COMEAP

COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS

# *The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom*

A report by the Committee on the Medical Effects of Air Pollutants

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Chairman: Professor J G Ayres

Chairman of the Subgroup on Quantification of Air Pollution Risks II: J F Hurley

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# Foreword

That air pollution impacts on health is in no doubt and indeed has been the driver for improvements in air quality worldwide. The size of this impact has, however, been more difficult to determine, the issue being complex and attended by degrees of uncertainty. Total mortality is the easiest endpoint to tackle, cause-specific mortality less so and morbidity (hospital admissions, symptoms and so on), frankly, difficult.

COMEAP led the field in attempting to quantify the size of the health impact of air pollution with its first quantification report in 1998, limited though it was to quantifying the effects of short-term exposures to particles, sulphur dioxide and ozone on mortality and hospital admissions. At that stage we did not have sufficient information on the impact of long-term exposures to be able to quantify for the UK what we suspected to be a likely greater effect with any degree of certainty.

Over the last three years COMEAP has again been grappling with this area, this time addressing the impact of longer term exposures. In 2009 COMEAP produced a report on the effects of long-term exposures on mortality which recommended that formal quantification of the effects of such long-term exposures should be undertaken. Over the last decade, other groups and researchers have attempted to do this both in terms of 'impact' and 'burden' reporting differing results. One of the reasons for these apparent differences is the way in which these effects are expressed and this has been a focus of this current insightful and challenging report, which addresses these issues in detail.

The report more than matches earlier COMEAP reports in terms of its ability and willingness to tackle difficult areas and to move thinking forward rather than simply reiterating what is already known. As a consequence, I believe that this report will not only generate interest and stimulate discussion in the short term but will be used in the longer term as a starting point for further research and as a guide to policy development.

Any report such as this represents many months of work; this is no exception. I am particularly grateful to Fintan Hurley for leading on this with such rigour and energy and to the Secretariat and Members of COMEAP, and the QUARK II subgroup of COMEAP, who have contributed their time so generously. This is the work of a truly independent, truly expert group and a remarkable example of all that is good in providing scientific advice to Government. I look forward to seeing how this is received and how the report itself impacts on policy development and consequent cleaner air in the UK.

Professor Jon Ayres

Chairman of the Committee on the Medical Effects of Air Pollutants

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# Contents


Foreword iii

Acknowledgements iv

Executive Summary 1

1 Introduction 9

1.1 Context, scope and purpose of this report 9

1.2 The detailed questions we propose to answer 10

1.3 Structure of the report 12

Part I Questions about Impact of Pollution Reduction 13

2 Basic Concepts: Impact Questions 15

What Happens to Deaths and Life Expectancy when Death Rates are Reduced

2.1 Introduction: how mortality impacts are described 15

2.2 Core concepts of mortality and survival analysis – a more technical description 18

2.3 Inputs to the calculations 22

2.4 Outputs of the calculations – life-table differences between baseline and impacted scenarios 25

2.5 Distribution of impacts over time 26

3 Specification of the Impact Calculation 30

3.1 Calculations and parameters 30

3.2 Pollution reductions 33

4 Results of the Impact Calculations 38

4.1 Main results 38

4.2 Sensitivity analyses – 'impact' or 'benefit' questions 41

5 Discussion of Impact Calculation 48

5.1 Validity of the metrics used 48

5.2 Uncertainties in the results 49

5.3 Impact or benefit of reductions in anthropogenic $PM_{2.5}$ – methodological issues 50

Part II Questions about Burden of Air Pollution 55

6 Basic Concepts: Burden Questions 57

6.1 Comment on the questions 57

6.2 Burden of mortality 60

7 The Burden Calculation 65

7.1 Methodology of burden calculations 65

7.2 Results 66

7.3 Sensitivity analyses – burden question 68

8 Discussion of the Burden Calculation 69

8.1 Introduction 69

8.2 Context for burden results 70

8.3 Uncertainties in the estimates 74

8.4 What questions about pollution at 2008 levels does the burden question answer? 75

Part III Integrated Aspects 79

9 Overall Discussion 81

9.1 Distinguishing the mortality burden of current anthropogenic $PM_{2.5}$ from the benefits of eliminating it 81

9.2 Expressing and communicating the results 83

9.3 Public health significance 86

10 Conclusions and Recommendations 90

10.1 Conclusions 90

10.2 Recommendations 91

11 References 94

Appendix

Membership of the Committee on the Medical Effects of Air Pollutants 97

Membership of the Committee on the Medical Effects of Air Pollutants Subgroup on Quantification of Air Pollution Risks II (QUARK II) 98

# Executive Summary

1 This report deals with answering what, at first glance, appear to be relatively simple questions regarding the effects of particulate air pollution on mortality in the UK. We have tried to explain not only the approaches we have used to answer the questions, but also the limitations of the interpretations that can be put on the results. We anticipate that it will be useful to policy makers and elected representatives, and hope also that it will make a helpful contribution to public awareness and understanding of the health effects of air pollution. In summary:

a Airborne particles comprise an anthropogenic component and a natural component.

b There is an interest in the effects of air pollution on mortality in terms of the impact that policies for reduction would have, or the current burden in terms of public health.

c These effects can be expressed at the population level in terms of life expectancy, and on loss or gain in life years. The burden can also be expressed in terms of deaths occurring in a specified year across the population.

d As everyone dies eventually no lives are ever saved by reducing environmental exposures – deaths are delayed resulting in increased life expectancy.

e These measures are averages or aggregates across the population; it is not known how the effects are distributed among individuals.

2 We conclude that:

a Removing all anthropogenic ('human-made') particulate matter air pollution (measured as $PM_{2.5}$ [1]) could save the UK population approximately 36.5 million life years over the next 100 years and would be associated with an increase in UK life expectancy from birth, i.e. on average across new births, of six months. This shows the public health importance of taking measures to reduce air pollution.

b A policy which aimed to reduce the annual average concentration of $PM_{2.5}$ by 1 $\mu g/m^3$ would result in a saving of approximately 4 million life years or an increase in life expectancy of 20 days in people born in 2008.

c The current (2008) burden of anthropogenic particulate matter air pollution is, with some simplifying assumptions, an effect on mortality in 2008 equivalent

[1] $PM_{2.5}$ is defined as the mass per cubic metre of airborne particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 μm. In practice, $PM_{2.5}$ represents the mass concentration of all particles of less than 2.5 μm aerodynamic diameter.

to nearly 29,000 deaths in the UK at typical ages and an associated loss of total population life of 340,000 life-years. The burden can also be represented as a loss of life expectancy from birth of approximately six months.

d The uncertainties in these estimates need to be recognised: they could vary from about a sixth to double the figures shown.

## Background and purpose

**3** In 2009, the Committee on the Medical Effects of Air Pollutants (COMEAP) published a report on the effect on mortality of long-term exposure to air pollution. The report focused on particulate matter, represented as $PM_{2.5}$, as the air pollutant most strongly associated with increased risks of mortality and recommended coefficients expressing the relative risks of mortality associated with a 10 $\mu g/m^3$ increase in $PM_{2.5}$. It did not include calculations of the overall size of the potential effects of particulate air pollution on mortality in the UK. The Committee has now carried out these calculations and, in this report, presents not only the calculations but also an overview of the methods used, assumptions made and the relationships between different ways of expressing the results.

**4** We have sought to address two types of questions about the effect on mortality of air pollution in the UK. One type of answer provides information about the usefulness of policy measures to reduce air pollution (the 'impact' of policies) while the other represents the scale of the problem (the 'burden' imposed on public health).

**5** COMEAP has addressed three questions in this report:

Questions about impact of policies:

**Q(a)** What are the benefits expressed as an effect on mortality of a sustained reduction in annual average air pollution across the UK by a small fixed amount, e.g. by 1 $\mu g/m^3$ $PM_{2.5}$?

**Q(b)** If anthropogenic air pollution in 2008 were to be removed and pollution sustained at low non-anthropogenic levels, what would be the benefits in terms of effects on mortality?

Questions about burden on public health:

**Q(c)** What is the effect of air pollution on mortality in the UK today? In particular, what is the effect of air pollution at current (2008) levels on mortality in the UK in 2008?

**6** While it may seem that the second and third questions are equivalent, they require different assumptions to be made in addressing them. One purpose of our report is to explain why this is so.

## Air pollution, death rates, population survival and numbers of deaths

**7** A reduction in air pollution impacts on future patterns of survival and death in the population by decreasing the mortality risk and associated age-specific death rates, leading to fewer deaths initially and a sustained increase in life expectancy. However,

because everyone dies eventually, the total number of deaths in a given population cannot be changed by reducing levels of air pollution. Instead, a reduction in air pollution postpones deaths, so that on average people live longer. This leads to more people surviving year-on-year, and so to an increase in total population survival time, in terms of total years of life lived. The population becomes larger and older than it would have been if death rates had not been reduced, and this in turn changes the numbers and age distribution of annual deaths dynamically. As a result the mortality benefit of sustained pollution reduction needs to be looked on as two-fold:

a an immediate benefit in terms of fewer deaths in the first and early years after the change,

b a longer-term benefit of increased life expectancy, with associated greater survival time (life years lived) across the population as a whole.

**8** Consequently, the long-term mortality benefits of pollution reduction are best reflected either in terms of life expectancy or in terms of gains in population survival time ('life-years'), rather than in terms of annual deaths. It is on this basis that we address questions (a) and (b) above, using life-table methods which take account of the dynamics between death rates, population size and age structure. There is likely to be some delay in the reduction of mortality risk following a pollution reduction, the so-called cessation lag, and this has been incorporated into calculations of the impact of reductions in air pollution.

**9** In contrast, the calculation for question (c) is more straightforward, though some simplifying assumptions, related to lag, are required in specifying the pollution question that is being answered. Additionally, the result expressed, in terms of number of attributable or additional deaths, may easily be misunderstood or misrepresented. This calculation is *not* an estimate of the number of people whose untimely death is caused entirely by air pollution but a way of representing the effect across the whole population of air pollution when considered as a contributory factor to many more individual deaths.

**10** While many people are used to considering burden only in terms of the number of excess deaths in the population, COMEAP considers these deaths as indicators of total population survival, and we have calculated this explicitly by considering what loss of life is associated with each of these additional or attributable deaths. This has allowed us to describe the mortality burden as equivalent to a number of attributable deaths along with their associated loss of life. It has also allowed us to speculate about how the overall loss of population survival time might in reality be distributed across the whole population.

**11** All of these measures – impact on mortality equivalent to a number of deaths with given age distribution, population survival time or life-years and life expectancy at birth (which can also be calculated) – are characteristics of the population as a whole and cannot be applied at an individual level. This is because air pollution acts in combination with many other causes to affect mortality, so that we do not know how the changes in survival are distributed across individuals. Consequently, we consider it unrealistic to view air pollution as the sole cause of death in a number of cases equal to the population attributable deaths.

## Mortality benefits of reducing 'human-made' pollution

**12** We calculated the mortality effects, including effects on those born after 2008, of sustained reductions in air pollution based on 2008 levels of $PM_{2.5}$, over 106 years from 2008 as well as the difference in the life expectancy of the 2008 birth cohort. The risk coefficient used was for all-cause mortality of 1.06 (6%) per 10 $\mu g/m^3$ change in annual average airborne $PM_{2.5}$ concentration, which was derived from the American Cancer Study (ACS) cohort as recommended by COMEAP in 2009. We used the cessation lag distribution used by the US Environmental Protection Agency and incorporated sensitivity analyses for relevant factors.

**13** COMEAP estimates that, in the unrealistic scenario where all anthropogenic particulate air pollution were removed (a reduction of 8.97 $\mu g/m^3$ $PM_{2.5}$ in the UK):

a over the next 106 years, more than 36.5 million life years would be saved in the UK population, including new births;

b the increase in life expectancy for a birth cohort would be in the region of six months.

**14** Using the expert elicitation (COMEAP, 2009) which suggested a 75% chance that the risk coefficient lies between 1% and 12% implied that the true value lies between 5.8 and 66.2 million life years in England and Wales, or effects on life expectancy of between one month and one year.

**15** For a 1 $\mu g/m^3$ reduction, about 4 million life years could be saved in the UK over the next 106 years (range: 670,000 to 8 million life years), associated with an increase in life expectancy of around 20 days (range: 3 to 40 days). The results are relatively insensitive to changes in assumptions about how quickly mortality risks reduce following reductions in pollution (cessation lag). However, our sensitivity analysis showed that benefits are strongly sensitive to assumptions about discounting of future values.

**16** All these results scale in proportion to population-weighted mean concentrations of anthropogenic $PM_{2.5}$, and (approximately) to the risk coefficient. In addition, results that depend on population size scale in relation to the size of the population 30 years and above, other things being equal.

## Burden of current pollution levels on mortality today

**17** Using the same risk coefficient for all-cause mortality, as recommended in the 2009 report, the Committee has now calculated the mortality difference in terms of deaths, reduction in total population survival and reduction of life expectancy of the 2008 birth cohort between two scenarios which have the same population in 2008 but death rates that differ in 2008:

a In one (*baseline*) scenario, these are the observed age-specific death rates in 2008 (which were influenced by pollution).

b In the other (*alternative*) scenario, these age-specific deaths rates are reduced by an amount attributable to 2008 levels of anthropogenic particulate air pollution (8.97 $\mu g/m^3$ $PM_{2.5}$ in the UK).

These assumptions about population and death rates in one specific year are the same as what is usually assumed in discussions of the burden of current air pollution. The results have an easy interpretation in terms of air pollution only if the full effects of air pollution on mortality were immediate (i.e. without any time lags), which is known to not be the case. The results can also be interpreted as the effect of past and current air pollution on mortality in 2008 (i.e. taking account of latency and cessation), if it is assumed that pollution in previous years was similar to those levels measured in 2008, and we ignore any effects of past pollution on the size and age structure of the population in 2008.

**18** As a central estimate we conclude that anthropogenic $PM_{2.5}$ at 2008 levels (8.97 $\mu g/m^3$ in the UK) is associated with an effect on mortality equivalent to nearly 29,000 deaths at typical ages of death in 2008 in the UK and an associated loss of total population survival of 340,000 years and an average loss of between three and four months of life expectancy in Scotland and Northern Ireland and between six and seven months in England and Wales, reflecting differences in the levels of anthropogenic $PM_{2.5}$ to which these populations are exposed.

**19** These results are subject to many of the same uncertainties as stated in paragraph 14. Using the 75% plausibility interval suggested by the expert elicitation in COMEAP (2009) this means a range of effects on mortality equivalent to 4,700–51,000 deaths with a loss of 55,000–597,000 years of life in 2008, or effects on average life expectancy of between 1 month and one year, for England and Wales.

**20** Overall, we believe that the most important description of the burden of air pollution is in terms of years of total survival time lost to the current population. Simply stating a number of deaths does not allow for the ages at which these deaths occur, or for the fact that the loss of life associated with them varies with age. This is the basis of the calculation of total survival time, and some very valuable information about the burden is lost if this is not calculated.

**21** It is not known how this population-wide burden is spread across individuals in the population, but we can speculate between various possibilities. Our results are consistent with an average loss of life ranging at one extreme from 11½ years if air pollution was solely responsible for 29,000 deaths to, at the other extreme, six months if the timing of all deaths was influenced by air pollution. We believe both of these extremes to be extremely unlikely. Given that much of the impact of air pollution on mortality is linked with cardiovascular deaths, it is more reasonable to consider that air pollution may have made some contribution to the earlier deaths of up to 200,000 people in 2008, with an average loss of life of about two years per death affected, though that actual amount would vary between individuals. However, this assumption remains speculative.

## Expressing and communicating the results

**22** After careful consideration of the different kinds of questions that are asked about the effects of air pollution on mortality (what we have called impact questions and burden questions), and the different ways in which population survival and mortality can be and are discussed, we have reached the following conclusions and recommendations.

### *General points*

a In expressing these results there is a trade-off between full accuracy and accessibility.

b The Committee stresses the need for careful interpretation of these metrics to avoid incorrect inferences being drawn – they are valid representations of population aggregate or average effects, but they can be misleading when interpreted as reflecting the experience of individuals.

c There is also the need to communicate uncertainties openly and fairly.

*Life expectancy at birth* is a valid and meaningful expression of mortality effects for both the impact of reduced pollution and the burden of current pollution. It has particular advantages for communication and for comparison with other risks. However, it is incomplete as an expression of the mortality effect in the current population as it does not cover effects on other ages.

*Total population survival time (life-years gained or lost)* is also a valid and meaningful way of expressing mortality effects of both the impact and burden questions, and is the most comprehensive way of capturing the full effects. There are difficulties in communication. The concept of a 'life-year' is not a difficult one to grasp, but it is difficult to interpret the very large numbers of life-years involved in total population survival. However, it is the most relevant index for policy analysis.

*Number of attributable deaths* is a valid and meaningful way of capturing some important aspects of the mortality burden, across the whole population in any one particular year, of current levels of pollution, if we set aside some of the complexities of how quickly air pollution affects mortality risks. To emphasize that the number of deaths derived are not a number of deaths for which the sole cause is air pollution, we prefer an expression of the results as "an effect equivalent to a specific number of deaths at typical ages". It is incomplete without reference also to associated loss of life. The Committee considered it inadvisable to use annual numbers of deaths for assessing the impacts of pollution reduction, because these vary year by year in response to population dynamics resulting from reduced death rates.

## Public health significance

**23** Having an established methodology for assessing the benefits of proposed policy interventions is important for public health. It allows policies to reduce air pollution to be optimised so that the best possible improvement in the impact of air pollution on public health is achieved, given the available funds. It can also be used for other purposes, such as illustrating what role reductions in pollution could play in reducing health inequalities, in combination with other measures.

**24** We note that this methodology is also applicable to assessment of other public health burdens, where the implications of changes in population dynamics are not always taken into account. Wider use of this approach would allow more comparisons of our results with other public health burdens using a life-years or life-expectancy metric, with less scope for misinterpretation than numbers of deaths.

**25** The Committee's findings confirm that outdoor air pollution at current levels makes a significant contribution to mortality in the UK today, in terms of total population survival time, which has been estimated separately as a greater burden than the mortality impacts of environmental tobacco smoke or road traffic accidents. Correspondingly, reductions in population exposure to air pollution expressed as annual average $PM_{2.5}$ can have appreciable benefits in terms of reduced death rates and the associated increase in life expectancy and in terms of the total years lived by the population as a whole.

# Chapter 1
# Introduction

## 1.1 Context, scope and purpose of this report

The following general questions are often asked in discussions about the public health impacts of outdoor air pollution in the UK:

> 'What is the effect of air pollution on mortality in the UK today?'
>
> 'What are the effects on mortality of reducing air pollution?'

Both questions are important, for policy development and public debate. The first question reflects an attempt to understand the scale of the problem, as a motivation for action; the second calls for the provision of important information to assess the usefulness of actions or policy measures, relative to their costs.

The present report is the second of two linked reports that together attempt to answer these questions comprehensively, and to explain what is involved in answering them. In the first of these (COMEAP, 2009), the UK Committee on the Medical Effects of Air Pollutants (COMEAP) reviewed the international evidence on the extent to which long-term exposure to air pollution, expressed as annual average fine particles ($PM_{2.5}$[2]), adversely affects the age-specific risks of dying. That report focused on evidence for quantifying the relationship, rather than, for example, on explaining the mechanisms by which long-term exposure to polluted air can lead to earlier deaths from cardiorespiratory causes, and from lung cancer. To that end, the COMEAP (2009) report recommended concentration–response functions (CRFs), also known as risk coefficients (these terms will be used interchangeably), for quantifying the $PM_{2.5}$-mortality relationship, together with uncertainty bounds around the recommended principal coefficient. In this report we have focused on $PM_{2.5}$ and when we speak of the effects of air pollution on health we mean (unless qualifications are provided) fine particulate air pollution measured as $PM_{2.5}$.

The choice of CRF is both important and often contested, and it is one very important element in answering the questions posed above. However, it is by no means the only important issue, and it does not, of itself, answer those questions.

The present report is concerned with the other issues, information and assumptions that need to be made, to provide answers to the questions posed and will discuss:

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[2] $PM_{2.5}$ is defined as the mass per cubic metre of airborne particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 μm. In general terms it is the mass per cubic metre of particles of less than 2.5 μm aerodynamic diameter.

a what these issues are;

b how important they are; and, in particular,

c the set of apparently simple but sometimes confusing issues concerning estimating and expressing the mortality impacts of particulate air pollution using CRFs such as those given in COMEAP (2009).

Many of these issues are associated with different ways of measuring and expressing mortality impacts, of which there are two main variants: those that focus principally on survival, life expectancy, and total gain or loss of years lived by a defined population; and those that focus principally on numbers of deaths, which, it is often said, are easier to understand than life expectancy or population survival. All these indices are different ways of expressing the implications of differences in death rates, in the present context differences that arise because of differences in exposure to air pollution represented by annual average $PM_{2.5}$; it will be seen that the various indices are differently useful according to the specific question being asked and answered.

Our specific objectives are to:

a calculate, using the risk coefficients and other conclusions of COMEAP (2009), the implications for mortality of the UK population of:

- reductions in fine particulate air pollution attributable to human activity, i.e. anthropogenic particulate matter ($PM_{2.5}$), – both small policy-relevant reductions of 1 $\mu g/m^3$ annual average $PM_{2.5}$, and the unrealistic but interesting possibility of eliminating anthropogenic $PM_{2.5}$,
- current levels of anthropogenic $PM_{2.5}$;

b describe and explain the methods used to make these calculations;

c make transparent and understandable the assumptions that underlie the calculations, and to discuss their importance;

d clarify the relationships between different ways of expressing mortality impacts and to comment on the appropriateness of their use.

It is not our intention to re-visit the recommendations about risk coefficients of COMEAP (2009), and other strategic conclusions reported there, e.g. about the (non-)existence of a threshold, or the role of gaseous pollutants, or the relative toxicity of $PM_{2.5}$ from different sources. Evidence that has appeared since 2007, when the consultation draft of the COMEAP (2009) report was published, broadly supports the conclusions of that report.

## 1.2 The detailed questions we propose to answer

COMEAP (2009) recommended that, in estimating mortality impacts, we should focus on the effects of changes in air pollution that fall within current levels of anthropogenic (i.e. human-made) particulate air pollution. In that spirit, one useful question to answer, which we address, is as follows:

**Q(a)** What are the benefits expressed as an effect on mortality of a sustained reduction in annual average air pollution across the UK by a small fixed amount, e.g. by 1 μg/m$^3$ $PM_{2.5}$?

In line with the general questions posed in Section 1.1, and the specific objectives of this report described there, we consider also two other questions:

**Q(b)** If anthropogenic air pollution in 2008 were to be removed and pollution sustained at low non-anthropogenic levels, what would be the benefits in terms of mortality?

**Q(c)** What is the effect of air pollution on mortality in the UK today? In particular, what is the effect of air pollution at current (2008) levels on mortality in the UK in 2008?

While we recognise that interest is in current air pollution, we have specified the questions as relating to 2008 because this is the latest year for which we have population-weighted mean pollution concentration data and relevant population data. Thus when discussing 'current air pollution' or the 'current population' in this report, we consider current to mean 2008.

### 1.2.1 The 'impact' or 'benefits' questions

The first and second questions, (a) and (b) above, seek to assess the benefits to health, as expressed via improved survival and reduced death rates, that would result from policy measures targeted at reducing levels of air pollution in the UK. In this context, the terms 'benefit' and 'impact' are used to reflect the beneficial effects of policy measures. We recognise that the term 'benefit' clearly implies a value judgement, whereas 'impact' is a more neutral word. We use 'benefit' because a longer life span, especially if that implies a longer life in good health, is generally considered to be desirable.

Question (a) relates to a small change in anthropogenic particulate air pollution, and as such is directly relevant to policy development and evaluation. Question (b) considers the benefits of eliminating all anthropogenic particulate air pollution, and as such is unrealistic in current policy timeframes. We include it because it puts a long-term upper limit on the public health benefits of reducing air pollution, and because it provides results which can be compared with those from question (c), the burden question.

### 1.2.2 The 'burden' question

In Question (c), the general question ('What is the effect of air pollution on mortality in the UK today?') is made specific to mortality in 2008, and specifically refers to the effect on mortality in 2008 of anthropogenic particulate air pollution at *current* (i.e. 2008) levels. The wording 'at current levels' has been carefully chosen so as to specify the amount of air pollution, but not when it occurred – this could be in 2008, or in earlier years.

Complexities underlying the wording of the question, and how these relate to the results we provide, are discussed in detail in Sections 6.1 and 8.4.

The 'burden' question makes no suggestion of any change in levels of air pollution in the future.

## 1.3 Structure of the report

We will address these questions as we go through the different sections of the report. Part I (Chapters 2–5) focuses on questions (a) and (b). It explains the basic concepts involved in answering these impact questions and outlines the method used for the calculations. Then the results and some discussion of these are presented. Later chapters of the report, Chapters 6–8 (Part II), concern question (c) looking at the current burden of particulate air pollution. A similar layout is used for these chapters, i.e. concepts, methods, results and some discussion. Following this, Part III contains an overarching discussion, in Chapter 9, of these different questions, the validity and usefulness of the various mortality indices in answering them, and how the results might best be presented and communicated to their audiences whether they are policy makers or the general public. Chapter 9 also addresses the public health implications. Finally, we present our conclusions and some recommendations for future work.

# Part I

# Questions about Impact of Pollution Reduction

# Chapter 2
# Basic Concepts: Impact Questions

## What Happens to Deaths and Life Expectancy when Death Rates are Reduced

## 2.1 Introduction: how mortality impacts are described

This chapter describes how life-table methods can be used to make robust and consistent predictions of how changes (reductions) in mortality risks and associated age-specific death rates, such as those that might follow introduction of policies and measures that influence (reduce) $PM_{2.5}$ air pollution, impact future patterns of survival and death in a population of interest. In this report, the change in mortality risk is determined from the concentration–response function and other recommendations in the COMEAP (2009) report. As reducing air pollution affects the temporal pattern of deaths, this leads to an increase in years of life lived by the population concerned, i.e. an increase in population total survival time. The long-established statistical method of life-table analysis can be used to predict survival patterns following changes in age-specific death rates, from which effects on deaths, life expectancy and total survival time (life-years) can be calculated.

We begin with an introduction to the concepts, indicate their complexity, and then proceed to a more technical description of the issues which, we hope, will both help explain and help the reader to find a way through that complexity.

### 2.1.1 Survival

One approach to quantifying and expressing the mortality effects of lowering air pollution is based on the obvious fact that, in general and on average, people will live longer if age-specific death rates are lower. The size of the gain in survival is on average greater for those who are young, as they will experience the benefits of cleaner air, and the associated lower age-specific death rates, over most of their lifetime. So, if annual average $PM_{2.5}$ levels are reduced, people of all ages will gain in life expectancy, but older people will gain less than younger ones.

As explained later, these age-related variations in gains of survival are best shown via a survival curve that illustrates effects across all ages. It is impossible to express the gains in life expectancy simply, accurately and comprehensively in a single index. Consequently, there are numerous expressions such as 'gain in life expectancy from birth', or 'total years of life gained across or by the population', or 'average years of life gained across the population' or 'changes in the percentage of the population reaching age 60, 65 or 70, etc.', all of which attempt to capture and summarise relevant aspects of the underlying survival curve (or the differences between survival curves, when the impacts of reduced fine particulate air pollution are being assessed).

With these many indices, the issue is not one of validity – all are valid as arithmetically correct and legitimate expressions of the consequences of lower age-specific death rates and associated changes in survival curves. The issue is, rather, of the relevance and appropriateness of different ways of expressing longer survival that may be best for different purposes, e.g. expressions that are best for use in policy development may not be best for communicating benefits to the general public, and vice versa.

One purpose of the present report is to explain the various ways of expressing that the population on average lives longer, so that these various summary measures can be understood not only individually, but also in relation to one another.

### 2.1.2 Numbers of deaths

A second way of expressing the benefits of lower age-specific death rates is in terms of numbers of deaths per year or, more exactly, (though this is rarely made explicit), in terms of the differences between two scenarios – one with higher particulate air pollution and higher age-specific death rates; the other with lower air pollution and lower age-specific death rates.

All discussion needs to take account of the fact that death at some point is inevitable and, thus, that commonly used expressions such as 'lives saved by reducing air pollution' are strictly wrong. The 'saving' is temporary – length of life is increased: death is postponed but in the end not avoided. So ultimately the total number of deaths in a given population will not be changed by reducing air pollution. But it is possible to say 'in a specified year *X* deaths could be saved (i.e. temporarily avoided, postponed) following a specified reduction in air pollution' and indeed there are arithmetic calculations that are simple and straightforward and do just that.

There are, however, two kinds of complications that need to be taken into account. Firstly, it is likely that exposure to $PM_{2.5}$ acts in conjunction with other risk factors to cause earlier death; certainly we cannot, in principle or in practice, identify a group of people whose death was caused solely by exposure to particulate air pollution. This leads to difficulties in interpreting numbers of deaths postponed following reduced air pollution.

The second complexity relates to the fact that the same number of deaths will not continue to be postponed or temporarily avoided in each subsequent year that an air pollution reduction policy is in place. This will be illustrated explicitly later (Section 2.5). The reasons, however, are relatively straightforward: reductions in pollution lead to reduced age-specific risks of mortality, and, assuming other determinants of mortality remain unchanged, lead over time to more people surviving year-on-year, and so to larger populations that live longer than would be the case if death rates had not been reduced. The reduction in death rates and associated delay in death affect the overall demographics of the pattern of death in the population in the following ways:

a **The size of the population is increased (assuming other factors remain unchanged).** This occurs because there are fewer deaths in the first and immediately subsequent years as a result of a reduction in air pollution. This initial reduction in deaths leads over time to more people being alive (i.e. to a larger population) than would have been the case if air pollution and associated death rates had not been reduced.

b **The age structure of the population is on average older**. This is a consequence of lower age-specific death rates and thus a delay in deaths and longer life expectancy.

Put together, action to remove or reduce current levels of anthropogenic $PM_{2.5}$ in the UK would result in a larger and older population, other things being equal. These are the population dynamic implications of reduced death rates. It is an aspect which, increasingly, is understood and taken into account, but nevertheless is often ignored in general discussion. It has important implications, because the size and age structure of the population affects the number of deaths per year, and it is this dynamic population aspect which affects some of the mortality estimates.

In fact, if we only look at the population alive at the time of implementation of a policy, while initially the number of deaths occurring each year will be reduced, eventually more deaths will occur annually under lower pollution levels. This is because everybody will die at some point; and the lower initial numbers of deaths in the reduced pollution scenario must eventually be compensated by higher numbers of deaths in later years – people will be living longer and will therefore die later than in the baseline scenario. This is illustrated in Section 2.5.

*Summary*

Sustained pollution reduction leads (other things being equal) to sustained reductions in age-specific death rates, resulting over longer time periods to a larger and older population, which in turn change the numbers and age distribution of annual deaths dynamically (i.e. the number of deaths per year is not constant).

### 2.1.3 Implications of the above and our ambitions in this report

The mortality benefit of sustained pollution reduction, and associated sustained lower death rates, can be looked on as two-fold:

a an immediate benefit in terms of '*X*' fewer deaths in the first year (and different numbers in subsequent years);

b a longer-term benefit of prolonging life or increasing life expectancy by delaying death.

The latter is important when assessing the long-term benefit of policy action. But, as explained above, the reduction in deaths is not sustained year on year. Population total survival time ('life-years') better reflects the benefit to health of policy action as it is based on an assessment of the total survival of the population under a given scenario. For these reasons COMEAP has over the years considered expressing the mortality implications of reduced long-term exposure to air pollution in terms of life expectancy and the number of life-years gained over the population, as more informative than annual reductions in numbers of deaths sometimes described as 'lives saved'. As such, in the UK, the benefits of policy measures targeted at reducing levels of $PM_{2.5}$ have been expressed in terms of 'total life-years' rather than reductions in numbers of deaths (e.g. in the report by the Interdepartmental Group on Costs and Benefits (IGCB), 2007).

We recognise, however, that the effects of air pollutants on health are often expressed in terms of 'numbers of attributable deaths' and we support the use of this term insofar as its use is valid

and informative. So another purpose of this report is to explain the various ways of expressing mortality effects in terms of deaths rather than life expectancy, and to show the relationship of these to one another. This issue is addressed in Part I of this report, in the context of pollution reduction, and again in Part II, where it will be seen that the number of deaths can be useful as a way of representing the current mortality burden of pollution, though care needs to be taken with interpreting the results.

## 2.2 Core concepts of mortality and survival analysis – a more technical description

We begin with a description of the concept of life expectancy, and then adapt that to considerations of change in a current population with a distribution of ages. We highlight the many assumptions required in this or any calculation predicting future impacts, compare results with those from some widely-used approximations, discuss the implications of differences and make recommendations on how future impact assessments in this area might best be carried out.

### 2.2.1 Death rates, survival curves and life expectancy

*Life expectancy*, the expected or average length of life in a given population, cannot be observed directly; what can be observed and recorded are the fact of a death, the date (and time) of its occurrence and the certifying physician's opinion as to the underlying cause. Since date of birth is also recorded at death certification, age at death can be calculated.

Death data collected across a population can be summarised by sex, date of birth and/or age at death (and by any other demographic data available regarding the deceased subjects, e.g. socioeconomic status). The numbers of deaths divided by estimates of the size of the (sub-)populations from which they arise give *mortality rates*, i.e. the risk of death. These are usually available from national bodies as sex- and age-specific mortality *(hazard)* rates.

Estimation of life expectancy from these age-specific rates requires an assumption that they are the rates that will be experienced by members of a population as they age. The calculation combines the rates in order to predict the probability of survival to each age, and from this the average life expectancy can be derived. The formulae for the 'life-table' calculations are given in a supporting paper to this report (Miller and Hurley, 2010). The formulae also allow for the prediction of age-specific life expectancies: that is, the expected remaining life expectancy given survival to each age. This decreases with age, but by less than a year per year aged. For example, in anyone who has lived to be 60 years old, the total life expectancy is better than at 20 years old because the risks of dying between 20 and 60 years are no longer relevant.

Figure 2.1 shows some mortality hazard rates, by age, for males and females in England and Wales and in Scotland in the year 2008, and averaged over 2006–2008 in Northern Ireland. The *y*-axis is logarithmic: in adulthood, in developed countries, the risk of death increases exponentially with age, and so the hazard rates are approximately linear on the logarithmic scale, especially from about age 40 years onwards.

One summary measure of the impact of a change in mortality rates (e.g. due to changes in levels of particulate air pollution) is the implied change in life expectancy. Life expectancy is calculated from one (baseline) set of rates and then from a set of impacted (changed) rates, and

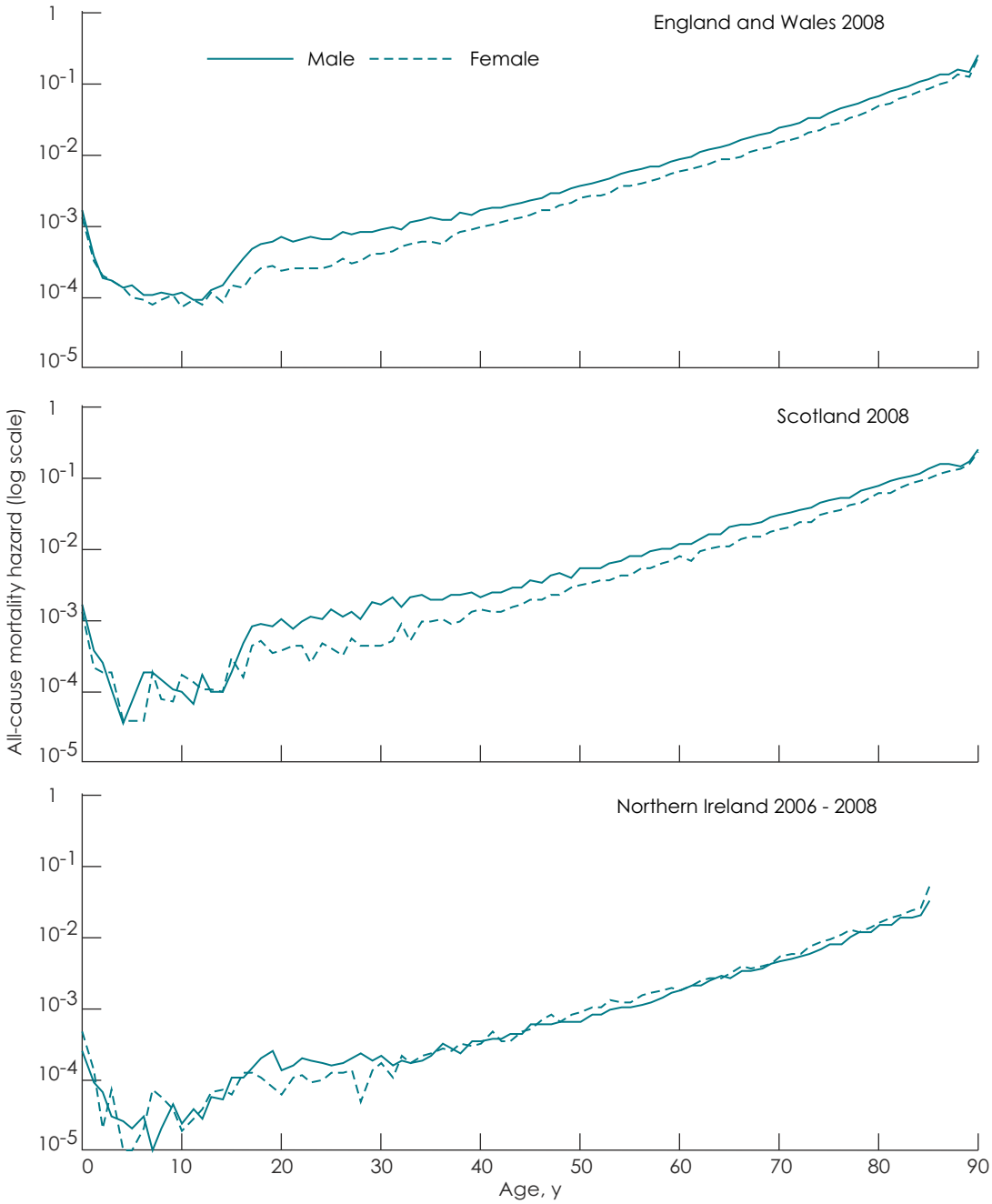


**Figure 2.1: All-cause mortality hazard rates in 2008 by sex and age for England and Wales, Scotland and in 2006–2008 for Northern Ireland**

the results compared. If the changes in rates are a mixture of reductions and null change, i.e. if there are no increases, then the change in life expectancy must be positive, i.e. a gain.

The life expectancy calculations combine the hazard rates, projected forward in time, to predict a survival curve. Figure 2.2 shows survival curves derived from the hazard rates shown for 2008 in England and Wales, in Figure 2.1. The difference in life expectancy between the sexes is equivalent to the area between the two curves, at least as far as age 90 years. Survival curves also enable calculation and comparison of other summary measures of survival, for example:

a The age to which a given proportion of the population will survive, or will have died, e.g. Figure 2.2 shows that, in a birth cohort with 2008 death rates for England and Wales, about 70% of women will survive to age 79 years, and 70% of men to age 74 years;

b The proportion of the population surviving to a given age, e.g. in the same birth cohort, about 78% of men, and 86% of women, would survive until age 70 years.

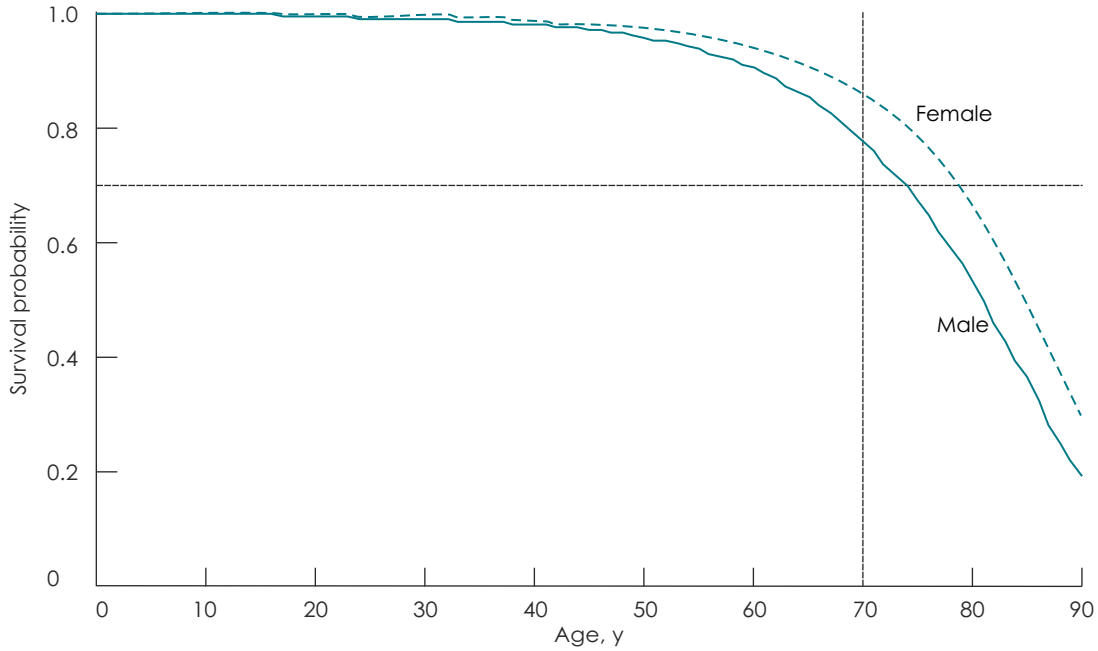


**Figure 2.2: Predicted survival curves for males and females, based on mortality rates for 2008 in England and Wales**

## 2.2.2 Impacts on whole populations

The life expectancy calculations can quantify impacts on new birth cohorts, but policies on air pollution impact on whole populations, at all ages. Thus, a prediction of impacts on a real population, as distinct from some theoretical future one, must be based on a series of life expectancy calculations, one for each age group affected. This is greatly facilitated by identifying separately the dimensions of age and calendar year (Miller and Hurley, 2003). The data required for the calculations can then be laid out in those two dimensions, as shown in Figure 2.3. Here the current population is stratified by age, from 0 to a maximum age, here 105 years. The body of the table is populated with mortality hazard rates (h) (here assuming that the 2008 rates will apply also in all succeeding years). Life-table calculations for the birth cohort, $e_0$, are carried out down the main diagonal of the table, shaded in grey, while those for other ages are followed up for the remainder of their lives down other diagonals, in the yellow shaded area. New birth cohorts are included in each year, and can be followed up similarly down their own diagonals, in the unshaded area.

This layout and formulation provides a consistent method for health impact assessments involving mortality, and specifically for quantifying the effects of future changes in age- and period-specific mortality rates, as are assumed to happen if changes occur in annual average particulate air pollution, represented as $PM_{2.5}$. More detail is provided in a supporting paper to this report (Miller and Hurley, 2010). Essentially, the calculations are carried out for a baseline (current) scenario, and the outputs compared with another set of calculations carried out with altered hazard rates (reduced hazard rates, if the impact of reducing air pollution is being assessed). These methods have been developed and refined over a number of years and in several projects, including some contracted by the UK Department of Health, and results have also been produced for European projects such as ExternE (External cost of Energy), Clean Air For Europe (CAFE) and most recently Health and Environment Integrated Methodology and Toolbox for Scenario Assessment (HEIMTSA).

| **Age (years)** | **Entry pop$^n$** | **Year** | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 2008 | 2009 | 2010 | … | j | … | 2111 | 2112 | 2113 |
| | | Births | $b_1$ | $b_2$ | … | $b_j$ | … | $b_{103}$ | $b_{104}$ | $b_{105}$ |
| 0 | $e_0$ | $h_0$ | $h_0$ | $h_0$ | | $h_0$ | | $h_0$ | $h_0$ | $h_0$ |
| 1 | $e_1$ | $h_1$ | $h_1$ | $h_1$ | | $h_1$ | | $h_1$ | $h_1$ | $h_1$ |
| 2 | $e_2$ | $h_2$ | $h_2$ | $h_2$ | | $h_2$ | | $h_2$ | $h_2$ | $h_2$ |
| ⋮ | | | | | | | | | | |
| i | $e_i$ | $h_i$ | $h_i$ | $h_i$ | | $h_i$ | | $h_i$ | $h_i$ | $h_i$ |
| ⋮ | | | | | | | | | | |
| 103 | $e_{103}$ | $h_{103}$ | $h_{103}$ | $h_{103}$ | | $h_{103}$ | | $h_{103}$ | $h_{103}$ | $h_{103}$ |
| 104 | $e_{104}$ | $h_{104}$ | $h_{104}$ | $h_{104}$ | | $h_{104}$ | | $h_{104}$ | $h_{104}$ | $h_{104}$ |
| 105 | $e_{105}$ | $h_{105}$ | $h_{105}$ | $h_{105}$ | | $h_{105}$ | | $h_{105}$ | $h_{105}$ | $h_{105}$ |

**Figure 2.3: Schematic layout of demographic inputs required for a full health impact assessment on mortality**

### 2.2.3 Cause-specific mortality

Evidence suggests that long-term exposure to particulate air pollution principally affects mortality from non-malignant cardiorespiratory causes and from lung cancer. It is usual, however, to compute population mortality impacts in terms of how air pollution affects risks of mortality from all causes, because this is easier computationally, the underlying risk coefficients are estimated more precisely, and results in terms of population survival are similar. COMEAP

(2009) recommends primary analyses in terms of all-cause mortality, with supplementary cause-specific analyses.

In the present report we deal only with all-cause mortality, because our aim is to illustrate core concepts of calculating and communicating mortality impacts. However, to indicate what is involved in carrying out cause-specific analysis, we note that cause-specific mortality rates from all the different causes of death should add up to give the all-cause mortality rate in a population. As this is the case, it is possible to focus the impact of air pollution on only a selection of causes of death.

To carry out cause-specific analysis, a separate input table for each cause must be set up; the mortality rates have to cover all causes and have to add up to the all-cause rates. Then a set of impact factors for the separate cause hazards will be defined, according to how air pollution affects each cause. Analyses can be undertaken for individual causes of death, or the impacted hazards can be summed to give new impacted all-cause hazard rates and the calculations proceed as before. More details are given in Miller and Hurley (2006).

### 2.2.4 Effects of baseline death rates

Absolute levels of death rates vary between women and men, between countries, and over time. An interesting and important result, illustrated on the basis of countries in the results section (Chapter 4), is that for a given percentage reduction in mortality hazards, the gains in life expectancy and in life-years per 100,000 are similar in different populations, even when underlying hazard rates (i.e. age-specific death rates) clearly differ. This is in accordance with theoretical results from Leksell and Rabl (2001): if the underlying mortality rates follow the log-linear pattern of Figure 2.1, then a small percentage change in them produces the same impact on life expectancy regardless of the baseline level.

This fact has two very important consequences, which are discussed in detail later. One of these concerns spatial transferability of results: because the same percentage changes in death rates lead to similar changes in life expectancy, even if the underlying population age structure and death rates are different, then life-table results from one country can be transferred to another, with suitable scaling for actual population size.

The other consequence concerns transferability over time: results are robust to assumptions about how death rates will change over time in the future. It is not necessary to have correct or even realistic estimates of future 'baseline' death rates – results regarding the effect on life expectancy and life-years of a change in death rates will not be sensitive to decreases (or increases) in baseline rates over time, as long as both the true future death rates and those assumed for the analysis follow the general log-linear shape with age of Figure 2.1.

## 2.3 Inputs to the calculations

In any practical situation, the results will depend on a number of assumptions, which should be stated explicitly. In practical applications some of these assumptions are made explicit, while others are treated as 'obvious' or as a consequence of the questions being asked. We think, however, that it is useful to make all assumptions explicit. We discuss them here in general terms, and summarise how we deal with the simpler ones. How we deal with more complex ones is summarised in Chapter 3.

### 2.3.1 Population

a What is the population of interest and for which the impacts will be estimated?

This depends entirely on the questions asked. Our analyses deal with the UK as a whole. We have carried out life-table analyses variously for the populations of (i) England and Wales, (ii) Scotland and (iii) Northern Ireland, because demographic data (population sizes, death rates) are readily available for these three populations.

b Will impacts be estimated separately by sex?

All our analyses are carried out separately for women and men as death rates are published separately by sex.

c Are any other stratifying factor(s) used?

No, in these analyses we have not attempted to stratify by other factors, e.g. socioeconomic status.

d What is/are the age distribution(s)?

In these analyses we have used age distributions and death rates based on individual years rather than, say, the published five-year age groups available online from the Office for National Statistics.

e Are adjustments made to the size and age distribution of the population?

A sustained reduction in pollution will affect the health and survival chances not only of those people who are currently alive, but also of those not yet born. For that reason, we include new births into the population when assessing the impact of a pollution reduction. For simplicity, we assume a constant number of new births annually.

We do not adjust population size and age distribution to take account of migration.

### 2.3.2 Baseline death rates

f What baseline hazard rates for mortality should be used?

The analyses reported here use baseline rates for 2008 for England and Wales and for Scotland, and averaged across the three years of 2006–2008 for Northern Ireland.

g Will the analyses be cause-specific or all-cause?

Following COMEAP (2009), we give priority to analyses of all-cause mortality, but cause-specific analyses could also be carried out.

h Will assumed baseline rates in the future be constant or changing?

We assume that baseline death rates will not change in the future. This assumption that rates will not change is clearly unrealistic, but is widely used to produce 'period' life tables and to calculate life expectancy. Some others, e.g.

UK Government actuaries in the Office for National Statistics, calculate alternative 'cohort' estimates based on age-specific mortality rates extrapolated to continue falling at the same rate as in recent years (Office for National Statistics, 2008).

However, estimated impacts of an air pollution-related change in mortality rates are insensitive to the levels of baseline rates (see Section 2.2.4) and so this assumption of no change gives valid results.

### 2.3.3 Pollution-related changes in death rates: impact factors

i What percentage changes in death rates will be examined?

The percentage change in mortality hazards is a consequence of two factors:

- the percentage change per $\mu g/m^3$ reduction in $PM_{2.5}$, based on the selected CRF,
- the magnitude of the reduction in $PM_{2.5}$ being examined – this depends on the questions being addressed.

These combine to produce factors by which the baseline hazard rates will be impacted by the change in pollution. We call them 'impact factors'[3] and denote them $k$. It is possible that these may vary by age or with calendar time, or both; their pattern will depend on the question to be answered and the assumptions made.

j Will the impact factor, $k$, vary by age?

Following COMEAP (2009), No, except that, to be consistent with the American Cancer Society (ACS) study populations (Pope *et al*, 2002), we will assume that there are effects only in ages 30 years and over. Other work has shown that relaxing this assumption, i.e. by applying the same percentage change to death rates at ages under 30 also, has only a small effect on the final answers, because death rates at ages under 30 years are low.

k Will the impact factor, $k$, reflect a time delay from intervention to hazard change?

Yes, this is the issue of time lag or cessation lag between reductions in the annual average $PM_{2.5}$ and consequent changes in mortality risks. There are a number of ways in which the overall impact on mortality rate may occur or be implemented in a life-table approach; a selection of these are illustrated in Figure 2.4. Further explanation of cessation lag can be found in Section 3.1.4 and a supporting paper to this report (Walton, 2010).

l Will the impact factor, $k$, vary over time?

Although we allow for cessation lag in questions of impact, we do not assume time-dependent, future, variation in the underlying risk coefficients.

[3] Impact factors are multipliers used to adjust the mortality rates; for example, for a 1% reduction in hazards, the impact factor is (100 – 1)/100 = 0.99.

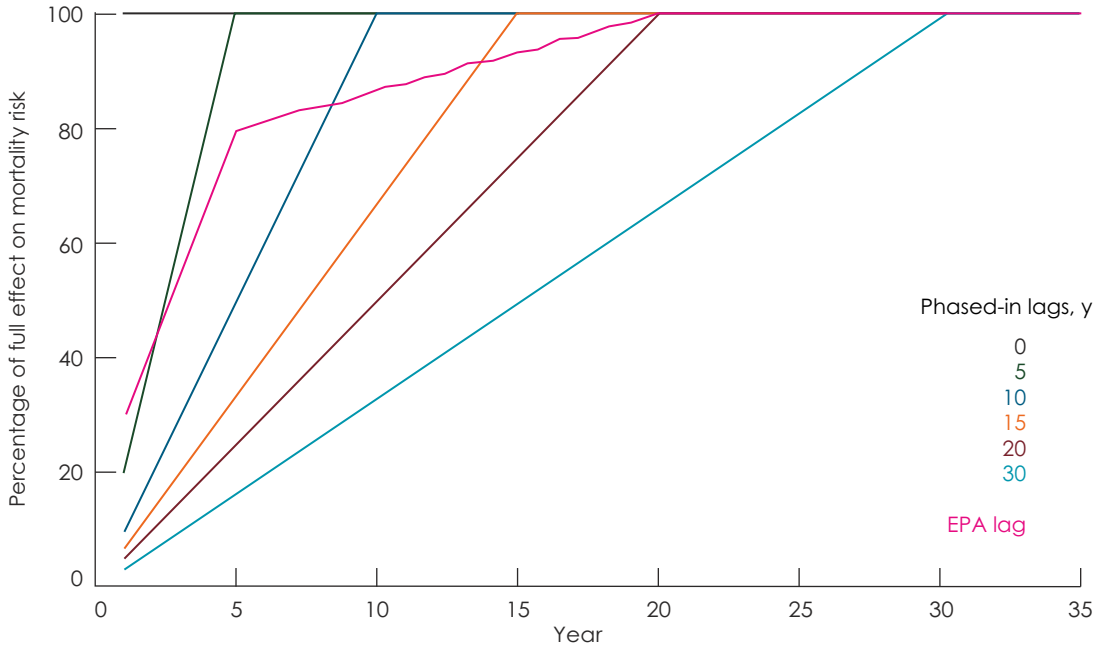


**Figure 2.4: Examples of cessation lag of implementation of mortality risks (adapted from Walton, 2010). Note the first year is designated as year 1 and is marked at point 1 at the end of the first year, thus the graph does not start at zero**

## 2.4 Outputs of the calculations – life-table differences between baseline and impacted scenarios

The life-table calculations produce, as primary outputs, predictions of the differences between the baseline and alternative scenarios, in terms of both numbers of deaths and total years of life lived. As shown in Figure 2.5, these are calculated for all combinations of age and calendar year. It is likely that users will want these to be summarised in some way, e.g. over given time periods, or over all age groups. The shaded triangle in Figure 2.5 delimits the outcomes for the population alive in 2008, when the change in hazards took place, and we can summarise over this triangle to quantify the effects on the population current in 2008. It is easy to include the additional impacts in the cohorts created by new births in the years after 2008; these will lie in the portion above the shaded triangle. With a permanent change in hazards, the benefits to survival continue to accrue every year, so it is usual to stop accumulating them at some point. We have tended to count impacts up to the year after which the 'current' (here 2008) population is extinct (zero survival), i.e. to the end of 2113, but again the choice is with the user.

If the health impact assessment (HIA) is to provide evidence that forms the basis of cost–benefit analyses (CBA), then it will be necessary to attach monetary values to the impacts. Having them itemised by age and calendar year allows great flexibility in how this is done – for example, if life-years are valued differently at different ages. In addition, many CBAs will require both costs and benefits, if they accrue at future times, to be subject to a discount rate. In order that the values of the benefits can be correctly discounted, it is necessary to know their distribution over time, and the life-table analyses supply them in this form.

Other forms of weighting may also be facilitated by the disaggregated layout of the life-table results, as described in a supporting paper (Miller and Hurley, 2010) – for example, if it is

| Age (years) | Year | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 2008 | 2009 | 2010 | … | j | … | 2111 | 2112 | 2113 |
| 0 | $d_{0,2008}$ $y_{0,2008}$ | $d_{0,2009}$ $y_{0,2009}$ | $d_{0,2010}$ $y_{0,2010}$ | | $d_{0,j}$ $y_{0,j}$ | | $d_{0,2111}$ $y_{0,2111}$ | $d_{0,2112}$ $y_{0,2112}$ | $d_{0,2113}$ $y_{0,2113}$ |
| 1 | $d_{1,2008}$ $y_{1,2008}$ | $d_{1,2009}$ $y_{1,2009}$ | $d_{1,2010}$ $y_{1,2010}$ | | $d_{1,j}$ $y_{1,j}$ | | $d_{1,2111}$ $y_{1,2111}$ | $d_{1,2112}$ $y_{1,2112}$ | $d_{1,2113}$ $y_{1,2113}$ |
| 2 | $d_{2,2008}$ $y_{2,2008}$ | $d_{2,2009}$ $y_{2,2009}$ | $d_{2,2010}$ $y_{2,2010}$ | | $d_{2,j}$ $y_{2,j}$ | | $d_{2,2111}$ $y_{2,2111}$ | $d_{2,2112}$ $y_{2,2112}$ | $d_{2,2113}$ $y_{2,2113}$ |
| ⋮ | | | | | | | | | |
| i | $d_{i,2008}$ $y_{i,2008}$ | $d_{i,2009}$ $y_{i,2009}$ | $d_{i,2010}$ $y_{i,2010}$ | | $d_{i,j}$ $y_{i,j}$ | | $d_{i,2111}$ $y_{i,2111}$ | $d_{i,2112}$ $y_{i,2112}$ | $d_{i,2113}$ $y_{i,2113}$ |
| ⋮ | | | | | | | | | |
| 103 | $d_{103,2008}$ $y_{103,2008}$ | $d_{103,2009}$ $y_{103,2009}$ | $d_{103,2010}$ $y_{103,2010}$ | | $d_{103,j}$ $y_{103,j}$ | | $d_{103,2111}$ $y_{103,2111}$ | $d_{103,2112}$ $y_{103,2112}$ | $d_{103,2113}$ $y_{103,2113}$ |
| 104 | $d_{104,2008}$ $y_{104,2008}$ | $d_{104,2009}$ $y_{104,2009}$ | $d_{104,2010}$ $y_{104,2010}$ | | $d_{104,j}$ $y_{104,j}$ | | $d_{104,2111}$ $y_{104,2111}$ | $d_{104,2112}$ $y_{104,2112}$ | $d_{104,2113}$ $y_{104,2113}$ |
| 105 | $d_{105,2008}$ $y_{105,2008}$ | $d_{105,2009}$ $y_{105,2009}$ | $d_{105,2010}$ $y_{105,2010}$ | | $d_{105,j}$ $y_{105,j}$ | | $d_{105,2111}$ $y_{105,2111}$ | $d_{105,2112}$ $y_{105,2112}$ | $d_{105,2113}$ $y_{105,2113}$ |

**Figure 2.5: Schematic layout showing pattern of predicted output from mortality simulations, and subgroups for summaries: d = number of deaths, y = total person years**

desired to express impacts in terms of disability- or quality-adjusted life-years (DALYs or QALYs). Such impacts may also be combined with economic discounting.

For our analyses, we investigate the effect of discounting on the results by having given each life year a nominal value of one. This does not imply that the health effects themselves discount, but rather that if a value were associated with the health effects this shows how the value would be affected.

## 2.5 Distribution of impacts over time

It may be of interest to understand how the impacts of a change in pollution are distributed over time; results from the full life-table calculations allow examination of this question. Figure 2.6 shows the time-pattern of the differences in life-years and deaths following a sustained reduction in pollution of 1 μg/m³ $PM_{2.5}$ from 2008, based on 2008 rates for England and Wales. It shows results for the 2008 population based on two different assumptions: (i) those alive in 2008, without new births – results from this population are interesting, because the life-table analysis follows up the population until everybody has died; and (ii) the 2008 population supplemented by new births in subsequent years – this is more appropriate for benefit assessment because a sustained reduction in pollution will benefit not only those alive in 2008, but new generations also.

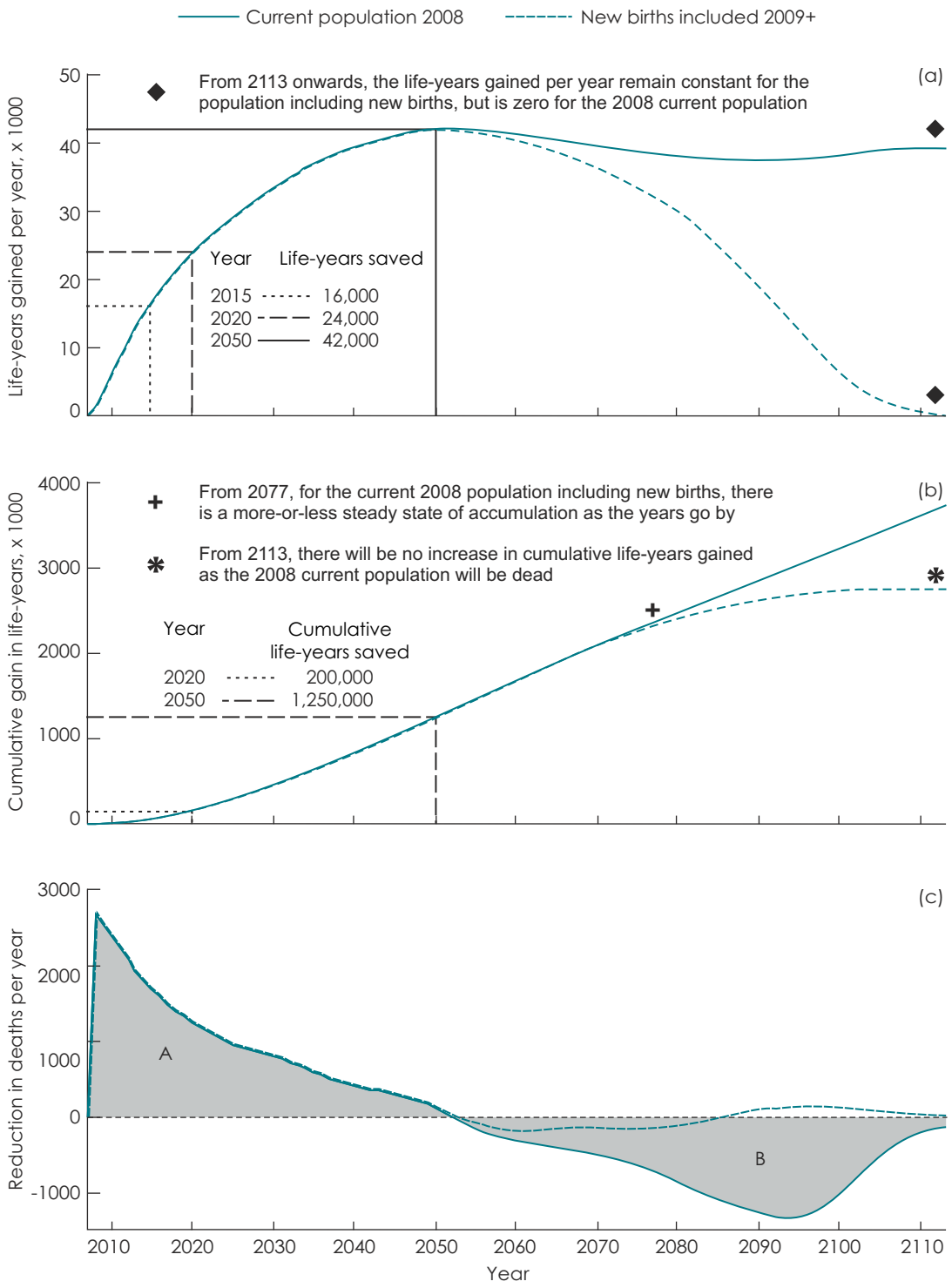


**Figure 2.6: Patterns of impacts following a permanent reduction of 1 µg/m³ in annual average $PM_{2.5}$ concentrations, impacting on all-cause mortality hazard rates for England and Wales; impacts expressed as annual (a) and cumulative (b) gains in numbers of life-years, and as annual reductions in numbers of deaths (c)**

In order to illustrate the methodological issues as clearly as possible we assume, for the purposes of this illustration, that reductions in mortality risks follow immediately from reductions in annual average $PM_{2.5}$, i.e. without cessation lag; we realise this is an over-simplification. In fact, when we carry out the calculation we do assume a cessation lag, see Section 3.1.4. Also, this report focuses on mortality in adults, with mortality risks changing only at age 30 years and older – i.e. the age range of the ACS cohort study – and so the graphs of the difference in deaths and life-years between the baseline and alternative scenarios are identical for both the 2008 population and the 2008 population supplemented by new births for the first 30 or 40 years, but then diverge quite markedly over time.

The graph of the differences in deaths for both populations (current and current with new births, Figure 2.6c) shows a sharp reduction in deaths in the first year, when under both scenarios (i.e. without and with pollution reduction) the population at risk is the same size and the same age. Over time, the population with reduced death rates, due to reduced pollution, has more survivors than the baseline population, i.e. it has more people surviving, and on average they are older. Consequently, the difference in numbers of deaths annually between the two scenarios reduces gradually over time. But all the current population have to die some time, and from around 2050 the number of deaths in the current population is higher in the lower-pollution scenario with the reduced hazard rates. Eventually, the whole current population has died, and the number of further deaths in each scenario is zero, so their difference, at the right-hand end of Figure 2.6c, must be zero. When we include new births, it is also the case that the difference in deaths eventually converges to zero. This is because a population that experiences constant mortality rates and a constant birth rate, as assumed here, will eventually develop an age distribution that mirrors its implied survival curve, and the number of deaths will equal the number of new births. Thus, the numbers of deaths becomes the same (and their difference zero) in the baseline and alternative (reduced pollution) scenarios, but the populations have stabilised to different age distributions: in the reduced pollution scenario the population is larger and on average older, and thus generates more life-years annually (as is seen for the population including new births in Figure 2.6a).

It can be seen clearly from the deaths graph (Figure 2.6c) that assuming that the reduction in the number of deaths in the first year (almost 3000 for the 1 $\mu g/m^3$ change in England and Wales illustrated) applied in all subsequent years would result in a major overestimate of the total reductions in numbers of deaths over the overall period. This, however, is what is implied when it is assumed that one number can be given for the annual number of deaths that would be saved if air pollution was reduced.

The situations differ after about 2050, according to whether new births are included or not.

a **Without new births,** the annual number of deaths under the two scenarios continues to diverge, with more deaths annually under the scenario of lower age-specific death rates due to reduced pollution, as the population reaches extinction under both scenarios; until the time when the population under both scenarios has died out fully, there are no more deaths, and so no difference in deaths between the two scenarios. Without new births, the reduced deaths initially (i.e. up to about the year 2050), indicated by shaded area A in Figure 2.6c, under a reduced pollution scenario are counterbalanced exactly by increased deaths after about 2050, indicated by shaded area B in Figure 2.6c, reflecting that everybody in the population dies in due course – what changes is length of survival and age at death.

b **With new births,** the number of deaths annually in the two populations is similar after 2050 although, and this is not evident from the graph, those who die under the reduced pollution scenario are on average older than their counterparts without pollution reduction.

# Chapter 3
# Specification of the Impact Calculation

To carry out these analyses, a number of parameters needed to be defined. This section sets out these parameters, their allocated values and the reasoning for the decisions made. It describes a core analysis framework, with specific values of key parameters. We then assessed to what extent the results were sensitive to a number of key assumptions. We did this by varying the input values of a number of key assumptions, one at a time; these sensitivity analyses are also presented.

## 3.1 Calculations and parameters

### 3.1.1 Method: life-table analyses

The calculations reported here were carried out using the IOMLIFET spreadsheet tool. This can accommodate virtually any set of input assumptions, which are discussed further below, and offers a similarly diverse set of options for summarising the impacts, depending on the exact question posed, and the kinds of output considered useful for answering that question.

### 3.1.2 Population

For these analyses population data from 2008 were used and, as outlined in Section 2.3.2, baseline mortality hazard rates from 2008 were used for England and Wales and for Scotland, while averaged 2006-2008 data were used for Northern Ireland, where the smaller population leads to more instability in annual rates. Results for the present report were based on separate analyses for the populations of England and Wales, of Scotland and of Northern Ireland.

An alternative approach, where the necessary data are not available for a target population, would be to carry out impact calculations from available data for (say) England and Wales, and scale these to other countries – or to England alone, to Wales alone or to specific regions within them – by total population and using region-specific population-weighted pollution concentrations. Theoretical and empirical results show that estimated impacts are insensitive to local levels of baseline mortality, provided that the age-specific mortality hazards have the same approximate log-linear shape – Figure 2.1 suggests they do.

Ensuring the appropriate population was assessed was also important. For assessing the impact of a reduction policy, the current 2008 population plus new births during the follow-up period was used because people as yet unborn in 2008 would also benefit from sustained pollution reductions. The analysis did not attempt to take account of patterns of migration or changes in

birth rate and it was assumed that the same number of new births occurred annually throughout the follow-up period as in 2008.

The impact of pollution reduction on life expectancy has been expressed in terms of life expectancy from birth of the cohort born in 2008. This impact on life expectancy was calculated by comparing the predicted life expectancy based on 2008 mortality rates with the predicted life expectancy when mortality rates have changed with the reduction in particulate air pollution. This is also discussed in Section 2.2.1.

For this assessment, a follow-up period of 106 years was used, i.e. a period long enough to allow the current (2008) population to die out. This ensured the full extent of mortality benefits to those alive in 2008 was reflected. A shorter follow-up period would fail to include benefits that only occur later on, even though they may be a significant contributor to the overall impact.

The reduced hazard rates were applied to the population from the age of 30 years because the ACS study (Pope *et al*, 2002), from which the coefficient was derived, only included adults aged 30 years or more and so is not directly informative of effects in people younger than this.

### 3.1.3 Coefficient

#### *Principal coefficient used*

A principal risk coefficient of 1.06 change in all-cause mortality hazard per 10 μg/m$^3$ $PM_{2.5}$ (annual average concentration) was used, as recommended by COMEAP (2009). The 95% confidence interval for this coefficient is 1.02–1.11. However, using a relatively simple expert elicitation, COMEAP (2009) derived a plausibility distribution to take account also of other aspects of uncertainty, such as strength of evidence for causality and confidence in transferability of the coefficient from the USA where the underlying study was carried out. This plausibility distribution gives rise to a number of intervals which can be used for quantification; COMEAP proposed the interval of 1.01–1.12, based approximately on the 12.5$^{th}$ and 87.5$^{th}$ percentiles of the overall range of COMEAP Members' consolidated views of the probability, for quantification purposes, while suggesting that a wider interval of 1.00–1.15 should also be included in reports on quantification of risk such as this one.

#### *Range of coefficients used*

Consequently, as a sensitivity analysis, we have investigated results for the removal of all anthropogenic $PM_{2.5}$ using a range of coefficients, specifically assuming risk coefficients of 1.00, 1.01, 1.06, 1.12 and 1.15 per 10 μg/m$^3$ increase in $PM_{2.5}$. One reason was to provide substantive results corresponding to the COMEAP-recommended uncertainty ranges. The other was to investigate, and illustrate, to what extent results were linear in relation to the risk coefficient, i.e. to what extent a given percentage change in risk coefficient implied also the same percentage change in mortality impacts.

#### *Scaling for different concentration changes*

The main risk coefficient from the ACS study, as reported by Pope *et al* (2002) and recommended by COMEAP (2009), is for a 10 μg/m$^3$ increase in $PM_{2.5}$; it needs to be scaled when a different concentration change is used. Because the ACS study (Pope *et al,* 2002)

derived the coefficients from study of the relationships between the logarithm of the relative risk and concentration, we used logarithmic (multiplicative) scaling[4] in the calculations reported here.

### 3.1.4 Cessation lag

Cessation lag is a term used to denote the time pattern of reductions in mortality hazards following a reduction in pollution. There is little direct evidence about cessation lags and clear-cut evidence-based recommendations are not possible. COMEAP (2009) considered that while in principle it might take 40 years for all benefits to be achieved, in practice benefits were likely to occur significantly earlier, with a noteworthy proportion in the first five years. For the present impact calculations we decided to use a recommendation of the US Environmental Protection Agency (US EPA). This was initially agreed by the US EPA in 2004 (US EPA, 2004) and was re-affirmed for use in analyses in 2010 (US EPA, 2010). In this distribution, 30% of the risk reduction occurs in the first year after pollution reduction, 50% occurs across years 2–5 (i.e. 12.5% per year) and the remaining 20% of the risk reduction is distributed across years 6–20 with smoothed annual values. These three components of the distribution reflect short-term, cardiovascular and lung cancer effects, respectively.

In addition, we have reviewed further the evidence on cessation lag; a separate paper on lags has been written and is provided as a supporting paper to this report (Walton, 2010). This sets out various alternative lag structures based on evidence in the literature and concludes that a categorical evidence-based choice between them is not possible. The suggestions for lag structures are encompassed by a range from no lag to a 30 year phased-in lag and the US EPA lag structure also lies well within this range.

The effect of alternative assumptions about cessation lags has been assessed as a sensitivity analysis using no lag and 5, 10, 20 and 30 year phased-in lags. Under these lag patterns, changes to the hazard rates are applied gradually in a linear fashion until the reduction is fully implemented over the full time period of the specified lag. For the present calculations, this linear phase-in was approximated using a yearly step function where the step was the mid-year point on the straight line between the start date and the lag end date.

### 3.1.5 Discounting (weighting factors)

Some analyses discount future mortality impacts by applying to them weighting factors which reduce their value. Most commonly, this is done when monetary values have been attached to the relevant future deaths, or life-years lived, and the resulting values are discounted to take into account the future value of life-years. While technically this is not difficult to do using the life-table methods underlying the present report, the process can be controversial with respect to the choice of discount rate, if any.

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[4] The equation for scaling is based on multiplicative scaling of the relative risk, i.e. 1.06 for all-cause mortality for a concentration increase of 10 $\mu g/m^3$ $PM_{2.5}$. If the change in population weighted-mean concentration is $-x$ $\mu g/m^3$ (with a negative sign for reductions in concentration), then the new relative risk is calculated as $1.06^{-x/10}$. For convenience, people may simply scale the coefficient on a linear basis (e.g. the percentage change in mortality rates would be halved for a 5 $\mu g/m^3$ change). This is a reasonable approximation in many circumstances but the methods diverge increasingly when using larger coefficients (e.g. 12%) and large concentration changes (e.g. elimination of anthropogenic pollution).

We did not consider here what discounting (weighting) factors, if any, should be used – this report primarily focuses on health impacts *per se*, i.e. un-weighted. However, we did explore to what extent results are sensitive to discounting and how important discounting may be relative to other factors which affect the mortality calculations. Consequently, a limited number of illustrative examples of the effects of different weighting factors are provided; and rates of 1.5%, 3% and 6% per year were used in sensitivity analyses, in addition to the default and principal assumption of zero weighting or discounting.

## 3.2 Pollution reductions

The population, coefficient and cessation lag assumptions described in Section 3.1 were applied to the 1 μg/m$^3$ reduction in $PM_{2.5}$, to address question (a): 'What are the benefits expressed as an effect on mortality of a sustained reduction in annual average air pollution across the UK by a small fixed amount, e.g. by 1 μg/m$^3$ $PM_{2.5}$?'.

The same assumptions were also used in the calculation of the impact of removing all anthropogenic $PM_{2.5}$, i.e. to answer question (b): 'If anthropogenic air pollution in 2008 were to be removed and pollution sustained at low non-anthropogenic levels, what would be the benefits in terms of mortality?'. Answering this question also required the population-weighted annual average concentration of anthropogenic $PM_{2.5}$ to be modelled.

### 3.2.1 Modelling of $PM_{2.5}$ concentrations across the UK for 2008

Annual mean $PM_{2.5}$ concentrations have been estimated at a spatial resolution of 1 km × 1 km grid squares across the UK for 2008. The Pollution Climate Mapping (PCM) model has been used to calculate these estimates. The methods used to model $PM_{10}$ concentrations[5] in 2008 for the UK have been described in detail by Grice *et al* (2010) and the models used to calculate maps for 2004 have previously been described by Stedman *et al* (2007). The model for $PM_{2.5}$ is consistent with the model used for $PM_{10}$. For each component the concentrations of the fine ($PM_{2.5}$) and coarse ($PM_{10}$–$PM_{2.5}$) fractions have been calculated separately.

The modelling approach adopted was a pragmatic attempt to get close to understanding and accounting for all of the measured mass concentration (this is known as 'mass closure') for ambient concentrations of $PM_{2.5}$. The model included contributions from a number of different sources. The resulting ambient concentrations were then summed to calculate the total at each location. These components are listed below:

a Secondary inorganic aerosol (interpolated from sulphate ($SO_4^{2-}$), nitrate ($NO_3^-$) and ammonium ($NH_4^+$) measurements and scaled for counter-ions and bound water and size fractions);

b Secondary organic aerosol (estimated using the chemical transport model, HARM/ELMO);

c Large point sources of primary particles (explicitly modelled in the air dispersion model, ADMS, and with estimates of the emissions from the National Atmospheric Emission Inventory, NAEI);

[5] $PM_{10}$ is defined as the mass per cubic metre of particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 10 μm.

d Small point sources of primary particles (calculated with a small points model using dispersion kernels[6] derived in the air dispersion model, ADMS, and with estimates of the emissions from the NAEI);

e Regional primary particles (calculated using the chemical transport model TRACK model, NAEI emissions for the UK and EMEP emissions estimates for the rest of Europe);

f Area sources of primary particles (calculated with dispersion kernels derived from the air dispersion model, ADMS, and maps of emissions from the NAEI, and calibrated with measurements from the UK national monitoring networks);

g Rural calcium-rich dusts from soil resuspension (estimated using a resuspension model, emission rates depend on land cover and hour-by-hour meteorology);

h Urban calcium-rich dusts (modelled using a surrogate spatial distribution based on population);

i Regional iron-rich dusts (assumed constant 0.33 $\mu g/m^3$ $PM_{2.5}$);

j Iron-rich dusts from vehicle-related resuspension (estimated using a resuspension model, emission rate depends on heavy goods vehicle flow on major roads and hour-by-hour meteorology);

k Sea salt (interpolated from chloride ($Cl^-$) measurements and scaled for counter-ions);

l Residual (assumed constant 0.75 $\mu g/m^3$ $PM_{2.5}$).

Figure 3.1 shows the resulting map of annual mean ambient $PM_{2.5}$ concentrations for 2008. Figure 3.2 illustrates the source apportionment for $PM_{2.5}$ summarised as population-weighted means for different parts of the UK. The source apportionment information can be used to estimate the contributions to total concentrations from anthropogenic and non-anthropogenic sources. For many sources the distinction is reasonably clear cut. The contribution from sea salt is clearly non-anthropogenic, while the contribution from point source emissions from industries is clearly anthropogenic. It is less clear for some other sources such as dusts from soil resuspension, which could be considered as non-anthropogenic but are strongly influenced by land use and have therefore not been considered as non-anthropogenic in our analysis. The only contributions assigned as non-anthropogenic are sea salt and the residual. The composition of the residual is by definition not known but has been included in this category since other components which could arguably have been described as non-anthropogenic have been included as anthropogenic.

Contributors to the non-anthropogenic $PM_{2.5}$ fraction in the atmosphere include not only sea salt, which is expected to be of very low toxicity, but also sulphate and methylsulphonate formed from oxidation of biogenic dimethylsulphide releases from the oceans, and the fine fraction of wind-blown soil and dust, including Saharan dust. Other than sea salt, these are difficult to quantify even for a single location, but while the estimation by the PCM model is relatively crude, it is expected to be a reasonable and unbiased guide to non-anthropogenic concentrations.

[6] Running the dispersion model once for unit emissions and then applying to all of the grid squares in the country.

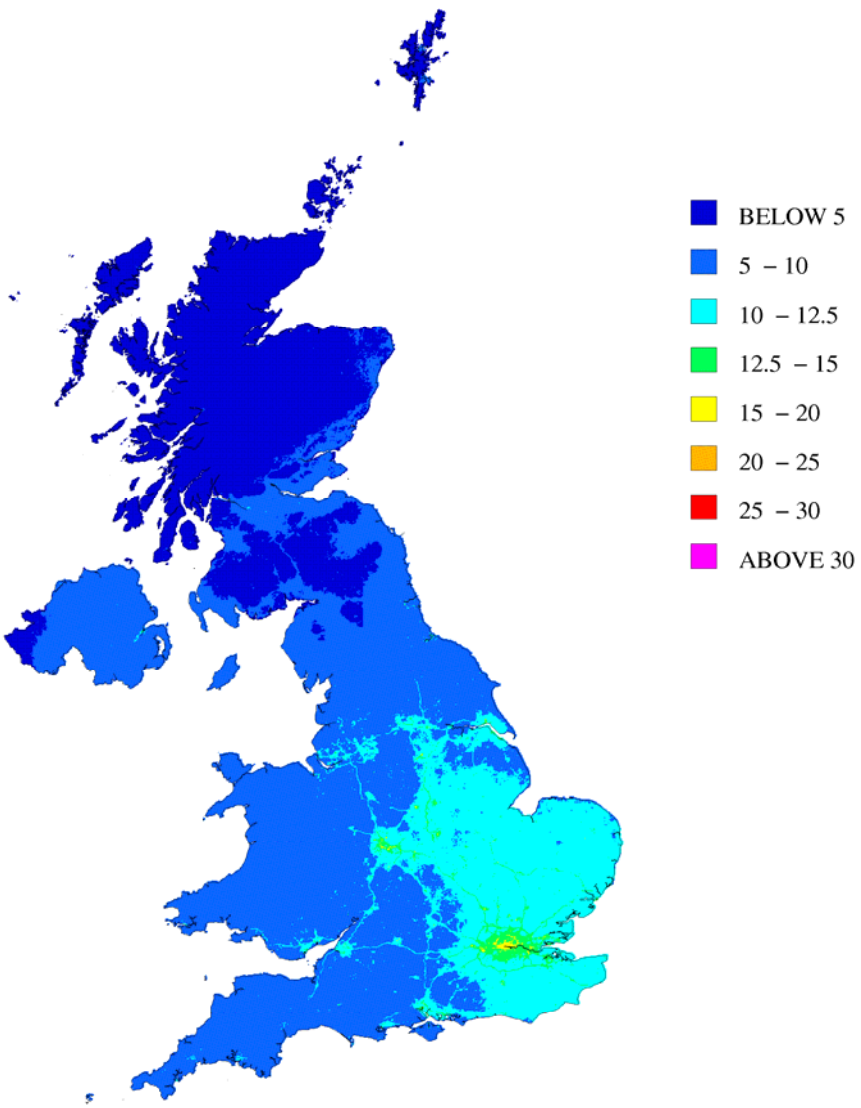


**Figure 3.1: Estimated annual mean background $PM_{2.5}$ concentration in 2008 (µg/m³, gravimetric)**

### 3.2.2 Pollution parameters for assessing the effects of air pollution

The population-weighted mean is a useful summary statistic, which greatly simplifies the calculation of human health impacts if the concentration–response function used is linear with no threshold. The population-weighted mean was calculated by multiplying the 1 km × 1 km concentration values by 1 km × 1 km population statistics from the 2001 census. The values for all of the grid squares were summed and then divided by the total population to calculate the population-weighted mean.

The most recent population-weighted mean $PM_{2.5}$ levels, those from 2008, were used. These are presented in Table 3.1. For impact assessment in England and Wales, the combined pollution data from England and Wales were used.

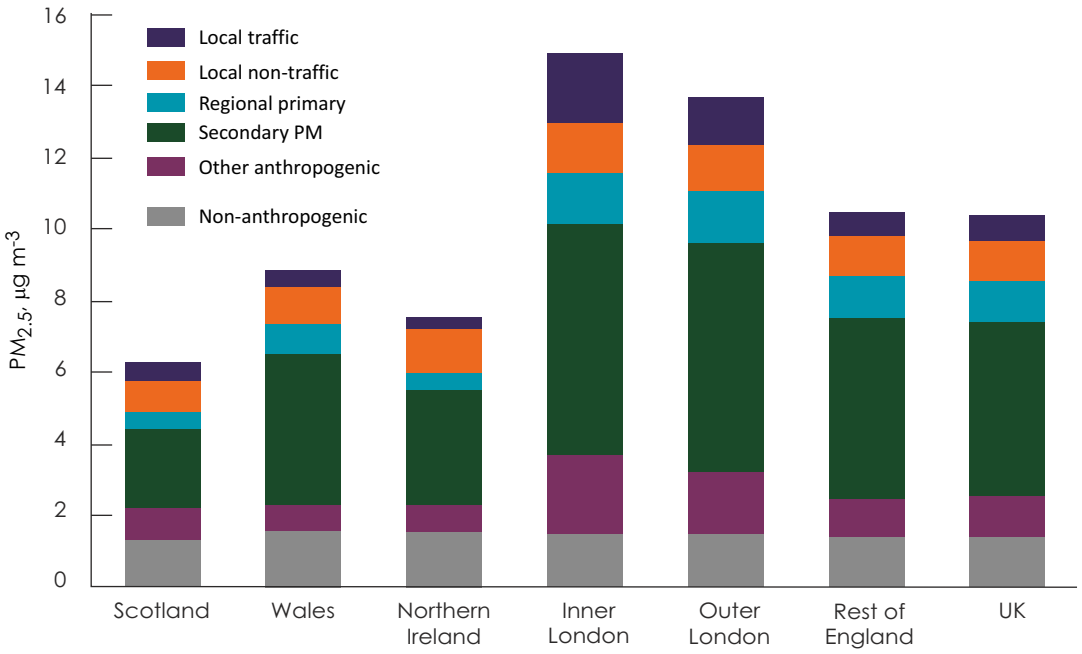


**Figure 3.2: Source apportionment of $PM_{2.5}$ around the UK for 2008**

Estimating the mortality benefits of removing all anthropogenic $PM_{2.5}$, i.e. reducing the total $PM_{2.5}$ (e.g. 10.88 µg/m³ for England and Wales) to non-anthropogenic $PM_{2.5}$ levels (e.g. 1.43 µg/m³ for England and Wales), involves extrapolating ACS risk estimates beyond the range of concentrations in the ACS study. In the study, the lowest annual average $PM_{2.5}$ concentration of any city was 7 µg/m³ (Pope *et al*, 2002). This extrapolation introduces additional uncertainties into the impact estimates. Therefore, the impact of reducing $PM_{2.5}$ from the total $PM_{2.5}$ to 7 µg/m³ was also calculated.

**Table 3.1: Population-weighted mean $PM_{2.5}$ concentrations (µg/m³) from 2008**

| | Total | Non-anthropogenic | Anthropogenic | Above 7 µg/m³ |
|---|---|---|---|---|
| Inner London | 14.91 | 1.47 | 13.43 | 7.91 |
| Outer London | 13.68 | 1.47 | 12.21 | 6.68 |
| Rest of England | 10.47 | 1.41 | 9.06 | 3.49 |
| Wales | 8.83 | 1.57 | 7.26 | 1.88 |
| *England and Wales combined* | 10.88 | 1.43 | 9.46 | 3.90 |
| Scotland | 6.28 | 1.31 | 4.97 | 0.21 |
| Northern Ireland | 7.55 | 1.53 | 6.02 | 0.91 |
| UK | 10.39 | 1.42 | 8.97 | 3.50 |

There are some points to note in this table. The total and anthropogenic $PM_{2.5}$ levels for London are high compared with other areas. This is because the other population-weighted means reported represent an average across a mixture of urban and rural areas. Pollution in other large cities, e.g. Cardiff and Manchester, are encompassed in the reported concentrations for each country or region, e.g. Wales and Rest of England, respectively, and therefore appear lower, but may in fact be of a similar level.

It can also be seen from Table 3.1 that the concentration above 7 $\mu g/m^3$ is not simply a subtraction of 7 $\mu g/m^3$ from the total. This is because the summary concentration data presented are the population-weighted mean of the values in individual 1 km × 1 km grid squares. In some of these grid squares the annual mean $PM_{2.5}$ concentration was below 7 $\mu g/m^3$. The result of the subtraction of 7 $\mu g/m^3$ was therefore zero for all the grid squares with a concentration below this value, irrespective of the concentration.

It is worth noting that for all the calculations made in this report, we assumed an immediate reduction in pollution. Also, it is possible, and easy, to modify the methods described here to take account of ongoing pollution reduction strategies phased in over time. These were not considered in this report because, while they increase the complexity of the argument, they do not raise any fundamentally new conceptual issues.

# Chapter 4
# Results of the Impact Calculations

Except where stated otherwise, calculations of the impact of pollution reduction measures were based on the 2008 population including new births; the baseline population and life expectancies are shown in Table 4.1. Follow-up was for 106 years and a risk coefficient of 1.06 (6%) per 10 μg/m$^3$ $PM_{2.5}$ was used. The cessation lag distribution proposed and used by the US EPA (2004 and 2010) was used and results presented as the health impacts *per se* with no monetary valuation included.

**Table 4.1: Baseline 2008 population data (population rounded to the nearest 1000)**

| Country | Total population | Population aged 30 years and over | Life expectancy (years) | |
|---|---|---|---|---|
| | | | Males | Females |
| England | 51,465,000 | 32,116,000 | not used | not used |
| Wales | 2,990,000 | 1,893,000 | not used | not used |
| *England and Wales combined* | 54,455,000 | 34,008,000 | 78.17 | 82.12 |
| Scotland | 5,169,000 | 3,302,000 | 75.46 | 80.14 |
| Northern Ireland | 1,775,000 | 1,038,000 | 76.71 | 81.44 |
| UK | 61,399,000 | 38,348,000 | not used | not used |

## 4.1 Main results

Two questions have been posed to assess impact or benefits. The first, question (a), asks

> 'What are the benefits expressed as an effect on mortality of a sustained reduction in annual average air pollution across the UK by a small fixed amount, e.g. by 1 μg/m$^3$ $PM_{2.5}$?'

The answer to this first question demonstrates the effects of a small change in levels of anthropogenic particulate air pollution of the magnitude associated with policies for pollution reduction. The second, question (b), asks

> 'If anthropogenic air pollution in 2008 were to be removed and pollution sustained at low non-anthropogenic levels, what would be the benefits in terms of mortality?'

Unrealistic as it is that all human-made air pollution could be eliminated, answering this question has allowed us to explore the population dynamics of a sustained large reduction in annual average $PM_{2.5}$. Taking account of population dynamics provides a more correct and therefore more realistic assessment of the impact of reducing air pollution than is provided by calculating the burden of current air pollution and implying that this is the same as the benefits of removing it. In the context of the present report, it allows results from the two approaches to be described and compared. This comparison is discussed in Chapter 9.

This second question is answered both on the basis of removal of all anthropogenic $PM_{2.5}$ and reducing levels down to 7 µg/m³ as explained in Section 3.2. The life-years gained as a result of these reductions are presented in Table 4.2, while the impact on life expectancy is given in Table 4.3. For ease of comparison between the different countries, the last column of Table 4.2 gives the life-years gained per 100,000 population aged 30 years and over.

For a 1 µg/m³ reduction, these results show that the impacts per 100,000 population aged 30 years and over are similar across England and Wales, Scotland and Northern Ireland, and so illustrate that the full population impacts scale approximately on the basis of population size.

The same is not true of the removal of all anthropogenic $PM_{2.5}$ and removal down to 7 µg/m³; this is because the population-weighted mean concentrations of $PM_{2.5}$ being removed under these scenarios differ significantly by country. As an example, for England and Wales anthropogenic $PM_{2.5}$ is 9.46 µg/m³, whereas in Scotland it is 4.97 µg/m³; the impacts of the removal for these two populations are affected not only by the difference in population size but also by the difference in the level of pollution. Nonetheless, if this difference in population-weighted mean is taken into account, it is possible to scale the results from one country to another.

**Table 4.2: Life-years gained (rounded to the nearest 1000) over 106 years, by population, including new births, following specified reductions in $PM_{2.5}$. UK totals are aggregated from the individual results presented**

| Pollution reduction | Country | Population-weighted mean concentration | Life-years gained | Life-years gained per 100,000 people aged 30 years and over |
|---|---|---|---|---|
| 1 µg/m³ | England and Wales | 1 µg/m³ | 3,604,000 | 10,597 |
| | Scotland | 1 µg/m³ | 353,000 | 10,687 |
| | Northern Ireland | 1 µg/m³ | 128,000 | 12,302 |
| | **UK total** | **1 µg/m³** | **4,084,000** | **10,651** |
| All anthropogenic | England and Wales | 9.46 µg/m³ | 34,059,000 | 10,0151 |
| | Scotland | 4.97 µg/m³ | 1,754,000 | 53,113 |
| | Northern Ireland | 6.02 µg/m³ | 769,000 | 74,063 |
| | **UK total** | **8.97 µg/m³** | **36,582,000** | **95,394** |
| Removal down to 7 µg/m³ | England and Wales | 3.90 µg/m³ | 14,048,000 | 41,308 |
| | Scotland | 0.21 µg/m³ | 76,000 | 2,297 |
| | Northern Ireland | 0.91 µg/m³ | 116,000 | 11,179 |
| | **UK total** | **3.50 µg/m³** | **14,240,000** | **37,134** |

**Table 4.3: Increased life expectancy for UK populations following reductions in $PM_{2.5}$**

| Pollution reduction | Country | Population-weighted mean concentration | Increased life expectancy (days) for the 2008 birth cohort | |
|---|---|---|---|---|
| | | | Males | Females |
| 1 µg/m³ | England and Wales | 1 µg/m³ | 21 | 20 |
| | Scotland | 1 µg/m³ | 23 | 21 |
| | Northern Ireland | 1 µg/m³ | 22 | 21 |
| | UK* | 1 µg/m³ | 21 | 20 |
| All anthropogenic | England and Wales | 9.46 µg/m³ | 200 | 185 |
| | Scotland | 4.97 µg/m³ | 112 | 102 |
| | Northern Ireland | 6.02 µg/m³ | 131 | 124 |
| | UK* | 8.97 µg/m³ | 191 | 177 |
| Removal down to 7 µg/m³ | England and Wales | 3.90 µg/m³ | 82 | 76 |
| | Scotland | 0.21 µg/m³ | 5 | 4 |
| | Northern Ireland | 0.91 µg/m³ | 20 | 19 |
| | UK* | 3.50 µg/m³ | 74 | 69 |

* Calculated by weighting the England and Wales, Scotland and Northern Ireland results by the relevant birth cohort size.

Indeed, as one would expect, the size of the population-weighted mean concentration has a major impact on the results to the extent that, for a given population and set of baseline assumptions, the change in life-years scales almost exactly with the size of the population-weighted mean reduction, at least to within the rounding error. Thus, for example, in England and Wales, the estimated gains in life-years per 100,000 people aged 30 years and over are 10,597, 41,308 and 100,151 for population-weighted reductions of 1, 3.90 and 9.46 µg/m³ $PM_{2.5}$, respectively.

It is interesting to compare the results of total life-years, which are dependent on population size, with those for life expectancy, which are not. Here again, as for the life-years per 100,000 of the population, the differences between the different countries for removal of anthropogenic $PM_{2.5}$ are mostly determined by the difference in level of pollution; and again, results for a 1 µg/m³ change in $PM_{2.5}$ are similar across different populations, whether differentiated by country or by sex.

There are also variations in anthropogenic particulate air pollution between and within England and Wales as indicated by Table 3.1 in Section 3.2.2. In particular, annual average anthropogenic $PM_{2.5}$ in inner London is nearly 50% higher than in England and Wales as a whole, implying an improvement of life expectancy, i.e. the average across 2008 births there of about nine months, rather than the England and Wales average of approximately six months. There also will necessarily be parts of England and Wales with a lower than six months effect on life expectancy. This is an improvement in the life expectancy across each 2008 birth cohort; it is not possible to determine from these results how this would be distributed between individuals within the cohort.

## 4.2 Sensitivity analyses – 'impact' or 'benefit' questions

### 4.2.1 Coefficient

The results in Section 4.1 above are based on the central estimate of the effect of air pollution on all-cause mortality expressed as a risk coefficient of 1.06 per 10 μg/m³ increase in $PM_{2.5}$. In the 2009 report, COMEAP also recommended use of a plausibility interval of 1.01–1.12 for sensitivity analysis and that a wider interval of 1.00–1.15 be included in any report on quantification of risk. The results for a reduction of all anthropogenic $PM_{2.5}$ (9.46 μg/m³) in England and Wales, using different coefficients are provided in Table 4.4.

**Table 4.4: Effect of varying the coefficient on the estimation of the health impact over 106 years of a reduction of all anthropogenic $PM_{2.5}$ (9.46 μg/m³) in England and Wales**

| Coefficient | | Life-years gained | Increased life expectancy (days) for the 2008 birth cohort | |
|---|---|---|---|---|
| | | | Males | Females |
| 1.00 | (0%) | 0 | 0 | 0 |
| 1.01 | (1%) | 5,820,000 | 34 | 32 |
| 1.06 | (6%) | 34,059,000 | 200 | 185 |
| 1.12 | (12%) | 66,192,000 | 390 | 361 |
| 1.15 | (15%) | 81,601,000 | 481 | 445 |

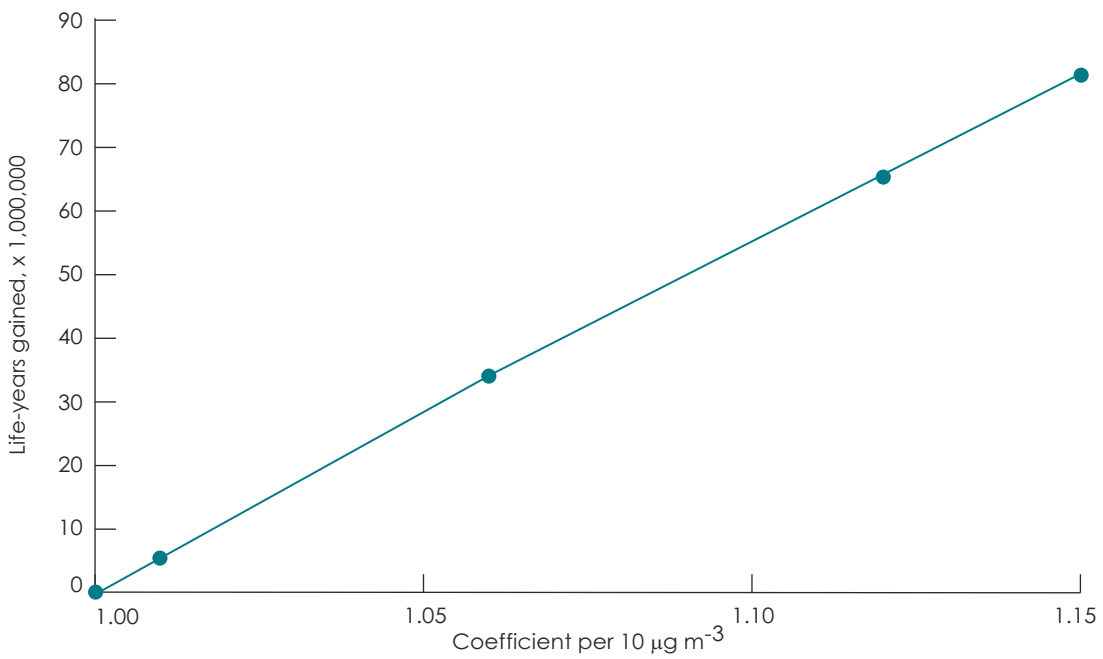


**Figure 4.1: Gain in life-years for the population of England and Wales including new births following removal of anthropogenic $PM_{2.5}$ depending on the risk coefficient for deaths from all-causes**

Figure 4.1 shows the gain in life-years of this reduction, for the population of England and Wales including new births, as the risk coefficient increases. As can be seen this is an approximately linear relationship. In fact it is actually very slightly curved (convex upwards), something which is also clear from Table 4.4, which shows some non-linearity in the relationship with percentage change of risk coefficient. For example, the life-years for the 12% coefficient is less than double the life-years for the 6% coefficient. This is because (see Section 3.1.3) the proportionality is multiplicative, or log-linear, on relative risks, and linear scaling in terms of the risk coefficient as a percentage is an approximation to that. The difference is of little importance unless results from very different coefficients are compared, e.g. 1.01 and 1.15 in Table 4.4.

### 4.2.2 Cessation lag

As noted in Section 3.1.4, it was agreed that for the purposes of this report the cessation lag distribution used by the US EPA would be applied here for calculation of the mortality impacts but that the effect of making different assumptions regarding cessation lags would be considered. To illustrate the effect of varying the cessation lag assumptions, the health impacts of a 1 $\mu g/m^3$ reduction in $PM_{2.5}$ and removal of all anthropogenic $PM_{2.5}$ on the population of England and Wales have been assessed using no lag and 5, 10, 20 and 30 year phased in lags as a sensitivity analysis. The results are presented in Table 4.5 and Figure 4.2 illustrates the effect for the removal of anthropogenic $PM_{2.5}$.

For both reductions the 30 year phased lag decreases the result by around 15% compared with the no-lag result, as can be seen in Figure 4.2 showing life-years gained with increasing time over which the lag is phased in. Estimates for other lags can be derived from this linear relationship, e.g. using a 25 year phased-in lag would give results of approximately 3,290,000 life-years gained for a 1 $\mu g/m^3$ $PM_{2.5}$ reduction and 31,100,000 life-years gained for the removal of anthropogenic $PM_{2.5}$.

It is worth noting that the use of different lag times has no effect on the life expectancy of the 2008 birth cohort, as the hazard rate reduction is only applied to this population at the age of 30 years upwards, by which time all the lags assessed here are fully implemented.

The impact of different lags on the shape of the curves of life-years gained per year and deaths fewer per year for a 1 $\mu g/m^3$ reduction is illustrated in Figures 4.3 and 4.4.

**Table 4.5: Health impact over 106 years of reductions in $PM_{2.5}$ for the population of England and Wales using different lag times**

| Coefficient | Life-years gained | |
|---|---|---|
| | 1 µg/m³ | All anthropogenic (9.46 µg/m³) |
| No lag | 3,730,000 | 35,266,000 |
| US EPA lag | 3,604,000 | 34,059,000 |
| 5 year phased lag | 3,649,000 | 34,504,000 |
| 10 year phased lag | 3,565,000 | 33,706,000 |
| 20 year phased lag | 3,387,000 | 32,019,000 |
| 30 year phased lag | 3,198,000 | 30,221,000 |

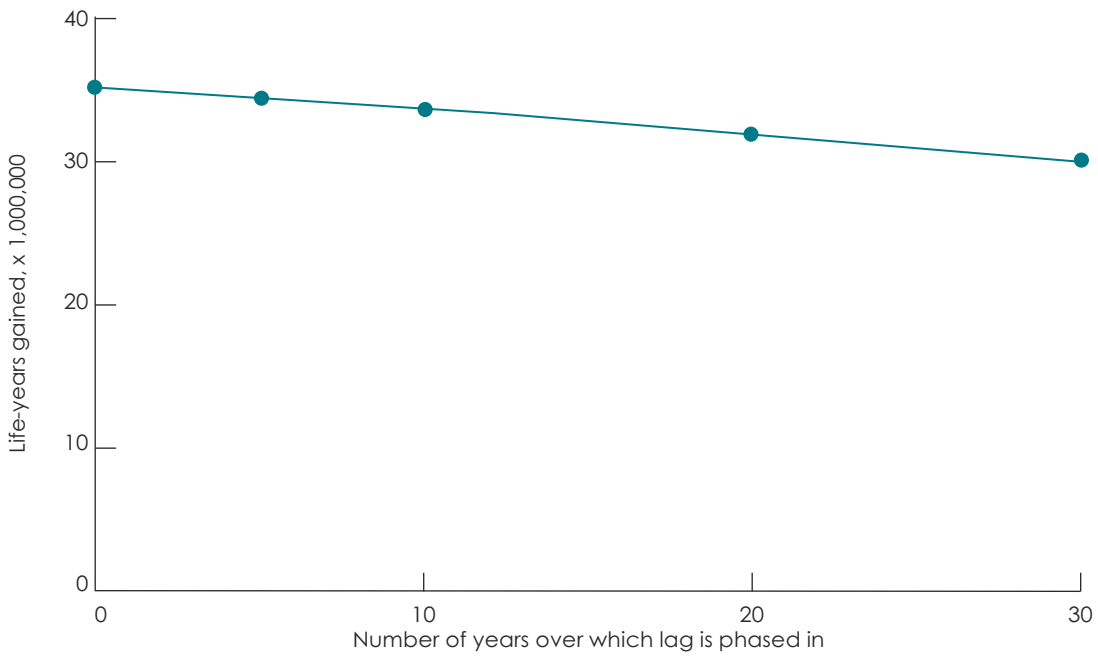


**Figure 4.2: Gain in life-years for the population of England and Wales including new births following a reduction of all anthropogenic $PM_{2.5}$ (9.46 µg/m³) with different phased in lag times**

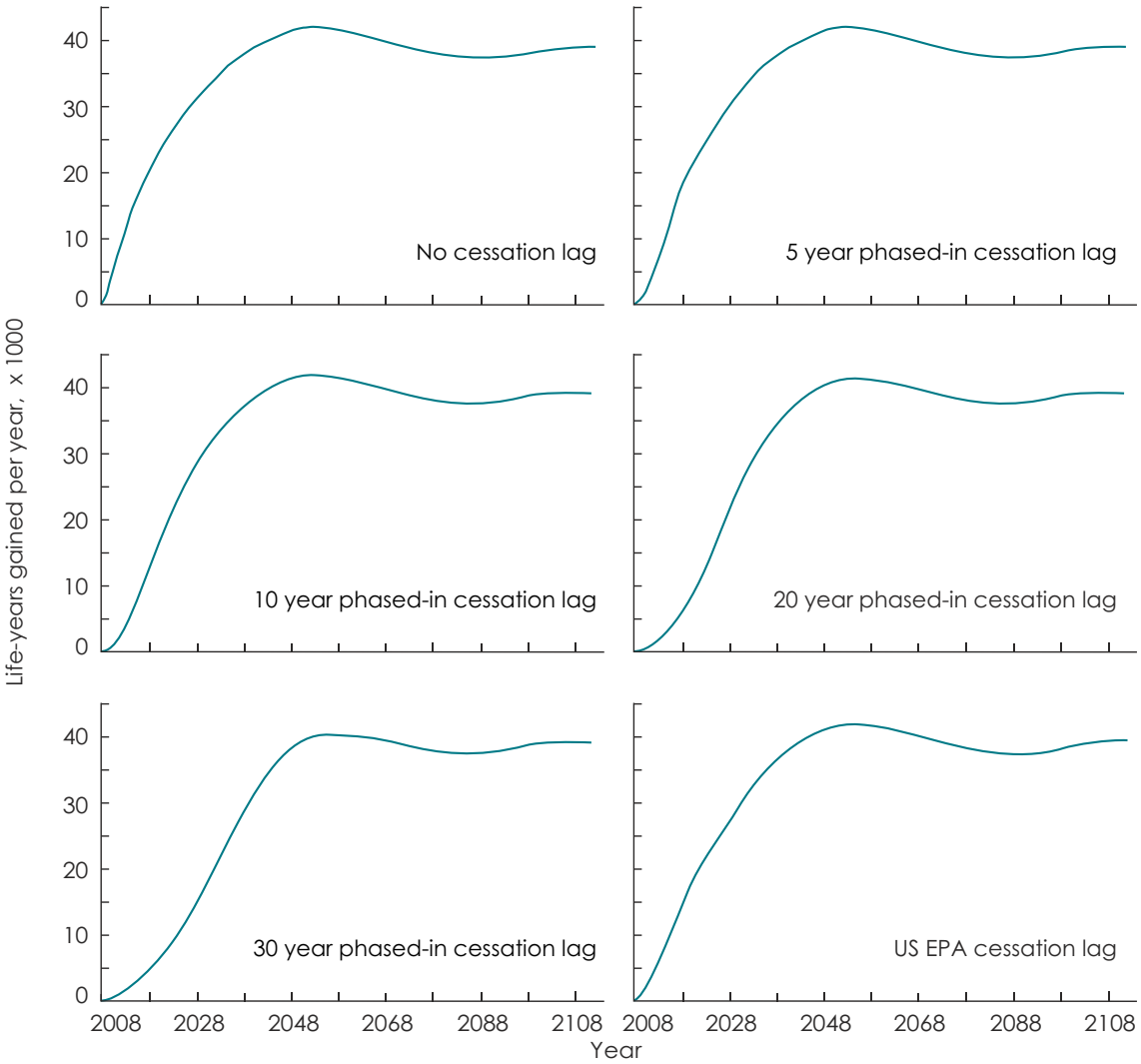


**Figure 4.3: Life-years gained per year for the population of England and Wales, including new births, following a 1 µg/m³ reduction in $PM_{2.5}$ with different lag times**

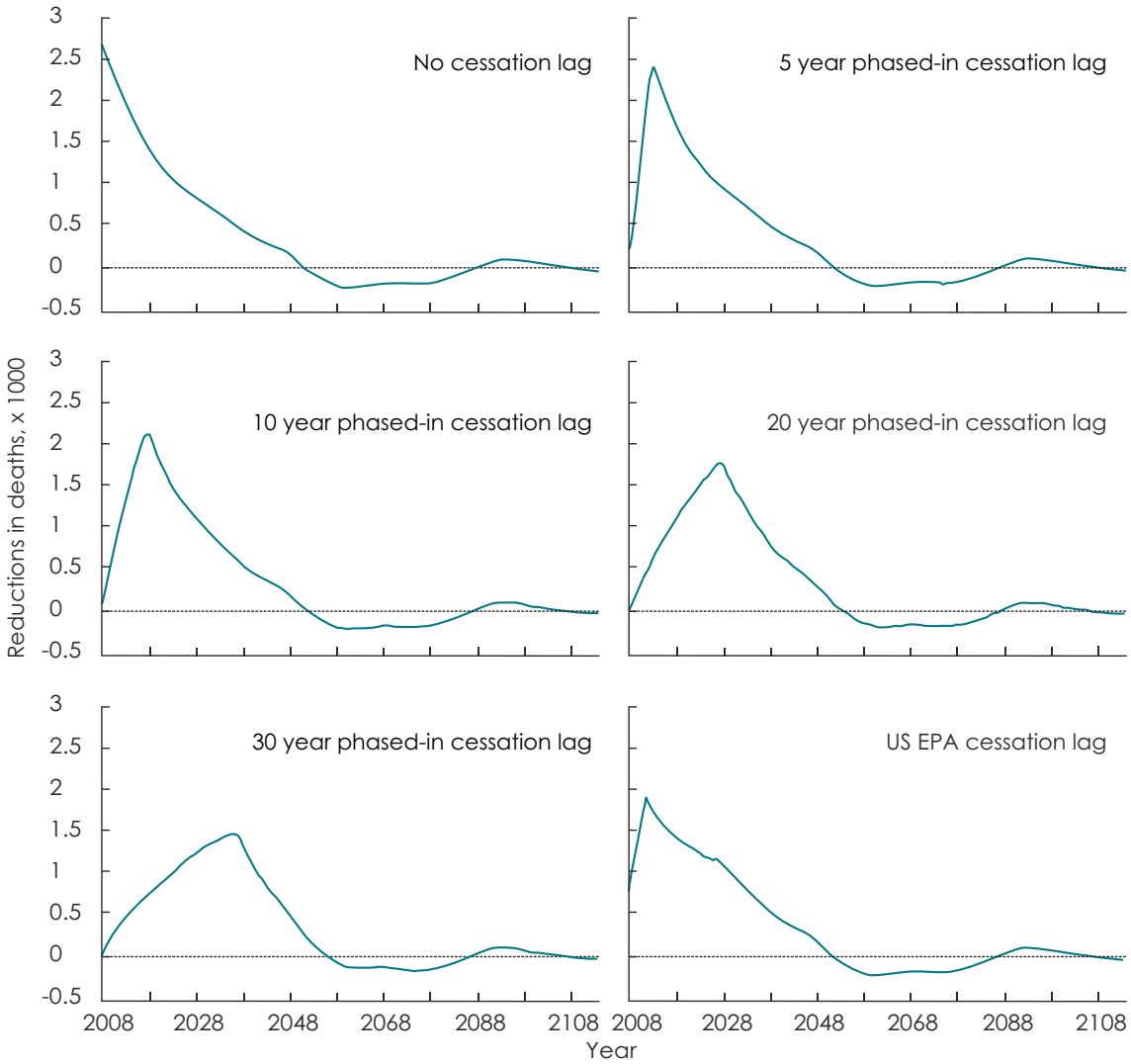


**Figure 4.4: Number of deaths fewer per year for the population of England and Wales, including new births, following a 1 µg/m³ reduction in $PM_{2.5}$ with different lag times**

### 4.2.3 Weighting factors

The effect of discounting on the valuation of health impacts following removal of all anthropogenic $PM_{2.5}$ (9.46 µg/m$^3$) in England and Wales was assessed and the results are presented in Table 4.6. In this case a life year has been given a nominal monetary value of 1 as this report does not cover issues around monetary valuation. However, the numbers can easily be multiplied by an appropriate monetary value if needed. The total life-years gained *per se* in this scenario for the population, including new births, is 34,059,000.

Part of the interest in discounting was to evaluate the combined effect of any discounting and cessation lag. Table 4.6 shows the results for the US EPA cessation lag. Figures 4.5 and 4.6 give the results for other lags and illustrate that assumptions regarding discounting have a much greater effect than those regarding lags, within the range of values explored here.

**Table 4.6: Effect of discounting rate on the value of life-years gained over 106 years following a reduction of all anthropogenic $PM_{2.5}$ (9.46 µg/m$^3$) in England and Wales**

| Discount rate | Value of life-years gained |
|---|---|
| 0% | 34,059,000 |
| 1.5% | 15,173,000 |
| 3% | 7,821,000 |
| 6% | 2,912,000 |

These data have been included to illustrate the effect of discounting on the results of the impact calculations and as a comparison of the importance of discounting relative to other factors which affect the mortality calculation; this example is not intended as a recommendation of appropriate monetary values or discount rates.

It can be seen that the use of a discount rate substantially changes the result, reducing – sometimes markedly – the apparent benefit of the pollution reduction. The current recommendations of the Interdepartmental Group on Costs and Benefits (IGCB), who oversee the economic analysis of the health impacts of air quality policies, have a net effect approximately equivalent to a discount rate of 1.5% or less (IGCB, 2007).

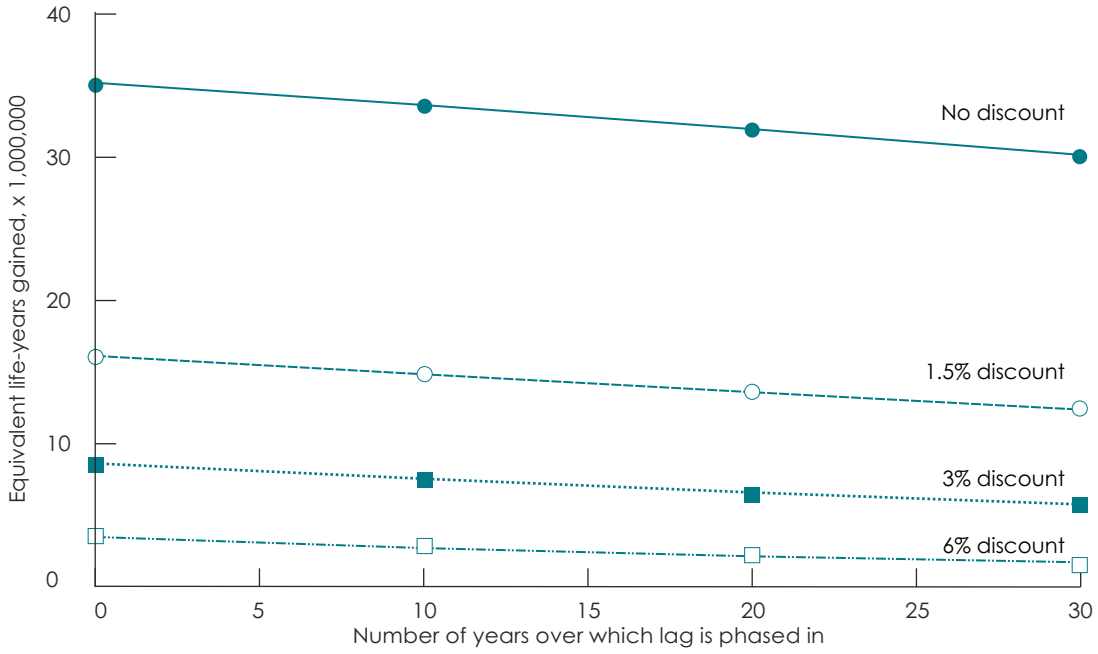


**Figure 4.5: Effect of phased-in cessation lag at different discount rates on the value of the impact of a reduction of all anthropogenic $PM_{2.5}$ (9.46 µg/m³) in England and Wales**

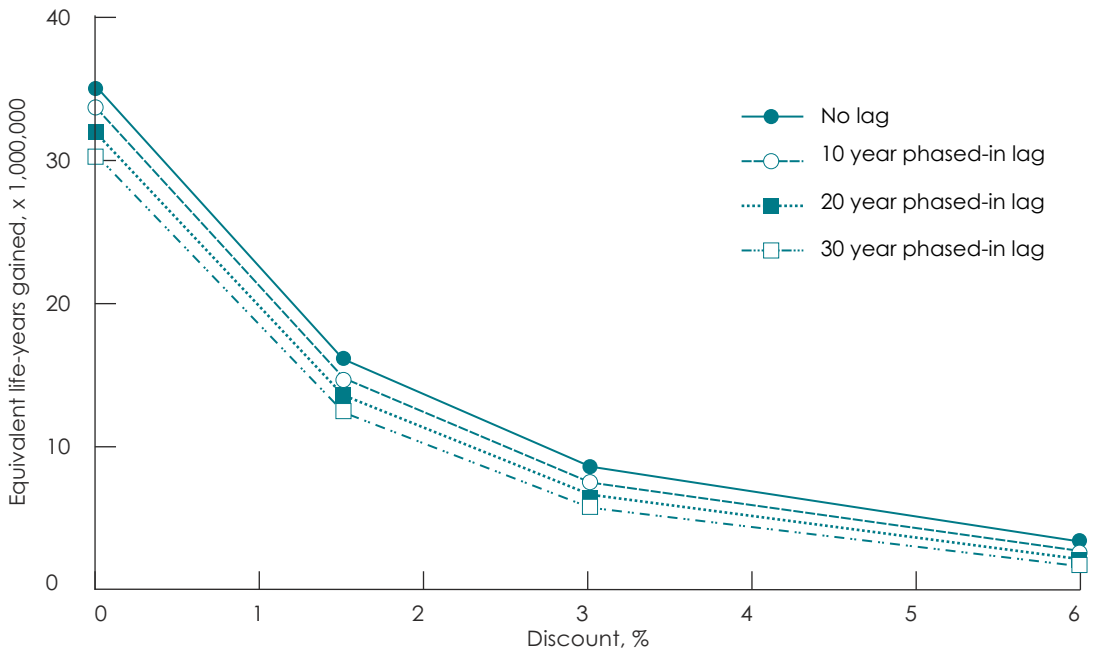


**Figure 4.6: Effect of discount rates with different cessation lags on the value of the health impact of a reduction of all anthropogenic $PM_{2.5}$ (9.46 µg/m³) in England and Wales**

# Chapter 5
# Discussion of Impact Calculation

## 5.1 Validity of the metrics used

Questions relating to the impacts and benefits of reducing or removing anthropogenic particulate air pollution are relevant when considering future policy options.

Sustained reductions in air pollution lead to sustained reductions in age-specific death rates (at ages 30 years or more), and these reduced death rates affect both the size and the age distribution of the population. We have gone to considerable lengths to take account of and explain the resultant population dynamics, and what they imply for representing the mortality impacts of a sustained reduction in pollution.

### 5.1.1 Total population survival

Briefly, the impacts of sustained pollution reduction are best represented in terms of total population survival, as measured in total years of life lived by the population, because the gains expressed in terms of life-years lived continue to accumulate over time.

The 'life year' is not, in our view, a difficult concept to grasp – we celebrate a year of life lived each time we celebrate a birthday. The metric is appropriate for policy evaluation and cost–benefit analysis, in that monetary values can be given to life-years and incorporated into the analysis. The concept is often extended nowadays to take account not only of survival, but of health and well-being also, using concepts such as Quality Adjusted Life-Year (QALY) measurement (National Institute for Health and Clinical Excellence, 2010).

Once these years are aggregated across a population, it can be difficult to make sense of the resulting numbers, because we have nothing with which to compare them and thus cannot easily interpret them as 'large' or 'small'. Nonetheless, this approach is useful for the evaluation of policy options.

### 5.1.2 Life expectancy at birth

Life expectancy at birth is a measure that depends only on death rates, not on population size. Here, its main use is in communicating the mortality benefits of a sustained reduction in air pollution, and as such it can be helpful in comparing the mortality benefits of reducing different kinds of risks to mortality. Also, it is relatively easy to calculate.

Other than in its communication uses, it is of limited use for policy analysis, because it captures the impacts in new births only, not in the full current population of all ages.

We sound, however, a word of caution about interpretation. *Life expectancy at birth* describes the length of time that someone would expect to survive if they experienced exactly the same age-specific risks of death as the population whose life expectancy is being described. As such, it is an average value, in that the age-specific death rates it uses reflect on average the experience of the population as a whole whose life expectancy is being described. In the present context, the population whose life expectancy is being described is the population of England and Wales, or of Scotland, or of Northern Ireland, or sub-populations of these. In each case, the population as a whole is stratified by factors such as sex, which is known to affect life expectancy.

The same is true of *changes* in life expectancy – by definition this is a concept that describes the average experience of the population, under different scenarios with different death rates. It is a mistake to interpret the average experience as applying to individuals, or even as being 'typical'. It follows that assuming that a change (increase or decrease) in life expectancy means that everybody in the population experiences the same actual change (increase or decrease) in life-years, is also a mistake.

### 5.1.3 Deaths per year

The change in the number of deaths in a specified calendar year is also a valid measure, in that by using life tables it can be calculated correctly, taking account of population dynamics, as we have shown in Chapter 2. However, as shown there, information on change in the number of deaths in a year has limited usefulness in policy analysis because the benefits, in terms of reduced deaths following a reduction in pollution, are not sustained over time: everybody dies eventually and reduced pollution cannot affect the fact of death, only its timing (age at death). So a figure of 'annual' deaths may mislead if these changes are not understood and taken into account.

However, the need for metrics which can illustrate the burden of air pollution in a way that is more immediately accessible and readily interpreted than population survival aggregated over time across the population has been highlighted (e.g. House of Commons Environmental Audit Committee report on Air Pollution, 2010). We address this issue in Part II of this report, explaining the metrics we have used and presenting calculations of the current burden of particulate air pollution. We also discuss other possible metrics, and some of the misconceptions that have sometimes led to inappropriate inferences being drawn from the use of such metrics.

## 5.2 Uncertainties in the results

### 5.2.1 Risk coefficient

As can be seen from the sensitivity analysis on the risk coefficient, there is quite a difference in the results depending on the value used, because results are almost proportional to the risk coefficient. The central estimate for England and Wales is 34.1 million life-years gained (36.5 million for the UK) and an increase in life expectancy of about 6 months. The uncertainties are described in detail in COMEAP (2009), but the range of the result for the 75% plausibility interval is a gain of 5.8–66.2 million life-years gained and an increase in life expectancy of one month to one year across the population of England and Wales following removal all anthropogenic $PM_{2.5}$. This range is greater, 0–81.6 million life-years gains and 0–15 months increased life expectancy for the widest interval, 0–15%, of the risk coefficient.

Note that the plausibility distribution of COMEAP (2009) clusters round the central value of 6%, i.e. is not distributed equally across the ranges quoted here; it is important not to treat all values within the range as equally likely. To limit the chances of such a misinterpretation, COMEAP (2009) recommended that the full plausibility distribution be used.

### 5.2.2 Pollution reduction

The reduction in $PM_{2.5}$ included in the calculations clearly and inevitably affects the results. Uncertainty increases for the calculation of the impact of removing anthropogenic pollution as the proposed reduction leads to levels lower than 7 μg/m³ (the lowest annual average concentration recorded in the cities studied by Pope *et al*, 2002) i.e. extending beyond the range of the data used to derive the coefficient. Estimates shown in Section 4.1 of this report indicate that more than 60% of the total benefits for removing anthropogenic pollution, expressed as life-years gained or increased life expectancy, derive from concentrations below 7 μg/m³.

To some extent this increased uncertainty is already signalled by the Pope *et al* (2002) results, where the confidence intervals splay outwards as the data become more sparse as we approach 7 μg/m³. If we assume that the central estimate line carries on without changing slope as we pass below 7 μg/m³, we should accept that the confidence intervals will also continue to splay, in effect indicating the greater uncertainty at these lower annual average concentrations.

There are also some uncertainties in the modelling of annual average $PM_{2.5}$ across the UK, and in the attribution of that as anthropogenic or not. But both the estimated risk coefficient and the modelled $PM_{2.5}$ annual average concentrations come from a strong base of evidence, and we are confident that the estimate of the effect on mortality and associated estimates of uncertainties are of the right order of magnitude.

## 5.3 Impact or benefit of reductions in anthropogenic $PM_{2.5}$ – methodological issues

Estimating the mortality impact or benefit of reductions in annual average anthropogenic $PM_{2.5}$ raises a number of issues.

### 5.3.1 Proportional scaling of results

Changes (i) in population-weighted mean concentration, given the same risk coefficient, or (ii) in risk coefficient, given the same population-weighted mean concentration, raise identical issues about whether changes in the impacts are proportional, because both sets of changes express themselves via percentage changes in age-specific mortality hazards. Indeed, the determining factor is the product of risk coefficient and population-weighted mean concentration. Impact results, in terms of life-years and life expectancy, vary almost proportionally with either factor, and so with the product of the two together. This result is exact when relative risks are scaled multiplicatively (i.e. linearly on the logarithmic scale), as described in Section 3.1.3, and we have used logarithmic (multiplicative) scaling in this report as this may be important for sensitivity analyses using larger coefficients or analyses using large concentration changes.

A frequently used approximation is to work linearly in percentage change of risk coefficient, and then the linear scaling of results is approximate. Within the ranges of risk coefficient and

population-weighted mean concentration of interest to policy making in the UK, the non-linearity is very small compared with other sources of uncertainty within the whole impact assessment, and so results from a change of 1 μg/m$^3$ in $PM_{2.5}$ with a risk coefficient of 1.06 can be scaled to give results for other population-weighted mean concentrations, and/or other values of the risk coefficient. This can simplify both analysis and reporting of the main results for straightforward applications.

### 5.3.2 Follow-up for how many years?

The life-table results in the present report are based on a follow-up of the population, and its mortality, over a period of 106 years. This length of follow-up was chosen so that follow-up, of the population alive in 2008, would be long enough to allow all that population to die.

However, there is something arbitrary about the length of follow-up, and results are sensitive to it. For example, under sustained pollution reduction, and with new births included in the target population, the gain in deaths postponed increases annually, though by a decreasing amount, for between 40 and 50 years, and then stabilises (see Figure 2.6 in Section 2.5). Longer follow-up has little or no impact on difference in deaths between the baseline and the reduced pollution scenarios. The situation is different, however, for the total life-years gained as a result of the reduction in anthropogenic $PM_{2.5}$ pollution. Here, as is also shown in Section 2.5, the difference between baseline and reduced pollution scenarios stabilises to a non-trivial number of additional life-years gained annually.

The implication is enormous - there is no limit to the public health benefit of a sustained pollution reduction when that benefit is expressed in terms of life-years gained across the population as a whole indefinitely into the future.

However, the benefits appear to be no longer without limit if the value of future life-years is discounted (discounting is considered further in Section 5.3.3 below). Under those circumstances, the increase in life-years in the later years of the analysis eventually carries little or no weight, i.e. discounting imposes a *de facto* time limit on the analysis. Exactly how early this happens depends on the rate of discounting and whether there is also an uplift to account for increases in monetary values due to economic growth or inflation. However, it will happen at some point.

It is interesting nevertheless to recognise that it is only by discounting the future gains in life-years that these gains can be limited. In terms of health *per se* they are limitless.

### 5.3.3 Weighting factors/discounting

Results in Chapter 4 show that applying weighting factors to future life-years, as is done when discounting is applied, has a major impact on the results. For example, applying a discount rate of 1.5%, which might be considered modest, reduces the estimated gains in life-years over a 106 year period by more than 50%, i.e. its impact on the final results is the same as would result from a no-discount analysis but with the risk coefficient reduced from 6% (per 10 μg/m$^3$ $PM_{2.5}$) to 3%.

Use of a high discount rate, e.g. 6%, reduces the estimated benefits to less than 10% of their no-discount value.

We have not investigated to what extent these reductions are sensitive to the length of mortality follow-up via the life tables.

The issues of discounting – whether or not to do it, and if so at what rate of discount – are complex ones, involving economic issues, and ethical issues of inter-generational justice. We do not wish or intend to enter here into discussion of how they might be resolved. But we do think it important to highlight that whatever judgements are made about discount rates will have relatively large impacts on the answers, and that this in turn highlights the importance of transparency in who makes such judgements, and on what basis.

### 5.3.4 Cessation lag

Cessation lag is the lag time between the reduction in pollution and consequent reduction in mortality rate[7]. As noted earlier, this is a difficult issue because there is limited knowledge to inform the choice of cessation lag; such evidence as there is, and much of it is indirect, is summarised in a supporting paper (Walton, 2010).

We have used a pattern of cessation lag proposed by the US EPA because it broadly reflected the views of the COMEAP second Quantification of Air pollution RisKs (QUARK II) sub-group, as endorsed by COMEAP, and it seemed an advantage not to develop a different pattern unless there were good grounds for doing so. We were aware, however, that other patterns of cessation lag could reasonably have been selected, and so we also looked at some results using a range of different lag periods. The cessation lags examined all had the same simple structure: following reduction in annual average anthropogenic $PM_{2.5}$, the resultant reductions in age-specific death rates were phased in gradually, from year 1 until a set time later; in our analyses full risk reduction was attained respectively at 5, 10, 20 or 30 years after pollution reduction. This range of lags encompasses the suggestions for possible lag structures laid out in the supporting paper (Walton, 2010).

This sensitivity analysis gave two interesting results. Firstly, assuming different patterns of cessation lag from among those studied here did not lead to large differences in results; for example, the reduction in life-years gained from no lag to a lag phased in over 30 years was less than 15%. To some extent this reflects the 106 year length of the life-table analysis – follow-up of the population over such a long period reduces the relative contribution of what happens in the years immediately following reduced $PM_{2.5}$ concentrations. For this reason, there will be more difference between lags for analysis of policies which only change pollution for a short time – follow-up will still be for 106 years but there will only be a marked difference between scenarios in the early period when lags are also phasing in at different rates. This is somewhat similar to the idea, not investigated here, that the benefits to younger members of the starting population are likely to be insensitive to lag, while the older part of the population is likely to be relatively more affected by different assumptions. The reason for this is simply because for younger people there is longer for the time-course of the analysis to work its way through and therefore the effects in the first 30 years (for a 30 year phased-in lag) after a reduction in pollution are diluted by averaging over a longer remaining life-time.

[7] The cessation lag is different to the onset lag which is the time from first exposure and the occurrence of the effect.

Secondly, for the simple phased-in lags of the sensitivity analyses, results are almost linear in respect to the length of lag. Thus it is possible to estimate values by interpolation and extrapolation. The total impact calculated using the US EPA lag is equivalent, with a long follow-up via the life tables, to that with a simple lag phased in over about seven years using the approximate linearity of results.

In looking at the combined effect of cessation lag and discounting, it can be seen that the results in terms of value are strongly sensitive to any discounting applied but not greatly affected by the choice of lag structure, given the follow-up over 100+ years. However, discounting puts relatively more emphasis on events in the near future compared with the more distant future, and so will increase rather than decrease the importance of assumptions about cessation lag. For a 9.46 $\mu g/m^3$ reduction, the reduction in life-years gained from no lag to a lag phased in over 30 years was around 15% with no discounting but around 30% for a 1.5% discount rate.

# Part II

# Questions about Burden of Air Pollution

# Chapter 6
# Basic Concepts: Burden Questions

## 6.1 Comment on the questions

### 6.1.1 Why does COMEAP study the mortality burden of outdoor air pollution?

Part I of this report has considered questions relating to the impacts or benefits of reducing or removing anthropogenic fine particulate air pollution. These are relevant when considering future policy options – they help in predicting the mortality benefits of reduced air pollution.

It might seem that this is sufficient for a report such as this, because it deals with the effects of policy changes, and COMEAP has in the past focused on impact questions such as these – it has seen its role as providing methods and results which will inform the development of health protective policy, and questions such as these were the relevant ones to answer.

Why then consider burden of disease, which we do not use (or advise using) for assessing the benefits of protective policy?

There are two main reasons why we have considered the mortality burden of outdoor air pollution. One is that this is an issue which has now become a topic of general discussion, in the UK, Europe and elsewhere. The underlying purpose seems to be to assess the significance of outdoor air pollution as a public health problem, so that by highlighting its importance, the impetus for action can be increased. A variant of this is to see the relative ranking of various factors influencing health and mortality; such a ranking is implicit in the World Health Organization (WHO) Global Burden of Disease (GBD) project (WHO, 2010). Although COMEAP sees its main role as providing assessments, methods, tools and opinion to inform the development of policy, as in Part I, it also has a public education role, and we do not wish to stand back from this debate on the health, disease and mortality burden of air pollution.

The second reason is in some way linked, and concerns the ease of interpretation of the metrics we use in discussing the mortality effects of pollution reductions. Some commentators consider that total population survival, life-years and life expectancy are difficult to understand; the recent report on air pollution of the House of Commons Environmental Audit Committee (2010) has asked for use of number of deaths, which it considers to be a more intuitive measure, and more easily understood by the general public.

We have seen in Part I that there are serious problems with using the number of deaths as a metric in the context of sustained reductions of particulate air pollution. This is because the resultant sustained reductions in death rates lead to people living longer and, if no other changes occur, to populations which are on average older and larger (i.e. include more people).

The most relevant metrics for expressing these gains in population survival are total survival time of the population (in years of life gained) and life expectancy of a birth cohort, because these measures show sustained gains from reduced death rates. We have shown in Part I that the annual number of deaths is not an appropriate metric because, although there is some evidence of a benefit in that lower death rates lead to lower annual numbers of deaths initially, this gain is not sustained over time because of the changes in population size and age, and because everybody dies eventually. This works against use of the number of deaths in the context of sustained pollution reductions, no matter how easy it is to understand the concept of numbers of deaths.

The annual number of deaths is, however, the way in which the burden of outdoor air pollution is typically described, and so a second reason why we address the question of burden is to help to see if there is a legitimate use for this index. In this section, as well as explaining the metrics we have used and presenting calculations of the current burden of particulate air pollution, we also discuss other possible metrics, and some of the misconceptions that have sometimes led to inappropriate inferences being drawn from the use of such metrics.

### 6.1.2 The 'burden' question

We begin with some discussion of what we have called the question of air pollution burden. In Section 1.1 we introduced this by asking a non-specific general question: 'What is the effect of air pollution on mortality in the UK today?', and we clarified that: (i) we are interested in anthropogenic air pollution and (ii) we are treating 'today' as the year 2008 because that is the most recent year for which we have relevant data.

#### *Relationships in time between exposure to air pollution and risks of mortality*

However, these clarifications are not sufficient to give us a clearly formulated question that can be answered unambiguously. This is because the relationship in time between exposure to air pollution and age-specific risks of mortality is unknown, and may be complex. That relationship has two main dimensions:

a The first of these relates to how, in a temporal sense, particulate air pollution contributes to the development or acceleration of the chronic diseases (cardio-respiratory and lung cancer) which long-term exposure to air pollution has been shown to affect. This is conventionally described as *latency* or *onset lag*.

b The second relates to the distribution over time of how age-specific risks of mortality change following sustained reduction in air pollution. This is called *cessation lag* and has been discussed already in Part I.

Some things are known about air pollution, mortality, latency and cessation lag, but a lot is unknown. It is well established that there is some immediate effect on death rates in the days immediately following higher or lower air pollution levels; these are the effects detected by time-series studies. However, most of the mortality effects of air pollution take longer to occur; these are the effects detected by cohort studies such as the American Cohort Society (ACS) study (Pope *et al*, 2002). As explained in COMEAP (2009), cohort studies do not tell us directly how quickly the risks in mortality change following changes in air pollution, or indeed to what extent pollution from many years ago rather than in recent years is responsible for increased mortality now. This is because cohort studies principally examine differences in mortality

between cities with different levels of annual average pollution; they have limited information about changes over time within cities.

### *Implications for the question of burden*

Latency relates to how, over time, disease develops in response to air pollution and other factors. Cessation lag relates to how people recover following reduction in, or cessation of, exposure to air pollution. We will not discuss in detail the similarities and differences between the two concepts but we note that they are different, and that both aspects complicate answering an apparently simple question such as 'What are the mortality effects of anthropogenic air pollution in 2008?'. In fact, the question is not well formulated because it can be interpreted to mean either of two things, depending on whether we focus on mortality in 2008, or on pollution in 2008.

**Focus on mortality in 2008 leads us to ask:** 'What is the effect on mortality in 2008 of anthropogenic air pollution regardless of when that air pollution occurred – in 2008, or in any of the previous years where exposure to air pollution affected mortality in 2008?'

In this formulation the focus is on *current mortality*, but we must look backwards, in principle many years, to the air pollution levels that contribute to it. Consequently, in practice, any estimated burden will reflect exposure to *both* 2008 and earlier levels of air pollution. Focus on effects of pollution on mortality in a particular year is what is usually understood in discussions about mortality burden; and for purposes of comparison with other work we follow that convention and focus on versions of that question.

**Focus on pollution in 2008 leads us to ask:** 'What is the effect of anthropogenic air pollution in 2008 on mortality, regardless of when that mortality occurs – in 2008, or in any future year thereafter whose mortality patterns are affected by 2008 pollution?'

In this formulation the focus is on *current pollution* but we must look forwards, in principle many years, to assess its effects on mortality; any estimated burden will reflect impacts on mortality in *both* 2008 and subsequent (future) years. It is clear that, at least in principle, these two questions may well give different answers.

### *Re-formulation of the burden question in terms of population and death rates*

Our proposed way of dealing with this complexity is to do what others do, and re-formulate the burden question in a simple way that does not involve any complex calculations to take account of latency or cessation lag. Like others, in addressing this burden question we have calculated the mortality difference between two scenarios which have the same population in 2008 but death rates that differ in 2008:

a In one (baseline) scenario, these are the observed age-specific death rates in 2008 (which were influenced by pollution).

b In the other (alternative) scenario, these age-specific deaths rates are reduced by an amount attributable to 2008 levels of anthropogenic particulate air pollution.

Part II of this report is concerned principally with investigating, reporting and discussing the mortality implications of this difference in 2008 death rates. Some additional assumptions about future death rates are needed when we consider the total population survival time; we

discuss these later (Section 6.2.2). Part II also includes a calculation of the difference in life expectancy of the 2008 birth cohort attributable to anthropogenic particulate air pollution at 2008 levels; this does not require any assumptions about the 2008 population, but does require assumptions about death rates in the future. These are also described later (Section 6.2.1).

### *Implications of the re-formulation*

Re-formulating the burden question in this simple way allows for some simple calculations and answers, in terms of mortality burden in 2008, of the kind that are done by others. It does not, however, remove of the complexity in terms of latency and cessation lag, because these issues now appear in how the answers we obtained relate to anthropogenic pollution in 2008 and earlier years. We discuss these issues in Section 8.4. (The wider discussion of the mortality burden of air pollution generally ignores the issues.)

a Briefly, our results would be easy to interpret in terms of air pollution, if the full effects of air pollution on mortality were immediate, which we know is not the case.

b We think that the comparison of the scenarios specified above also can be interpreted as the effect of past and current air pollution on mortality in 2008, i.e. taking account of latency and cessation, if we assume that pollution in previous years was similar to levels in 2008, and we ignore any effects of past pollution on the size and age structure of the population in 2008. We know, however, that in real populations, past pollution does affect population size and age structure.

Either way, however, in order to answer the apparently simple burden question, we need to make some simplifying assumptions that ignore some of what we know about time lags, or about how sustained differences in death rates affect population dynamics. These issues are discussed in Section 8.4.

The formulation we have adopted in terms of 2008 population and death rates largely allows the two questions – the effect on mortality in 2008, and the effect of pollution in 2008 (under an assumption of no latency and no cessation lag) – to coincide in that death rates in 2008, and in 2008 only, differ between scenarios for both questions. This is perhaps a complicated way of saying that the usual calculations of burden ignore the issues of latency and cessation lag and so do not distinguish between the effect on mortality in 2008 and the effect of pollution in 2008.

## 6.2 Burden of mortality

In this section we discuss the various metrics which are, or can be, used to express the mortality burden of air pollution, understood as the differences between two scenarios ('baseline' (2008 levels of pollution) and 'alternative' (non-anthropogenic levels of pollution)) which differ only in respect of their death rates in 2008, as described in Section 6.1.

As we saw in Part I, there are several ways in which the mortality impacts of different death rates can be expressed – in terms of numbers of deaths, total population survival or loss of life (expressed in life-years lived), and life expectancy. All three approaches are meaningful in describing the burden of pollution, *provided that they are interpreted – as they are intended to be interpreted – as measures of population aggregate or average effects*. It is *not* appropriate to use these

results to express how the burden is distributed across individuals in the population. This is discussed more fully in Section 8.2; however, because of its importance, we emphasise it several times in this chapter also.

### 6.2.1 Deaths

The burden of air pollution on current mortality is usually discussed in terms of the number of additional or attributable deaths. This is easy to calculate, but can easily be misinterpreted and it needs, also, information about age at death in order to represent the mortality impact unambiguously.

Given a concentration–response function that relates concentrations of particulate air pollution to changes in population risk, expressed as changes in age-specific population mortality rates, the calculation of the number of additional deaths associated with the presence of air pollution becomes a simple matter of allocating a proportion of observed deaths as attributable to those changes, and hence to the pollution, provided that the issue of time lags can be taken into account or bypassed, and that the effects of past pollution on population size and age-structure can be ignored. As described above, this is most easily envisaged as comparing two scenarios in the same current population, the first with current mortality rates and the second with mortality rates that would apply in the absence of pollution at current levels. If the latter scenario had mortality rates 10% lower than the former, then we call that difference in death rates the attributable risk, and a rough estimate would be that 10% of deaths are 'attributable' to air pollution. More correctly, given a relative risk, RR, the attributable fraction, AR, is calculated by the formula

$$AR\% = 100 \times (RR - 1)/RR$$

Thus if we are interested in the proportion of deaths attributable to 10 μg/m$^3$ of $PM_{2.5}$ air pollution, and associate that with a relative risk of 1.06, then the fraction of the total number of deaths that is attributable to the air pollution is

$$100 \times 0.06/1.06 = 5.7\%$$

It should be noted that 'attributable' risk and 'attributable' deaths are epidemiological concepts relating to effects at the population level and, as such, they are an important part of calculating the total population mortality burden caused by current air pollution. Interpreted in this way, this simple calculation is meaningful in answering the total burden of mortality question as posed in this report, in that, taken in conjunction with the age at death, it gives one way of expressing the total mortality impact on the population as a whole – the overall population mortality burden is equivalent to the number of attributable deaths, taking account of their age at death. Indeed, the number of deaths is of limited value, unless we know about and take account of age at death also.

However, it can be seriously misleading to interpret in terms of individuals the number of additional deaths, and associated age distribution, computed as attributable to air pollution. Specifically, the attributable deaths are *not* an estimate of the number of individuals in whose earlier death air pollution has played some part. As discussed in Section 8.2, that number could be much bigger. Thus, we consider it more appropriate to express the results of such calculations as 'an effect on mortality equivalent to '*X*' deaths'.

Here we apply the calculation to mortality impacts in 2008 of current (2008) pollution levels.

Where we have age-dependent mortality rates, the calculations can be done separately by age group, and for this report we consider effects on mortality rates only for those aged 30 and above. This is how the concentration–response function from the ACS study (Pope *et al*, 2002), as recommended by COMEAP (2009), is usually applied. It avoids extrapolating beyond the ACS cohort in terms of age.

It is also possible to calculate the burden for scenarios in which mortality rates differ by an amount related to only a part of the total pollution concentrations, rather than to all particulate air pollution, and we apply that approach here also, i.e. to calculate the burden of anthropogenic $PM_{2.5}$ only (see, for example, Armstrong and Darnton, 2008).

Although we have not directly investigated this, the burden of deaths estimated in this manner is likely to be broadly similar in different years close in time, i.e. the burden as estimated for 2008 is similar to what would have been estimated in 2007 or 2006 – there would be small differences because of differences in pollution levels or in baseline death rates. These differences would be greater for years further in the past.

Problems arise when 'attributable' deaths calculated in this static way are expressed as 'lives that would be saved' by pollution reduction measures where pollution would change over time: the danger is that these might be reported as if the number of 'lives saved' or 'deaths avoided' by an equivalent pollution reduction will repeat yearly when that reduction is sustained over time. As we have seen (Sections 2.1 and 2.5), the effect of ageing in the population defeats this; sustained reductions in death rates, as a result of reduced $PM_{2.5}$ pollution, lead over longer time periods to a larger and older population, which in turn changes the numbers and age distribution of annual deaths dynamically. The appropriate approach to address the benefits of pollution reduction/removing anthropogenic pollution is the impacts calculation described earlier in Part I. This is discussed further in Section 9.1.

### 6.2.2 Total survival time (years of life lost)

#### *Years of life lost per attributable death*

An associated question of interest concerns how much loss of potential life across the population is implied by the additional mortality associated with air pollution. In contrast to calculating a number of 'attributable' deaths in the current year (2008), answering it requires consideration of what might have happened in the future, and so it requires some assumptions about future death rates.

A number of methods can be found in use for addressing this question. Almost all involve the estimation of the loss of remaining life associated with each 'attributable' death at each age and then summing these to give a figure of the number of years of life lost by the population (i.e. the reduction in survival time).

a A simple version involves subtracting the age at each 'attributable' death from the average life expectancy (from birth) of the current population. Life-years lost are then aggregated to give the reduction in total survival time for the population as a whole. This may be a reasonable approximation for deaths at a young age (such as the majority of male road traffic accident victims), but

when considering the effects of air pollution leads to illogical results at the level of the individual, including negative figures at ages past the average life expectancy. This could easily be misinterpreted as suggesting that air pollution has a benefit at these older ages, and that clearly is not so. It may also lead to underestimation of the total population burden. Consequently, we do not recommend this approach be used in assessing the mortality effects of air pollution, some of which are manifested after the age of average life expectancy.

b The WHO Global Burden of Disease project uses a similar approach, but instead of national life expectancy, it uses life expectancy from Japan, which gives high values of 80 years for men and 82 years for women.

Instead, we regard as more justifiable an approach which sums the age-specific remaining life expectancy related to each 'attributable death' at each age. This gives a positive estimate of years of life lost for deaths at all ages. It does raise another methodological issue, concerning which age-conditional life expectancy to use to assess the loss of life per age-specific attributable death.

a Methods for loss of potential life tend to use estimates of remaining age-dependent life expectancy based on the forward projection of unaltered current mortality rates.

b We think that it is more appropriate, when considering the burden of air pollution, to use an age-dependent life expectancy based on the future mortality rates that would have applied in the absence of anthropogenic air pollution in the future. This is the method we have used to give the results reported in Chapter 7.

Where the difference between future mortality rates in the presence or absence or air pollution is small, this distinction is not very important – we have computed results both ways and confirmed this is so in the present context. But the issue highlights some of the logical difficulties in defining a current burden based on future events.

### *Total population survival time*

The total burden of air pollution, in terms of population survival time or population loss of life, is then the aggregate of these age-conditional years of life lost, aggregated over all attributable deaths. We emphasise once again that these numbers are population aggregate or average figures which are useful in helping compute or express the total mortality burden in the population; they are not meaningful as estimates of the number of individuals affected by air pollution, or of the size of the effect among individuals, even on average.

We have explored several different ways of calculating the total survival time, all involving the aggregation of age-specific remaining life expectancy associated with each death 'attributable' to air pollution. Despite slight methodological differences (life tables or proportional calculations) and underlying assumptions (e.g. using life expectancies based on current mortality rates or those that would apply in the absence of air pollution), all give similar results.

This suggests a stability in the total population survival time as an expression of mortality burden. It may be that this is the index that best reflects the burden of mortality, and that

'attributable' deaths and associated life-years lost per attributable death are best understood as convenient computational stepping-stones to arrive at the burden expressed as total population survival time. Indeed, though popular discussion focuses on the numbers of deaths, it is difficult to make sense of these numbers without some consideration, explicit or implicit, of age at death, and so of the loss of life expectancy associated with death at that age. The total population survival time, expressed in life-years, makes this aspect explicit and transparent and, by allowing exploration of these issues, allows a more meaningful discussion of mortality burden than can be captured by the numbers of deaths only.

### 6.2.3 Life expectancy

We have used one additional approach to look at the burden of air pollution: calculating the loss of life expectancy to the 2008 birth cohort. In doing this, we have calculated the loss of life expectancy that would be experienced on average by those born in 2008 if they were exposed throughout their lifetime to current (2008) levels of air pollution, rather than solely to non-anthropogenic particulate air pollution. This is an example of 'period life expectancy' for a birth cohort, defined as the life expectancy calculated by projecting forward the mortality rates experienced in a particular period, often a single year. A reduction in life expectancy is a different measure of the mortality burden of pollution.

When expressing the burden in terms of an effect on mortality equivalent to '*X*' deaths within the current year (2008), we are not concerning ourselves with future effects. But when we consider the burden in terms of life expectancy, we are interested in the burden on the 2008 birth cohort of the current and future anthropogenic particulate air pollution.

In the full life-table approach used to calculate impacts of a sustained change (reduction) in air pollution (see Part I), our baseline assumption is that mortality rates in the future will remain the same as currently. This implies that the calculated gain in life expectancy for a birth cohort will be the same for both the burden and the equivalent impact question, when the latter is calculated assuming no cessation lag[8].

[8] In this report the results are not identical. There are some small deviations because different methods are used to deal with the final, open-ended age interval: see section 3.1.2 in Miller and Hurley (2006).

# Chapter 7
# The Burden Calculation

## 7.1 Methodology of burden calculations

To calculate the burden associated with mortality in 2008 we have compared two scenarios:

**Baseline** a 2008 population that experiences 2008 age-specific death rates, i.e. that has been exposed to particulate pollution at 2008 levels,

**Alternative** a 2008 population that experiences age-specific death rates reduced by an amount implied by 2008 levels of anthropogenic air pollution,

and calculated the mortality differences between them as the burden of mortality in 2008 of particulate pollution. Note that the comparison is specific about the size of the pollution effect addressed, but it does not say when that exposure was experienced.

The coefficient of 1.06 per 10 μg/m$^3$ $PM_{2.5}$ was applied to the 2008 anthropogenic population-weighted mean concentration from Table 3.1, using the logarithmic scaling previously described in Section 3.1.3, to produce an impact factor[9], *k*, with which to modify the population hazard rates from the baseline scenario to derive those for the alternative scenario.

For reference, the baseline population data on deaths and life expectancies are summarised in Table 7.1.

**Table 7.1: Baseline 2008 population data**

| Country | Total deaths in the population | Total deaths in the population aged 30 years and over | Life expectancy (years) | |
|---|---|---|---|---|
| | | | Males | Females |
| *England and Wales combined* | 506,791 | 499,701 | 78.17 | 82.12 |
| Scotland | 55,532 | 54,647 | 75.46 | 80.14 |
| Northern Ireland | 14,669 | 14,322 | 76.71 | 81.44 |
| UK | 576,992 | 568,680 | N/A | N/A |

[9] Impact factors are multipliers used to adjust the mortality hazard rates. For example, an impact factor of 0.99 implies a 1% reduction in hazard rate.

The relevant age-specific deaths recorded for 2008 were then multiplied by the impact factor, *k*, to give numbers of deaths at each age attributed to the difference in particulate air pollution concentrations between the baseline and alternative scenarios. These age-specific deaths for males and females were then summed to give a total.

To calculate the total population survival time lost, the differences in expected deaths between the scenarios were combined with the remaining life expectancy at each age, using future mortality rates reflecting an absence of anthropogenic particulate air pollution. These were accumulated over all ages to give the sum of the age-specific remaining life expectancies associated with all the 'attributable' deaths and, therefore, interpretable as the total amount of life lost estimated as being 'attributable' to air pollution.

Period life expectancy (see Section 6.2.3) of the 2008 birth cohort was calculated from the 2008 mortality hazard rates (baseline current pollution scenario) and from rates for those aged 30 years and over reduced by the impact factor, *k*, (alternative, non-anthropogenic pollution only scenario). These were compared to estimate the implied change in period life expectancy attributable to anthropogenic $PM_{2.5}$ pollution.

A number of sensitivity analyses were performed, varying chosen input assumptions. These differed in some respects from those carried out for impact calculations: since our burden calculations were specified without adjustment for lag structures, no sensitivity analyses on lags were carried out; and discounting was not considered relevant for calculations based on 2008.

Sensitivity analysis was carried out, for burden calculations based on the England and Wales population, to investigate the effect of varying the coefficient, again giving results corresponding to the COMEAP-recommended uncertainty ranges. Coefficients of 1.00, 1.01, 1.06, 1.12 and 1.15 per 10 $\mu g/m^3$ were used.

Also, to avoid the uncertainties of extrapolating the ACS risk estimates beyond the range of concentrations in the ACS study as described in Section 3.2.2, the burden of anthropogenic particulate air pollution was investigated both as the effects of all anthropogenic $PM_{2.5}$ and as the effects of anthropogenic $PM_{2.5}$ above 7 $\mu g/m^3$.

## 7.2 Results

The total effect on mortality, expressed as the number of 'attributable' deaths, is presented in Table 7.2. For ease of comparison between the different countries, the last column of Table 7.2 gives the number of 'attributable' deaths per 100,000 population aged 30 years and over. The burden on total survival time in years is presented in Table 7.3 and the burden on life expectancy in Table 7.4.

As for the impacts question (Chapter 4), the burden of anthropogenic $PM_{2.5}$, expressed as an effect on mortality in terms of equivalent ('attributable') deaths and burden on total survival time depends both on the population size and the level of pollution under consideration. When considered on the basis of 'attributable' deaths per 100,000 of the population (aged 30 years and over) the differences between the countries are determined by the difference in pollution only. The same is true for life expectancy as seen in Table 7.4 below.

**Table 7.2: Effect on mortality in 2008 of anthropogenic $PM_{2.5}$ air pollution in the UK population. UK totals are aggregates from the individual results presented**

| Pollution included | Country | Population-weighted mean concentration | Number of 'attributable' deaths | Number of 'attributable' deaths per 100,000 people aged 30 years and over |
|---|---|---|---|---|
| All anthropogenic | England and Wales | 9.46 µg/m³ | 26,799 | 79 |
| | Scotland | 4.97 µg/m³ | 1,560 | 47 |
| | Northern Ireland | 6.02 µg/m³ | 502 | 48 |
| | **UK total** | **8.97 µg/m³** | **28,861** | **75** |
| Anthropogenic >7 µg/m³ | England and Wales | 3.90 µg/m³ | 11,228 | 33 |
| | Scotland | 0.21 µg/m³ | 67 | 2 |
| | Northern Ireland | 0.91 µg/m³ | 77 | 7 |
| | **UK total** | **3.50 µg/m³** | **11,372** | **30** |

**Table 7.3: Burden on total survival in years (rounded to the nearest 1000) of anthropogenic $PM_{2.5}$ air pollution in the UK population resulting from the mortality burden in 2008. UK totals are aggregates from the individual results presented**

| Pollution included | Country | Population-weighted mean concentration | Burden on total survival (life-years lost) |
|---|---|---|---|
| All anthropogenic | England and Wales | 9.46 µg/m³ | 315,000 |
| | Scotland | 4.97 µg/m³ | 19,000 |
| | Northern Ireland | 6.02 µg/m³ | 6,000 |
| | **UK total** | **8.97 µg/m³** | **340,000** |
| Anthropogenic >7 µg/m³ | England and Wales | 3.90 µg/m³ | 132,000 |
| | Scotland | 0.21 µg/m³ | 1,000 |
| | Northern Ireland | 0.91 µg/m³ | 1,000 |
| | **UK total** | **3.50 µg/m³** | **134,000** |

**Table 7.4: Burden on life expectancy of anthropogenic $PM_{2.5}$**

| Pollution included | Country | Population-weighted mean concentration | Difference in life expectancy (days) for the 2008 birth cohort | |
|---|---|---|---|---|
| | | | Males | Females |
| All anthropogenic | England and Wales | 9.46 µg/m³ | 203 | 190 |
| | Scotland | 4.97 µg/m³ | 113 | 104 |
| | Northern Ireland | 6.02 µg/m³ | 134 | 132 |
| | UK* | 8.97 µg/m³ | 194 | 182 |
| Removal down to 7 µg/m³ | England and Wales | 3.90 µg/m³ | 83 | 78 |
| | Scotland | 0.21 µg/m³ | 5 | 4 |
| | Northern Ireland | 0.91 µg/m³ | 20 | 20 |
| | UK* | 3.50 µg/m³ | 75 | 70 |

* Calculated by weighting the England and Wales, Scotland and Northern Ireland results by the relevant birth cohort size.

## 7.3 Sensitivity analyses – burden question

### 7.3.1 Coefficient

For this burden question, the importance of the coefficient on the estimation of the effects of air pollution has also been explored in line with the recommendations in the 2009 COMEAP report. The data for all anthropogenic $PM_{2.5}$ (9.46 µg/m$^3$) in England and Wales have been used in this sensitivity analysis and the results are presented in Table 7.5.

Other uncertainties, which were not analysed, are discussed in Chapter 8.

**Table 7.5: Effect of varying the coefficient on the estimation of the burden of all anthropogenic $PM_{2.5}$ (9.46 µg/m$^3$) in England and Wales**

| Coefficient | | Number of 'attributable' deaths | Burden on total survival (life-years lost) | Difference in life expectancy (days) for the 2008 birth cohort | |
|---|---|---|---|---|---|
| | | | | Males | Females |
| 1.00 | (0%) | 0 | 0 | 0 | 0 |
| 1.01 | (1%) | 4,682 | 55,000 | 35 | 32 |
| 1.06 | (6%) | 26,799 | 315,000 | 203 | 190 |
| 1.12 | (12%) | 50,801 | 597,000 | 396 | 372 |
| 1.15 | (15%) | 61,887 | 728,000 | 489 | 460 |

# Chapter 8
# Discussion of the Burden Calculation

## 8.1 Introduction

Underlying what we have called the burden question is a very reasonable wish, namely to understand and make explicit the effect of current levels of pollution in the UK (or in specific parts of the UK, such as London) on the resident population. In addition, there is an associated wish: to find, if practicable, ways of expressing mortality results that are not only valid but are easy to communicate, and in particular to find valid expressions in terms of the numbers of deaths per year attributable to outdoor air pollution at current levels.

However, two factors work against a simple answer. Firstly, pollution changes over time. Secondly, the relationships between when exposure to pollution occurs and when the consequent risks to mortality are increased, are complex: pollution in previous years affects mortality in 2008, and pollution in 2008 affects mortality in later years among the population of those alive in 2008.

For these reasons, and as discussed in detail in Section 6.1, we converted the usual and apparently simple, but ambiguous, burden question 'What is the effect of air pollution on mortality in the UK today?' into a more specific question: 'What is the effect of air pollution at current (2008) levels on mortality in the UK in 2008?'. In addition, we based our calculations of mortality on a simple comparison exactly like that used more widely in discussion of mortality burden.

Basing our calculations on a simple comparison has a disadvantage: it means that we need to be explicit in describing how the comparisons we have made – and that others conventionally make – relate to the levels of anthropogenic $PM_{2.5}$ that people in the UK actually experienced in 2008 and former years. This aspect is usually ignored in discussions of mortality burden; we discuss it in Section 8.4.

The simple comparison also has important advantages, however, in that it gives answers, in terms of an effect on mortality equivalent to '*X*' attributable deaths at ages mirroring deaths in the general population, similar to those usually reported in discussions of burden. It also allows us to express the implications of differences in 2008 death rates in other ways, and to describe the relationships between the different indices. We discuss these aspects in Section 8.2. In particular, we highlight that results are valid as descriptions of aggregate or average effects across the population, but it is not valid to see them as informative about how particulate air pollution affects individuals. The relationship between burden calculations such as these, and the impact calculations of Part I, is discussed in Section 9.1.

In Section 8.3, we discuss some uncertainties underlying the results. The public health significance of the results is discussed later, in Section 9.3.

## 8.2 Context for burden results

### 8.2.1 Individuals and populations

We have shown how, under a set of stated assumptions, calculations can estimate figures to represent the burden of particulate air pollution on mortality in a particular year. The measures we have estimated comprise differences in the numbers of ('attributable') deaths, life expectancy from birth, and burden on expected total survival time. We consider that these apparently simple and often-used measures are valid and appropriate for what we understand is one of the main functions of asking, and answering, the burden question: highlighting the current magnitude of the effect on mortality caused by current levels of outdoor particulate air pollution. However, if incorrect inferences are to be avoided, care must be taken not to make inappropriate extrapolations of these statements of effects at the population level to statements of effects at the individual level.

The raw ingredients of our calculations are population sizes and mortality rates, estimates of the concentration of airborne $PM_{2.5}$ to which the population is exposed, and a risk coefficient that predicts how the mortality rates change with changes in $PM_{2.5}$ concentrations. All of these inputs refer to whole populations, and average across individual variations the distributions of which we do not know.

As an example, there is almost certain to be variation between individuals in the extent to which their mortality risks are increased by exposure to outdoor particulate air pollution, and there is limited evidence on how the impact of air pollution on risks varies across the population as a whole. There are many ways in which those impacts could be distributed across the individuals of a new birth cohort and produce the same average gain in life expectancy, and in reality we do not (and probably cannot) know the true pattern. The numbers of people who are affected or unaffected and the change in expected survival at an individual level are unknown. But there is no basis in evidence to support the view that everybody is affected equally, and there is plenty of evidence, direct and suggestive, to indicate otherwise.

The warning that population burden estimates should not be interpreted at the individual level has implications for different aspects of each of our burden estimates, and we discuss these in the following sections.

### 8.2.2 Life expectancy

As discussed in Section 5.1.3, *life expectancy at birth* is an average value; it is the average of the ages at death of a whole birth cohort, and these range from deaths in the first year of life to the age of the last death in the cohort. It is therefore obviously a mistake to interpret this average expectation of life as applying to all individuals in a birth cohort, or even as being 'typical'. Similarly, changes in life expectancy can describe only the average experience of the population, under different scenarios with different age-specific risks of death. It is therefore also a mistake to assume that a change in life expectancy means that everybody in the population experiences the same actual change in life expectancy.

### 8.2.3 'Attributable' Deaths

The number of 'attributable' deaths calculated as the mortality burden of anthropogenic particulate air pollution is an aggregate, across the population as a whole, of an 'attributable' risk that affects the age-specific chances of death or survival of the population as a whole.

A common misinterpretation comes from assuming that the entire mortality effect of air pollution is borne by a small subset of the population, sometimes described as the 'statistical victims' of outdoor air pollution.

The figure for 'attributable' deaths represents a population-wide difference between the numbers of deaths expected to occur under higher and under lower death rates. It does not represent an actual group of people, in principle identifiable, whose early and untimely deaths are caused solely by exposure to outdoor air pollution. Such a 'silver bullet' view of causality may be appropriate to deaths from some external causes, such as through road traffic accidents or gun crime, or to particular diseases, such as mesothelioma following exposure to asbestos. But air pollution has the potential to affect everyone who breathes the air. The effects are principally on mortality from non-malignant cardiorespiratory causes (and, in practice, principally from cardiovascular causes), and from lung cancer. These are complex diseases, with multiple established and likely causes at the population level, and almost certainly with a complex mixture of factors affecting initiation and progression at the individual level also.

Given this complexity, it is not plausible to think of the figure of 'attributable' deaths as enumerating an actual group of individuals whose death is attributable to air pollution alone, i.e. the "victims' of outdoor air pollution. The use of the qualified term 'statistical victim' may be intended to reflect that the group is in reality a fiction, and that the number of people in whose deaths air pollution has played a part might be much larger; but in our experience it leads to questions such as 'Who are the statistical victims?', 'What are their characteristics?' and – when considering reductions in air pollution and associated reductions in age-specific death rates – 'Who are the people who would not have died in 2008 if air pollution had not been present, i.e. whose lives would have been (temporarily) saved?'. These questions may appear reasonable, but they are based on a mistaken notion of causality, because they presume that results which are valid as population estimates also have a straightforward interpretation in terms of the individuals affected – and, in this case, they do not. (This problem is common to many applications of the notion of 'attributable' risk or death, particularly where the outcome has multiple contributing causal factors.)

We therefore also consider it inappropriate to use the term 'premature' deaths to express the outcome of the burden calculation. If only a small minority of people are susceptible to the long-term adverse effects of particulate air pollution, and air pollution is the only factor that contributes to their dying (i.e. diet, lack of exercise, smoking etc. have played no part), then the number of deaths calculated as being 'attributable' to air pollution could be regarded as the number of 'premature' deaths caused by air pollution. If, instead, air pollution impacts on everyone exposed and contributes, to some extent, to all deaths, the whole population might arguably be regarded as dying 'prematurely' – but the impact on the timing of death of each individual would be much smaller. We discuss this and related issues in Section 8.2.4.

### 8.2.4 Total population survival time and its distribution

#### *Validity and usefulness of the index*

Total population survival time is another valid way of representing the burden of air pollution on current mortality. This can be estimated in more than one way. The method we have used is to estimate the number of attributable deaths, for each death, to estimate (in terms of life-years) the conditional loss of life associated with death at that age, and to aggregate these values across the population as a whole. This approach links easily with the discussion of attributable

deaths, above. Indeed it can be understood as making explicit an idea implicit in considering attributable deaths, that to understand the mortality burden we need to understand also the age distribution of the deaths, and the loss of life implied by it.

Overall, we believe that the most important description of the burden of air pollution is in terms of years of total survival time lost to the current population. Simply stating a number of deaths does not allow for the ages at which these deaths occur, or for the fact that the loss of life associated with them varies with age. This is the basis of our calculation of total survival time, and some very valuable information about the burden is lost if this is not calculated.

### *Interpretation at the individual level*

Other commentators believe that other measures should be quoted, and some of these suggestions lead to discussion of how the total burden of survival is distributed. In particular, it has been suggested (e.g. In House Policy Consultancy, 2010) that 'years of life lost to victims' would be a helpful measure in communicating the burden of air pollution. We would agree, if the statement were a recommendation (as above) to quantify the total loss of survival in the population (although we would have concerns, already described, about the use of the term 'victims'). However, some commentators interpret it differently, and have introduced the concept, and calculation, of 'average years of life lost per statistical victim' – i.e. the years of life lost per 'attributable' death.

In one sense, this simply reverses our calculation of total survival impact, which was obtained by multiplying the deaths by age-specific remaining life expectancy. The average is simply the average of these values, over all the 'attributable' deaths. The danger is not in the calculation as such; it is in interpreting it as an attribute of the individuals who have been affected by air pollution. As discussed previously, this interpretation raises the same difficulties as considering the number of people whose timing of death has been impacted upon by air pollution.

It is not always realised that the 'years of life lost per statistical victim', can give only very limited information about the burden caused by air pollution, or indeed by any hazard. It is not dependent on the concentration of air pollution, but is a function of the age-structure of a population and the age-specific risk of death within that population. An increased age-specific risk of death, that applies equally across all ages 30 years and above (such as that related to $PM_{2.5}$), increases the number of age-specific deaths and the loss in the total population survival time, in direct proportion to each other. Therefore, each additional death at a particular age is associated with the same reduction in survival time. This means that the 'years of life lost per statistical victim' is constant for a static population and represents, simply, the average years of life lost associated with *each* death above the age of 30 years; this applies whether the death is 'attributable' to air pollution or not. Therefore, we prefer the term 'the average loss of life per death' as more appropriate; it is not specific to the additional deaths calculated as being 'attributable' to air pollution in any way.

### *Distribution of total survival time across the population*

We have repeatedly cautioned against identifying the population burden of air pollution as an impact on a group of individuals, the additional or attributable deaths caused by air pollution. It may be helpful to consider how the effect could be distributed. The various ways all amount to taking the same total burden in terms of life-years, and partitioning it in different ways across the population as a whole (to express risk), or across those who have died.

Given a burden estimate of reduction in total survival time for the whole population, distributing this total over any subset of the population is a matter of simple arithmetic. Table 8.1 demonstrates this, over a number of hypothetical assumptions about the distribution of the added risk to different subsets of the population. Given our estimate that the burden of pollution-related mortality in 2008 implies an associated loss of total survival (or life expectancy) of 340,000 years, this averages 3 days per member of the population. However, it is arguable that we should consider the burden as expressing itself only across deaths, not across those who do not die in 2008, even if their risk of death was increased. Shared over all the 569,000 deaths in those aged 30 years and over, the average life shortening would be ½ year.

Given that much of the impact of particulate air pollution on mortality is linked with cardiovascular deaths, it is also arguable that the maximum number of deaths to which air pollution would have contributed a part is likely to be nearer 191,000 than 569,000, in which case the average loss of life is calculated at around 2 years. Table 8.1 shows other examples of averages, depending on what proportion of all deaths are considered as affected. At the extreme, if the 'attributable deaths' were the only ones affected, that would imply an average loss of 11½ years.

**Table 8.1: Hypothetical average years of life expectancy lost in 2008 due to the contribution of anthropogenic particulate air pollution, averaged over different sections of the UK population**

| Hypothetical population affected | Number affected | Hypothetical average loss of life expectancy |
|---|---|---|
| Whole population (ages 30+) | 38,348,000 | 3 days |
| All deaths (ages 30+) | 569,000 | ½ year |
| 50% of deaths (30+) | 290,000 | 1 year |
| Deaths from CV causes (30+) | 191,000 | 2 years |
| 20% of deaths (30+) | 116,000 | 3 years |
| 10% of deaths (30+) | 58,000 | 6 years |
| 7% of deaths (30+) | 40,000 | 8½ years |
| 'Attributable' deaths (30+) | 29,000 | 11½ years |

One view of a calculation of 'years of life lost per death' is that it provides an illustration of the maximum average loss of life that could be associated with deaths affected by air pollution, and a graphic and/or eye-catching way of communicating something complex. As such, it might serve a useful purpose, provided that its limitations are widely understood. In our experience, however, communication is not careful, the limitations are not expressed clearly; and the dangers of discussing these intended-to-be-convenient fictions are not avoided.

## 8.3 Uncertainties in the estimates

### 8.3.1 Dealing with uncertainties

Our calculations in relation to the question of burden in 2008 estimate a reduction of more than six months in the average life expectancy of a birth cohort experiencing mortality hazard rates affected by anthropogenic particulate air pollution. Performing burden calculations for deaths among adults in each age group at age 30 years or more in the 2008 UK population, and aggregating over age groups, gave an estimated number of 29,000 additional or attributable deaths in the UK in 2008. Associating these with estimates of remaining life, we estimate a total burden of lost survival time of 340,000 years, representing the total number of years of future life that were not realised because of the excess mortality in 2008 attributable to air pollution.

We discuss in Section 9.3 the public health implications of these results. In this section we note that these are, of course, central estimates, and there are always uncertainties attached to such as these. We describe below some of principal sources of uncertainty, and the sensitivity of our results to alterations in the underlying assumptions.

### 8.3.2 Risk coefficient

As explained and discussed in COMEAP (2009), there are uncertainties in assessing the risk coefficient as a 6% change in mortality per 10 μg/m$^3$ change in annual average $PM_{2.5}$. In the 2009 COMEAP report there is a substantial description of the uncertainties surrounding the risk coefficient; this will not be repeated here. The confidence interval from the ACS study, a plausibility interval and a wider interval for sensitivity analysis are described (COMEAP, 2009) for the all-cause mortality coefficient. Because the size of impacts or burdens in the same population is close to proportional to the size of the relative risk coefficient, it is easy to calculate approximate results for other risk coefficients. For example,

a Expert elicitation suggested a 75% chance that the risk coefficient lies between 1% and 12%, implying (see results in Section 7.3) a range from about 4,700 to nearly 51,000 deaths in 2008 for England and Wales, while attributable deaths range from 0 to 62,000 in 2008 using the widest interval of 0–15% for the risk coefficient.

b The expert elicitation of probabilities in COMEAP (2009) suggested a 50% chance that the risk coefficient lies between 2% and 9%, implying deaths linked with anthropogenic $PM_{2.5}$ across the UK of between about 9,000 and 40,000.

The estimates of the effect on life expectancy and total survival time are subject to uncertainties exactly corresponding to those that affect calculations of 'attributable' deaths. For example, risk coefficients of, respectively, 1% and 12% (change in age-specific death rates per 10 μg/m$^3$ change in $PM_{2.5}$), rather than the 6% value used, give an estimated burden expressed as life expectancy of between one month and one year, for England and Wales. The corresponding range in loss of population survival time is 55,000 to 597,000 years.

### 8.3.3 Pollution estimate used

The uncertainties with respect to all anthropogenic particulate air pollution are as described in Part I, Section 5.1.2, when assessing the impact of reducing all anthropogenic air pollution. Namely, the confidence in the estimates of effects of $PM_{2.5}$ below 7 μg/m³ is lower than that of effects above 7 μg/m³. The results presented here for burden indicate that more than 60% of the total deaths, of the shortened total population survival and of the decreased life expectancy, derive from concentrations below 7 μg/m³.

Additionally, the uncertainties in attributing the $PM_{2.5}$ as anthropogenic or not apply equally here.

## 8.4 What questions about pollution at 2008 levels does the burden question answer?

### 8.4.1 The issue

We described in Section 6.1 that there are complex relationships in time between long-term exposure to particulate air pollution and risks of mortality, and that, consequently, the apparently simple burden question 'What is the effect of air pollution on mortality in the UK today?' is more complicated than it seems, because death rates in 2008 are affected by air pollution in earlier years, and air pollution in 2008 affects death rates in later years.

We put the issues to one side by defining two scenarios, one baseline and one alternative, that examine mortality in 2008, using the same 2008 population, but with different death rates in 2008. The baseline uses actual 2008 death rates; the alternative uses these, reduced by an amount attributable to 2008 levels of anthropogenic particulate air pollution. This led us to comparing the implications, for mortality in 2008, of a difference in mortality hazard rates equivalent to that derived from the coefficient of 1.06 per 10 μg/m³ $PM_{2.5}$ and 2008 levels of anthropogenic $PM_{2.5}$. This is the difference between the 2008 population experiencing hazard rates equivalent to those for 2008 levels of pollution and a hypothetical population of the same size and age experiencing lower hazard rates equivalent to those for non-anthropogenic levels of pollution.

In this section we re-visit the issue, touched on briefly in Section 6.1, of exactly what question about pollution these scenarios answer, or what assumptions about pollution levels and lags are consistent with the burden question we have answered and the results we have reported. The difficulties in interpretation arise because the population response to air pollution is an ongoing and inherently dynamic process, spread over many years, and it is difficult to capture a snapshot of this, as the burden question does, via the impact of air pollution on mortality in one particular year.

### 8.4.2 Interpretation that ignores lags

The question we have answered is consistent with an assumption that the full mortality effects of pollution are immediate, i.e. that there are no time lags. One pragmatic and apparently common sense view is to say we have examined a hypothetical situation of no lags, recognising that this is an approximation to a more complex reality.

This straightforward (though strictly incorrect) formulation does not require any assumptions about past concentrations of $PM_{2.5}$, because under an assumption of no lags, these have no impact on 2008 death rates. A subtle variation might note that in reality, even under an assumption of no lags, past levels of pollution do have some impact on the numbers of deaths in 2008, in that they will have influenced the size and age structure of the 2008 population, but this is a detail that does not affect the calculations. In other words, the issue of past pollution levels is irrelevant, if we assume no lags.

### 8.4.3 Interpretations that take account of lags

There are other explanations that allow a more realistic view of time lags in relation to the death rates used in 2008. These require additional assumptions about pollution levels prior to 2008, e.g. for the baseline scenario, that annual average $PM_{2.5}$ in the past had been constant at 2008 levels, and $PM_{2.5}$ in the past had been constant at non-anthropogenic levels for the alternative scenario. Then, whatever the patterns of latency or cessation lag, effects of past exposure (or absence of anthropogenic particulate air pollution) in various years will combine to give, respectively, the baseline and alternative death rates in 2008 that we have examined.

As a hypothetical example, to illustrate the concept, if the lag were spread over two years, 2008 pollution levels would only have 50% of the full effect on mortality in 2008. However, in addition, 50% of the full effect of pollution in 2007 would affect mortality in 2008. If pollution levels had been the same in 2007, then this effectively adds up to the same answer as assuming 100% of the full effect of 2008 pollution in 2008.

There remain some difficulties of interpretation, however, because of how different levels of pollution, and consequent different death rates, in past years might affect population age and structure over time, a phenomenon we described and discussed in Part I.

a From one viewpoint, these population dynamics are irrelevant, because the burden question is defined as applying to the population in 2008, without consideration of how that population occurred.

b From another viewpoint, it is unrealistic to assume pollution at 2008 levels in the past without taking into account the effects of pollution-impacted death rates in the past on population size and age structure, compared with an alternative scenario of only non-anthropogenic particulate air pollution. It is, however, difficult to take account of the population dynamics, because a comparison between the baseline and alternative scenarios in this way requires a choice of population at some time in the past to use as the common starting point, and it is unclear when that point should be.

In general, taking account of population dynamics from some point in the past would lead to a larger and older population in 2008 under the alternative scenario compared with the baseline as a result of the lower age-specific death rates (see Section 2.1.2). This means that there would be higher numbers of deaths in 2008 in this alternative scenario, taking into account population dynamics, than in the alternative scenario we have used in our calculation, which is based on the existing 2008 population. Therefore, the difference in deaths in 2008 between the baseline and alternative scenarios would be smaller if this approach were adopted than the number of attributable deaths that we have calculated.

> A plausible starting point for such an analysis might be the earliest year at which past exposures were considered to impact on 2008 death rates. This is the earliest year about which we need to assume that past exposures are similar to 2008, and so it depends on what lag is assumed. For no lag, it gives the 2008 population. If, as is often assumed, the time period between relevant exposure and mortality is principally a matter of a few years, then the effect of population dynamics on the number of deaths in 2008 is small, assuming a common population at the start of the lag period; although with longer lag scenarios, the effect on deaths in 2008 is greater.

These considerations highlight that the burden question as usually addressed require some additional assumptions about how air pollution does, or does not, affect death rates and population size and age, in order to make the calculations we have made about mortality burden in 2008 – calculations which are typical of what is usually done – meaningful in terms of pollution at 2008 levels. These assumptions are not consistent with what we know about (i) the time course of pollution and mortality and (ii) how differences in death rates affect population size and age over the longer term. However, under some plausible circumstances, their overall practical impact may not be large.

# Part III

# Integrated Aspects

# Chapter 9
# Overall Discussion

Our specific objectives are to:

a calculate, using the risk coefficients and other conclusions of COMEAP (2009), the implications for mortality of the UK population of:

- reductions in fine particulate air pollution attributable to human activity, i.e. anthropogenic particulate matter ($PM_{2.5}$), – both small policy-relevant reductions of 1 μg/m$^3$ annual average $PM_{2.5}$, and the unrealistic but interesting possibility of eliminating anthropogenic $PM_{2.5}$,
- current levels of anthropogenic $PM_{2.5}$;

b describe and explain the methods used to make these calculations;

c make transparent and understandable the assumptions that underlie the calculations, and to discuss their importance;

d clarify the relationships between different ways of expressing mortality impacts and to comment on the appropriateness of their use.

## 9.1 Distinguishing the mortality burden of current anthropogenic $PM_{2.5}$ from the benefits of eliminating it

### 9.1.1 Deaths

One important theme of the present report, elaborated in Part I, is that the mortality benefits of eliminating current anthropogenic particulate air pollution do not reproduce themselves year on year, at least not in terms of annual numbers of deaths and life-years lived in the population as a whole. This is because changes in pollution levels affect death rates which, over the course of time, affect population size and age structure. These dynamic changes in population size and age distribution, in turn, affect annual deaths and life-years lived (this is described in Section 2.5). This fact greatly complicates the discussion of the benefits of reducing air pollution, when those benefits are expressed in terms of annual numbers of deaths and annual life-years lived.

As discussed in Section 6.1, the burden question is somewhat ambiguous in terms of the relationship between the timing of pollution and the timing of mortality impacts. However, we re-defined it to conform to what is usually done in assessing burden, and we focused on impacts on mortality in one year only, i.e. in 2008, and set up the comparisons of death rates and population in a way that ignores population dynamics. This gives results in terms of all indices – number of deaths, life-years and life expectancy – that are similar within the years

close to 2008, because in effect the burden calculation starts anew each year, and so it ignores consideration of population dynamics by virtue of how the question is framed.

An implication is that there is a need to distinguish between the mortality burden of current (anthropogenic) air pollution, which ignores population dynamics, and the mortality impacts or benefits of eliminating all anthropogenic particulate air pollution, which take account of them and so – for numbers of deaths per year and life-years – give results that vary year by year. In other words, answering the question of mortality burden of anthropogenic particulate air pollution does not, in general, give an answer to the question of the mortality benefits of eliminating anthropogenic $PM_{2.5}$.

Nonetheless, the number of deaths in 2008 attributable to current levels of air pollution is one meaningful measure of the mortality burden of air pollution. We re-iterate two characteristics of the result. Firstly, it is implicit in the calculations that the distribution of age at death of these attributable deaths mirrors the age distribution of deaths generally among adults at age 30 years or more, and the number of deaths, together with age at death, dispose towards thinking about population survival – see Section 9.1.3. Secondly, these numbers are valid as ways of calculating and communicating the magnitude of an effect at the population level; they should not be interpreted as expressing how air pollution impacts on individuals. There are many other, more plausible, ways that the population effect may be distributed across individuals. For these reasons we favour describing the mortality burden as *equivalent to* a number of attributable deaths, at usual ages.

There is one interesting connection between the number of deaths in the burden and impact analyses: the burden of current anthropogenic $PM_{2.5}$ on mortality in terms of equivalent or 'attributable' deaths, as described in Part II, is identical to the impact estimated in terms of the reduced number of deaths in the first year of a sustained pollution reduction without cessation lag, as estimated in Part I. This is because, in that first year, the population size and age structure have not been altered by pollution reduction.

Eliminating all current anthropogenic $PM_{2.5}$ would, for many years, lead to reduced annual deaths in the population as a whole. However, contrary to what is usually implied, the reduction in the annual numbers of deaths is, after year 1, not the same as the burden of current anthropogenic $PM_{2.5}$ expressed as 'attributable' deaths. Population-wide, the benefit of reduced pollution in terms of fewer deaths per year reduces progressively over many years. If new births are included, in time the number of deaths annually settles down to a number similar to that before the pollution reduction, but with an older age distribution. If new births are not included, in due course there will be more deaths per year under the scenario of a sustained reduction in pollution.

This is one of the reasons why COMEAP and IGCB have focused on life expectancy and gains in life-years rather than deaths when dealing with questions relating to the mortality impacts of long term exposure to air pollution. A supporting paper provides an overview of previous UK estimates of the impact of long-term exposure to fine particles (COMEAP Secretariat, 2010).

### 9.1.2 Life expectancy at birth

However, this difference between the burden question and the impact question does not apply to the mortality benefits of reducing air pollution when these effects are expressed in terms of the changes in life expectancy of a new birth cohort. This is because life expectancy is an average figure: changes in life expectancy depend only on changes in death rates and not on population size or age structure. The percentage changes in death rates remain constant over time, for a given sustained reduction in pollution. This implies constant changes in death rates, and so the burden of current anthropogenic $PM_{2.5}$ on life expectancy is the same as the life expectancy benefit of eliminating current anthropogenic $PM_{2.5}$, in the absence of lag, or with lags that are less than 30 years.

### 9.1.3 Total population survival

The total population survival, in terms of life-years, is a valid and important metric for answering both questions. For impacts of a sustained reduction in pollution, it is the best metric for capturing the full mortality implications on the population as a whole, and so is the principal metric used in policy analysis. In the case of burden, the discussion is usually in terms of additional or attributable deaths, but implicit in this discussion is the notion of age at death and associated loss of life which, when aggregated across all attributable deaths, gives a measure of burden as total population survival, in life-years. It can be difficult to make sense of the resulting numbers of life-years because people are not used to thinking in these terms, but population survival time is important because the different ways of apportioning the burden across individuals (see Section 8.2.4) all amount to different ways of distributing the same total population survival time – and we do not know which distribution across individuals is actually valid, even though some representations are more plausible than others.

For the purposes of IGCB, measures of life expectancy and gains in life-years are appropriate for assessing the monetary benefits of proposed policy interventions. Having an established methodology to do this is important for public health because it allows policies to reduce air pollution to be optimised so that policies with the maximum health benefits for the minimum costs are chosen. This ensures that the best possible improvement in the impact of air pollution on public health is achieved, given the available funds. It can also be used for other purposes, such as illustrating what role reductions in pollution could play in reducing health inequalities, in combination with other measures.

For cost–benefit analysis, the use of life tables allows easy incorporation of monetary value and weighting of the results, e.g. by discounting the value of future life-years. In addition, life tables give flexibility due to the results being itemised by age and calendar year. This monetary valuation and discounting is not relevant for the contribution of air pollution to the burden in terms of deaths in 2008 as these occur now. It might be possible to consider for the loss of total survival as this metric looks into the future and, just as for the results of an impact calculation, it is likely to have a significant effect on the results.

## 9.2 Expressing and communicating the results

After careful consideration of the different kinds of questions that are asked about the effects of air pollution on mortality (what we have called the impact question and the burden question), and the different ways in which population survival and mortality can be and are discussed, we have reached the following conclusions and recommendations.

### 9.2.1 General points

a In expressing these results there is, to some extent, a trade-off between full accuracy and accessibility.

b We stress the need for careful interpretation of these metrics to avoid incorrect inferences being drawn – they are valid representations of population aggregate or average effects, but they can misleading when interpreted as reflecting the experience of individuals

c We stress also the need to communicate uncertainties openly and fairly.

### 9.2.2 Life expectancy at birth

Effects on life expectancy at birth are one valid and meaningful expression of mortality effects for both the impact of reduced pollution and burden of current pollution.

a *Communication:* life expectancy can be a very helpful measure in communicating about air pollution.

- It can be used to compare the effects of air pollution on mortality with those of other risk factors such as smoking, obesity or poverty and so is a concept with which people increasingly are familiar.
- Also, the gains in life expectancy at birth from eliminating anthropogenic particulate air pollution are the same as the burden on life expectancy at birth from current levels of anthropogenic particulate air pollution – this simplifies communication and discussion.
- It should be remembered and emphasised that life expectancy is an average concept, and in practice the amount actually gained will differ between individuals.

b *Policy analysis:* life expectancy at birth is incomplete as an expression of the mortality effect in the current population as it does not cover effects on other ages and so is not really useful for analysis of policy options, e.g. via cost–benefit analysis.

### 9.2.3 Total population survival time

The total population survival time (life-years gained or lost) is also a valid and meaningful way of expressing mortality effects. COMEAP has for many years proposed and used this measure as the most accurate and complete way of capturing the mortality effects of air pollution reductions (the impact question); we think it is the most accurate and complete way of capturing the mortality burden of air pollution also. The impact and burden questions give different results in terms of life-years because they ask different questions – burden is about mortality in one year only, whereas impact deals with mortality for many years in the future.

a *Communication:*

- The concept of a life year is not difficult or unusual – it is what we celebrate each birthday. But total population survival and the large numbers of life-years involved are difficult to grasp, and burdens associated other risk factors tend not to be reported in terms of total population survival and life-years.

- It would be helpful to discussions about air pollution, and we think helpful more generally, if effects on total population survival were more prominent in discussions about the public health burden of other risks also (e.g. smoking, obesity, poverty and social inequalities), and in discussion of the public health benefits of reducing them.
- Meantime, we need to develop ways of making the language of total population survival and life-years more accessible to policy makers and the general public.
- Our hypothetical illustrations of how the burden may be distributed across the population are an attempt in that direction; these can be useful, provided that any one way of dividing up the population total is taken as a way of illustrating the overall effect, and not as a literal description of what is actually happening to individuals.

b *Policy analysis:* the total population survival, in terms of life-years, is by far the single most relevant metric for policy analysis.

### 9.2.4 Attributable deaths

Attributable deaths are a valid and meaningful way of capturing some important aspects of the mortality burden in one year, e.g. 2008, of current levels of pollution, if we set aside some of the complexities of how quickly air pollution affects mortality risks. It is inadvisable to use annual numbers of deaths for assessing the impacts of pollution reduction, because these vary year by year in response to population dynamics of reduced death rates.

a *Communication:* there is a widely held view that communication in terms of numbers of deaths is to be encouraged, because people understand what a death is. Of course they do, but it is unclear whether or not the population understands the notion of 'attributable deaths' as used by epidemiologists and as carried over into public debate about air pollution.

We support discussion of the burden of mortality in terms of attributable deaths, with some important *caveats*.

- The numbers of deaths alone capture only some of the information needed about mortality burden. So, for example, it is not straightforward to compare in terms of numbers of deaths the burden of air pollution, which mostly affects older people, and of road traffic accidents, suicide, or HIV/AIDS, which by comparison affect younger people. Implicit in any communication about deaths is some understanding of age at death or, equivalently, the loss of life implied by death at various ages. This is best captured explicitly – which, in effect, means discussion in terms of total population survival time, even if that discussion appears to be in terms of attributable deaths and associated years of life lost.
- The number of attributable deaths (at various ages) is a concept that applies to the population as a whole, and as such is valid and meaningful. It is not right to interpret it as a valid or meaningful representation of the number of individuals in whose earlier death air pollution played some part. It is important to maintain this distinction in communicating about attributable deaths and therefore we prefer an expression of the results as 'an effect equivalent to '*X*' deaths'.

- Communication should take into account that the number of deaths works differently for the question of burden and the question of impact. There is a very short-term equivalence: the burden of current anthropogenic $PM_{2.5}$ on mortality in terms of equivalent or attributable deaths is identical to the reduced number of deaths in the first year of a sustained pollution reduction, without cessation lag. This is because, in that first year, the population size and age structure has not been altered by pollution reduction. However, this equivalence is not sustained over time, because everybody dies eventually.

b *Policy analysis:* it is difficult to see a useful role for the numbers of deaths in policy analysis, because in the long run air pollution does not affect the number of deaths, only the age at which death occurs.

## 9.3 Public health significance

As we consider the public health implications, it is important to note that the present report addresses only one aspect of the public health significance of outdoor air pollution, albeit the aspect which various impact analyses (e.g. IGCB, 2007) have shown to be the dominant one, namely, mortality in adults following long-term exposure to outdoor particulate air pollution. Because of the nature of the studies on which these estimates are based, they include at least some of the short-term mortality effects of particles on those aged 30 years and over, as detected via time-series studies. Because the mortality effects of long-term exposure are far greater, they are given precedence; and to avoid double-counting, the results presented here should not be associated with additional calculations of mortality as a result of short-term exposure.

It is important to recall that outdoor air pollution also damages public health in additional ways: for example, there is strong evidence that

a exposure to $PM_{2.5}$ affects mortality in infants;

b short-term changes in other pollutants, notably daily variations in ozone, are related to daily mortality;

c air pollution carries a morbidity burden over a very wide range of cardiorespiratory health effects.

These aspects need consideration for a complete assessment of the health effects of air pollution, but this report quantifies the dominant effect namely of long term exposure to outdoor particulate matter.

### 9.3.1 Impact or benefit of reductions in anthropogenic $PM_{2.5}$

As suggested, it is unlikely that all anthropogenic $PM_{2.5}$ will be removed, even in the medium term. However, it is likely given the exposure reduction targets in the EC Air Quality Directive (2008) that small reductions will occur. Here we consider life-years gained not only for removal of all anthropogenic $PM_{2.5}$, where the numbers are very large and consequently not easy to appreciate, but also for a 1 $\mu g/m^3$ reduction, where the numbers will be smaller and possibly somewhat easier to comprehend. As discussed in Section 5.2.1, the results for a 1 $\mu g/m^3$ reduction can be scaled to other changes in population-weighted mean concentrations.

For the UK, a total of about 36.5 million life-years could be gained across the population as a whole, over a period of little more than 100 years if all anthropogenic $PM_{2.5}$ is eliminated. Assuming an average life expectancy of the order of 80 years, this is approximately equivalent to over 450,000 lifetimes. For a sustained reduction of 1 μg/m³ $PM_{2.5}$, the impact is about 4 million life-years gained, or 50,000 lifetimes. To put the overall figure in context, a similar calculation (for England and Wales rather than the UK) estimated that the removal of all motor-vehicle traffic accidents would lead to a gain of about 8.1 million life-years and the elimination of the mortality risks of passive smoking to a gain of about 13.2 million life-years (Miller and Hurley, 2006). It is important to realise that while ambient air pollution might be less potent than cigarette smoke or environmental tobacco smoke, everyone in the population is exposed to air pollution and this increases the overall public health impact.

Another way of getting some perspective on this is to look at the monetary equivalent, using, for example, the value of a life year of €40,000, as used by the European Commission in its cost–benefit analysis for Clean Air for Europe (CAFE, 2010). Then the full value (over more than 100 years) of reducing anthropogenic $PM_{2.5}$ by 1 μg/m³ would be €160 billion, which is substantial; eliminating all anthropogenic $PM_{2.5}$ would have a corresponding monetary value, over 100 years, of about €1.5 trillion.

As noted earlier, these numbers continue to grow year on year and, in the absence of discounting, can be made, by extending the length of the follow-up period, as large as anyone would wish.

These estimates of effect on life-years are, of course, also subject to uncertainties similar to those discussed earlier, plus some additional ones related to cessation lag and discounting. For example,

a For elimination of anthropogenic $PM_{2.5}$, risk coefficients of, respectively, 1% and 12% rather than a 6% value (change in age-specific death rates for a 10 μg/m³ change in $PM_{2.5}$) would give for England and Wales an estimated effect of between 5.8 and 66.2 million rather than 34.1 million life-years;

b For different lags, removal of anthropogenic $PM_{2.5}$ for England and Wales gives 34 million life-years for the EPA lag and a range from 30 million life-years for a 30 year lag to 35 million for no lag;

c As noted earlier in Section 5.2.4, for the US EPA lag, discounting could reduce the result for elimination of anthropogenic $PM_{2.5}$ in England and Wales by 50% for a 1.5% discount rate and by 90% for a 6% discount rate. This effect may differ for different lags.

### 9.3.2 Public health burden of 2008 air pollution levels

The results indicate that the mortality burden associated with current air pollution is a serious public health issue, whether expressed in terms of equivalent deaths, total population survival or reduction in life expectancy.

#### *Effect on mortality ('attributable' deaths)*

Our calculations estimate that the effect on mortality linked to anthropogenic particulate air pollution (expressed as anthropogenic $PM_{2.5}$) was equivalent to nearly 29,000 deaths in 2008 in the UK as a whole. Most of this burden is in England and Wales – this is where the great

majority of the UK population resides, and average population-weighted concentrations of $PM_{2.5}$ are much higher there than in Scotland and Northern Ireland.

A burden expressed in terms of deaths in 2008 could be lower than this, if it was felt that the effect of lags on changes in population size and age structure should be taken into account. However, it could also be larger, if a higher coefficient were used. The range for a 1% and a 12% coefficient around a central estimate for England and Wales of around 27,000 attributable deaths would be from 5,000 to 62,000 deaths, although these outer estimates have a lower probability. The result would be about 11,000 deaths if it was felt that the calculation should not apply to pollution levels below the lowest concentration in the study used to derive the coefficients (7 $\mu g/m^3$).

As we discussed in Section 8.2.3, the results do not necessarily mean that 29,000 deaths in the UK in 2008 were solely caused by air pollution. It may be that air pollution contributed a smaller part to the deaths of a larger number of people – perhaps a small part to all deaths of people aged 30 years or more in 2008. While air pollution may not necessarily be the sole cause of any deaths, this does not mean that its effect is not important. Many familiar major risks to health (such as active or passive smoking, diets high in fats or sugar and the lack of exercise) are also contributory factors that act in combination with each other to progress disease and, ultimately, lead to death. We note that the complex issues surrounding estimation of the burden of air pollution that we have discussed in this report apply just as much to other public health burdens, though they are not always communicated in these other contexts.

It is not possible to compare this result directly with a figure for an impacts calculation, as following the impacts of a change in pollution forward in time changes the number of deaths every year. It is only possible to derive a single figure for deaths for the burden calculation by taking a pragmatic view of the question, i.e. by assuming no lag or by incorporating artificial assumptions such as pollution being constant at 2008 levels in the past and ignoring changes in population size. The result needs to be taken in the spirit of giving a general feel for the size of the problem rather than a precise prediction.

### *Total population survival time (life-years lost)*

Our central estimate of the years of life lost by those who died in the UK in 2008 as a result of anthropogenic particulate air pollution is approximately 340,000 years. This represents the total number of years of future life that were not realised because of the excess mortality in 2008 attributable to air pollution. This figure is obviously smaller than the estimated 36.5 million life-years gained as a result of removing anthropogenic $PM_{2.5}$ pollution in the impacts calculation. This is because the burden calculation is based on taking a snapshot over a year, whereas the sustained removal has effects in the long-term both because we are considering an effect of long-term reduction in exposure and because those born in the future, as well as those currently alive, benefit from reductions in pollution.

### *Life expectancy (months lost)*

Our main calculations imply that current (2008) anthropogenic particulate air pollution in the UK causes a reduction in average life expectancy at birth of more than six months across the populations of England and Wales. Lower estimates of more than four months in Northern Ireland and between three and four months in Scotland reflect lower levels of anthropogenic particulate air pollution in Scotland and Northern Ireland. Within each country, there will also be local variations according to population-weighted annual average $PM_{2.5}$ concentration, which

is highest in cities. So, for example, we have estimated that the average loss of life expectancy to the 2008 birth cohort in London is about nine months.

The differences in life expectancy burden between England and Wales, Scotland and Northern Ireland are smaller than the corresponding differences in mortality burden expressed as 'attributable' deaths, because the life expectancy results reflect how death rates are affected by population-weighted annual $PM_{2.5}$, but not differences in population size – i.e. they are averages at the level of the individual. .

As mentioned previously, the estimate of the average amount of life expectancy lost or gained in the burden calculation or impact calculation, respectively, is the same. This makes it a useful metric for communication.

In trying to evaluate the significance of a loss of average life expectancy of six months, it could be argued that, at the population level, it is not dramatic; for example, the average loss of life expectancy *among smokers*, due to active smoking, is ten years (NHS Choices, 2010). The differences in life expectancy associated with socioeconomic status are similarly large: for example, seven years in England (The Marmot Review, 2010). A point to note with these comparisons is that only some of the population are active smokers; the NHS Choices website gives a 2007 figure that one in five people smoke in England. That suggests a loss of life expectancy due to active smoking of closer to two years across the population as a whole, if we ignore the complexities linked to people smoking for a number of years and then quitting. Exposure to air pollution is an involuntary and unavoidable risk, in contrast to active smoking which, at least initially, is considered to be voluntary.

The effect of anthropogenic outdoor particulate air pollution on life expectancy is larger than that of several other established mortality risks. It is approximately 2.5 times the loss of life expectancy previously calculated (Miller and Hurley, 2006) for males in England and Wales due to either road traffic accidents or exposure to environmental tobacco smoke, and more than six and three times, respectively, for females.

### *Summary*

Our results demonstrate that outdoor air pollution is a serious public health issue.

# Chapter 10
# Conclusions and Recommendations

## 10.1 Conclusions

### 10.1.1 Benefits of pollution reduction

We have concluded that in the unrealistic scenario where all human-made particulate air pollution ($PM_{2.5}$) is removed, as a central estimate, 36.5 million life years could be saved across the UK population, including new births, over the next 106 years. This means that the total survival of the UK population would increase by 36.5 million years over that time, corresponding to an increase in life expectancy at birth, i.e. on average across new births, of six months. These results depend on a number of simplifying assumptions, and are subject to uncertainties, principally about the value of the risk coefficient of 1.06 (6%) per 10 $\mu g/m^3$ increase in $PM_{2.5}$. Expert elicitation (COMEAP, 2009) suggested a 75% chance that the risk coefficient lies between 1% and 12%, implying a range of 5.8–66.2 million life-years for England and Wales and an average increase in life expectancy of one month to one year.

A pollution reduction of 1 $\mu g/m^3$ of $PM_{2.5}$ would lead to an increased UK total survival of approximately 4 million life years, or 20 days increased life expectancy from birth, using the central estimate for the risk coefficient. These results are relatively insensitive to changes in assumptions about how quickly mortality risks reduce following reductions in pollution, an issue reviewed in detail by Walton (2010). But they are strongly sensitive to assumptions about discounting of future values, an issue we have not addressed other than to highlight its importance.

### 10.1.2 Current mortality burden of pollution

As a central estimate, we conclude that anthropogenic $PM_{2.5}$ at 2008 levels had an effect on mortality equivalent to nearly 29,000 deaths in 2008 in the UK and an associated loss of total population survival of 340,000 years. These results are consistent with an average loss of life ranging from 11½ years, if air pollution was solely responsible for 29,000 deaths, to six months if the timing of all deaths was influenced by air pollution. We believe both of these possibilities to be extremely unlikely. Given that much of the impact of air pollution on mortality is linked with cardiovascular deaths, it is more reasonable to think that air pollution may have made some contribution to the earlier death of up to 200,000 people in 2008, with an average loss of life of about two years per death affected, though that actual amount would vary between individuals. However, this assumption remains speculative.

Little is known about how the risk from air pollution is distributed across the population, but the extent to which individuals are affected is likely to be highly variable. Thus, whatever the number of people whose timing of death is affected, the extent of loss of life will probably be very different for each individual. The burden can also be expressed as a loss of life expectancy to the 2008 birth cohort of about six months (ranging from three to four months in Scotland and Northern Ireland to six to seven months in England and Wales).

These results are subject to many of the same uncertainties as stated above for the impact calculations. Using the 75% plausibility interval suggested by the expert elicitation in COMEAP (2009) this means a range of effects on mortality equivalent to between 4,700 and 51,000 deaths with a loss of between 55,000 and 597,000 years of life in 2008, or effects on life expectancy, i.e. the average across 2008 births, of between one month and one year, for England and Wales. Additionally, if changes in population size and age structure were taken into account, the answer would be smaller for longer lags.

### 10.1.3 Methodological conclusions

We show that all these results scale in proportion to population-weighted mean concentrations of anthropogenic $PM_{2.5}$, and (approximately) to the risk coefficient. In addition, results that depend on population size, i.e. attributable deaths and life-years lived, scale in relation to the size of the population aged 30 years and above, other things being equal.

## 10.2 Recommendations

### 10.2.1 Metrics to express results

The Committee has looked at expressing and communicating the results and concluded that:

a. To some extent, in expressing these results there is a trade-off between full accuracy and accessibility.

b. There is a the need for careful interpretation of these metrics to avoid incorrect inferences being drawn – they are valid representations of population aggregate or average effects, but they can misleading when interpreted as reflecting the experience of individuals.

c. There is a need to communicate uncertainties openly and fairly.

*Life expectancy at birth* is a valid and meaningful expression of mortality effects for both the impact of reduced pollution and the burden of current pollution. However, it is incomplete as an expression of the mortality effect in the current population as it does not cover effects on other ages.

*Total population survival time (life-years gained or lost)* is also a valid and meaningful way of expressing mortality effects of both the impact and burden questions. It is the most comprehensive way of capturing the full effects. However, there are difficulties in communication. The concept of a 'life-year' is not difficult to grasp, but it is difficult to interpret the very large numbers of life-years involved in total population survival. It is the most relevant index for policy analysis.

*The number of attributable deaths* is a valid and meaningful way of capturing some important aspects of the mortality burden, across the whole population in any one particular year, of current levels of pollution, if we set aside some of the complexities of how quickly air pollution affects mortality risks. To emphasise that the number of deaths derived is not a number of deaths for which the sole cause is air pollution, we prefer an expression of the results as 'an effect equivalent to a specific number of deaths at typical ages'. It is incomplete without reference also to associated loss of life, and there is a danger that the burden is misinterpreted as representing how air pollution affects an individual. It is inadvisable to use annual numbers of deaths for assessing the impacts of pollution reduction, because these vary year by year in response to population dynamics.

In this report we have applied a rigorous approach to calculating and discussing questions relating both to the impact of pollution reduction and, as far as we can, to the burden of current levels of particulate air pollution (given the question represents only a snapshot in time of what is a dynamic process). The assumptions that underpin calculations of the public health significance of other risk factors are not always so transparently laid out and discussed. We consider it important that, if calculations of impact or burden are used to compare and rank the public health importance of different risks, similar methods and the same degree of rigour, are used in the calculations.

### 10.2.2 Public health significance

Clearly air pollution is a significant public health issue. We recommend that protection of the public continues to be a significant driver of policy in this area.

Public discussion has highlighted the need to communicate the mortality effects of air pollution in ways that can be easily understood by the general public as well as by policy makers. There is a need for accuracy as well as accessibility. We recommend that those who want to have an objective and evidence-based discussion take account of the communication guidelines of Section 9.2.

### 10.2.3 Further work

#### *Methods to predict the mortality impacts of policies to reduce air pollution*

**1** Following COMEAP (2009), this report has used all-cause mortality to estimate the public health impacts of air pollution. However, evidence on air pollution and cause-specific mortality is increasing and additional insights may be gained in the future by cause-specific analyses. These insights may include a better understanding of lags because the lag structure will vary by cause of death. The present report has indicated how a cause-specific analysis might be done; there is scope and arguably a need to do this, at some point in the future.

**2** Our work has highlighted that discounting of future values can change the results very significantly. There is a need for further clarification of how the mortality indices addressed here link with monetary valuation in theory and in the practice of cost-benefit analysis.

### *Methods for calculating the burden of air pollution*

**3** The Committee's work has highlighted some interesting connections between the three principal mortality indices of total population survival, deaths and life expectancy. We are aware that there is more to be understood about these connections, and about similarities and differences between the impact question and the burden question. COMEAP recommends that these connections be investigated further with a view to bringing even greater clarity to the discussion.

**4** We noted some difficulties in taking account of time lags on the usual burden calculations, especially if attempts are made to take account of associated changes in population size and age structure. Because of this, we recommend further work on the burden question in the context of longer lags, to clarify concepts and to see if burden results are sensitive to different assumptions about how pollution affects death rates over time.

### *Better understanding of the public health significance of air pollution*

**5** These figures derived for air pollution would be given context if comparable figures (i.e. shortening of life measures) for other known causes of disease (such as cigarette smoking) or widely applied interventions (such as cancer screening) were derived.

**6** It would be of interest to analyse the contribution of historical improvements in air pollution control to the steady, but largely unexplained, improvement in average life expectancy across most Western populations in recent decades.

### *Public understanding of the mortality burden of air pollution*

Public discussion has highlighted the need to communicate the mortality effects of air pollution in ways that can be easily understood by the general public as well as by policy makers.

**7** The Committee considers that our report makes a significant contribution to clarifying what can be said accurately. There is a need for further work to make key concepts such as total population survival accessible.

**8** There are many opinions about what 'the public' can and does understand, about deaths, life expectancy and life-years lived, but a lack of empirical evidence. We recommend investigation to better address the needs of the public.

# Chapter 11
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# Appendix

## Membership of the Committee on the Medical Effects of Air Pollutants

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**Members**

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Professor Robert L Maynard CBE FRCP FRCPath FFOM (Medical)
Dr Heather Walton BSc DPhil (Scientific) *(until March 2010)*
Ms Alison Gowers BSc MSc (Scientific) *(from March 2010)*
Dr Karen Exley BSc(Hons) MSc PhD (Scientific)
Miss Britta Gadeberg BSc(Hons) MSc (Scientific) *(from April 2010)*
Miss Inga Mills BSc(Hons) MSc (Scientific)
Mrs Isabella Myers BSc(Hons) MSc (Scientific)

# Membership of the Committee on the Medical Effects of Air Pollutants Subgroup on Quantification of Air Pollution Risks II (QUARK II) (Mortality)

**Chair**

Mr J Fintan Hurley MA*

**Members**

Professor H Ross Anderson MD MSc FFPH FRCP FMedSci
Mr Ben Armstrong BA MSc PhD
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Professor Jon G Ayres BSc MD FRCP FRCPE FFOM FRCPSG
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**Secretariat**

Professor Robert L Maynard CBE FRCP FRCPath FFOM (Medical)
Dr Heather Walton BSc DPhil (Scientific) *(until March 2010)*
Ms Alison Gowers BSc MSc (Scientific) *(from March 2010)*
Dr Karen Exley BSc(Hons) MSc PhD (Scientific)
Miss Britta Gadeberg BSc(Hons) MSc (Scientific) *(from April 2010)*
Miss Inga Mills BSc(Hons) MSc (Scientific)
Mrs Isabella Myers BSc(Hons) MSc (Scientific)

* Members of the Working Group tasked with undertaking this work and drafting this report on behalf of QUARK II and COMEAP.

*COMEAP Secretariat*
*www.comeap.org.uk*

*c/o Health Protection Agency*
*Chemical Hazards and Poisons Division*
*Chilton, Didcot, Oxfordshire OX11 0RQ*

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