show the likely range of uncertainty. For each pathway considered, the low and high confidence-interval values have also been included in the tool. This allows the user to adjust the CRFs used between the low, central and high values, producing low, central and high estimates of the health impacts (and associated costs) respectively. The range of impacts around the central estimate obtained by adjusting only this parameter is shown in Table 6.6.

Table 6.6 - Sei	nsitivity analysis	around the CRF
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	Valuation of total cost (PV, 2012 prices)							
Analysis type	Low CRF	Central CRF (central estimate)	High CRF					
Cost-benefit analysis	939	1120	1308					
All impacts assessed	1963	2710	3438					

#### 6.4.2 Average life-years lost

As discussed in the section on chronic mortality impacts above, COMEAP (2010) encourage the use of total population survival time to measure the impacts of chronic mortality. However, there are many combinations of "number of deaths" and "average LYL per death" that aggregate to the same total population survival time lost.

Our central estimate of the number of life years lost takes an average LYL per death of 2 years. In reality the number of affected individuals is likely to fall between two extremes: the number of "attributable" deaths representing a lower bound estimate of the number of deaths; and all deaths (at age over 30) being affected (with a lower associated average LYL). COMEAP (2010) noted that it is not known how this population-wide burden is spread across individuals in the population but we can speculate between various possibilities.

	Impacts across all ages			Impacts in under 65's			Impacts in over 65's			
Average LYL per death	Deaths (000's)	LYL (000's)	Working LYL (000's)	Deaths (000's)	LYL (000's)	Working LYL (000's)	Deaths (000's)	LYL (000's)	Working LYL (000's)	Value of impact £m
11.8	35.6	420.4	31.9	4.9	37.1	24.1	30.7	383.4	7.8	775
8	52.6	420.4	37.0	7.2	43.2	28.2	45.3	377.2	8.7	951
4	105.1	420.4	43.5	14.5	51.3	33.6	90.7	369.1	10.0	1120
2	210.2	420.4	47.0	28.9	55.6	36.5	181.3	364.8	10.5	1209
1	420.4	420.4	48.8	57.8	57.8	38.0	362.6	362.6	10.8	1263

#### Table 6.7 – Impact of different LYL per chronic fatality on health impacts and value

The impacts of changing the average number of LYL per chronic death on intermediate chronic mortality health impacts and their subsequent valuation is shown in Table 6.7. This

displays how different numbers of deaths and average LYL per death can be combined to create the same overall level of population survival time lost, with consequent effects on the size of impacts assessed.

	Valuation of total cost (PV, 2012 prices)					
Analysis type	Low deaths (high average LYL 11.8 years)	High deaths (low average LYL 2 years) (central estimate)				
Cost-benefit analysis	1120	1120				
All impacts assessed	2149	2710				

In the tool we have developed the functionality to vary the number of deaths through chronic mortality affects. Holding the overall number of life-years lost across the population constant, we have included the option to calculate impacts where the average LYL assumed per death is 11.8, hence calculating impacts associated with the number of "attributable" deaths. This allows the user to test what impact changing this assumption around the average life-years lost per death has on the final valuation of productivity impacts.

The range of impacts on final aggregate costs in the model through changing this parameter is shown in Table 6.8. This sensitivity has no impact on the value of impacts to be included in CBA as chronic mortality impacts have been excluded due to concerns regarding the overlap of these impacts with existing valuation of the reduction in risk of death using WTP in Defra's appraisal guidance.

#### 6.4.3 Inclusion of SDL

In the core proposal for impacts to be included in CBA and GDP impacts, we propose that impacts on productivity through dependents being absent from school are excluded from the analysis. This is due to uncertainty in the applicability of the CRFs to the UK: the CRF developed for PM was estimated in the context of high concentrations of PM and other studies have not found evidence of a non-zero impact; and the UK has relatively low concentrations of ozone in comparison to the US where the CRF for this pollutant was developed.

Although including these CRFs is likely to over-estimate the impact of air pollution on SDL in the UK, excluding both CRFs is likely to lead to an under-estimate in the total impact: the real impact is likely to be somewhere between the two. Hence the tool includes the functionality to switch the impacts of air pollution on SDL on or off. This allows the user to gain a sense of the size of the likely impacts of SDL under these relatively 'high' CRFs. The range of impacts flexing only this parameter is shown in Table 6.9 below.

Analysis type	Valuation of total cost (PV, 2012 prices)					
	SDL not included (central estimate)	SDL included				
Cost-benefit analysis	1120	1175				
All impacts assessed	2710	2765				

#### 6.4.4 Valuation of productivity impacts

The tool values the health impacts using unit values for the cost of absence from the CBI survey. As discussed above, these values were chosen such that the marginal cost of a unit of labour included both non-wage and indirect costs of absence alongside more direct costs.

However, there is some uncertainty around the use of this valuation. The costs are based on survey data from employers who are asked to estimate the costs of absence ex-post. Hence the reliability of these estimates depends on how accurately employers are able to value all costs associated with absence (e.g. including less tangible impacts such as on customer satisfaction) at the end of the year for the whole period.

Alternative sources are available with which productivity impacts can be valued. Average wage rate data are readily available from ONS. Although this inherently excludes non-wage costs to employers these data provide a relatively robust estimate of the wage element of marginal cost. Further, average wage is used to value impacts in other air quality assessments (e.g. US EPA and DEC NSW assessments).

An additional measure would be to take average wage and add 30% to account for nonwage costs. This is the approach recommended in UK Government's Standard Cost Model and is used by DWP in their assessment of the costs of work-absence (DWP, 2013).

Lastly, top-down estimates of the marginal productivity of workers can be derived from aggregate GDP data. In theory, the top-down and bottom-up approaches should produce similar estimates of the marginal value of productivity of workers. In practice, given the different measurement techniques, there are differences in the values produced, with the top-down values being much higher than bottom-up.

Analysis type	Valuation of total cost (PV, 2012 prices)				
	Average wage	CBI values (central estimate)	Average wage plus 30%	GDP per worker per day	
Cost-benefit analysis	916	1120	1179	2168	
All impacts assessed	2535	2710	3175	4949	

Table 6.10 – Sensitivity analysis around the valuation of productivit	y impacts
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For the central estimate of impacts, we have proposed using the CBI values as these are central between average wage and top-down values. Further, a similar approach is taken in the EU CAFE methodology. As with the other sensitivities above, flexibility has been built into the model to allow the user to explore what impact changing the unit valuation has on the overall estimated impacts. The range of impacts is shown in Table 6.10.

#### 6.4.5 Baseline sickness absence in WDL calculation

Some caution has to be applied in relation to the application of the CRF for WDL to current "all-cause" sickness absence rates in the UK. As discussed in Section 4.4 above, further consideration of cause-specific absence data provided by ONS suggests that the number of "attributable" cases could be over-estimated by using an all-cause baseline.

The ONS data indicate that of the total 134 million sickness absence days in 2012, respiratory sickness accounted for 4.5 million WDL and cardiovascular sickness accounted for 5.7 million WDL. Minor illnesses (including coughs, colds and other minor respiratory illnesses) contributed a further 25.6 million sickness absence days.

Our central estimate of impacts suggests around 6.5m WDL could be attributed to current levels of air pollutants. Relative to a stricter baseline of 'air pollution related' sickness absence days (35.8m per annum), this suggests a CRF of around 17% per 10  $\mu$ gm<sup>-3</sup> increase in PM<sub>2.5</sub> would be required to produce the same level of attributable WDL. This is higher than the predicted impact of long-term exposure to PM<sub>2.5</sub> on cardiovascular mortality which would be anticipated to parallel the impact on the development of disease. It also implies a much greater impact on respiratory health than implied by the results of most individual studies of acute respiratory endpoints, which we have assumed would dominate as a cause of respiratory WDL.

Given this uncertainty, we have added sensitivity to the tool to allow the user to test the impact using a lower baseline rate of sickness absence which focuses on that component of sickness absence that could plausibly be related to air pollution. This produces a lower bound estimate of WDL. The range of impacts is presented in Table 6.11.

Table 6.11 – Sensitivi	ty analy	sis around/	baseline	sickness absence
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	Valuation of total cost (PV, 2012 prices)				
Analysis type	'Air pollution related' absence	All-cause sickness absence (central estimate)			
Cost-benefit analysis	560	1120			
All impacts assessed	2149	2710			

#### 6.4.6 Sensitivity of results to CRF choice and thresholds

It is apparent from the overall results that the majority of productivity impacts are associated with PM. However, it should be noted that this result is likely to be in part a consequence of the CRF's and the thresholds for pollutant metrics selected for use in the modelling tool. The balance of impacts between different pollutants would be different had the impacts of chronic exposure to other non-PM pollutants on mortality been included explicitly rather than excluded due to concerns regarding overlaps with the CRF used for PM.

As mentioned in section 4.3.2.2, HRAPIE indicate that additional analysis for  $O_3$  concentrations above 10 ppb (20 µgm<sup>-3</sup>) should also be performed. We have therefore carried out additional calculations for pathways for which we have used a 35 ppb threshold for ozone as our central case. In these calculations we use a 10 ppb threshold instead, in order to investigate the impact of using the lower threshold. Using a 10 ppb threshold produces a higher valuation than using a 35 ppb threshold. The results are shown in Table 6.12 below.

Impact	Pollutant	Population Affected	2012 concentration	used in tool (ugm <sup>-3</sup> )	Valuation of cost for the specific pathway (£m, PV, 2012 prices)	
			Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70 μgm-3 (i.e. 35 ppb) cut off	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 20 μgm-3 (i.e. 10 ppb) cut off	Valuation using a threshold of 35 ppb	Valuation using a threshold of 10 ppb
Acute mortality	O <sub>3</sub>	Employed persons	6.2	44.4	3.0	21.3
Presenteeism	O <sub>3</sub>	Employed persons	6.2	44.4	50.6	360.0

# **7 Conclusions and recommendations**

Ricardo-AEA, alongside its partners IOM and Metroeconomica, were commissioned to identify the links between air quality and productivity and where possible, develop a methodology with which productivity impacts could be assessed, quantified and valued.

The project carried out a wide-ranging critical review of published research on the assessment and valuation of air pollution impacts from UK, EU and other international sources. Starting from the basic economic production function, a long-list of conceivable impact pathways was identified through which air pollution could influence productivity. From this list, a number of pathways were taken forward for quantification under the project where:

- There was evidence of a clear link between air pollution and productivity impacts
- These pathways were considered significant in a UK context
- Sufficient evidence was available with which a quantitative estimated impact could be developed.

This project developed a methodology to assess the productivity impacts of air pollution in the UK and estimate the associated economic cost. This methodology was captured within a modelling tool for use in policy appraisal.

An initial assessment of the productivity burden associated with levels of pollution in 2012 found a significant economic cost. Pollution levels in 2012 had an estimated total cost of  $\pounds 2.7$ bn through its impact on productivity (over the lifetime of impacts)<sup>35</sup> and an impact in 2012 equivalent to a reduction in GDP of 0.11%. The ability to capture these productivity impacts in policy or project appraisal going forward will increase the comprehensiveness of the analysis and reduce the likelihood that the benefits of reducing pollution are underestimated.

The methodology developed under this project provides a reasonable indicative estimate of the impacts of air pollution on productivity, avoiding potential overlaps with the benefits captured under the current air quality appraisal guidance. However, estimating the impacts of air pollution on productivity is uncertain and necessarily based on a number of assumptions. Important caveats around the methodology have been identified in the discussion of results in Section 6.3 above.

The project team recommend that Defra consider the following issues going forward to improve the methodology developed here:

- The methodology developed is based on the expert judgement of the project team and has included evidence which has not yet been considered fully by COMEAP. Defra should be aware of any discussions and conclusions of COMEAP related to the evidence base on which this methodology has been developed or where there is potential to include further evidence in the methodology
- The methodology developed under this project could be integrated into the existing damage cost tool such that productivity impacts can be assessed alongside effects already captured by the damage cost methodology
- A number of issues arose during the development of the valuation approach regarding the assumptions taken to underpin the approach. Specifically, these issues

<sup>&</sup>lt;sup>35</sup> Note only £1.1bn of these impacts are considered additional to those included under Defra's appraisal guidance.

were assumption around replacement of workers, overlap of direct productivity assessment and inclusion of consumption impacts. It was necessary to take a judgement on these issues under this project to facilitate the development of the approach. The judgements taken on each of these issues were deemed most appropriate by the project team based on the knowledge and experience of the project team and the context of the literature reviewed. However, different approaches may have been taken for different valuation methodologies in other contexts. As such, it would be beneficial if these issues were discussed on a crossdepartmental basis to reach agreement regarding these issues in the appraisal and evaluation of policy. This would in turn improve the comparability of CBA across Government departments and its effectiveness as a tool for policy appraisal.

# Appendices

Appendix 1: References

- Appendix 2: Wider impact pathways
- Appendix 3: An indirect estimation of WDL via other health outcomes
- Appendix 4: Concentration response function options included in tool

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# **Appendix 2 – Wider Impact Pathways**

#### Overview

The objective of this project was to identify the links between air pollution and productivity. To identify these links, we started with a simple economic production function to identify potential impact pathways against each of the factors of production. The output of this exercise was a long list of potential pathways as set out in Section 3.2 above.

This long-list of potential impact pathways was then reduced to a set of pathways to be taken forward for analysis. These pathways included only those where the project team considered: the evidence was sufficient around the existence of the pathway, the pathway was significant in the UK context and quantitative information existed with which an estimated impact of the pathway could be quantified. These pathways focused on the direct impacts of air pollution on human health through ingestion of pollutants via inhalation.

Under this process a number of potential pathways were not taken forward for quantification. This appendix discusses in more detail those pathways which were de-prioritised, setting out the evidence gathering undertaken for each pathway.

These 'wider' pathways are grouped according to the factor of production upon which they could impact. For each pathway, we construct a hypothetical scenario through which air pollution could feasibly impact on the relevant factor of production. We then discuss the evidence for the pathway and present our rationale for excluding this pathway from future quantification. A summary of our conclusions under each pathway is included in Table A2.1.

#### Impact pathways via capital

Capital as a factor of production is defined as covering all fixed inputs into the production process; e.g. buildings and machinery.

#### Wider pathway 1: Air pollution curtails operation of sensitive capital assets

Where air is used as an input into a production process using fixed capital or machinery, it is feasible that air pollution could therefore reduce the effectiveness (and hence the productivity) of the machinery used. For example, internal combustion engines used in cars and some industrial processes require air as an input.

Air pollution could reduce the potential output per machine or directly reduce the total stock of machinery available (where some machines are unable to work due to air pollution). This pathway could impact acutely, placing a machine out of action, or could impact through gradual build-up of pollutants over time, leading to a requirement to clean machinery (whilst the machine is being cleaned, production using the machine is halted). Alternatively, the impact could occur through a general loss of performance of fixed assets.

The potential for air pollution to directly impact machinery could be reduced in practice through the application of filters. Where air is required as an input, it is likely that firms already apply filters or other devices to ensure a supply of air is provided and production is not interrupted. For example, vehicles using internal combustion engines have air filters to prevent particulate matter and other air-borne contaminants from entering the engine and reducing its performance. Therefore, current levels of air pollution may not have a significant prohibitive impact on machinery (and consequent impact on productivity) given technological advancements are already widespread. However, the fact that filters are applied to machinery or engines (or other mechanisms) to clean air represents a direct cost that air pollution (in part) places on businesses; if air pollution was reduced, it could be argued that this could reduce the requirement for air cleaning equipment (or the regularity with which filters are replaced), in theory reducing the cost of machinery.

A wider but connected issue is that air pollution could also have a conceivable impact on the effectiveness of machinery through the acidification of water. Acidification has more obvious

impacts on ecosystems, but could also in theory impact on production where clean water is used as an input. Again, machinery may be needed at additional cost to clean and/or filter water. However, this review found no evidence to suggest this is a significant effect.

#### What is the strength of evidence underpinning this pathway?

This review has found limited evidence regarding the potential impact of air pollution on the effectiveness of machinery in UK context or wider. It is evident that air filters are widely applied to internal combustion engines to prevent abrasive particulate matter from entering engines. Filters are applied to prevent particulate matter causing mechanical wear and oil contamination. However, it is not clear what types of air pollution are particularly problematic or the potential impacts of removing filtration (i.e. there is no evidence of the potential impacts of current levels of air pollution).

This pathway is not included in the current Defra guidance. Nor is it considered or noted as a potential impact in either the EU CAFE or US EPA studies.

#### What is the likely significance of the pathway?

How significant this pathway is will depend on the stock of machines that are susceptible to the impacts of air pollution. The vast majority of road vehicles in the UK use internal combustion engines (in 2013 there were 35m vehicles licenced to use roads in the GB; see DfT, 2013), alongside some industrial machinery (e.g. small scale power generation). Further, filters are also applied in air conditioning systems which need to be replaced or washed regularly to avoid losses in efficiency or useful life (a report in 2010 suggested there were around 2m mobile and stationary air conditioning units in the UK; see ICF International, 2011). If we assume all these machines are susceptible to PM, the impacts (or the preventative costs incurred to avoid negative impacts) of air pollution could be reasonably significant.

However, it is unclear what impact changes in particular pollutants will have on the need for air filters or the regularity with which these need to be replaced. In theory, reducing air pollution should impact on how often filters need to be replaced but marginal changes in air pollution may only have a negligible (if any) impact on the rate of replacement given the need to filter other air-borne contaminants (e.g. organic matter). Further, some studies suggest that for engines, once fitted, regular replacement may not necessarily be required, reducing the cost associated with air pollution (Norman et al, 2009).

#### Does information exist with which this pathway can be quantified?

No evidence has been found that this pathway is significant: hence no information is available with which this pathway can be quantified and monetised.

#### Conclusion: Can we consider this pathway further?

Air pollution could in theory impact productivity through this pathway. However, we are unable to consider this pathway further under this project given lack of evidence regarding the potential impact of air pollution on engines and other machinery which require clean air as an input. Further, there is also no available information with which impacts can be estimated. Based on the discussion above, any changes in air pollution (in particular marginal changes) are likely to have only a negligible impact as it is unlikely that the application of filters is sensitive to small changes in air pollution.

#### Wider pathway 2: Buildings and other fixed productive assets are corroded

Air pollution has a well-documented impact on buildings: it can have a corrosive or soiling impact. Consequently, air pollution could be considered to have an impact on productivity through its impact on buildings, where pollution affects the stock of useable buildings or productive assets. Air pollution could directly 'reduce' the stock of capital through the corrosive effects of acid rain. This could lead to buildings needing to be subsequently replaced sooner or face the cost of necessary repair work to maintain their productive potential.

Buildings are a relatively indirect source of capital in the production process. Even though the exterior of a building may be (gradually) affected by air pollution, the productivity of the processes within the building is unlikely to be affected in the short-term. However, where buildings are repaired or maintained on an ongoing basis, this effectively represents a willingness to pay on behalf of the firm to avoid possible (more significant) losses in production of the processes contained. This repair has a direct cost to the business.

Where buildings are not repaired, air pollution could have a direct impact on productivity where buildings need to be fully refurbished or even replaced, which could have a consequent effect of halting productive activities. This is likely to happen over a longer time period: a building will be corroded over time but the processes within are likely to remain unaffected until a particular threshold is reached where refurbishment, replacement or other rectifying action is required. Even then, the productive output of the processes may continue but with additional costs (e.g. through relocation to a different building) and the effects are unlikely to be permanent.

Building soiling on the other hand would not have a direct impact on productivity as this does not have a corrosive impact, only an amenity impact<sup>36</sup>.

Thinking more widely, air pollution could also have a possible impact on the fixed amount of capital where natural resources (e.g. natural stone) at different stages of the mining process (i.e. pre, during and post) are exposed to acidification. This could directly reduce the stock of the useful resource available.

#### What is the strength of evidence underpinning this pathway?

The pollutants most implicated in acid damage are SO<sub>2</sub> (most importantly), H+ and NO<sub>2</sub>. In the current Impact Pathway Approach (IPA) guidance, material damage from ozone on rubber (based on a quantification of impacts by: Holland et al, 1998) and SO<sub>2</sub> on natural stone and zinc coated materials is captured. The approach values the impacts on buildings using building repair values (AEA, 2006). Soiling of buildings is also included in the current guidance valued according to the associated cleaning costs.

The Climate Cost study (Holland et al, 2011a) included an assessment of the damage to building materials from acidic deposition using the Atmospheric Long-Range Pollution Health/Environment Assessment (ALPHA) model. Holland et al (2012) also monetises damage to buildings from acid deposition using this model. The ALPHA model includes an approach to monetise impacts of acid corrosion (trends are included for SO2, NO2 and H+) of stone, metals and paints on 'utilitarian' buildings and ozone damage to polymeric materials (e.g. natural rubbers). The impacts are valued once corrosion is assumed to have reached a critical thickness loss, applying values to materials used in the building trade.

In the US EPA's study of air pollution impacts (2011a), it notes that acidic deposition has been shown to have an effect on a number of materials including zinc, galvanised steel and other metals. This study quantifies the impacts of sulphur oxides on carbonate stone, galvanised steel, carbon steel and painted wood in commercial and residential buildings. The impact of ozone on rubber is noted but not quantified alongside other impacts. Materials damage is valued as cost of changes in future materials maintenance activities.

Hence the link from air pollution to a corrosive impact on buildings is well studied and included in existing impact assessments. However, there is little evidence of the link to raw (or unmined productive) materials. AEA (2006) note that the impact of acidification is worse in areas of northern Europe where bed rock is harder and weathers too slowly to counteract deposited acidity. But this does not specifically refer to stone (or other mined materials) as a potential source of commercial viable raw material. The impact of air pollution on reserves of

<sup>&</sup>lt;sup>36</sup> However, if we were to take a wider definition of productivity, it could be argued that given cleaning has an associated time cost, this could fall under the boundary of a productivity impact if it is argued that the time cost of the cleaner reduces the amount productive labour: without information regarding the proportion of buildings cleaned and those left dirty, it is difficult to quantify what proportion of the impacts from AP should be allocated to amenity or cleaning costs

natural material such as stone will depend on the type of natural material, how it is situated naturally (i.e. is it exposed) and the methods of extraction (i.e. whether it is exposed to potential corrosive forces).

#### What is the likely significance of the pathway?

Defra's IPA notes that the benefits of reduced impacts on buildings are relatively low. The Climate Cost project estimated the benefits of reducing damage to buildings in its pollution reduction scenario to be around 0.7% of total health, building and crop benefits. Holland et al (2012) estimate benefits to materials and crops under its emissions reduction scenario are around 1.3% of health, crop and building costs.

In terms of impacts on raw materials, the significance of impacts would depend on the size of relevant extractive industries in the UK and the susceptibility of their extraction methods to potential corrosive impacts of air pollution.

#### Does information exist with which this pathway can be quantified?

The impact of air pollution on damage on buildings is well studied and quantitative data exist with which an impact can be identified. There is insufficient evidence that any impact on natural resources is apparent hence no information exists with which an impact can be estimated.

#### Conclusion: Can we consider this pathway further?

Existing Defra IPA guidance already captures the impact on buildings by valuing impacts at the cost of repair. These impacts could be classified as productivity impacts if buildings were considered as a physical capital input into the production process. Hence the inclusion of any additional impacts on buildings under this project would risk double counting with the impacts already captured under existing guidance and as such no further impacts are taken forward for quantification.

There is no evidence of any potential impacts of air pollution on raw materials or extractive industries. Further consideration could be given to potential impacts where amenity of tourists is affected, impacting on the value of the UK tourist industry.

# Wider pathway 3: Increasing returns to capital incentivises additional investment in the long-term

Reducing air pollution has positive health impacts (e.g. by increasing life expectancy) which subsequently improve productivity in the short-term through increasing the stock of labour. Improving health could also have a secondary impact on productivity through increasing the pension age (and hence number of years worked) in the longer-term.

In economic theory, if the supply of labour increases in an economy, all other things being equal, this would also increase the return to capital in that economy. This in turn may incentivise an increase in investment in capital over the longer term, to take advantage of these greater returns to capital investment. If this investment occurs, this would increase the overall stock of capital (and hence production) in the economy.

#### What is the strength of evidence underpinning this pathway?

This effect is not mentioned in existing air pollution impact studies.

#### What is the likely significance of the pathway?

The likely significance will be determined by the improvements in health gained through reductions in air pollution and the response of firms to this impact. However, the size of these long-term impacts is likely to be less significant than the in-year valuation of the direct health impacts, in particular when considering discounting in cost-benefit analysis.

#### Does information exist with which this pathway can be quantified?

Any impact through this pathway will be in the long-term. Further, a number of factors will influence investment in capital over time, which makes any relationship between labour and
investment in capital difficult to define in this context. Hence it is not surprising that this review did not find any evidence to capture this impact. An economy will accumulate capital over time and this will be captured in the overall rate of growth; however, what proportion is associated with (and hence what the impact of) increasing life expectancy will be at best very difficult to disaggregate. Deciphering what factor of this is subsequently associated with improving air quality will be even more complex.

#### Conclusion: Can we consider this pathway further?

Although this pathway could exist in theory, given the difficulties with attempting to produce a robust estimate of this impact and the lack of evidence that this is a significant impact of air pollution, we have not taken this pathway forward for quantification.

# Impact pathways via labour

#### Wider pathway 4: Impact of absenteeism on longer term productivity growth

Where an improvement in health reduces absence, it is feasible that this could have a positive impact on the productivity of person in the longer term. Lower levels of absence would lead to greater hour's worked and subsequently greater learning on the job. Further, longer life and lower absence would lead to greater returns to investment in training, which may encourage higher investment from employers. Where improved health increases attendance of school, this could also have a positive impact on productivity through the future productivity of a child.

#### What is the strength of evidence underpinning this pathway?

This is not covered by EU studies using the ALPHA model. The ALPHA model uses a valuation of absenteeism based on the CBI surveys. These surveys look at static costs to business (e.g. lost output for that day) rather than defining a cost of absence over a longer-term perspective. The US EPA study also overlooks this potential impact: in this study the impacts on productivity are quantified using average wage. As with the ALPHA model, this is a static value of impacts and does not take into account any impact on the future wage of the person (and hence longer-term productivity growth). In addition, in the US study school loss days are valued in a similar way to working days lost, valuing the impact of parent absence to care for the child. This therefore does not capture any indirect cost of the child's absence which could include the impact of absence on the child's future productivity.

Looking outside existing air quality impact assessments, there does not appear to be a great deal of evidence regarding a possible link between sickness absence and long-term productivity improvements of employees.

Evidence does exist that greater life expectancy encourages human capital investment in children, in particular for developing countries (Jayachandran, S. and A. Lleras-Muney, 2009). Further, the link between level of schooling attained and future productivity and wage rates is well documented. Hence in theory, additional school attendance could have a direct impact on improving a child's future productivity through additional learning. Although evidence regarding a general link exists, information is not available such that (potentially minor) absence of varying lengths associated with air pollution can be linked to future changes in productivity.

A recent study by Isen et al (2014) considers the long-term impacts of in-utero and early childhood exposure to ambient air pollution on adult labour market outcomes. Rather than focusing on reductions in learning through absenteeism, this study focuses on the impacts of air pollution through a general reduction in the ability of children to learn and hence their cognitive/physical development, and the impact of continuing ill health on the ability to work in future life. This study estimated the impact to be \$6.5bn (2008 prices) for the annual cohort of births in 1972. Whilst this study suggests these impacts could be significant, no similar study has been undertaken of the likely impacts in the UK.

#### What is the likely significance of the pathway?

The impact on productivity will be determined by the length of absence, the length of employment before absence and the type of employment. Minor or short-term absences could have relatively minor impacts on on-the-job learning as learning could simply be deferred. Further, where a person has been in a job for a long period of time (or where a job involves relatively little learning), there may be little or no potential for further productivity improvements which could be affected by absence.

Although there may be greater incentives for employers to invest in staff when they live longer, this could be tempered by other factors, such as the possibility that employees could move jobs or the fact that time taken for training itself incurs productivity losses in the short-term. Both these factors could curtail the incentive to invest in further training.

For school absences, the impact on educational attainment is likely to be relatively minor where absences are short: schools are likely to have processes in place to ensure absent pupils catch up on missed learning. Further, the link from formal education attainment to future wages (and hence productivity) is influenced by a number of other more significant factors (e.g. further education attainment, etc.).

#### Does information exist with which this pathway can be quantified?

No specific evidence has been found with which an estimate of the impact of air pollution on productivity through this impact pathway can be made. As noted above, it is likely that other factors have a stronger impact on learning (either at school or on the job) and hence future wage attainment and growth.

#### Conclusion: Can we consider this pathway further?

The impact of short-term absences on work and school attendance, and subsequently on learning, is likely to be small given learning can be deferred. The impact of longer term absences could be more significant. However, there is insufficient information to link air pollution to absence and different levels of educational attainment, with consequent impacts on productivity, which is not surprising given the long-term nature of this impact pathway.

#### Wider pathway 5: Air pollution and Visibility

Visibility relates to reduction in visual range through the presence of air pollutants. The link between air pollution and visibility has long been explored by analysis in US where visibility problems are particularly associated with PM and NO<sub>2</sub> (EC4MACS, 2013). This has mainly been explored through impacts on visual amenity. Poor visibility could also have an impact on productivity through both the productivity and stock of labour:

- It could reduce the productivity of days worked in outside jobs or of non-market productive activities where outside and reliant on visibility: e.g. voluntary work.
- It could reduce the stock of labour by increasing travel time to work hence reducing working time/non-working productive time, reducing the number of working days directly (e.g. where an outside job cannot be performed due to poor visibility), reducing the amount of non-market productive activities where outside the home and reliant on visibility voluntary work or through flight/other travel cancellations where visibility is poor causing missed work days.

The impact of air pollution on visibility has been a problem in the UK historically (e.g. the smog's of the 1950s) but these impacts have not been included in recent air quality impact assessments in the UK. This is also true for the assessment of impacts at an EU level: This could be because of recent significant improvements in visibility across much of UK and Europe. However, there are still relatively recent examples where economic activity has been disrupted through poor visibility, for example disruption to flights due to eruption of Icelandic volcano (although this was not a problem caused in the UK) and dense fog (e.g. December 2013 although no evidence found in this review has linked these episodes to air pollution).

For visibility to have an impact, it needs to last for a sufficient period of time so as not to simply have the effect of deferring work.

#### What is the strength of evidence underpinning this pathway?

Holland et al (1999) used data on behalf of UNECE Task Force on Economic Aspects of Abatement Strategies to calculate substantial damages from visibility at EU level. However, given the lack of concern over impacts and the restraints on analysis (in particular related to short-term fluctuations in pollution levels) they concluded the results were not reliable.

Following this, Holland et al (2005) performed a comprehensive review of quantification methods for visibility impacts and concluded there was an inadequate base of UK or European data on which to base a credible assessment. When formulating the UK damage costs, AEA (2006) concluded that this issue was not regarded as significant in Europe, as the loss of visibility due to air pollution was considered less of a problem now than historically. The impacts of air pollution on visibility also do not form part of appraisal at the EU level.

Studies in the US do include assessment of impacts on visibility. The US EPA (2011a) considers air pollution impairing visibility in both residential and recreational settings. The benefits of improving residential visibility relate to the impacts on an individual's daily life (e.g. at home, work, etc.), whereas the recreational impacts capture the effect on people attending national parks or wilderness areas.

In the US studies, changes in visibility are valued using an individual's WTP for improvements in both settings which are based on contingent valuation studies. However, it is unclear whether these studies requested that people include valuation of productive activities in their estimates of WTP: the paper (and supporting studies on which WTP estimates are based) suggest that WTP may capture only the amenity impacts of poor visibility for both 'residential' and 'recreational' valuation, and hence may exclude estimation of impacts on earnings and hence productivity.

#### What is the likely significance of the pathway?

The US EPA (2011a) study estimated that the total benefits through improved visibility under CAAA to be \$67bn in 2020 (\$48bn is residential valuation – this is equivalent to around 4% of all mortality benefits in 2020).

#### Does information exist with which this pathway can be quantified?

There is no evidence that the link between air pollution and poor visibility is significant in the UK. Further, although CRFs for this impact pathway exist from the US studies, there are serious concerns regarding the applicability of these CRFs in a UK context.

#### Conclusion: Can we consider this pathway further?

Generally there is low concern regarding visibility problems in UK. The link between visibility and air pollution has not been identified and specified for current UK conditions. Data may exist on current levels of visibility, but there is difficulty in linking this data to cause and the contribution of air pollution. Further, although CRFs are available from US studies, previous work has suggested that applying them in a UK context may not be appropriate.

Hence in the absence of data linking air pollution to changes in visibility (with significant impact on poor visibility episodes which would have productivity impacts), we propose that this pathway is not pursued under this project.

#### Wider pathway 6: Indirect impacts on human health

Alongside the direct risks of air pollution to health via inhalation, air pollution may also cause a risk to health through the food chain. For example, air pollutants could impact on humans through impacts on crops, livestock, poultry and fish. Further, it could also have an impact on the suitability of drinking water. As with direct impacts on human health, this in turn would have an impact on productivity by reducing the total number (absenteeism) or effectiveness (presenteeism) of days worked.

#### What is the strength of evidence underpinning this pathway?

In the supporting documentation for the ALPHA model (EC4MACS, 2013) it is noted that reducing emissions could reduce the costs of providing clean drinking water. This implies that there is a preventative cost currently incurred to reduce the impact of air pollutants on drinking water and hence a cost saving associated with reducing pollutants. Further, it notes a study from Netherlands (van der Velde et al, 2004) which investigated the benefits of reduced acidification in terms of: lower costs of treatment of groundwater, longer lifetimes for wells/pipelines and lower maintenance costs for wells/pipelines.

In previous work for ExternE (2005), dose-response functions for lead were explored to link lead exposure of infants to changes in IQ level. In the development of its air pollution assessment, the EC4MACS project considered these benefits to be too small for inclusion relative to other effects.

In the latest US EPA (2011a) assessment of air pollution impacts, it notes hazardous air pollutants (e.g. mercury) could accumulate in the food chain with sub-lethal impacts. It noted that people can be exposed to toxic air pollutants from: eating contaminated food (e.g. fish from contaminated waters), consuming meat, milk or eggs from animals that have fed on contaminated plants, eating crops grown in soil where air pollutants have been deposited and from drinking water contaminated with toxic air pollutants (see US EPA, 2011b; and US EPA, 2012b). This study does not estimate potential impacts through this pathway.

Considering the wider literature, a report by the WHO notes that air pollution can affect health through contamination of food and water. The emission of heavy metals such as cadmium, lead and mercury, contributes to the deposition and build-up of these heavy metals in soils and subsequent exposure of humans via digestion (WHO, 2007). Cadmium exposure is associated with kidney and bone damage and is a potential human carcinogen, lead exposure can have detrimental impacts on the development of infants and children and mercury can also be toxic. The report goes onto note that food is the predominant source of lead uptake amongst the general population however lead levels have significantly decreased in Europe over recent years.

Alongside the impacts of crop consumption, the WHO notes that methylmercury can also enter the body readily through the dietary route, in particular through fish consumption (this route found to be significant in sub-populations in Scandinavia). Further, in a summary of evidence of air pollution impacts, the Climate and Health Alliance (2013) note studies have explored the potential exposure of humans to heavy metals through consumption of fish.

In a study looking in more detail at human exposure to mercury in the EU (EC, 2001), it is noted that mercury intake through water is normally very low and daily intake through diet is difficult to estimate accurately. Mercury is known to bioconcentrate in aquatic organisms which can then be passed onto other food stuffs where fish is used to feed cattle or poultry. This study concluded that in areas of Europe where fish consumption represents a considerable part of diet, the US EPA recommended limits of mercury levels could be considerably exceeded.

#### What is the likely significance of the pathway?

In terms of drinking water, Van der Velde (2004) explored the current cost associated with cleaning. This study estimated a total benefit between 1990 and 2040 for the Netherlands of reducing pollution of €45m through reduced cleaning costs (amongst other benefits).

In terms of wider health impacts, there is evidence that this is a concern internationally. However, underlying rates of exposure to mercury and lead that are noted in the studies found in this review seem to suggest potential impacts in the UK are small. Further, given the international nature of the UK food chain, it is uncertain to what extent changes in pollutant levels in the UK would have on the exposure to health impacts through this pathway.

As part of the evidence gathering under the project, we contacted the FSA to enquire as to the potential significance of this impact pathway for the UK. The FSA noted that

contamination of crops from pollution via soil was likely to be more significant than direct contamination by air. Further, common behaviours such as washing food before use are likely to further reduce the likely health impacts of direct contamination by air. In addition, even though contamination through soil could occur, there are policies in place which would reduce the potential impact: controls are currently placed on levels of certain contaminants in food which apply irrespective of source.

#### Does information exist with which this pathway can be quantified?

Although there is evidence that this pathway could exist, there is no evidence or information on which a quantitative estimate of the impact of air pollution on health could be based. No CRF has been specified to link changes in air pollutants to health impacts via indirect exposure through food or water consumption.

#### Conclusion: Can we consider this pathway further?

Although the link from heavy metals to human health is recognised, in particular by the US EPA, a methodology has yet to be developed to value impacts. To fully understand potential impacts in UK, an understanding would need to be developed of the UK food chain and to what extent changes in air pollution in the UK could lead to changes in concentrations of pollutants in the chain. Further work would be required to develop baseline estimates of background concentrations and specific health related CRFs. However, given current policy regarding the testing of food in the UK, this impact pathway could be relatively insignificant.

#### Impact pathways via natural resources

In this paper, we have defined natural resource inputs as any naturally occurring inputs which are used in the production process. This includes the use of raw materials and does not overlap with man-made capital considered above (with the possible exception of mined building materials discussed above). Where we consider natural resource inputs, it is important to be aware of potential overlaps with the assessment of air pollution impacts on ecosystem services. Work is ongoing to quantify impacts on ecosystems in Defra's IPA<sup>37</sup>. It is intended that any solution would apply an ecosystem services approach to valuing impacts.

#### Wider pathway 7: Impact on animal health

Air pollution could have a strong impact on animal health as it does on human health and so could subsequently impact on the output of the agricultural or fisheries industries.

Air pollution could impact animal health directly via inhalation, or indirectly through high levels of chemicals in contaminated feed or degradation to pastoral land. In the same way as for humans, ozone, sulphur dioxide and  $NO_2$  could affect the respiratory system in animals; and heavy metals and dioxins can affect respiratory, circulatory, gastrointestinal and central nervous systems of animals. There could also be an indirect effect on the productivity of livestock from changes in the surrounding ecosystem.

Alongside impacts on livestock, air pollution could also impact on the abundance fish in coastal or terrestrial water bodies through nitrogen run off into water sources (EC4MACS, 2013). This could have a subsequent impact on the fishing industry.

#### What is the strength of evidence underpinning this pathway?

The supporting documentation for the ALPHA model notes the potential impacts that air pollution could have on livestock (ibid). However, it notes that these impacts can only be assessed qualitatively as quantification is not yet practicable on European scale, but research is continuing. This guidance also implicates air pollution as a cause of loss of salmon and trout from large numbers of rivers in northern Europe. The main effects are toxic impacts on fish from metals released into water as a result of acid rain (e.g. aluminium).

<sup>37</sup> See http://uk-air.defra.gov.uk/research/ecosystem-research

The US EPA (2011a) highlights that emissions of hazardous air pollutants (e.g. mercury) could cause direct toxic effects in animals. This could have reproductive and developmental effects. Its latest assessment of air pollution impacts notes that the impacts of ozone on faunal species have been little studied, but limited research has shown variety of pulmonary impacts to specific mammalian and avian species.

This study also recognises that acidification can change water chemistry, which in turn can impact on sensitive species and change community composition. While many fish-species are acid-sensitive, the main lethal agent is the increase in dissolved aluminium which occurs with falling pH levels. The report sets out a case study through which the US EPA considers the value of fish decline by using WTP for improvements in recreational fishing. Further, the US EPA also notes that coastal waters are generally nitrogen limited, hence air pollution and associated increases in nitrogen can lead to significant eutrophication of habitats in these areas, greatly affecting the productivity of the marine environment.

Outside of air quality impact assessments, the effect of air pollutants on animals is also noted, in particular in case of significant incidents of emissions (Catcott, 1961).

#### What is the likely significance of the pathway?

The total potential significance of this pathway will depend on the size of the relevant sectors of the UK economy (i.e. agriculture and fisheries). The impact of air pollution on livestock could be relatively small in the UK, as livestock are predominantly located in rural areas away from large urban centres where the impacts of air pollution are worst (with the exception of ozone). In terms of fisheries, a study in Norway (Narvud 2002) estimated a yearly benefit from an increased number of lakes with undamaged fish associated with reductions in air pollutants to be in the range of  $\in$ 80m to  $\in$ 134m.

#### Does information exist with which this pathway can be quantified?

In the course of developing the original damage cost estimates, AEA (2006) noted that  $SO_2$ ,  $NO_x$  and secondary pollutants have impacts on ecosystems. Emissions of  $NO_x$  are known to be responsible for a range of impacts through their contribution to acidification and eutrophication, with the latter noted as widespread in Europe. AEA conclude that appropriate models and evidence are not currently available with which these impacts can be captured.

Further, the supporting documentation for the ALPHA model concludes that estimation is possible in principle but further work is needed first .Some studies have been carried out to investigate these impacts, but there are significant issues around quantification due to the need to account for effects over longer timescales and the variability of conditions pertaining to climate, soil, species, ecological structure, human pressures, etc.

The review carried out under this project has not found any further evidence to suggest any of the conclusions of more detailed reviews above have changed. No CRF is available linking changes in air pollution to a change in livestock numbers or fish stocks.

#### Conclusion: Can we consider this pathway further?

The link between air pollution and health of livestock and fish-stocks is well documented. However, there is a lack of evidence around the potential significance of this problem in UK. Further, there is little evidence on which a robust estimate of impacts can be made.

#### Wider pathway 8: impact on outputs of commercial crops

Air pollution has direct impacts on crop yields. This can apply to all commercial crops, including agricultural crops grown for food, bioenergy or commercial forests. Any impacts on crop yields can be considered a direct impact on economic production and hence could fall under the definition of 'productivity' adopted in the present analysis.

#### What is the strength of evidence underpinning this pathway?

In the existing Defra appraisal guidance, ozone is recognised as the most serious air pollutant problem for the agriculture and horticultural sectors. Changes in crop yield are

assessed in the guidance using international crop prices. This captures the impacts of a range of crops, but impacts on some commercial crops (e.g. trees) are currently excluded. Further, the guidance does not include impacts of ozone on visible damage to crops and non-ozone impacts on crops (e.g. through acid deposition, nutrient deposition, interactions with pests/pathogens, etc.)<sup>38</sup>.

Studies at the EU level also include a valuation of the impacts on crop yield. The Climate Cost and IIASA studies included an assessment of impacts from ozone exposure using the ALPHA model. However, this does not consider the productivity of grassland and consequent impacts on livestock nor impacts on forests. Regarding the latter, the documentation notes that some papers are available depicting acidification impacts on forests but the methods used are judged invalid given the lack of a single unifying damage mechanism. Further, there is still considerable uncertainty around link between air pollution and timber production and dose-response relationships are not well established. The ALPHA model only provides qualitative consideration of other factors, such as the impacts on the tolerance of crops to other stresses (e.g. drought or cold), performance of pests and pathogens and acidification of agricultural soils. It does note that where air pollution impacts on timber and tree health, this could also have a multiplier impact on the prevalence of pollution due to the role trees play in filtering the air.

The US EPA study estimates the impact of changes in tropospheric ozone on crop and tree growth. It notes that acidification can have adverse effects on forest populations and increases in reactive nitrogen can limit plant growth, but moderate increases in nitrogen can have a fertilising effect.

In the wider literature, air pollution (in particular SO<sub>2</sub>) has been linked to forest decline in north America and a number of northern European countries, e.g. English Pennines around industrial cities (EC4MACS, 2013). A study by Karlsson et al (2005) investigated the response of forest stand in Sweden to ozone concentrations. This study found ozone had the potential to reduce forest growth by 2.2% and economic returns by 2.6%.

#### What is the likely significance of the pathway?

The significance of the potential impacts will again be determined by the size of the relevant industries in the UK. Existing estimates of the size of impacts suggest they are not insignificant, but are small relative to other air quality impacts. In the Climate Cost project, the benefit of reducing damage to crops in pollution reduction scenario is €113m in 2020 (around 1.2% of total health, building and crop benefits). In IIASA's work, the benefits to materials and crops under emissions reduction scenario are around 1.3% of health, crop and building costs. The US EPA study estimates the total reduction of impact of ozone on crops and forest land to be \$10.7bn in 2020 under the CAAA (equivalent to around 0.6% of all mortality impacts of policy). Further, an AEA assessment (1999) of air pollution impacts estimated the impact of moving to protocol ceilings directive in EU on timber production was worth around 10% of value of impacts on crops.

#### Does information exist with which this pathway can be quantified?

Relevant information on the impacts on most crops is available and valuation of impacts is already included in existing guidance. One key sector not currently included is the impact on timber production. These impacts are likely to be included through other ongoing projects.

#### Conclusion: Can we consider this pathway further?

A methodology to value impacts on crop yields from wide range of crops is included in current IPA guidance. Hence no further quantification is proposed under this study to avoid the risk of double counting. For timber, Defra have already noted this could be an area for future quantification as a result of ongoing projects.

<sup>&</sup>lt;sup>38</sup> Some air pollutants other than ozone have been linked in the literature to crop damage (e.g. SO2, NO2, NH) but generally at higher levels than are currently experienced in the UK. The IPA guidance assumes direct impacts of these on agriculture are small.

# Table A2.1 - Summary of evidence regarding wider pathways

Pathway	Strength of evidence	Potential scale of impact	Ability to model	Carried forward for further analysis
Air pollution curtails operation of sensitive capital assets	Evidence that engines have filters attached to avoid negative impacts of particulates; no evidence of impact on other machinery No detailed study of which particulates are problematic (if specific) or how current levels of air pollution impact machinery	Applying air filters incurs a preventative cost for all engines Changes in air pollution unlikely to have significant impact – filters would still need to be applied	No evidence found linking changes in air pollution on productivity of machinery No evidence linking changes in specific air pollution to application of filters	No: Not possible given lack of evidence regarding impacts But impacts likely to be insignificant given need to apply filters anyway
Buildings and other fixed productive assets are corroded through acid rain	Strong evidence linking impacts on buildings No evidence of potential impacts on raw materials	Corrosive impact on buildings is found to be significant but much less important than health impacts, particularly in case of UK No evidence of scale of impacts on raw materials	CRFs estimated for buildings and impact already captured in IPA	No: Impacts on buildings already included No evidence to suggest air pollution is an issue for mining
Increasing returns to capital incentivises additional investment	Impact theoretical No evidence this is an impact of air pollution: Not captured in existing studies	No evidence on potential scale of impact Impact is likely to be marginal given impacts of air pollution: majority of valued health benefit likely to be captured by static valuation	Impacts, if any, are long-term; incentives to invest in capital influenced by many factors, making linkage between capital and labour difficult to define, in particular for air pollution No evidence linking changes in air pollution to capital formation	No: No evidence that there is a link: hugely difficult to define robust linkage, in particular with respect to air pollution impacts
Impact of absenteeism on longer term productivity growth	No evidence that this is a significant effect of air pollution: Impact is not captured or noted in existing air pollution studies – use static value Links are reasonably well accepted but any impacts are longer term	Impacts are likely to increase with length of absence; impacts of short absences likely to be small given lost learning will be deferred Further, where air pollution impacts older persons, learning benefits lost more likely to be small. Majority of health benefits captured in static	No evidence linking changes in air pollution to lost learning rates and longer term productivity improvements Other factors could have stronger influence than air pollution	No: No evidence of air pollution impacts and other factors imply any impacts are likely to be insignificant

Pathway	Strength of evidence	Potential scale of impact	Ability to model	Carried forward for further analysis
		valuation		
Air pollution and Visibility	Strong evidence linking air pollution and visibility	Visibility could have a range of impacts on stock and productivity of labour	No evidence around current link from air pollution to visibility in UK	No: Link is proven but impacts considered insignificant in UK
	In particular, the US EPA capture and value impacts on amenity (but not productivity)	Given recent improvements in visibility, previous reviews of potential impacts have suggested impacts are insignificant in UK	No CRF available linking air pollution to productivity impacts via visibility - could not apply US CRF	Also lack of applicable CRF
Indirect impacts on human health	Impacts through food and water consumption are documented Impacts have been noted in the ALPHA model and US EPA work	Where impacts occur, these could be significant, either through severe health impacts or water cleaning costs	No evidence linking changes in air pollution to impacts on food/water in UK context	No: Valuation could be undertaken once CRF available using methodology for wider health impacts
	No evidence this is a problem in the UK	Change in air pollution likely to have negligible impact on need to clean water and on UK food given international nature of food chain	No CRF from indirect impacts to lost productivity Further work would be required to better understand UK food chain (and water supply)	No evidence to suggest this is a significant impact in UK
Impact on animal	Impacts on livestock and fish are noted	Scale of impact depends on overall size	No evidence that this is an issue in	No: Not possible given lack of
health	in current air pollution studies, in particular through high local emissions on livestock	of UK livestock and inland (or close to shore) fisheries industries Impact on livestock likely to be relatively small given location away	the UK and no data on current levels of water pollution or lost livestock	evidence providing quantitative link to animal health, in particular in UK
	However, impacts are not quantified and no evidence that this is a significant problem in UK	from polluted urban centres	No CRF available linking change in air pollution to change in livestock or fish stock in UK	Once a link is established, valuation would be relatively straight forward through use of market values
Impact on outputs of commercial crops	Impact on crops well documented and already included in appraisal of air pollution policy	Impact on crops and timber is significant, but not on same level as health impacts	Further research is required to develop dose-response functions specifically applicable to UK	No: Impacts on crop yield already captured in existing Defra guidance
	Impact on timber also well documented and included in US appraisal of air pollution policy	Impact depends on scale of crop agriculture and timber industries in UK: crop agriculture likely to be greater than timber value	Functions are available for US but may not be appropriate to apply in UK given difference in land area covered and species type	Impacts on timber production could be included in future valuation using commercial price for timber but lack of UK-specific CRF

# Appendix 3 – An indirect estimation of WDL via other health outcomes

# Overview

In our proposed methodology for estimating the productivity impacts of air pollution, we have included an estimate of the number of working days lost (WDL) attributable to air pollution based on a concentration response function (CRF) developed under the US HIS programme by Ostro (1987). However, given this CRF was developed nearly 25 years ago, there is uncertainty as to whether this CRF is still appropriate and applicable to the UK today.

Alongside our estimate of WDL based on the specific CRF for WDL, we have also investigated 'bottom-up' approaches to estimating WDL using CRFs for other individual health endpoints and sickness absence data relevant to air pollution. The intention was to use this bottom-up approach to validate the estimate of WDL based on the published CRF specific to WDL. Our approach to and results of this exercise are presented in this appendix.

A fairly rapid estimate of potential air pollution impacts on sickness absence was made on the basis of deriving single CRFs for respiratory and cardiovascular absences respectively, based on published CRFs for a range of respiratory and cardiovascular health endpoints. These CRFs for respiratory and cardiovascular sickness absence (without a detailed breakdown of cause) were then combined with sickness absence data for respiratory and cardiovascular causes in order to estimate impacts.

An in-depth literature review was beyond the scope of this project, CRFs were largely derived from the recent WHO-led review for the European Commission (HRAPIE), the CAFÉ study and the US EPA CBA of the Clean Air Act. Some further information was sought by searching PubMed (free to access online database of the medical literature maintained by the US National Institutes of Health Library). Specific searches were made for PM/PM<sub>2.5</sub>/PM<sub>10</sub>/ozone/O<sub>3</sub>/NO<sub>2</sub>/nitrogen dioxide and respiratory symptoms/school absence/sickness absence/working days/primary care consultations/GP consultations/restricted activity days/RADs.

As in our assessment of mortality effects discussed in the main report, it is important to note that there is uncertainty in the application of CRFs based on whole population studies to the working population. The working population are likely to be on average fitter and therefore less susceptible to the adverse effects of air pollution than the long-term unemployed or those with disabilities/chronic illness that prevent them from working. The working population is also likely to be less susceptible to air pollution than older people who have retired from the workplace.

# **CRF for PM impacts on respiratory absences**

It is sometimes stated that the numbers of people that experience adverse effects as a result of exposure to air pollution increases as health endpoints of decreasing severity are examined (see Fig.1 from the HEAL (2012) report for example). This might be expected to reflect a steepening of the CRFs for respiratory health endpoints of decreasing severity. The proportional increase in the number of emergency hospital admissions might be expected to be less than the proportional increase in respiratory symptoms.

If such a relationship existed, the CRFs for all sickness absence would probably be closer to that for respiratory symptoms than emergency admissions. In practice, reported CRFs for respiratory admissions are highly variable and there is only weak evidence that CRFs are steeper for less severe endpoints. The larger anticipated impact on less severe endpoints is likely to be due to greater background incidence of these health conditions relative to more severe health outcomes.

Table A3.1 below shows the CRF per 10 µgm<sup>-3</sup> increment in PM for a range of health endpoints. The source studies have not been reviewed in detail given this bottom-up

approach is only used to sense-check our central estimate of WDL. For now, the values can be considered indicative, and sufficient for the present discussions.

Endpoint	PM Metric	Population group	% change per 10 µgm <sup>-3</sup>	Source
Emergency hospital admission	PM <sub>2.5</sub>	All age	1.9	HRAPIE
A&E visits	PM <sub>10</sub>		1.0	Central Scotland study
GP consultation	PM <sub>10</sub>	All age		Central Scotland study
Asthma			3.6	
Lower respiratory symptoms			0.4	
Upper respiratory symptoms		15-64	5.7	Hajat et al (1999)
		65	3.3	
Respiratory symptoms	PM <sub>10</sub>	Adult with asthma	0.1676 days per adult	Central Scotland study
Respiratory symptoms	PM <sub>10</sub>	Child with asthma	0.1335 days per child	Central Scotland study
RADs	PM <sub>2.5</sub>	All age	4.7	HRAPIE
Wheeze	PM <sub>2.5</sub>	Child with asthma	20	Gent et al (2009)
Respiratory symptoms	PM <sub>10</sub>	Child	5	Ward & Ayres (2004)
Respiratory symptoms	PM <sub>2.5</sub>	Child with asthma	2.8	HRAPIE (Weinmayr et al., 2010)
Hospitalisation	PM <sub>10</sub>	Child with asthma	1.7	Romeo et al (2006)
Wheezing			6.3	
Coughing			2.6	
Medication use			3.3	
Any respiratory symptom	PM <sub>10</sub>	Child with asthma	10	Mar et al (2004)
	PM <sub>2.5</sub>		23	
	PM <sub>10-2.5</sub>		10	
	PM <sub>1</sub>		24	
Any respiratory symptom	PM <sub>10</sub> PM <sub>2.5</sub>	Adult with asthma	No	Mar et al (2004)
	PIM <sub>10-2.5</sub>		relationship	
Respiratory symptoms	PM <sub>10</sub>	Adult with COPD	9.3	Peacok et al (2011)
Respiratory symptoms	PM <sub>10-2.5</sub>	Adult with COPD or asthma	0.6-0.7	Karaiatsani et al (2012)
Limitation in walking			7.6	
Wheezing	PM <sub>10-2.5</sub>	Child with asthma	7.5	Mann et al (2010)
Wheezing	PM <sub>2.5</sub>	Child with asthma	5.1	Escamilla-Nuñez et al (2008)
School absenteeism	PM <sub>10</sub>	Child	No association	Gilliland et al (2001)
School absenteeism	PM <sub>10</sub>	Child	No association	Chen et al (2000)
School absenteeism	PM <sub>10</sub>	Child	4	Ransom and Pope (1992)
School absenteeism	PM <sub>10</sub>	Child	1.4	Park et al (2002)

Endpoint	PM Metric	Population group	% change per 10 µgm <sup>-3</sup>	Source
Asthma symptoms	PM <sub>10</sub>	Adults 18-80 years	No association	Willers et al (2013)
Asthma medication			Na	
Blocked nose/hay fever			association	
Chest tightness/cough			Not significant	
RADs			1.25	
			1.25	

Whereas the CRF for emergency hospital admissions is well established, the CRFs for less severe endpoints are highly uncertain. There have been many fewer studies of the impacts of air pollution on acute respiratory symptoms than of the impacts on mortality, which probably partly reflects the relative difficulty of investigating respiratory symptoms impacts relative to death rates.

There is considerable variability in the CRFs reported by different individual studies for similar effects. These differences are likely to reflect differences in the susceptibility of the individuals included in different panels, differences in symptoms definition, differences in medication use and the importance of confounding factors such as infection and exposure to aeroallergens such as pollen and moulds.

Many of the available studies have been conducted in groups of potentially vulnerable individuals such as children and/or individuals with asthma or COPD. These individuals are likely to be much more sensitive to the impacts of air pollution than typical working adults. It is probable, however, that working adults with asthma or COPD would be more likely to be absent from work for respiratory illness than other working adults and may dominate the respiratory sickness absence statistics. It is therefore likely that CRFs for these vulnerable groups would be fairly representative in terms of potential impacts on respiratory sickness absence.

Despite the constraints associated with the available CRFs, they are sufficient to enable an estimate of a generalised respiratory CRF that could be used in conjunction with respiratory sickness absence rates to estimate air pollution impacts. A relatively low estimate of impact would be an increase of 2% in respiratory absence per 10  $\mu$ gm<sup>-3</sup> increase in PM<sub>2.5</sub> and a higher estimate of impact would be an increase of 7% in respiratory absence per 10  $\mu$ gm<sup>-3</sup> increase in PM<sub>10</sub>. The difference in metric arises because of the variable use of PM<sub>2.5</sub> and PM<sub>10</sub> as the PM metric in the source studies and the likelihood that in addition to PM<sub>2.5</sub>, the coarse fraction of PM<sub>10</sub> is harmful to respiratory health.

# CRF for PM impacts on cardiovascular absences

The development of a CRF to estimate air pollution impacts on acute cardiovascular sickness absence is likely to be based on the CRF for emergency hospital admission as there is a paucity of less severe endpoints to consider. HRAPIE indicate that a 10  $\mu$ gm<sup>-3</sup> increase in daily mean PM<sub>2.5</sub> is associated with a 0.91% increase in emergency hospital admissions for all cardiovascular disease (all age) including stroke.

It seems likely that a substantial proportion of cardiovascular sickness absence would be due to people being off work waiting for, undergoing and/or recovering from surgery. Even where absence is due to acute effects such as a stroke, it is likely that the typical length of absence would be much greater than for most respiratory illness. It is suggested that the CRF for chronic mortality from cardiovascular causes (11% per 10  $\mu$ gm<sup>-3</sup> increase in PM<sub>2.5</sub>) would give a better estimate of an appropriate CRF to apply to cardiovascular sickness absence data for this bottom-up sense check. As discussed above, this would be consistent with the approach taken in the Global Burden of Disease study for most risk factors and endpoints but

may give an over-estimate of CV morbidity impact associated with particulate air pollution (Lim et al, 2012).

### CRF for NO<sub>2</sub> impacts on sickness absence

There is relatively little CRF information for the short-term effects of  $NO_2$  on health other than those relating concentrations of  $NO_2$  to emergency hospital admission.

HRAPIE identified two CRF for emergency hospital admissions for respiratory illness but recommended that only the CRF based on 24-hour mean concentrations be used in the calculation of total effects. Two studies on wheezing in children with asthma provide some limited additional CRF information. There are, however, insufficient data to indicate whether the CRFs for respiratory health endpoints of lesser severity than hospital admission are likely to be similar to or steeper than those for hospital admission.

In the absence of alternative CRF information, it is suggested that the CRF for emergency hospital admission indicating a 1.8% increase per 10 µgm<sup>-3</sup> increase in 24-hour mean NO<sub>2</sub> concentrations is used as a proxy for the CRF for an increase in respiratory absence.

# Table A3.2 – Summary of published CRFs for respiratory morbidity associated with $NO_2$

NO <sub>2</sub> Metric	Health endpoint	Percentage increase per 10 $\mu gm^{-3}$ increase in NO <sub>2</sub>	Source study
NO <sub>2</sub> , daily maximum 1- hour mean	Hospital admissions, respiratory diseases, all ages	1.50	HRAPIE - APED meta-analysis of four studies published before 2006; coefficient from single- pollutant model
NO <sub>2</sub> , 24-hour mean	Hospital admissions, respiratory diseases, all ages	1.80	HRAPIE - APED meta-analysis of 15 studies published before 2006; coefficient from single-pollutant model
NO <sub>2</sub>	Wheezing in children with asthma	6.11	Mann et al (2010)
NO <sub>2</sub> , maximum 1 hour mean	Wheezing in children with asthma	1.42	Escamilla-Nuñez et al (2008)

The REVIHAAP project report (WHO, 2013a) recommends including the effects of short-term NO<sub>2</sub> exposure on hospital admissions for respiratory diseases in all ages in the "core" cost– benefit analysis, suggesting that a risk coefficient adjusted for at least PM mass should be used. HRAPIE, however, concluded that it was not possible to calculate a CRF that was adjusted for PM effects.

It seems likely that the combined impacts of PM and  $NO_2$  on hospital admission would be less than the sum of the effects predicted by the CRF information for the two pollutants but greater than that calculated for either pollutant alone. Similarly it seems likely that the effect of air pollution on respiratory sickness absence would be greater than that calculated for either pollutant alone but less than the sum of the effect that might be calculated for the two pollutants separately.

# **CRF for O<sub>3</sub> impacts on sickness absence**

HRAPIE identified CRFs for emergency hospital admissions for cardiovascular and respiratory illness and for minor restricted activity days. HRAPIE did not include minor restricted activity days in their recommended core set of CRFs for effects quantification and for the purpose of estimating total  $O_3$  effects, recommended that effects were calculated only for days when the maximum 8-hour mean exceeds 70 µgm<sup>-3</sup>.

Based on this limited information, a CRF of a 1% increase in respiratory and cardiovascular sickness absence per 10  $\mu$ gm<sup>-3</sup> increase in daily maximum 8-hour mean ozone concentrations for days when this exceeds 70  $\mu$ gm<sup>-3</sup> is recommended for this bottom-up calculation.

O₃ metric	Health endpoint	Percentage increase per 10 μgm <sup>-3</sup> increase in O3	Threshold for calculation of effects	Source of CRF
O <sub>3</sub> , daily maximum 8- hour mean	Hospital admissions, CVDs excluding stroke respiratory diseases	0.89 0.44	>35 ppb (>70 µgm <sup>-3</sup> )	HRAPIE - APHENA study based on data from eight European cities; coefficients adjusted for PM <sub>10</sub> in two- pollutant model
O <sub>3</sub> , daily maximum 8- hour mean	Hospital admissions, CVDs excluding stroke respiratory diseases	0.89 0.44	>10 ppb (>20 µgm <sup>-3</sup> )	HRAPIE - APHENA study based on data from eight European cities; coefficients adjusted for PM <sub>10</sub> in two- pollutant model 65+ years
O <sub>3</sub> , daily maximum 8- hour mean	Minor restricted activity days (mRADs), all ages	1.54	>35 ppb (>70 µgm⁻³)	HRAPIE - Ostro and Rothschild's (1989) six separate analyses of annual data 1976–1981 of the United States National Health Interview Survey
O <sub>3</sub> , daily maximum 8- hour mean	mRADs, all ages	1.54	>10 ppb (>20 µgm <sup>-3</sup> )	HRAPIE - Ostro and Rothschild (1989)
O <sub>3</sub> daily maximum 8- hour mean	School loss days in 4th grade due to respiratory illness	15.7		USEPA calculation of benefits of Clean Air Act - Gilliland et al 2001
O <sub>3</sub> 1-hour maximum	School loss days	0.13		USEPA calculation of benefits of Clean Air Act - Chen et al 2000
O <sub>3</sub> daily	School absence	2.6		Park et al (2002)

#### Table A3.3 – Summary of CRFs for respiratory morbidity associated with O<sub>3</sub>

# **Detailed bottom-up model of WDL**

The construction of a more detailed bottom-up model with which to estimate WDL by specific endpoints presented a number of significant challenges. This includes the fact that the impact of the health endpoints on WDL for which CRF information exists is not known. Each emergency hospital admission would presumably lead to a number of WDL, whereas only a proportion of days on which increased respiratory symptoms are experienced would result in sickness absence.

Further, this bottom-up approach to estimating WDL by endpoint may potentially underestimate the impacts on sickness absence due to cardiovascular disease that would potentially be attributable to air pollution because there is no CRF information for endpoints such as hospitalisation for cardiovascular surgery. In addition, such endpoints would be related to long-term rather than short-term exposure to air pollution. Respiratory symptoms and emergency hospital admissions are discussed in more detail below.

#### Respiratory symptoms

A major challenge in estimating WDL due to respiratory symptoms is the absence of wellestablished CRFs in adults. HRAPIE present a CRF for reduced activity days (RADs) based on the same set of studies as the WDL CRF where RADs were not exclusively due to respiratory illness. The source studies failed to find a relationship between air pollution and respiratory RADs. Other more recent European studies have largely focussed on children and there are a small number of studies that have investigated specific groups of adults considered vulnerable to air pollution (Peacock et al, 2011; Karakatsani et al, 2012). Willers et al (2013) provide CRFs for chest tightness/cough and RADs (the Willers et al CRF for RADs implies a smaller PM impact than that recommended by HRAPIE).

Another challenge involved in undertaking a bottom-up estimate of WDL is that information about the baseline incidence of respiratory symptoms is scarce. Although baseline incidences can be estimated from the source studies from which CRFs are drawn, differences between the study populations and the UK working population lead to an uncertainty about the relevance and reliability of these baseline estimates.

Asthma UK (2005) estimated that on average on any day 1.5% of adults in Wales experience asthmatic symptoms. Separately, the prevalence of asthma in children based on "severe asthma" in the International Study on Asthma and Allergies in Childhood (ISAAC) (Lai et al., 2009) was reported to be 4.9% in western Europe and the daily incidence of symptoms in this group was estimated as 17% (interpolation from several panel studies). This gives a slightly lower estimate of the daily incidence of asthmatic symptoms in children than the estimate derived for adults in Wales.

HSCIC (2010b) estimated that the prevalence of lifetime doctor-diagnosed asthma was 16% among men and 17% among women and decreased with age for both sexes. But the proportion reporting asthma within the last 12 months was constant across age groups (9% of men and 10% of women). In the group with current symptoms, 22% of men and 9% of women reported that their symptoms had interfered with their usual activities in the last week. The implied daily incidence is somewhere between 3 and 22% in men with current asthma and between 1.3 and 9% in women with asthma. These figures suggest that about 1.5-2% of UK adults might experience asthmatic symptoms on any one day.

Clearly, respiratory symptoms are not confined to those with asthma. Further, as those with asthma normally use a preventative inhaler in order to minimise symptoms, the incidence of respiratory symptoms in people with asthma may not be very different from the incidence in the wider population. This might imply a baseline incidence of respiratory symptoms on an average day across the general UK population of about 5%. The incidence of symptoms in the working population would be lower as it would be anticipated that people in work would be in better health than those not in the workplace and chronic respiratory illness is more prevalent in older age groups.

A further challenge in estimating WDL due to respiratory symptoms is that not all symptom days will lead to work absence and there is no basis for determining the proportion of symptom days that will equate to WDL.

Overall, the high level of uncertainty in estimating the incidence of respiratory symptom days in the working population and in the proportion of symptom days that equate to work absence means that this approach to WDL cannot be considered in detail and could only provide a highly uncertain estimate of impact.

#### Emergency hospital admissions

In principle WDL should capture WDL lost to hospital admissions for respiratory and cardiovascular illness, although this is not explicit in the source studies. The absence of an explicit reference to hospital admission is probably due to the comparative rarity of emergency respiratory or cardiovascular (CV) admissions in people who are working. The addition of emergency hospital admissions to WDL would be anticipated lead to an element of double counting for those who are in work. A high proportion of sickness absence for CV causes would be associated with planned and unplanned hospital admission.

In terms of developing a bottom-up approach to estimating WDL, the impact of emergency hospital admissions would be anticipated to encompass the impact of severe cardiovascular events such as myocardial infarction that may be "attributable" to air pollution. CRFs are available from the HRAPIE study for emergency admissions for respiratory and cardiovascular causes and baseline admission rates are available from HSCIC. However, the

length of stay associated with each admission and spent recovering at home are not known. This issue was previously considered by Hurley et al (2005) who suggested that:

"Air pollution affects the numbers both of daily deaths and daily hospital admissions. It seems likely therefore that it also affects the severity of outcome of some people who would in any case have been admitted to hospital but who survive.... That severity might be marked, for example, by length of stay in hospital. We know of no studies which have examined this issue and so severity of condition post-hospitalisation is not quantified (except insofar as death may result soon afterwards)".

This was also considered subsequently:

"Between endpoints for particles, assume that all respiratory hospital admissions (RHA), congestive heart failure admissions (CHF), and cerebrovascular admissions (CVA) also involve restricted activity days (RAD). In adjusting RADs to take account of hospital admissions, it is arguable whether or not to convert each admission into equivalent hospital days. On balance, we have decided to do so using approximate average length of stay:

Net RAD = RAD - (RHA \* 10) - (CHF \* 7) - (CVA \* 45)".

The text does not give any references for the estimates of days and members of this project team consider this was an expert's guess. Looking at recent UK information about hospitalisation for respiratory and cardiovascular causes provides another route for estimating the WDL associated with emergency hospital admission. However, the HSCIC summary statistics for hospital admissions are difficult to unpick.

The HSCIC indicate that in 2012-13 in England there were 221,197 admissions for respiratory illness, of which 136,949 were emergencies, the average length of stay (all admissions) was 6.7 days; and 164,069 of all admissions were in people 16-64 years. Further, for cardiothoracic surgery only 5937 of 76,438 admissions were due to an emergency and the mean length of stay was 9.8 days. These data also show that 142,002 of 601,678 admissions for cardiology were due to an emergency and the mean length of stay (all admissions) was 5.1 days: 239,090 of all admissions were in the 16-64 age group.

In the existing Defra appraisal guidance, the impact of air pollution on respiratory and cardiovascular hospital admissions is captured. This guidance assumes an average duration of stay in hospital of 8 days for respiratory and 9 days for cardiovascular admissions (Defra, 2013a). It is important to note that focussing on only the length of stay in hospital may underestimate the productivity impact given affected persons could be absent from work after leaving hospital during recovery time spent at home.

The results of a CBI study (CBI, 2013) suggest that about 60% of CV absence in surveyed companies is due to chronic conditions and ONS estimate that a total of 4.6 million working days were lost to CV illness in 2011. Most absence due to CV illness is likely to arise from serious illness requiring treatment in hospital at some point within the total period of absence either to assess medication needs and/or because surgery is required.

Given an estimated hospital admission rate (emergency and planned) of perhaps 200,000/year for CV illness in people in work across the UK (based on age-related hospital admission rates published by the HSCIC), the average length of hospital related absence might be about 18 days per admission. The emergency admission part of the CV impact on WDL due to air pollution is likely to be relatively small and the overall WDL "attributable" to air pollution effects on CV health may mostly arise from chronic effects leading to a requirement for planned treatment. There is no CRF for chronic effects of air pollution on the requirement for hospital treatment for CV illness. As discussed above, the chronic mortality CRF provides a potential starting point but may give an over-estimate of impact.

In the absence of other alternative evidence, we have assumed that hospital admissions for any condition after higher air pollution days last typically as long as admissions for the same causes at other times. The WDL while in hospital is likely to represent a small proportion of total WDL associated with each emergency admission. The US EPA attributed 5 years loss of earnings to each case of Myocardial Infarction (MI) (the 1990 reference cited by the US EPA is not in the reference list) which seems a high estimate in the light of current advice given to CV patients in the UK. The NHS advise: "Following an MI, most people can go back to work within 2-3 months. However, each person is different. For example, some people who have a small MI and feel well go back sooner. On the other hand, some people with ongoing symptoms or complications such as angina or heart failure may take longer to go back or may not be able to go back to work"<sup>39</sup>. The British Heart Foundation (2005), in an information leaflet aimed at patients, stated that two-thirds of those under 65 who have a heart attack are in work and about half return to work. In other leaflet they advise that the average time taken to return to work after heart surgery is 2-3 months.

Overall it seems likely that a high proportion of CV emergencies would result in a number of weeks of WDL: probably 2-3 months for a serious event versus up to a week for a minor scare. Other emergency admissions would, however, involve only a few days absence from work while an individual's medication regime was reviewed. In contrast, the number of days spent recovering at home following an emergency respiratory admission is probably much smaller: at most 2-3 weeks.

#### Conclusions and a bottom-up estimate of WDL

Overall, it has not been possible to develop a full bottom-up model of WDL given the data available regarding appropriate CRFs, baseline illness rates and impacts of each type of illness on WDL. As such, we have retained the use of the WDL specific CRF as part of our central impact estimation using the tool. However, using the information we have gathered, it has been possible to derive an illustrative estimate for the possible WDL due to some specific causes such as emergency hospital admissions. We have done this to provide a further sense check on the estimation of WDL. A preliminary estimate of potential impact of respiratory symptoms can be made as follows.

Based on a working population of 30 million, a baseline symptoms incidence of about 5% (based on the whole population) and  $PM_{10}$  levels of 15-20 µgm<sup>-3</sup>, the number of symptom days over a 221 working day year attributable to air pollution based on a CRF of 1.25% per 10 µgm<sup>-3</sup> increase in  $PM_{10}$  for chest tightness/cough as representative of cold type symptoms, might be of the order of 6 million days. This might be lower if the incidence of respiratory ill health in working people is assumed to be less than that of the whole population (given the steep rise in chronic respiratory illness in older age groups). The implied sickness absence would be much lower, say less than 1.5 million days.

If RADs are considered (not specifically respiratory), based on a  $PM_{2.5}$  levels of about 11  $\mu gm^{-3}$  and a CRF of 4.7% per 10  $\mu gm^{-3}$  increase in  $PM_{2.5}$ , about 19 million RADs might occur in working people on working days (based on a baseline of 31 million attributable RADs on average per annum across all persons). Say half of those days led to absence, then the WDL impact would be less than 10 million days but it is likely that for a substantial proportion of the workforce, the proportion of RADs that translate to WDL is less than half.

For hospital admissions, we take the information provided by the HSCIC regarding the number of hospital admissions for different causes each year. This data suggests that 74% and 40% of respiratory and cardio vascular hospital admissions each year are between the ages of 16-64. Considering only those admissions due to emergency, this produces around 102,000 and 56,000 admissions for respiratory and CV reasons respectively. Combining this baseline of admissions with levels of pollutants in 2012 and relevant CRFs, around 84,000 and 9,800 respiratory and CV admissions respectively each year can be attribute to air pollution. If we take a lower bound of likely WDL per admission (say 10 days for respiratory

<sup>&</sup>lt;sup>39</sup> See http://www.patient.co.uk/health/Myocardial-Infarction-After-the-MI.htm. (Webpage Accessed March 2014)

and 40 days for CV admissions), this produces a total illustrative estimate of WDL of around 1.2m.

If we combine the estimated number of WDL for respiratory symptom days and hospital admissions together, the bottom-up estimate of the number of WDL attributable to current levels of air pollution already adds to around 2.7m WDL. Given that these are conservative estimates of these two potential health outcomes; a wide-range of other health outcomes are not considered here that are related to air pollution (e.g. CV illness that does not entail hospital admission); and the impacts of pollutants other than PM are excluded, this suggests that our central estimate of the number of WDL based on the WDL specific CRF (although high) could be considered not an unreasonable estimate of the total WDL associated with the current levels of all air pollutants.

# Appendix 4 – Concentration response function options included in tool

As discussed in the main body of the report, there are a number of concentration response functions available for different health impacts from the literature. In a response to a request from the Project Steering Group, we added a functionality to the tool such that alongside the core set of CRF's that are recommended for inclusion by the project team, the user could also test the change in estimated productivity impacts when using CRFs: that are already included in existing IGCB appraisal guidance and that are recommended by HRAPIE. These three sets of CRFs are set out in the tables below.

Impact	Pollutant	Metric	CRF per 10 µg	per 10 µg/m3 change in pollutant (%) Low Central High			
impact	Tonatant		Low				
Chronic Mortality	PM <sub>2.5</sub>	Annual mean concentration	4	6.2	8.3		
Acute Mortality	NO <sub>2</sub>	Annual mean of the daily maximum 1-hour mean concentrations	0.16	0.27	0.38		
Acute Mortality	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70µg/m3 (i.e. 35ppb cut-off)	0.14	0.29	0.43		
Absenteeism (WDL)	PM <sub>2.5</sub>	Annual mean concentration	3.9	4.6	5.3		
Restricted activity days (RADs)	PM <sub>2.5</sub>	Annual mean concentration	4.2	4.7	5.3		
mRADs	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70µg/m3 (i.e. 35ppb cut-off)	0.6	1.53	2.49		
SDL	PM <sub>10</sub>	Annual mean concentration	2.5	4	6.4		
SDL	O <sub>3</sub>	Annual mean of the daily maximum 1-hour mean concentrations	0.06	0.13	0.249		
Chronic bronchitis	PM <sub>10</sub>	Annual mean concentration	4	11.7	18.9		

Table A4.1 – CRF's recommended	ded by the project team
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Impact	Pollutant	Metric	CRF per 10 µg	/m3 change in p	B change in pollutant (%)     Central     High     6		
inipact	Fondtant	Methe	Low Central High				
Chronic Mortality	PM <sub>2.5</sub>	Annual mean concentration	1	6	12		
Acute Mortality	NO <sub>2</sub>	Annual mean of the daily maximum 1-hour mean concentrations	N/A	N/A	N/A		
Acute Mortality	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70μg/m3 (i.e. 35ppb cut-off)	0.6	0.6	0.6		
Absenteeism (WDL)	PM <sub>2.5</sub>	Annual mean concentration	N/A	N/A	N/A		
Restricted activity days (RADs)	PM <sub>2.5</sub>	Annual mean concentration	N/A	N/A	N/A		
mRADs	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70µg/m3 (i.e. 35ppb cut-off)	N/A	N/A	N/A		
SDL	PM <sub>10</sub>	Annual mean concentration	N/A	N/A	N/A		
SDL	O <sub>3</sub>	Annual mean of the daily maximum 1-hour mean concentrations	N/A	N/A	N/A		
Chronic bronchitis	PM <sub>10</sub>	Annual mean concentration	N/A	N/A	N/A		

Table A4.2 – Cl	RF's already	included in	IGCB	guidance
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# Table A4.3 – CRF's recommended by HRAPIE

Impact	Pollutant	Metric	CRF per 10 μg	RF per 10 μg/m3 change in pollutant (%) Low Central High			
impact	i onatant		Low				
Chronic Mortality	PM <sub>2.5</sub>	Annual mean concentration	4	6.2	8.3		
Acute Mortality	NO <sub>2</sub>	Annual mean of the daily maximum 1-hour mean concentrations	0.16	0.27	0.38		
Acute Mortality	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70μg/m3 (i.e. 35ppb cut-off)	0.14	0.29	0.43		
Absenteeism (WDL)	PM <sub>2.5</sub>	Annual mean concentration	3.9	4.6	5.3		
Restricted activity days (RADs)	PM <sub>2.5</sub>	Annual mean concentration	4.2	4.7	5.3		
mRADs	O <sub>3</sub>	Annual mean of the daily maximum of the running 8-hour mean concentrations using a 70µg/m3 (i.e. 35ppb cut-off)	0.6	1.53	2.49		
SDL	PM <sub>10</sub>	Annual mean concentration	N/A	N/A	N/A		
SDL	O <sub>3</sub>	Annual mean of the daily maximum 1-hour mean concentrations	N/A	N/A	N/A		
Chronic bronchitis	PM <sub>10</sub>	Annual mean concentration	4	11.7	18.9		



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