SOCIAL COSTS OF MORBIDITY IMPACTS OF AIR POLLUTION - ENVIRONMENT WORKING PAPER No. 99

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ABSTRACT

Outdoor air pollution is a major determinant of health worldwide. The greatest public health effects are from increased mortality in adults. However, both PM and O\textsubscript{3} also cause a wide range of other, less serious, health outcomes; and there are effects on mortality and morbidity of other pollutants also, e.g. nitrogen dioxide (NO\textsubscript{2}) and sulphur dioxide (SO\textsubscript{2}). These adverse health effects have economic consequences; OECD (2014) suggests that the social costs of the health impact of outdoor air pollution in OECD countries, China and India was approximately USD 1.7 trillion and USD 1.9 trillion, respectively, in 2010. However, the study highlights that though the social costs of premature mortality account for the majority of these totals, the social costs of morbidity remain poorly estimated.

The objective of this paper is to inform the development of improved estimates of the social costs of human morbidity impacts resulting from outdoor air pollution in two components; namely to develop a core set of pollutant-health end-points to be covered when estimating the costs of morbidity, and to review current estimates of the cost of morbidity from air pollution.

The paper recommends a core set of pollutant-health (morbidity) combinations, for application in OECD countries, China and India, that meets the criteria of being unbiased (i.e. not systematically under-estimating or over-estimating the effects), credible (i.e. based on recognised expert reviews) and implementable (i.e. for which the necessary input data, including concentration-response functions (CRFs) and background rates of morbidity) are available or can be estimated. The review has identified five pollutant-health pairs for consideration. These are:

1. Respiratory Hospital Admissions (RHA) and Cardiovascular Hospital Admissions (CVHA) in relation to PM and to ozone.
2. Restricted Activity Days (RADs) and associated Work Loss Days (WLDs) in relation to PM and/or ozone.
3. Chronic Bronchitis in adults in relation to PM.
4. Acute Bronchitis in children aged 6-18 years, defined as “bronchitis in the past 12 months” resulting from PM
\textsubscript{10}; and
5. Acute Lower Respiratory Illness (ALRI) in children aged <5 years, resulting from PM\textsubscript{10}.

Whilst the empirical evidence for these pollutant-health outcomes remains limited, they are judged to be sufficiently robust to be considered in policy analysis.

The paper presents a list of unit values for the relevant health end-points. The unit values are comprised of three broad components: “resource costs” (which includes avertive expenditures, e.g. relocation to area of lower air pollution, staying inside, etc., and mitigating expenditures, e.g. the direct medical and non-medical costs associated with treatment for the health impact), “opportunity costs” (which includes costs related to loss of productivity and/or leisure time due to the health impact) as well as “disutility costs” (which includes pain, suffering, discomfort and anxiety linked to the illness).

A pragmatic approach to calculating air pollution-related morbidity effects might be to assume that they are a (near) constant fraction of the total health impact. On preliminary investigation the ratio of mortality to morbidity effects in recent EU and US evaluations is very different. However, the apparent
differences are strongly influenced by different approaches to valuation of mortality; the differences are much less when EU mortality valuations are based on VSL rather than VOLYs and, under some valuations, morbidity costs become <10% of the mortality ones. Given that the evidence points to under-estimation of morbidity impacts in quantitative HIA, marking up mortality costs by 10% to 15% may give a broadly correct estimate, at low cost. However, since there are many real differences between countries and regions with regard to pollutant mix, valuation of health treatment costs, productivity losses and pain/suffering, as well as cultural factors, this approach should be complemented by the bottom-up approach that compiles CRFs and unit values for the given context, whenever possible.

**JEL Codes:** I180 (Health: Government Policy; Regulation; Public Health); Q530 Air Pollution; (Water Pollution; Noise; Hazardous Waste; Solid Waste; Recycling), Q58 (Environmental economics: Government policy).

**Key words:** Air quality regulation, non-market valuation, health impact assessment
RÉSUMÉ

La pollution de l’air extérieur est l’un des déterminants majeurs de la santé dans le monde. Elle a pour principal effet d’accroître la mortalité chez les adultes. Cependant, les particules (PM) et l’ozone (O₃) ont aussi tout un éventail d’autres effets moins graves pour la santé, et d’autres polluants, tels que le dioxyde d’azote (NO₂) et le dioxyde de soufre (SO₂), ont également des répercussions sur la mortalité et la morbidité. Or, ces effets néfastes pour la santé ont des conséquences économiques : selon OCDE (2014), en 2010, les coûts sociaux découplant des effets sanitaires de la pollution de l’air extérieur ont avoisiné 1 700 milliards USD dans les pays de l’OCDE et 1 900 milliards USD en Chine et en Inde. Mais cette étude montre aussi que, si les coûts sociaux imputables à la mortalité prématurée représentent la majeure partie de ce total, l’estimation des coûts sociaux liés à la morbidité reste cependant très insuffisante.

Le présent rapport vise à améliorer l’estimation des coûts sociaux de la morbidité liée à la pollution de l’air extérieur en recourant à une double approche qui consiste, d’une part, à établir un ensemble de liens polluants-effets sanitaires qu’il conviendra de prendre en compte pour estimer le coût de la morbidité, et, d’autre part, à examiner les estimations existantes du coût de la morbidité associée à la pollution extérieure.

Le rapport propose un ensemble d’associations polluants-effets sanitaires (morbidité), qui peut être appliqué dans les pays de l’OCDE, ainsi qu’en Chine et en Inde, et qui satisfait à plusieurs critères : il est non biaisé (c’est-à-dire ne sous-estimant ni ne surestimant systématiquement les effets), crédible (c’est-à-dire fondé sur les examens d’experts reconnus) et applicable dans la pratique (c’est-à-dire pour lequel les données nécessaires, et notamment les fonctions concentration-réponse et les taux de base de morbidité, sont disponibles ou peuvent être estimées). Cinq paires polluants-effets sanitaires ont ainsi été identifiées :

1. Admissions hospitalières pour causes respiratoires ou cardiovasculaires associées aux particules et à l’ozone.
2. Journées d’activité restreinte ou journées de travail perdues du fait de la pollution aux particules et/ou à l’ozone.
4. Bronchite aiguë chez l’enfant de 6 à 18 ans, définie comme « bronchite au cours des 12 derniers mois » causée par les particules (PM₁₀) ; et
5. Infections aiguës des voies respiratoires inférieures chez l’enfant de moins de 5 ans, imputables aux particules (PM₁₀).

Bien que les données empiriques relatives aux effets sanitaires de ces polluants restent limitées, on estime cependant qu’elles sont suffisamment robustes pour être intégrées à l’analyse.

Le document présente une liste de valeurs unitaires associées aux différents effets sanitaires. Ces valeurs sont le résultat de trois grandes composantes : les « coûts en ressources » (qui incluent les dépenses d’évitement, c’est-à-dire occasionnées par un déménagement dans une région où l’air est moins pollué, ou par le fait de rester à l’intérieur, par exemple, et les dépenses d’atténuation, qui recouvrent les coûts médicaux et non médicaux directement liés au traitement du problème de santé), les « coûts d’opportunité » (qui correspondent aux coûts liés à la perte de productivité et/ou de temps de loisirs
imputables aux effets de la pollution sur la santé) ainsi que les « coûts de désutilité » (qui sont liés à la douleur, à la souffrance, à la gêne et à l’inquiétude provoquées par la maladie).

Une approche pragmatique du calcul des effets de la pollution atmosphérique sur la morbidité pourrait consister à partir du principe que ces effets représentent une part constante (ou presque) de l’impact sanitaire total. Il ressort d’un premier examen que, dans des études récemment menées dans l’Union européenne (UE) et aux États-Unis, le rapport entre les effets sur la mortalité et ceux sur la morbidité est très différent. Cependant, ces différences apparentes s’expliquent en grande partie par la disparité des méthodes de valorisation de la mortalité : ainsi, la différence est moindre quand les évaluations de la mortalité effectuées dans l’UE sont fondées sur la « valeur d’une vie statistique » (VVS) et non sur la « valeur d’une année de vie perdue », et, dans certains cas, le coût de la morbidité est inférieur de moins de 10 % à celui de la mortalité. Les impacts de la morbidité étant manifestement sous-estimés dans les évaluations d’impact sanitaire (EIS) quantitatives, le fait de majorer le coût de la mortalité de 10 % à 15 % pourrait donner une estimation globalement correcte à moindre coût. Toutefois, compte tenu de la multiplicité des différences réelles existant entre les pays et les régions du point de vue des combinaisons de polluants, du coût des traitements médicaux, des pertes de productivité et de la douleur/souffrance causée par la maladie, ainsi que des facteurs culturels, cette approche devrait être complétée, dans la mesure du possible, par une approche ascendante associant des fonctions concentration-réponse et des valeurs unitaires spécifiques au contexte.

Codes JEL : I180 (Politique de la santé ; régulation ; santé publique) ; Q530 (Pollution de l’air ; pollution de l’eau ; bruit ; déchets dangereux ; recyclage des déchets solides) ; Q58 (Économie de l’environnement : Politiques publiques).

Mots clés : Règlementation sur la qualité de l’air, attribution d’une valeur non marchande, évaluation d’impact sur la santé.
FOREWORD

This report was prepared by Alistair Hunt, (University of Bath), Julia Ferguson, (University of Cranfield), Fintan Hurley, (Institute of Occupational Medicine), Alison Searl, (Institute of Occupational Medicine), as a follow-up of the 2014 OECD book on The Cost of Air Pollution.¹

¹ The authors acknowledge helpful input from the OECD Secretariat and from members of the Working Party on Integrating Environmental and Economic Policies.
SOCIAL COSTS OF MORBIDITY IMPACTS OF AMBIENT AIR POLLUTION

1. Introduction

1.1 Background

Outdoor air pollution (often also referred to as “ambient air pollution”) is a major determinant of illness, disease and earlier death worldwide. The greatest adverse public health effects are from increased mortality in adults attributable to long-term exposure to outdoor particulate matter (PM), expressed as fine particles (PM$_{2.5}$). Recent analyses from the Global Burden of Disease (GBD) project estimated that long-term exposure to outdoor PM$_{2.5}$ was responsible for more than 3 million deaths worldwide in 2010 and, in terms of disability adjusted life years (DALYs), it was ranked 9$^{th}$ in severity of the 43 risk factors and clusters of risk factors globally for which rankings were presented (Lim et al., 2012). Mortality effects of long-term exposure to ozone (O$_3$) are less well established but were estimated by GBD as causing about 150,000 deaths worldwide in 2010. As evidenced by numerous reviews, e.g. the recent REVIHAAP project of the World Health Organisation, both PM and O$_3$ also cause a wide range of other, less serious, health outcomes; and there are effects on mortality and morbidity of other pollutants also, e.g. nitrogen dioxide (NO$_2$) and sulphur dioxide (SO$_2$) (WHO, 2013a).

These adverse health effects have economic consequences: OECD (2014) suggests that the welfare, or social, costs of the health impact of outdoor air pollution in OECD countries, China and India was approximately USD 1.7 trillion and USD 1.3 trillion and USD 0.6 trillion, respectively, in 2010, reflecting that outdoor air pollution continues to be a major public health hazard. However, that study highlights that though the social costs of premature mortality account for the majority of these totals, the social costs of morbidity remain poorly estimated. This is of particular significance not only because these estimates may influence the economic efficiency of air quality regulation, but also because two of the morbidity cost components – medical treatment costs and opportunity costs – have implications for health care planning and business planning across affected countries.

There are two main methodological components to the lack of agreement currently on the social costs of the morbidity effects of outdoor air pollution (OECD, 2014). One is because there is not an established common core set of pollutant-health combinations (and by implication a common core set of health endpoints) to be included in such social cost estimation exercises, or indeed an established common method for deciding what combinations to include. The other reflects a lack of an established common method underlying the practice of monetising the health end-points. Additionally, and as discussed later, there are legitimate reasons why the application of the same methodological principles can lead to different results in different contexts.

The objective of this paper is to inform the development of improved estimates of the social costs of human morbidity impacts resulting from air pollution in two components, by addressing the two principal methodological limitations identified above. The paper therefore recommends a core set of pollutant-health combinations and associated health endpoints to be included when estimating the costs of morbidity and explains how these were derived. It then reviews current estimates of the unit values that could be used to estimate the cost of morbidity from air pollution and suggests unit values to be adopted in subsequent analyses of this sort.
1.2 Health end-points to be covered when estimating the costs of morbidity

Aims

The specific focus of this health impact assessment (HIA) section of the paper is to identify a consistent and comprehensive core set of pollutant-health pairs and associated health end-points for the assessment of the morbidity costs of air pollution and so it addresses the relevant methods and results, and gives a discussion. In doing so, this report adopts a simple “in” or “out” approach to including pollutant-health combinations and associated health outcomes, informed by an attempt to elaborate criteria for inclusion (or not), and a discussion of the implications of implementing these criteria that recognises that there may not be a universal optimal set. This proved to be an ambitious programme of work. Nevertheless, something more comprehensive can be envisaged: once criteria have been agreed, it may be possible to develop and implement a more complete and nuanced approach to the analysis, going beyond an “in” or “out” dichotomy, for example by providing probability weights for inclusion, based on strength of evidence on causality and other criteria. Such an approach would need to take into account differences in different spheres of implementation (i.e. a possible lack of universality) and its acceptance would depend on the credibility of those who developed the probability weights. However, if successful it would allow a more nuanced approach for cost-benefit analysis (CBA) where the focus is on expected outcomes. The present report may be a suitable starting-point for such a more sophisticated approach.

Coverage: OECD countries plus China and India

The paper covers OECD countries plus China and India, in line with the coverage used in OECD (2014). In principle, however, the paper has a wider domain of application, e.g. to other countries in WHO Europe, or to countries in South-East Asia, because while it has not been practicable within the time and resources available to focus on these countries and regions specifically, the issues of relevance and of transferability of evidence that apply to these countries are similar in nature to those that apply to OECD countries generally, though maybe arising in a more extreme form (e.g. because of higher pollution levels in some cities and countries).

World Health Organisation (WHO) workshop 2014

In May 2014, the WHO office in Bonn organised a 2-day workshop on methods and tools for assessing the health risks of air pollution at local, national and international level (WHO, 2014). Regarding morbidity, its underlying viewpoint was similar to that of OECD (2014): it said that: “A main objective of air pollution health risk assessment (HRA) is to help optimize policies with respect to their health benefits and costs. All monetary valuations of air pollution impact assessment show that the impact of morbidity outcomes is small relative to mortality. However the quantification of morbidity estimates at local, national and international level remains very important information for policy-making and for public health.”

The WHO Report (2014) includes several background papers. One of these, by Laura Perez, concerned morbidity impacts (Perez, 2014). This is an informative overview of the state-of-the-art and the present report draws on it especially for its lists of plausible morbidity impacts that are not quantified. Also, some comments on the similarities and differences between the conclusions of the two projects are given in the discussion below. Otherwise, the present report was carried out independently of Perez (2014), except of course that the underlying source material is the same.

1.3 Outdoor air pollution and health: Methodology used

Considerations in selecting a set of pollutant-health pairs

There is no real consensus internationally on why some pollutant-health combinations get included in exercises quantifying the health impacts of outdoor air pollution and others do not. The selection process in this paper was guided by the ambition that any proposed set of pollutant-health combinations would, if implemented, (1) lead to estimated impacts that were in some sense “good”; (2) that it would be feasible to implement them; and (3) that the associated methods and results have sufficient credibility to be used. These somewhat vague general criteria are now discussed in more detail with a view to making them operational.

Provide “good” estimates

This derives from the two main uses of quantification of morbidity impacts, i.e. for Burden of Disease studies, whose purpose is to estimate the health impacts attributable to a risk factor, in this case outdoor air pollution, at current levels; and for health impact assessments, whose purpose is to make an a priori estimate of the health impacts of a policy, programme or measure, and of how these impacts are distributed (WHO, 1999) often, but not only, as input to cost-benefit analyses of proposed policies and measures. Because both of these uses are about making estimates, they lead to the same fundamental criterion: Will the final estimate be better or will it be worse if quantification of the relevant pollutant-health combination is included in the HIA?

One aspect of providing “good” estimates is to avoid bias in the estimates, i.e. to avoid approaches which either systematically under-estimate or systematically over-estimate the true, unknown health impacts. Both aspects are discussed in the following paragraphs, beginning with avoiding systematic under-estimation.

If a pollutant-health combination is not included in the analysis, then from the viewpoint of quantification that pollutant-health combination is treated as if it had no health impact at all, i.e. as if its impact is zero. This leads to an interesting question: Given the evidence, is there a better estimate than zero of the effect that the relevant pollutant has on the relevant health outcome, and if so, what is it?

Answering this question involves issues of judgement which in some instances may be easy, in others difficult. Experts may, and do, differ in their assessments in particular instances. These implementation difficulties do not, however, diminish the importance, from the viewpoint of avoiding systematic under-estimation of effects, of developing an analysis informed by this question.

Doing so contrasts with an alternative approach which is much more common; i.e. of including pollutant-health combinations if and only if there is “high confidence” or “reasonable certainty” in the resulting quantified effect from that pollutant-health combination. The idea underlying this approach is understandable – that it may detract from the quality of the HIA if the quantification of particular pollutant-health combinations can be contested. Also it seeks to avoid the charge that the benefits of outdoor air pollution reduction are being inflated. Both of these link to the issue of credibility, discussed later.

But it has a disadvantage whose significance will vary according to circumstances. This is because at least some of the other pollutant-health pathways, for which there is some, though less convincing, evidence are likely also to be real. (Lack of convincing scientific evidence does not mean that there is no effect.) Consequently, this policy of including only those pollutant-health combinations for which there is very strong evidence leads to a methodology and results that systematically under-estimate the true impact. This in turn leads to an anti-precautionary bias in the policy-making process, insofar as the policy process is based on or informed by the results of the HIA (Hurley and Vohra, 2010).
It is also necessary to avoid systematically over-estimating impacts. In practice, this means not including as real effects pollutant-health combinations that simply reflect association rather than causation, and not double-counting health effects that are real, i.e. not attributing to two different pollutants, or to two different health outcomes, what is in reality the same effect, and then adding the answers.

There is another, more pragmatic, consideration in whether or not including a particular pollutant-health combination will lead to better estimates; that is, will the inclusion (or not) of the relevant pathway have any influence on the final answers and resulting decisions? (“Does it matter?”). This has different implications in quantitative and qualitative analyses.

Outdoor air pollution has a wide range of effects on human health; and in practice this wideness of effect, across all ages, from pollutants which are pervasive, is one of the reasons why its reduction matters. Qualitatively, therefore, it matters to identify all the pollutant-health combinations that are supported by evidence.

From the viewpoint of quantification, however, an important consideration is whether or not inclusion makes a difference to the estimated health impacts. If included, are the estimated impacts big enough to make a difference? Or, put differently, if there is a bias from excluding something, is this likely to be large (relative to impacts that are quantified)? Clearly, it is more important to try to include those effects which have greater impact. In the present context, this implies that the impact on final answers of individual pollutant-health pathways will be measured especially in relation to the impact on mortality of long-term exposure to PM$_{2.5}$, the pathway that dominates most quantification projects currently.

Provide credible estimates

Credibility also has two aspects: It is desirable first that the pollutant-health combinations proposed have support from authoritative expert reviews and do not simply reflect the views of the present authors; and second, that the pollutant-health combinations are meaningful in those geographical locations where they are to be applied.

In practice, these two aspects, both desirable, often conflict with one another. This is because authoritative expert reviews are more readily available from particular countries or regions, notably North America and Europe; whereas the intended geographical coverage of this report is much wider internationally.

Provide estimates that are feasible to implement

Quantification requires several elements (Hurley and Vohra, 2010):

1. A set of pollutant-health combinations;
2. For each, a concentration-response function (CRF) to be used in quantification; in the present context, a CRF expresses quantitatively the relationship between ambient concentrations of pollutant, e.g. PM$_{2.5}$, and a health or health-related outcome, e.g. respiratory hospital admissions, by means of a relationship typically giving a per cent increase in outcome per unit change in pollution; e.g. a 1.9% increase in respiratory hospital admissions per 10 µg/m$^3$ increase in PM$_{2.5}$ concentration (WHO, 2013b);
3. Background knowledge of the population and circumstances in which the quantification is to be applied; and in particular:
   a. Knowledge of pollution levels (for burden of disease) or how they change in response to a policy (for health impact assessment of a policy)
   b. Knowledge of the population, possibly disaggregated by age, gender, and perhaps by socio-economic factors; and
   c. Knowledge of background rates (prevalence, incidence) of the health outcomes to be quantified.
Feasibility requires not only that the individual components of quantification are in place. It is essential also that these various inputs are aligned, e.g. that the modelling of pollution gives results in the same metrics as the pollutant component of the CRF; that the health outcome underlying the CRF has the same meaning as that underlying the background rates of morbidity, or the monetary valuation studies (Hurley and Vohra, 2010).

The focus of this paper is the first of these, a set of pollutant-health combinations, only, but as far as practicable, the work has been done with at least some consideration of the other elements also.

**The strategy adopted and associated plan of work**

The aim then was to propose a comprehensive, non-overlapping core set of pollutant-health pairs that would, on current understanding, generate usable estimates of the morbidity impacts of outdoor air pollution which hopefully could be implemented and which would have some credibility if implemented. The strategy adopted to meet these ambitions needed also to take account of some important limitations and in particular (i) lack of knowledge and consensus internationally on what pollutant-health combinations have real effects that matter, and (ii) the present authors’ limited knowledge of the specific circumstances of all of the countries, in OECD and more widely, where quantification may be carried out.

In order to develop recommendations which had some coherence across countries, facilitating both aggregation across countries and comparisons between them, a two-stage strategy was adopted. This involved first developing, from the international literature and existing practice in established high-level health impact assessments, a core set of pollutant-health combinations which could be considered for implementation throughout OECD, China, India and elsewhere; and then considering what is involved in applying such a core set in the OECD countries and beyond.

**1.4 A core set of pollutant-health outcome pairs**

The first stage focused on pollutant-health combinations where a real (causal) relationship is supported by current scientific evidence, as assessed by expert groups, especially those involved in major recent expert-driven evidence reviews. This include notably reviews by WHO (REVHAAP, WHO, 2013a; HRAPIE, WHO, 2013b) for the European Commission, by US EPA in its assessment of the costs and benefits of the US Clean Air Act, and in the Global Burden of Disease (GBD) project.

The selection of work by WHO for implementation in Europe and by US EPA for regulatory purposes is not intended to diminish the importance of work in other countries, for example in the UK, Canada, the Netherlands, Sweden and Finland. In these and in other countries also there is a strong and long-established tradition of both expert review of the health effects of air pollution and of resultant health impact assessment, often (though not always) linked to cost-benefit analysis. However, the recent WHO REVIHAAP and HRAPIE evaluations (WHO 2013 a, b) have an important status in policy making EU-wide and drew on experts from many countries in Europe and North America and they provide a sufficient up-to-date view of the situation in Europe. The assessments of US EPA are known for their comprehensiveness and rigour, and the HIA and associated cost-benefit analysis of the US Clean Air Act was developed over several years and with wide consultation.

In contrast to these two streams of HIA activity, i.e. HIAs that were for policy development EU-wide, or by US EPA, the Global Burden of Disease project was designed for application globally; and in this respect it meets directly a key need of the present report for wide geographical coverage. Additionally, the core team GBD team for air pollution HIA includes many leading international epidemiologists.
It was hoped that basing recommendations on these three sources would give a core set of pollutant-
health outcomes that reflected informed compromises between what is real, what matters, and what can be
implemented, as identified by expert groups that have high credibility internationally.

In order to achieve this, the present report identified and selected pollutant-outcome pairs that had
been used in quantification in at least one of these three sources. In practice, it was aimed for pollutant-
health combinations that had been selected (a) both by US EPA and by WHO for the European
Commission; or (b) selected by GBD. Given that the pollutant-health combinations of the present report
are being selected with a view to being used widely internationally, it was considered reasonable to require
that relevant expert groups agreed on their relevance for application in Europe and USA respectively. The
distinctive status given to GBD recommendations reflects that it was designed for application worldwide, and
not simply in the EU or USA; and that the fact of its implementation (Lim et al., 2012) shows feasibility.

In practice this meant limiting consideration of pollutants to particulate matter (PM expressed as
PM$_{2.5}$ or PM$_{10}$) and ozone (O$_3$), which were included in all three underlying major HIAs. Following
discussion with OECD during the project, morbidity aspects of nitrogen dioxide (NO$_2$) were also
considered, but less integrally than PM or O$_3$, because there is less consensus: of the major HIAs on which
selection was based, NO$_2$ was included only by HRAPIE, and then with limited pollutant-health combinations. US EPA did however consider direct effects of NO$_2$ in preparatory analyses for the NO$_2$
standard (US EPA 2009a); results are summarised in Section 2.1. Later, some comments were also
included about sulphur dioxide (SO$_2$) (not in any of the three underlying HIAs). The present project has not
considered evidence on proximity to roads (REVIHAAP, 2013; Perez, 2014), because of issues of
transferability and difficulty of implementation.

Some consideration was also given to the extent that these pollutant-outcome pairs might, if and when
included in HIA / CBA, give rise to impacts that are in some sense “substantial”; and to avoiding double-
counting. Double-counting can arise in at least two ways: (i) for a particular pollutant, when different
pollutant-health pairs are based on health outcomes that are related and at least in part overlapping; and
(ii) when the same or closely related health outcomes are linked with different pollutants, or different
metrics of the same pollutant.

The underlying core HIAs are described in more detail in Section 1.5; the selected core pollutant-
health combinations are given in Section 2.

1.5 What is involved in applying this core set in OECD countries and beyond

In practice, careful consideration needs to be given to whether, or with what degree of uncertainty and
extrapolation, the same core set can usefully and realistically be applied across all other OECD countries,
China, India, and indeed more widely. This is because:

1. The epidemiological evidence comes primarily from the USA, Canada and countries of the EU;
and while in principle the evidence applies internationally, in practice, the transferability of
relationships depends not only on the intrinsic similarities of humans everywhere, but also on a
wide range of other issues concerning the pollution mixture; population and its health, how health
is understood; how health care systems are organised, and other factors.

2. Implementation requires not only the relevance of the pollutant-health combination, but also of
the data necessary to quantify it, including background rates of morbidity in the countries to
which the quantification will refer. (Note that it was not an ambition of this project to assess and
propose suitable sources of background mortality rates throughout the OECD countries, China,
India and elsewhere.)
These and other aspects are discussed below. For the most part, that discussion is done irrespective of whether or not credible monetary values can be attached (issues of monetary valuation are considered separately, later in the paper) to enable understanding and discussion on their own merits of the strengths and weaknesses of the proposed pollutant-health combinations as a basis for how morbidity health impacts may be quantified.

1.5.1 Pollutant-health combinations based mostly on applications in the EU and North America

The core HIAs in more detail

Development of a core set of pollutant-health combinations focused on HIA projects intended for application in:

1. Countries of the EU, for which recommendations for quantification exist from CAFE (2005) and, more recently, the HRAPIE project (WHO, 2013b), co-ordinated and managed by WHO ECEH Bonn. HRAPIE drew on hazard assessment and some recommendations for quantification in an earlier, linked project, REVIHAAP (2013), also led and managed by WHO Bonn.

2. USA, for which relevant HIAs have been done by US EPA, in particular the HIA and CBA of the US Clean Air Act (2013) and regulatory impact analysis for proposed new air quality standards for ozone, sulphur dioxide, particulate matter and nitrogen dioxide. In addition, US EPA carries out very detailed assessments of the health effects of individual pollutants, via its series of Integrated Science Assessments.

Both HRAPIE and US EPA give not only pollutant-health combinations, but also (i) one or more recommended concentration-response functions (CRFs), typically showing per cent change in prevalence or incidence of health outcome per unit (e.g. 10 µg.m⁻³) ambient concentration; and (ii) background rates of prevalence or incidence of the recommended health outcome, or how to find them, in countries of the EU and in USA. (Note that while the CRFs are specific to pollutant-outcome pairs, the background rates relate to health outcomes only and also are specific to the domain of application.)

Wider application of these pollutant-health combinations

In order to consider issues that arise in applying the core pollutant-health combinations functions more widely, a preliminary grouping was made of other OECD countries, China and India as follows:

1. Norway, Iceland and Switzerland (considered as similar to existing EU countries)
2. Canada
3. Australia, New Zealand
4. Japan, Korea
5. Israel (including Palestine)
6. Turkey
7. Chile, Mexico
8. China
9. India

The transferability of CRFs to these groups of countries is considered below.

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8. www.epa.gov/ncea/isa/.
1.5.2 Pollutant-health combinations based on the work of the Global Burden of Disease study

As a final step in developing a core set of pollutant-health outcome pairs, the Global Burden of Disease (GBD) project was considered, to see if it suggested any additional pollutant-health combinations that could and arguably should be included in the core set and used for implementation in the various OECD countries, China and India. The GBD is a large-scale and authoritative project which from time to time, and notably for the years of 1990, 2000 and 2010, considers a wide range of health determinants and their effects on population morbidity and mortality in countries and regions across the world as a whole. Outdoor and indoor air pollution were among the clusters of 67 risk factors considered by GBD 2010 (Lim et al., 2012). The GBD assessment of the health effects of air pollution focused on mortality. It did, however, consider morbidity also, to a lesser extent. Compared with HIA work for the EU or USA it has, in relation to the present project, the great advantage that the assessments were conducted worldwide, with results for 21 regions. Consequently, the GBD authors needed to address, and did address, the extent to which their methodology was and is applicable in very different contexts of air pollution, population and health internationally.

2. A core set of pollutant-health combinations

This section presents a core set of pollutant-health combinations, based on major HIAs for EU, USA, and the Global Burden of Disease study.

2.1 Pollutant-morbidity combinations (and associated CRFs)

Europe: The European Union

The most recent comprehensive approach to effects quantification in Europe, implemented in the HRAPIE project (WHO, 2013b), led by WHO ECEH in Bonn, Germany, was to develop (i) a main set of CRFs that are believed to be associated with a relatively high level of certainty when used for quantification and (ii) a secondary set of less certain CRFs for other pollutant-health combinations. While this two-tier approach may be understood as recommending a core set, and a supplementary set for sensitivity analysis, this is not strictly accurate: having given a view on the uncertainties of both sets (the pollutant-health combinations and associated CRFs in the second set being less certain than in the first), HRAPIE leaves it to users to choose which set to use – or to construct a hybrid.

Thus HRAPIE proposed two sets of CRFs:

- Group A: pollutant-outcome pairs for which enough data are available to enable reliable quantification of effects;
- Group B: pollutant-outcome pairs for which there is more uncertainty about the precision of the data used for quantification of effects.

CRFs for morbidity were where practicable based on European studies; sometimes, but by no means always, there was sufficient evidence from studies in Europe to do this. The international evidence is, however, very relevant to assessment of causality, which for the present study is an important aspect, and was considered by WHO in its related and slightly earlier project REVIHAAP (2013a).

CRFs for pollutants and health end-points where there is limited evidence and a high level of uncertainty were not included in quantification of effects. It is very likely that some of the omitted pollutant-health combinations relate to health impacts which are in fact real but not yet supported sufficiently by evidence to include them. Or, put differently, it is highly unlikely that all the omitted pollutant-health combinations are spurious. Consequently, the omission of these pollutant-health
combinations is almost certainly associated with some under-estimation of effects of air pollution on health.

In addition, HRAPIE identified some pollutant-health combinations and associated CRFs from both Group A and Group B as suitable for having their individual effects added to give an aggregate effect and proposed some rules to reduce or eliminate double-counting among these. When these were included and aggregated accordingly, HRAPIE considered that using in addition the other pollutant-health combinations in Groups A and B would lead to significant double-counting. Table 1 below refers only to those combinations that HRAPIE proposed to include in the aggregate; following HRAPIE, the CRF recommended for quantification is indicated by a single asterisk in the final column of the table. The time dimension of the pollutants, typically relating to the characteristic of one day or one year, reflects whether the underlying study was based on short-term exposures (daily variations) or longer-term exposures (annual averages).

Holland (2014)\(^9\) reviewed the implementation of these HRAPIE recommendations for European air pollution cost-benefit analysis (CBA) work, including the derivation of appropriate baseline rates from European databases. In his conclusions he noted that the confidence intervals on CRFs classified as A or B were similar and that the narrowest confidence intervals were for CRFs based on a single study, suggesting the need for caution in using confidence intervals (CIs) as a measure of uncertainty, because results based on two or more good studies are more informative than those based on only one, even when the two or more studies give different results and hence a wider CI.

An earlier European assessment for the Clean Air for Europe (CAFE) programme 2004-6, also intended for application EU-wide, identified pollutant-outcome pairs and CRFs (Hurley et al., 2005) some, though not all, of which are the same as or similar to those recommended by HRAPIE. In case of doubt, precedence should be given to HRAPIE which is not only more recent, but also had a wider authorship and review. However, for completeness, the CAFE pollutant-outcomes are given in Annex 1.

### Table 1. Morbidity pollutant-health combinations recommended by HRAPIE for inclusion

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Health outcome</th>
<th>Applicability</th>
<th>Reliability / independent contribution to effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PM</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annual mean PM$_{10}$</td>
<td>Prevalence acute “bronchitis”</td>
<td>Children 6-12 (or 6-18 years); All concentrations</td>
<td>B*</td>
</tr>
<tr>
<td>Annual mean PM$_{10}$</td>
<td>Incidence of chronic bronchitis</td>
<td>Adults 18+ years; All concentrations</td>
<td>B*</td>
</tr>
<tr>
<td>Daily mean PM$_{2.5}$</td>
<td>Cardiovascular hospital admissions</td>
<td>All ages, all concentrations</td>
<td>A*</td>
</tr>
<tr>
<td>Daily mean PM$_{2.5}$</td>
<td>Respiratory hospital admissions</td>
<td>All ages, all concentrations</td>
<td>A*</td>
</tr>
<tr>
<td>2 week average PM$_{2.5}$ converted to annual mean</td>
<td>Restricted activity days (RADs)</td>
<td>All ages, all concentrations</td>
<td>B*²³</td>
</tr>
<tr>
<td>2 week average PM$_{2.5}$ converted to annual mean</td>
<td>Work loss days (WLD)</td>
<td>Adults 20-65 years, all concentrations</td>
<td>B*</td>
</tr>
<tr>
<td>Daily mean PM$_{10}$</td>
<td>Incidence of asthma symptoms</td>
<td>Asthmatic children 5-15 years, all concentrations</td>
<td>B*</td>
</tr>
<tr>
<td><strong>Ozone</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily maximum 8 hour mean O$_3$</td>
<td>Cardiovascular hospital admissions excluding stroke</td>
<td>Age 65+, &gt;35 ppb</td>
<td>A*</td>
</tr>
<tr>
<td>Daily maximum 8 hour mean O$_3$</td>
<td>Respiratory hospital admissions</td>
<td>Age 65+, &gt;35 ppb</td>
<td>A*</td>
</tr>
<tr>
<td>Daily maximum 8 hour mean O$_3$</td>
<td>Minor RADs (Restricted Activity Days)</td>
<td>All ages, &gt;35 ppb</td>
<td>B*</td>
</tr>
<tr>
<td><strong>NO$_2$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annual mean NO$_2$</td>
<td>Prevalence of bronchitic symptoms</td>
<td>Asthmatic children aged 5-14 years, all concentrations</td>
<td>B*</td>
</tr>
<tr>
<td>24 hour mean NO$_2$</td>
<td>Respiratory hospital admissions</td>
<td>All ages, all concentrations</td>
<td>A*</td>
</tr>
</tbody>
</table>

* Only residual Restricted Activity Days (RADs) to be added to total effect after subtraction of days in hospital, Work Loss Days (WLD) and symptom days.

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**USA**

US EPA (2011) reports on a major analysis of the benefits and costs of the *Clean Air Act* from 1990 to 2020. Nationally representative age-specific incidence and prevalence rates were used, where available, for each health end-point and that these were derived from a variety of sources, such as the CDC, the National Center for Health Statistics and the American Lung Association. Details of the CRFs and of the literature sources underlying them are given in a supplementary report (Industrial Economics, Incorporated, 2011) which also provides details of the baseline rates of incidence/prevalence for morbidity end-points in the US context. However, the present report draws only superficially on these details because the focus here is on pollutant-health combinations, not on specific CRFs or background rates.

The pollutant-health combinations, pollutant metrics, and population groups for which impacts were calculated for each pollutant-health pair, are shown in Table 2, below.

In its analyses of the costs and benefits 1990-2020 of the US *Clean Air Act*, US EPA has included a wider range of end-points than the EU HRAPIE assessments. CRFs are based exclusively on US studies. Quantification was for PM$_{2.5}$ and O$_3$ only, not for NO$_2$.

The earlier final regulatory impact analysis for NO$_2$ air quality standards (US EPA, 2010a) did not include any direct health benefits associated with reductions in NO$_2$ levels because of the unknown population exposure to NO$_2$ in near-road environments; the assessment was based on the co-benefits to health of reducing PM$_{2.5}$. However, in preparatory analyses, US EPA (2009a, Chapter 5) did evaluate directly the health benefits of NO$_2$ reduction. Its choice of pollutant-health combinations was guided by an
earlier, 2008, Integrated Science Assessment which concluded that “recent studies provide scientific evidence that is sufficient to infer a likely causal relationship between short-term NO₂ exposure and adverse effects on the respiratory system”. On that basis, and taking into account the feasibility of quantification, US EPA (2009) quantified the effects of short-term exposure to (or daily variations in) NO₂ in relation to hospital admissions for asthma and for chronic lung disease, asthma Emergency Room visits, asthma exacerbation, and acute respiratory symptoms. Health endpoints considered relevant to NO₂, but not quantified (because of lack of consensus on causality or difficulties in quantification), included premature mortality, pulmonary function, other respiratory emergency department visits and other respiratory hospital admissions.

Table 2. Pollutant-health pairings used by US EPA for cost-benefit analysis - US Clean Air Act

<table>
<thead>
<tr>
<th>Pollutant metric</th>
<th>Health outcome</th>
<th>Application sub-population</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM, all as PM₂.₅</td>
<td>Chronic bronchitis</td>
<td>&gt;26 years</td>
</tr>
<tr>
<td>PM₂.₅ annual average</td>
<td>Nonfatal myocardial infarction</td>
<td>Adults (&gt;18 years)</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Respiratory hospital admissions</td>
<td>&gt;64 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Respiratory hospital admissions</td>
<td>20-64 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Respiratory hospital admissions</td>
<td>&lt;65 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Cardiovascular hospital admissions</td>
<td>&gt;64 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Asthma-related Emergency Room visits</td>
<td>&lt;18 years</td>
</tr>
<tr>
<td>PM₂.₅ annual average</td>
<td>Acute bronchitis</td>
<td>8-12 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Lower respiratory symptoms</td>
<td>7-14 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Upper respiratory symptoms</td>
<td>9-11 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Asthma exacerbation</td>
<td>6-18 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Minor restricted activity days</td>
<td>18-64 years</td>
</tr>
<tr>
<td>PM₂.₅ 24 hour average</td>
<td>Work loss days</td>
<td>18-64 years</td>
</tr>
<tr>
<td>O₃ 8 hour maximum</td>
<td>Respiratory hospital admissions</td>
<td>&gt;64 years</td>
</tr>
<tr>
<td>O₃ 8 hour maximum</td>
<td>Respiratory hospital admissions</td>
<td>&lt;2 years</td>
</tr>
<tr>
<td>O₃ 8 hour maximum</td>
<td>Asthma related Emergency Room visits</td>
<td>5-34 years</td>
</tr>
<tr>
<td>O₃ 24 hour average</td>
<td>Minor restricted activity days</td>
<td>18-64 years</td>
</tr>
<tr>
<td>O₃ 8 hour average</td>
<td>School loss days</td>
<td>5-17 years</td>
</tr>
<tr>
<td>O₃ 8 hour maximum</td>
<td>Reduced worker productivity</td>
<td>18-64 years</td>
</tr>
</tbody>
</table>

The pollutant-health combinations (and associated CRFs) for ozone and PM₂.₅ were updated in the more recent regulatory impact assessment (RIA) of ozone, undertaken to support standard-setting (US EPA, 2014). The health impacts evaluated for O₃ in the core analysis (morbidity) were respiratory hospital admissions (65y+), asthma-related emergency department visits (all ages), asthma exacerbations (6-18y), school loss days (5-17y) and acute respiratory symptoms as minor restricted activity days (MRADs) (18-65y); i.e. were similar, but not identical to those used in the earlier analysis of the Clean Air Act.

Similarly, the health impacts evaluated for PM₂.₅ in the core analysis (morbidity) were almost exactly those used in the analysis of the Clean Air Act, i.e. non-fatal heart attacks, respiratory hospital admissions, cardiovascular hospital admissions, asthma-related emergency department visits, asthma exacerbations, Work Loss Days (WLDs), acute respiratory symptoms as MRADs, upper respiratory symptoms in children with asthma, and lower respiratory symptoms in children.

The one difference, and it is an important one, is that of chronic bronchitis in adults. For many years, the combination of PM and chronic bronchitis has been an important pathway for quantification both in USA and in Europe. However, in recent years US EPA has moved away from including it in its primary
(core) analyses, including it either as sensitivity (US EPA, 2012) or not at all (US EPA, 2014). The justification for this change is given in US EPA (2012) and is discussed later.

Global Burden of Disease evaluation

The main results from the 2010 Global Burden of Disease (GBD) evaluation (Lim et al., 2012) include effects of ambient air pollution on human health. Most of the pollutant-health pairings included relate to cause-specific mortality. Thus, for PM (in the metric of PM$_{2.5}$), GBD includes effects on mortality from ischaemic heart disease (IHD) and cerebrovascular disease, from chronic non-malignant lung disease (COPD: chronic obstructive pulmonary disease) and from lung cancer (cancer of trachea, bronchus and lung); and for ozone, GBD includes effects on mortality from COPD (Lim et al., 2012).

Effects of PM on morbidity were quantified using the pollutant-health pairing of annual average PM$_{2.5}$ and lower respiratory infections (LRI), sometimes also called acute LRI (ALRI). These were quantified in children only, and (as with mortality) with a “theoretical minimum risk exposure distribution” of 5.8 – 8.8 µg.m$^{-3}$ annual average PM$_{2.5}$. Details of the corresponding meta-analysis of studies linking PM$_{2.5}$ and ALRI are given in Mehta et al. (2013); this underpins the more detailed descriptions below. Effects of ozone on morbidity were not quantified (Lim et al., 2012).

As explained earlier, because of the wide geographical reach of GBD, its recommendations were given particular consideration for the present report, even when they were not directly supported by HIAs in Europe or USA; and consequently the combination PM$_{2.5}$ and ALRI was included as a potential core pollutant-health combination for people aged <5 years.

The proposed core set of pollutant-health combinations

The paragraphs above show that HIA work in Europe and by US EPA use some similar and some different pollutant-health combinations for morbidity. There are four main areas of similarity:

1. Respiratory Hospital Admissions (RHA) and Cardiovascular Hospital Admissions (CVHA) in relation to PM and to ozone;
2. Restricted Activity Days (RADs) and associated Work Loss Days (WLDs) in relation to PM and/or ozone;
3. Chronic Bronchitis in adults in relation to PM only; and
4. Acute Bronchitis in children aged 6-12 or 6-18 years, defined as “bronchitis in the past 12 months” (Hoek et al., 2012) based on responses to symptoms questionnaires.

Note that of these, only the pairings of PM and ozone with hospital admissions were considered by HRAPIE as being among the relationships that could be estimated with greater levels of confidence; and US EPA also has recently down-graded its confidence in the combination of PM and chronic bronchitis (see later).

In addition, from the GBD study, a fifth pollutant-health pair was considered, for reasons given above:

5. Acute Lower Respiratory Illness (ALRI) in children aged <5 years.

A question was whether to include both sets of respiratory conditions in children, bearing in mind that illness also implied days of restricted activity in children. Although ALRI was proposed by GBD only, it was considered relevant that the condition was often severe (see below), that “a substantial fraction of the burden is experienced by populations in Africa and Asia” (Mehta et al., 2013); and that GBD 2010 had already succeeded in quantifying this pollutant-health outcome pair in 21 regions globally.
The core set was therefore selected to include all 5 health outcomes, i.e. to include both ALRI in younger children and acute bronchitis in older children, principally because there is evidence to support each of the two pairs, one has been used by both WHO and USEPA and the other by GBD internationally, and there is not a risk of double-counting because they refer to non-overlapping age groups.

2.2 Additional notes on core morbidity outcomes and causality

Hospital admissions

There was no doubt, in the expert evaluations of either WHO / EU or USEPA, that some effects of air pollution on hospital admissions should be included. This is because there is a wealth of relevant evidence from well-conducted time-series studies in locations (typically in cities) in Europe, in North America, and in many locations elsewhere in the world; and the association is widely understood as causal. On the basis of that extensive evidence:

- HRAPIE and US EPA agreed on quantifying RHA and CVHA in relation to PM;
- They agreed also on quantifying RHA in relation to ozone;
- HRAPIE in Europe also included ozone-CVHA, but US EPA did not. Because of likely under-estimation elsewhere, the pairing O₃-CVHA was included also in the present report;
- HRAPIE also includes RHA in relation to NO₂ as noted previously, US EPA did not include NO₂ in its analysis of the US Clean Air Act.

Note however that while the epidemiological evidence in support of including hospital admissions is very strong, major HIAs conducted in Europe and in the USA have shown that it does not have major influence on the outcome of cost-benefit analyses. Specifically, the monetised impact of RHAs and CVHAs is but a very small proportion of the total monetised damage attributable to outdoor air pollution or, equivalently, the monetised benefits of outdoor air pollution reduction. In the US EPA analysis of the Clean Air Act, avoided emergency hospital admissions accounted for <1% of the total benefit; in an analysis of the Gothenburg Protocol for the European Union, avoided hospital admissions due to PM₁₅ accounted for 0.06% of the total health benefit, and in the cost-benefit analysis undertaken to support revision of the National Air Quality Strategy in the UK, avoided hospital admissions accounted for <1.2% of the total benefit (IGCB, 2008). It follows that the weight of epidemiological evidence in favour of including hospital admissions as a health outcome should not be confused with thinking that its inclusion, or not, will make much difference to cost-benefit analysis results. Effects on other health outcomes, such as chronic bronchitis or Work Loss Days, for which the supporting epidemiological evidence is much weaker, have (when quantified and monetised) proven to be much more influential than hospital admissions.

A partial explanation may be that hospital admissions are likely to capture only a small proportion of the total acute morbidity associated with air pollution. It would be expected that in addition to those who became ill enough to warrant hospital treatment, there would also be (as indicated by Tables 1 and 2; see also below) a population who experienced a range of adverse effects on respiratory or cardiovascular health, but of insufficient severity to require hospital treatment; and it is difficult to capture quantitatively these wider effects in ways that can be applied with confidence internationally.

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Restricted Activity Days (RADs) and Work Loss Days (WLDs)

The EU (or, more correctly, WHO in its HRAPIE [WHO, 2013b] evaluations in support of the EU policy development) has recommended CRFs for these end-points based on the findings of a set of studies undertaken in the United States during the 1980s (e.g. Ostro, 1987; Ostro and Rothschild, 1989). From the viewpoint of causality, their inclusion is based principally on an argument of coherence rather than on direct evidence: given that ambient air pollution adversely affects both more serious health outcomes, notably mortality and hospital admissions, and milder ones, such as lung function and heart arrhythmia, it is logical that it affects intermediate cardio-respiratory health outcomes also.

HRAPIE recommended quantification of RADs and minor RADs at all ages, even though the core underlying studies were based principally on adults up to age 64 only. For reasons to do with employment practice, HRAPIE recommended quantification at ages 15-64 only for WLDs. Quantification of all three was assessed by HRAPIE among the less reliable Group B of quantifiable health outcomes. The earlier EU-wide CAFE assessment had included these pollutant-health outcomes as core ones for the age group 15-64, though with comments on relatively high uncertainty. CRFs for RADs and MRADs extrapolated to people of all ages were included as sensitivity analyses only. Their exclusion from core analyses is likely to lead to some under-estimation of the total effect. These CRFs have been included in US EPA’s impact assessments in which uncertainty was assessed on the basis of the statistical significance of the findings in the source studies.

RADs are defined as days on which individuals felt sufficiently unwell not to carry out their normal range of activities and are not limited to respiratory or cardiac symptoms that might be directly attributable to air pollution. National surveys have investigated the background incidence of RADs in some countries and background rates of WLDs would be available for many economies. Background rates of both RADs and WLDs are likely to vary by location and may have varied through time. Differences in cultural attitudes, the type of activities people undertake and demographics are likely to lead to differences in the interpretation of and incidence of RADs. Similarly, background rates of WLDs are likely to be influenced by social security arrangements, economic conditions and types of employment. These considerations give rise to uncertainty as to the general applicability of CRFs for RADs and WLDs based on studies undertaken in the US in the 1980s to other parts of the world in the second and third decades of the 21st century.

Incidence of chronic bronchitis in adults

HRAPIE based its quantification on two studies, AHSMOG in California, USA (Abbey et al., 1995a and 1995b) and the SAPALDIA study in Switzerland in Europe (Schindler et al., 2009). Both AHSMOG and SAPALDIA had identified presence of chronic bronchitis from a questionnaire of self-reported respiratory symptoms and both had used the same definition: reported occurrence of chronic cough or chronic phlegm (rather than chronic cough and phlegm) for at least three months of the year for two years.

Because the SAPALDIA results estimated a higher relative risk than those of AHSMOG, the new EU HRAPIE recommendations are for a steeper CRF than used in CAFE or by the US EPA. On the other hand, HRAPIE assessed the reliability of the recommended quantification of chronic bronchitis as Group B only. There were three reasons for this. First, a then-recent review (Schikowski et al., 2014) found that the evidence for an effect of PM on incidence or prevalence of chronic bronchitis was suggestive, but not convincing. Second, choice of AHSMOG and SAPALDIA as the basis for quantification was not based on review or meta-analysis of the literature as a whole. And finally, there are differences between SAPALDIA and AHSMOG in how the relevant exposure periods are defined and combination of the CRFs is therefore not straightforward.
In recent years US EPA has also questioned the evidence-base underlying PM and chronic bronchitis and has moved away from including chronic bronchitis in its primary analysis. US EPA (2012) explains that whereas some studies in the 1990s provide evidence that long-term PM$_{2.5}$ exposure gives rise to the development of chronic bronchitis in adults in the United States, the absence of newer studies finding a relationship between long-term PM$_{2.5}$ exposure and chronic bronchitis argues for moving this endpoint from the core benefits analysis to a sensitivity analysis. Additionally, in their review of the scientific literature on chronic obstructive pulmonary disease (COPD), which includes chronic bronchitis and emphysema, the American Thoracic Society concluded that air pollution is “associated with COPD, but sufficient criteria for causation were not met” (Eisner et al., 2010).

**Acute bronchitis in children**

The pollutant-health outcome of annual average PM$_{10}$ and acute bronchitis in children was included by HRAPIE based on an analysis by Hoek et al. (2012) of data in the PATY study from over 45,000 children aged 6-12 years from ten European countries and North America. Acute bronchitis was defined as “bronchitis in the past 12 months” and was identified by responses to questionnaires. The exact wording of the relevant questions in the PATY study varied a little between countries but typically acute bronchitis referred to doctor-diagnosed bronchitis (Gehring et al., 2006, Appendix 1). Hoek et al. (2012) found that the main concentration-response function linking annual average PM$_{10}$ and acute bronchitis was almost statistically significant at the conventional 5% level. HRAPIE (WHO, 2013b) recommended using it at ages 6-12 or for older young people also, e.g. 6-18 years, if background rates were available only for this wider age group.

Additionally, HRAPIE recommended including, in relation to NO$_2$, the health outcome of (prevalence of) bronchitic symptoms in children 5-14 years with asthma. The age-group is very similar to that for PM, but the pollutant-health combination is to be applied to children with asthma only. Also, the definition of health outcome is different – it required bronchitic symptoms for at least three consecutive months in the past year (WHO 2013b, following McConnell et al., 2003). Implementation is complicated, because background data are needed on prevalence of asthma in children as well as prevalence of sustained bronchitic symptoms in children with asthma; effects focus on a relatively small (though important) number of people; and quantification of NO$_2$ internationally is not yet common in major HIAs. For these reasons, the present report did not focus further on this pollutant-health combination.

**Lower respiratory infections in children aged less than 5 years**

The GBD estimates were based on the meta-analysis by Mehta et al. (2013). There, ALRI is described as “including pneumonia and bronchiolitis of bacterial and viral origin”; as being “nearly always diagnosed clinically, based on severe respiratory symptoms” and as being “characterized by acute-onset cough or difficulty in breathing with fast breathing for age”; severe ALRI was defined as “acute cough or difficulty in breathing with in drawing of the lower chest wall necessitating hospital admission”. These definitions and descriptions indicate a range of severity and indeed ALRI is reported as causing “one fifth of deaths in children under the age of 5 years, with 90% of ALRI deaths being directly attributable to pneumonia”.

**Purpose, status and structure of the rest of this Chapter**

On the basis of the above discussion, the chapter now considers four main sets of issues. First, in Sections 2.3 to 2.6, it considers the transferability of the relevant pollutant-health combinations (and as appropriate associated CRFs) to other groups of OECD countries, to China, India and more widely. Whereas the focus of the present report is on pollutant-health combinations, these Sections consider also the more complex related issue of transferability of CRFs. The discussion in the Sections is intended to raise issues that local practitioners should consider when thinking of implementing a health impact
assessment using the pollutant-health combinations proposed here. It is not intended as a definitive discussion of all aspects of transferability of CRFs.

In order to derive estimates of impact, both CRFs and baseline rates for the health end-points of concern are required. Factors that may affect the transferability of CRFs include:

- Choice of pollutant and metric;
- Similarity or not of pollution mixture – similarity of sources, kinds of PM, interaction between air pollution and climate, concentrations;
- Similarity or not of population – demography and especially disease profile; also cultural issues affecting reporting of symptoms (for RADs and for Chronic Bronchitis)
- Similarity or not of healthcare systems.

Second, Section 2.7 reports results from a limited review of health impact assessments carried out in OECD countries. This was carried out to answer two specific questions: To the extent that HIAs of outdoor air pollution have been carried out in countries outside of the EU and USA, then:

- To what extent have the authors used the same pollutant-health combinations as proposed in the present report, i.e. to what extent have they considered these transferable and implementable? And
- Do these wider international HIAs suggest any other pollutant-health combinations that should be considered as priority for the present report?

The literature review to answer this question was not intended to be systematic or even comprehensive; for example, only publications in English were considered. Consequently, some relevant studies will have been missed. It is unlikely, however, that pollutant-health combinations of wide general applicability have been missed, and if so the limited review has achieved its main purpose. In addition, it gives an impression of the “state-of-the art” of air pollution HIA across the OECD countries, China and India, which could serve as a starting-point for a more comprehensive survey, if required.

Thirdly, Section 2.8 considers the likely level of under-estimation implied by (i) dis-regarding the combinations of pollutant and health outcome that are included in e.g. HIAs by and for US EPA but not in Europe; and conversely; and (ii) other aspects of likely under-estimation of morbidity effects of outdoor air pollution.

Finally, Section 2.9 draws some working conclusions and recommendations based on the work of the Chapter as a whole.

### 2.3 Transferability: the role of pollution metrics

**PM**

HRAPIE uses PM$_{2.5}$ where it can and sometimes uses a conversion factor to go from a CRF in PM$_{10}$ to one in PM$_{2.5}$, or conversely, “based on an estimated 65% of PM$_{10}$ being in the PM$_{2.5}$ size range. This PM$_{2.5}$/PM$_{10}$ ratio of 0.65 is considered an average for the European population; however, in specific locations the ratio may be in the range 0.4-0.8 and a local estimate would be preferable for the conversion”.\(^{11}\) The US EPA has recently used PM$_{2.5}$ where practicable, including in its analysis of the US Clean Air Act.

\(^{11}\) WHO Regional Office for Europe (2013), pp 3.
There is no threshold for the application of the morbidity CRFs in PM, in European applications or in regulatory impact assessments undertaken by the US EPA. However, in Europe, the CRFs are applied to anthropogenic PM only. In impact analyses where the US EPA is comparing the benefits of different scenarios, CRFs have effectively only been applied to anthropogenic PM also, because only anthropogenic PM can be affected by policies.

Considering now transferability to other countries in OECD, China, India and elsewhere, there is a major drawback in recommending a function in PM$_{2.5}$ if there are not enough background measurements in PM$_{2.5}$ to allow the CRFs to be used.

It would be necessary to establish if there are, throughout the countries of interest, sufficient data in PM$_{2.5}$ to be useful. This has not been checked specifically for the present project; and indeed a recommendation of CRFs is outside of the scope of the work. Almost certainly, however, in many relevant countries there is a better network of measurement in PM$_{10}$, because it has been highlighted as a problem and so has been regulated for much longer, with resulting greater measurement. Under those circumstances, it would be helpful if any recommendations for CRFs which in Europe and USA are implemented in the metric of PM$_{2.5}$ could be given in the metric of PM$_{10}$ based on conversion factors relevant to the geography of the source study.

In terms of time-averaging, effects of PM are quantified either on the basis of annual average or 24-hourly daily average concentrations. In practice, annual average is used because when (as here) the CRF is linear and without a threshold, then impacts over one year can be calculated based on annual average values – there is no need to calculate impacts for each day and then aggregate the results (Hurley et al., 2005).

Ozone

Regarding ozone, there are several issues, including metric, CRF and threshold (or not).

Ozone is created in the presence of sunlight and so concentrations can vary markedly within a day; choice of measurement and averaging time therefore do matter. In CAFE, CRFs were used in the metric of 8-hourly daily maximum O$_3$, i.e. the daily maximum surface ozone concentration, usually measured in parts per billion (ppb), backward averaged over 8 hours, although this involved “translating” some CRFs which originally were in the metric of 1-hourly daily maximum or 24-hourly daily average. While it was considered that there was no strong evidence of a threshold or “safe level” for the population as a whole, there was a lack of suitable studies when daily concentrations were low. Based on WHO guidance to another EU project (RAINS) developed at the Task Force on Health (TFH) of the UNECE Convention on Long-Range Transport of Air Pollution (CLRTAP), it was considered prudent not to quantify effects on days when daily 8-hourly maximum O$_3$ was lower than 35 ppb; and, on other days, to quantify only for that portion of daily 8-hourly maximum that exceeded 35 ppb. This advice was based on uncertainties in the shape of CRF at very low ozone concentrations and in the modelled estimates of ozone concentrations produced by atmospheric models at low concentrations. This led to an index known as SOMO35 which aggregates, over the course of a year, the relevant ozone concentrations (8-hour daily max) above 35 ppb (WHO, 2008).

HRAPIE (WHO, 2013b) recommended that O$_3$ effects in the main analysis are quantified only for days when the 8-hour maximum exceeds 35 ppb for the main analysis and that effects are quantified for days when the 8-hour maximum exceeds 10 ppb as part of the sensitivity analysis. The US EPA also uses

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8-hour daily maximum O\textsubscript{3} as the metric for CRFs (which involved conversion of some CRFs based on 1 hour and 24 hour mean concentrations of O\textsubscript{3}). Although thresholds are considered for the CRFs for mortality in US EPA’s Regulatory Impact Assessment (RIA) for an O\textsubscript{3} standard, the CRFs for morbidity are presented without thresholds, but this may be because of the nature of the impact assessment (quantifying the benefit of removing the highest exposures). For the purposes of calculating the benefit of removing exposures above different levels, it would not have been necessary to consider exposures as low as 35 ppb. It seems reasonable to maintain this convention of quantifying only when 8-hour daily maximum O\textsubscript{3} exceeds 35 ppb.

\textbf{NO\textsubscript{2}}

As for PM, the preferred averaging time for studies of the impact of short-term exposure on e.g. respiratory hospital admissions is 24-hourly average while that for longer-term exposure is annual average.

\textbf{2.4 Transferability: effects on CRFs of the pollution mixture}

\textit{Differences in air pollution mixture internationally and their implications for transferability}

Transport, principally road transport but shipping also, is a major source of emissions for the air pollution mixture across much of Europe and North America, contributing primary particles, NO\textsubscript{2} and other pollutants to local populations, and transboundary pollutants. However, climatic differences mean that photochemical secondary pollutants, such as ozone and aerosol acidity, are likely to be more important across large areas of North America than in Western Europe. Also, large areas of North East USA and South East Canada are affected by pollution from industrial sources, with sulphur dioxide (SO\textsubscript{2}) and sulphates being relatively more important than in, say, Western Europe, which has fewer industrial sources. There is, however, high industrial pollution in parts of Eastern Europe.

The air pollution mix in urban areas of other developed economies, such as Australia and New Zealand, is broadly similar to that in Western Europe. Traffic emissions and secondary pollutants, such as ozone and aerosol acidity, are also likely to dominate the air pollution mixture in Latin America, Turkey and Israel but with a potentially larger component of windblown dust and salt in arid climates than in the humid climates of Western Europe. The air pollution mix in India and China has a substantially bigger contribution from coal and biofuel combustion and less efficient pollutant abatement on all emissions sources than in Western Europe, giving rise to higher levels of particulate matter, sulphur dioxide and other pollutants than in more developed economies.

From the viewpoint of the present project, the pollutant-health combinations identified in relation to PM, O\textsubscript{3} and NO\textsubscript{2} remain relevant in terms of causality despite differences in air pollution mixture, though their significance will vary according to concentration levels and demographic factors. The “state-of-the-art” has not developed sufficiently to enable a reliable assessment of what effect such differences in air pollution mixtures might have on the transferability of CRFs for individual pollutant-health combinations and of how well a set of pollutant-health combinations might express the effect of an outdoor air pollution mixture from different sources. This is because the interactions between different components of the air pollution mix in giving rise to adverse effects are only partially understood, as is the relative potency of particulate matter of different composition or derived from different sources within a particular size range. On this very important topic it is the established position of various expert groups (WHO in REVIHAAP [WHO, 2013a]; US EPA in its HIA of the Clean Air Act [US EPA, 2011]; COMEAP in the UK [COMEAP, 2015]) that it is not possible, for PM of a given size-range, to differentiate reliably between components of PM in terms of their relative toxicity.
Results from time-series studies of mortality and hospital admissions in different countries and regions show a strong consistency in finding adverse effects of key pollutants, together with differences, often unexplained, in the size of actual coefficients estimated (see e.g. Atkinson et al., 2014, discussed later). There is, however, evidence from studies of both long-term and short-term exposure, especially of mortality, that percentage change per µg/m³ pollutant is smaller at higher concentrations of PM than at those typically found in USA or Western Europe, and this cautions against simple extrapolation of CRFs from Europe or the USA to conditions of much higher PM air pollution.

*Implications for pollutant-health combinations – a role for SO₂?*

SO₂ is an important component of the pollution mixture in some regions internationally and so it is reasonable to consider its impact on morbidity and the possibility of quantifying these. This issue has not been examined in detail in the present project, because it is not included in the major international HIAs on which the present work is based. There follows nevertheless a brief summary of US EPA practice on quantifying the health effects of SO₂.

In their impact analysis of proposed SO₂ air quality standards, US EPA (2010) quantified four morbidity health outcomes as “sufficient to infer a likely causal relationship”: respiratory hospital admissions (RHA), emergency room visits (ERVs) for asthma, asthma exacerbation and acute respiratory symptoms. The effects quantified are of short-term exposure (“daily variations”). Other likely effects of short-term exposure (on e.g. premature mortality, pulmonary function, other respiratory emergency department visits and other respiratory hospital admissions) were not quantified because either “there is not consensus on causality [or] causality has been determined but empirical data are not available to allow calculation of benefits” (US EPA, 2010). An earlier publication (US EPA, 2009) reviewed evidence from laboratory studies from very short, 5- or 10-minute, exposures.

<table>
<thead>
<tr>
<th>Health end-point</th>
<th>Application</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory hospital admissions</td>
<td>65-99</td>
</tr>
<tr>
<td>Emergency department visits for asthma</td>
<td>All ages</td>
</tr>
<tr>
<td>Asthma exacerbations</td>
<td>4-12</td>
</tr>
<tr>
<td>Acute respiratory symptoms</td>
<td>7-14</td>
</tr>
</tbody>
</table>

The exact SO₂ metric used in the impact assessment is not clearly stated but is likely to have been the 1-hour daily maximum, in order to provide consistency with the regulatory standard. US EPA (2010) notes, however, that the underlying epidemiological studies used a range of metrics for daily SO₂, including 24-hr mean, 3-hr mean, 8-hr max, and 1-hr max. PM₂.₅ co-benefits were also calculated because SO₂ is a precursor of PM.

### 2.5 Transferability: effects of differences in population and health

*Effect on CRFs of population differences and differences in healthcare systems*

Population differences (within countries as well as across countries) impact on the baseline rates for health, including the health or health-related outcomes of the core set of pollutant-health pairs recommended here; potentially they also may affect vulnerability to the effects of air pollution. It is also likely that the health end-points evaluated in HIAs in Western Europe and North America do not equate exactly to health end-points that may be measured elsewhere. Because of differences in healthcare systems and cultural differences in patterns of work, the equivalence may sometimes be poor, for example in assessing a Restricted Activity Day, and in reporting of respiratory symptoms.

The age-structure and baseline health of the population in Western Europe, North America, Australia and New Zealand would be expected to be broadly similar. The age-structure and baseline health of
populations elsewhere is different and this will affect the baseline incidence and prevalence of morbidity from conditions such as chronic bronchitis and heart disease that are linked with air pollution. There are likely to be cultural differences in the recognition of symptoms and RADs, and there are likely to be both cultural and economic differences in attitudes towards sickness absence that may impact on both baseline rates and the steepness of the CRF. In addition, differences in the type of work (e.g. higher levels of employment in manual work) might impact on both baseline sickness absence and on the likelihood that air pollution related ill health of a particular severity would result in a day off work.

Healthcare systems are organised differently across countries and do not necessarily involve a clear separation of primary versus secondary care such that hospital visits in some parts of the world may equate to a visit to a primary care practice in Europe or North America. Healthcare may or may not be free at the point of use. Both costs and cultural factors are likely to affect how populations use health care services, particularly in relation to minor ailments. Rates of healthcare usage are likely to be highly variable by country and region, and quality and availability of recording is likely also to be highly variable internationally. It is unclear whether in principle resultant differences in background rates of outcomes such as emergency hospital admissions will affect also the pollutant-outcome CRFs, typically expressed as the relative (i.e. percentage) change in usage in response to differences in daily pollutant concentrations. It does not necessarily follow that the CRFs will be affected, but the situation is strictly unknown unless relevant studies have been carried out. Fortunately, there is a wider evidence-base internationally on studies of air pollutants and hospital admissions than for any other morbidity endpoint, so that issues of transferability of CRFs (which are not the main focus of the present report) can be examined directly. As noted earlier, times series studies of daily PM$_{2.5}$ and hospital admissions do show important regional differences in estimated CRFs, though it is unclear whether or not these differences are associated with differences in background rates.

**Background rates**

One requirement for pollutant-health combinations (and associated CRFs) to be transferable is that baseline rates for the health outcomes of concern can be found or estimated well enough to be used. A detailed evaluation of the availability of background rates is outside the scope of the present study. The following remarks may, however, be helpful.

Whereas information about healthcare usage, such as hospital admissions, is likely to be collected in most countries, though not necessarily publicly available, information about symptoms, RADs and conditions such as chronic bronchitis is not routinely collected. In the HRAPIE (WHO, 2013b) project, sources of background rates were found for Western European countries, including health-related end-points such as RADs, but considerable variability between countries exists for some end-points and there may be significant uncertainty in transferring such background rates to outside Europe.

It would be very helpful if it were possible to access reliable information on background rates in the many OECD countries (+ China and India) where the CRFs might be applied. The present report has not attempted to access background rates (it was outside of the remit and resources available) but it is likely that at least some relevant data are available on hospital admissions, though the work involved in accessing them may be substantial. It may also be possible to estimate or guesstimate background rates of chronic bronchitis, though it may be that insofar as data are available for chronic respiratory disease, this relates to COPD of various severities. It will, however, be difficult to find useful data on RADs for many of the countries of OECD, China, India and in other parts of the World.

There is an alternative approach, though the uncertainties involved are difficult to estimate and may be large if population and health differences are large also. In the CAFE project, “impact functions” were derived by integrating across both CRFs and background rates in locations where the underlying
epidemiological studies were conducted (when, as is often but not always done, the underlying epidemiological studies reported the background rates in the populations studied), to give number of cases per year per µg.m\(^{-3}\) PM or O\(_3\). Transferring such an impact function rather than the CRF as percentage change could be very suspect if the background rates are likely to be very different in the populations originally studied and those where the HIA is to be conducted, but it may be the best that is possible, other than estimating a zero effect which would be known to be untrue.

**Commentary**

As noted in the previous paragraphs, it is not really known what effect these differences in demography, culture and health care practice have on the transferability of pollution-health outcomes, on associated CRFs and on background rates of the various health outcomes. From the viewpoint of causality, the pollutant-health combination should in principle be transferable: there are no strong reasons why the fundamental issue of causality would change from one region to another. Even though a particular pollutant-health combination (“pathway”) may be much more important and relevant in one location compared to another, it should nevertheless be valid in both. That is the key conclusion for the present report, with its primary aim of recommending pollutant-health combinations.

### 2.6 Choice of CRFs: Using local and international evidence

The present report focuses on international evidence and recommends pollutant-health pairs on that basis. Both the evidence itself and the major HIAs that use it are dominated by studies from North America and Europe. There are, however, many primary studies, especially in epidemiology, from other countries around the world. The present section considers briefly the nature of that evidence and how it might be used, focusing on the availability of CRFs based on local epidemiological studies performed within the country (or region), and on the choice of CRFs for use in HIA in OECD countries, China and India. It in particular considers, in general terms, the question: should one use local or international evidence for local HIA? Some empirical answers about what is done in HIAs in various countries is given in Section 2.7, following.

**The availability of CRFs based on local epidemiological studies**

Countries across the world vary greatly in the extent to which good local studies of air pollution impacts on health have been conducted, particularly in relation to morbidity rather than mortality. The studies that have been undertaken are generally confined to fluctuations in health-care demand: daily changes in (i) hospital admissions, especially from various respiratory and cardiovascular causes, and (ii) emergency room visits or walk-in outpatient clinics. These health-care end-points are relatively easy to ascertain and to analyse in time-series studies because they work with aggregate population numbers (e.g. daily number of hospital admissions in a particular city), without a need to track identified individuals.

Studies of the effects of air pollution on less severe health-related changes, such as respiratory symptoms, lung function and heart rate variability, have been less widely conducted, partly because they require follow-up over time of panels of identified individuals. Sometimes also the health end-points are less well defined and are more difficult to interpret for impact analysis. The impact of air pollution on other behavioural acute effects, such as RADs and WLDs, does not appear to have been widely investigated beyond a series of studies undertaken in California, USA during the 1980s, even though HIAs which include these outcomes generally show a much higher public health impact than that for the relatively highly researched morbidity outcome of hospital admissions.

Similarly, the long-term effects of air pollution on morbidity have been investigated in only relatively few studies, notably in the US from the 1990s onwards and in some locations in Europe. Studies of long-
term pollution-exposure impacts on morbidity are more expensive and difficult to undertake than studies of hospital admissions and the findings can be more difficult to interpret, even within the context of the country where the study was undertaken, because of the need to adjust for other determinants of health whose effect could be confounded with that of air pollution. (By studying the same population over time, time-series studies in effect have designed in that the population on different days throughout the study is comparable in terms of age, sex, ethnicity and various slow-changing socio-economic and other determinants of health. Cohort studies of long-term exposure, however, involve contrasts between populations between and/or within cities, with a consequent need to gather and in the analysis take account of information about a wide range of individual and other characteristics.)

Choice of CRFs for use in HIA – use local or international evidence?

Where only limited (or no) information is available from local epidemiological studies, there is little option but to use CRFs derived elsewhere for the purposes of predictive HIA. Even when there are doubts about transferability, the alternative to transferring pollutant-outcome pairs and CRFs (and then discussing the strengths, limitations and uncertainties of using them in another context) is not to quantify at all, i.e. to act as if that pollutant-health outcome had no effect in the target population and this almost certainly involves under-estimation of health impacts. Understandably therefore there is an interest in transferring relationships even if the resulting estimates are uncertain. Consequently, for example, the lack of availability of local CRFs for RADs and WLDs means that CRFs derived from the US studies have been widely applied elsewhere in the world, despite uncertainty as to the transferability of the end-point and more especially the CRF from 1980s California, USA to other times, geographical locations and cultures.

Even if local CRFs are available, typically they are based on a single or small number of studies. On the other hand, the CRFs for most pollutant-health outcomes that are recommended by WHO Europe or US EPA or GBD take account of multiple studies and the international evidence as a whole and might therefore be considered more reliable than locally derived CRFs based on (much) smaller datasets, despite the apparent attractiveness of using local specific results. On that basis, it is reasonable to use the international evidence for core HIA analyses and use local CRFs, where available, for supplementary analyses only.

There are of course uncertainties in any such approach, about how the international evidence applies quantitatively to local situations. For some pollutant-health combinations, especially time series studies of mortality and of hospital admissions, there is evidence from a wide range of studies internationally (though concentrated in North America and Europe) which at least gives indicative information on consistency of evidence worldwide. In a recent meta-analysis of time series studies of daily mortality and hospital admissions in relation to daily PM$_{2.5}$, Atkinson et al. (2014) found consistent evidence of positive relationships, and that % change in respiratory mortality was generally higher than that for cardiovascular mortality. However, for all-cause mortality, they reported substantial regional variation, from 0.25% to 2.08% increase in mortality per 10 µg/m$^3$ increment in PM$_{2.5}$, around an overall meta-analysis value of 1.04% (95% CI 0.52% to 1.56%). They concluded that the constancy of evidence qualitatively supported policy measures to control PM$_{2.5}$ worldwide, but that the (unexplained) differences in risk estimates regionally required further investigation. These differences are, of course, relevant to the transferability of CRFs internationally.

Many analyses of the health impacts of air pollution have been limited to quantification of the effects of particulate matter on mortality, without consideration of other pollutants or morbidity. This may reflect an understanding which has been found in many HIAs that the effect on mortality in adults of long-term exposure to outdoor air pollution, expressed as annual average PM$_{2.5}$, is the predominant effect of concern (in terms of economic value). It may also reflect the availability of widely accepted concentration-response
information linking PM and mortality and the anticipated transferability of the effect, together with the lesser evidence linking air pollution with specific morbidity outcomes.

2.7 Evidence about outdoor air pollution and HIAs from other OECD countries, China and India

As noted earlier, the following review was carried out to answer two specific questions: To the extent that HIAs of outdoor air pollution have been carried out in countries outside of the EU and USA, then:

- To what extent have the authors used the same pollutant-health combinations as proposed in the present report, i.e. to what extent have they considered these transferable and implementable?
- Do these wider international HIAs suggest any other pollutant-health combinations that should be considered as priority for the present report?

The literature review is intended to be indicative of local sources and sufficient to answer these questions; it is not a systematic review or necessarily even comprehensive. For example, in reviewing HIA practice in different world regions it has proved difficult to identify published examples of HIAs that provide sufficient methodological detail to determine exactly what pollutant-health end-point pairs have been evaluated and the source of the CRFs. There are also potential gaps in the information reviewed as the information-searches behind this paper have focussed on English-language sources. It does, however, summarise an evidence-base for answering the questions posed. In addition it gives an impression of the “state-of-the art” of air pollution HIA across the OECD countries, China and India, which could serve as a starting-point for a more comprehensive survey, if required. Its focus was on locally conducted HIAs, but relevant epidemiological studies have sometimes been indicated also, towards the ultimate aim of giving some guidance on the transferability of the core pollutant-health combinations of the present study to other countries and regions of OECD, China and India. It does not include HIAs conducted in individual countries of the EU, or individual states of the USA, on the grounds that these are within larger geographical areas underlying the recommendations of the present report.

Norway, Switzerland and Iceland

The HRAPIE (WHO, 2013b) recommendations were intended to support development by the European Commission of air quality policies EU-wide. The 28 countries of the EU, many of them OECD members, show a reasonable but not extreme diversity in terms of pollution mixture and levels, population characteristics, health care and demography. Applying the HRAPIE recommendations also to Norway, Switzerland and Iceland does not increase that diversity significantly: these three countries are expected to be reasonably similar to other countries of North and Central Europe that are already well represented in EU evaluations. There are, however, important primary epidemiological studies from these countries, notably SAPALDIA in Switzerland, and some specific air quality issues, like effects locally and internationally of volcanic eruptions in Iceland.

Switzerland also has a strong and long-established tradition of air pollution HIA (see e.g. Künzli et al., 2000) which used pollutant-health combinations and associated CRFs in the tradition of US EPA and the EU’s HIAs. These have now been superseded by the more recent EU and US HIA evaluations discussed earlier, but they support the idea of using general EU pollutant-health combinations and CRFs for these three countries.

Canada

Canada may be similar enough to the USA that the same pollutant-health combinations can be used, and possibly the same CRFs also. Clearly there are differences in climate but there are similarities in
pollution, especially between North-East USA and South-East Canada, similarities in the causes of death and disease, and very close co-operation between key air pollution epidemiologists in the two countries. In addition, there is an established tradition of HIA of outdoor air pollution in Canada, associated notably with Health Canada.

Environmental regulation is an area of shared provincial and federal jurisdiction in Canada. The Canadian Environmental Protection Act of 1999 (CEPA) provides legislative authority and a range of regulatory tools that the federal government can use to address environmental problems. The federal government has implemented a number of air quality regulations in recent years, and carried out cost-benefit analyses for those regulations. The health benefits of regulations are analysed using an air quality dispersion models (AURAMS) which predicts air quality changes and then the Air Quality Benefits Assessment Tool (AQBAT) is used to estimate the health impacts of these changes. AQBAT uses widely accepted concentration-response functions and economic valuation estimates (Judek et al., 2012). The model includes 11 morbidity-related health endpoints and five mortality endpoints. These endpoints are similar to ones used by the US EPA.

Health Canada uses the AQBAT model for all high-impact air pollution regulations. One published example showing the use of AQBAT is the regulations for carbon dioxide emissions reductions from coal-fired electricity generation in Canada. Over a period of 20 years, AQBAT results showed a health benefit of CAD 4.2 billion which were due to reduced risk of death and avoided morbidity impacts, including avoided emergency room visits, hospitalisations, bronchitis cases, asthma episodes and restricted activity days following reductions in PM and ozone.

**Australia, New Zealand**

A significant proportion of policy and regulation relating to environmental issues in Australia is devolved to individual states, although some environmental regulation and policy appears to be developed at national level. HIA appears to be widely used in the regulatory decision process.

In 2011, the Council of Australian Governments (COAG) decided to develop a National Plan for Clean Air to improve air quality, and community health and wellbeing. The first stage of the process was to undertake a health risk assessment of airborne particles, ozone, nitrogen dioxide and sulphur dioxide which was completed in 2013. The main analysis focussed on PM and on its effects on mortality and hospital admissions. The HRA drew extensively on the work of WHO-Europe. The morbidity impacts calculated for PM\(_{2.5}\) were cardiovascular hospital admissions and emergency department attendances for childhood asthma. The impacts calculated for PM\(_{10}\) were respiratory hospital admissions and admissions for acute bronchitis in adults aged 65+. Only acute effects and exposures above “background” levels were considered. Assessments were also made for short-term exposures above background of the effects of O\(_3\) on childhood hospital emergency department attendance, of NO\(_2\) on cardiovascular hospital admissions for adults aged 15-64 and 65+ years, respiratory hospital admissions for people aged 1-14, 15-64 and 65+ years and asthma hospital emergency department attendance, and of SO\(_2\) on respiratory hospital admissions in adults aged 65+ years.


Japan, Korea

An initial information search of English-language publications did not provide much information describing HIA practice in Korea or Japan. One relatively small recent HIA in Korea (air pollution effects on health in Suwon city; Jeong, 2013) was based on the WHO AirQ health impact assessment tool that calculated PM$_{10}$, NO$_2$, SO$_2$ and O$_3$ effects on mortality, PM$_{10}$ effects on respiratory and cardiovascular hospital admissions, O$_3$, NO$_2$ and SO$_2$ effects on COPD hospital admissions and NO$_2$ and SO$_2$ effects on acute myocardial infarction. A more recent HIA of particulate air pollution in 27 South-East and East Asian cities, including several in Japan and Korea, focused on the mortality effects of long-term exposure (Yorifuji et al., 2015). In Japan, annual large-scale primary epidemiology studies of 3- and 6-year old children have enabled cross-sectional and longitudinal analyses of asthma prevalence and incidence in relation to key pollutants such as NO$_2$, SO$_2$ and SPM (suspended particulate matter). Results in recent years have not shown relationships between prevalence or incidence of childhood asthma and the studied pollutants, although a significant relationship with SPM had been identified in some previous years.\textsuperscript{16}

Israel

There is in Israel an established air pollution and health research community. A search of bio-medical literature in PubMed returned several investigations of the association between air pollution and various aspects of children’s respiratory health. However, there does not appear to be sufficient local CRF information to support a full analysis of morbidity impacts.

An initial information search for English-language publications did not turn up any examples of HIA in Israel, but it is possible that any HIAs that have been undertaken would be published in Hebrew. However, Tel Aviv became a participating city in the highly-regarded European air pollution HIA network, APHEIS, as part of its expansion to 26 cities.\textsuperscript{17} This supports the view that it is reasonable to extrapolate to Israel pollutant-health pairs and associated CRFs from the EU (or USA) on the basis that Israel is an advanced industrial Mediterranean country. The population, health care system and air pollution climate in Palestine, however, is likely to be different and not similarly comparable with USA or Western Europe.

Turkey

There has been some limited research into the health effects of air pollution in Turkey. A search in PubMed returned a small number of investigations of the association between air pollution and various aspects of children’s respiratory health, asthma and rhinitis in all ages. An initial information search did not turn up any examples of HIA in Turkey, but it is possible that HIAs have been published in Turkish. It might be reasonable to extrapolate from the EU on the basis that Turkey is a Mediterranean country with some similarities to European societies.

Chile, Mexico, elsewhere in Latin America

The air pollution mix in urban areas of both Chile and Mexico is likely to be affected by local climatic factors, although the sources of air pollution would be anticipated to be broadly similar.\textsuperscript{18} Population characteristics would be expected to be broadly similar across much of Latin America, although levels of deprivation are highly variable, both within and between countries. There is a strong tradition of research


\textsuperscript{17}. See www.invs.sante.fr/publications/2005/apha_310505/apha_3yreport.pdf.

\textsuperscript{18}. The use of wood for heating purposes in households is an important source of air pollution in some parts of Chile.
on air pollution and health in Mexico and in Latin America, and there are primary epidemiological studies in both Chile and Mexico. There are abstracts in PubMed for studies of daily mortality for both Chile and Mexico and various aspects of children’s respiratory health and use of medical services. Studies in Mexico have, for example, investigated heart rate variability and effects on central nervous system development in children and there is ongoing research into potential effects on the unborn child. The pollutants that have been investigated in epidemiological studies include PM$_{2.5}$, PM$_{10}$, NO$_2$, SO$_2$ and O$_3$ but most studies have focused on PM$_{2.5}$ or PM$_{10}$.

Several HIAs for air pollution in Latin America have been published in English and it is likely that further examples could be found in Spanish or Portuguese. One example of a Latin America-wide HIA was found that was based on US CRFs: *Urban Air Quality and Human Health in Latin America and the Caribbean* – published by the Inter-American Development Bank in 2005.\(^\text{19}\)

A PubMed search turned up several HIAs published in the last 15 years.

- Bell et al. (2006) published an assessment of the avoidable health effects of air pollution – PM and O$_3$ – in three Latin American cities, Santiago, São Paulo and Mexico City, that used local CRFs where possible, the pollutant-outcome pairs were not listed in the PubMed abstract but analysis included asthma attacks, children’s medical visits and chronic bronchitis.
- McKinley et al. (2005) quantified local and global benefits from air pollution control in Mexico City. Their analyses of health focused on PM (as PM$_{10}$) and ozone. Regarding health outcomes: “A set of 11 health outcomes, including premature mortality, chronic bronchitis, hospitalizations, and emergency room visits for cardiovascular and respiratory disease, and minor restricted activity days (MRAD) are considered”. Their results table focuses on mortality, chronic bronchitis and minor Restricted Activity Days. Full details are not given, but it very much looks like an approach similar to that of US EPA was used. There are, however, some useful comments on transferability and the authors recommend a hybrid of international and local studies as a basis for quantification.
- Cifuentes et al. (2001) assessed the health benefits of urban air pollution reductions associated with climate change mitigation (2000-20) in Santiago, São Paulo, México City and New York City for PM and O$_3$. In addition to mortality, they considered PM$_{10}$ effects on chronic adult bronchitis, acute bronchitis in children, respiratory hospital admissions, emergency department visits, asthma attacks, WLDs, RADs and respiratory symptom days and O$_3$ effects on respiratory hospital admissions, emergency department visits, asthma attacks, RADs and symptom days. CRFs were largely drawn from US studies, but the HIA also incorporated CRFs from Chile.

**China**

There has been extensive epidemiological investigation of the health effects of air pollution in China, some in joint studies in partnership with US institutions. Most of these studies have focussed on mortality rather than morbidity, but some studies have established concentration-response information for morbidity effects following acute exposure (“daily variations”), including:

- PM$_{10}$, SO$_2$ and NO$_2$ and outpatient arrhythmia visits;
- PM$_{2.5}$ and children asthma admissions;
- PM$_{10}$, SO$_2$, NO$_2$ and total hospital admissions and cardiovascular hospital admissions;
- PM$_{10}$, SO$_2$, NO$_2$ and outpatient and emergency room visits.

Lai et al. (2013a) have published pooled Chinese-specific estimates of relative risks (RR) per 10 μg/m³ for PM₁₀, SO₂, NO₂ and O₃ that include effects on specific types of hospital admissions (pollutant-endpoint pairs are not clear from the abstract). They have also published RR for preterm birth and still birth.

Other studies have reported concentration-response relationships for the effects of long-term (“chronic”) exposure, including:

- PM₁₀ and respiratory symptoms in girls (effects not found in boys);
- PM₁₀, PM₂·₅, SO₂, O₃ and effects on blood pressure and hypertension.

A number of HIAs have been published in the peer reviewed literature, some of which have focussed entirely on mortality effects associated with exposure to PM₂·₅. Examples of HIAs published in the last decade that consider morbidity include:

- Tang et al. (2014) assessed the health benefits of improving air quality in Taiyuan. The analysis was based on PM₁₀ and in addition to mortality included new cases of chronic bronchitis, outpatient visits, emergency-room visits and hospital admissions.
- Voorhees et al. (2014) assessed the health benefits of improving air pollution in Shanghai using methods based on those of US EPA. Long-term mortality effects were assessed for PM₂·₅, but acute mortality and morbidity effects were assessed for PM₁₀. The end-points considered were hospital admissions, emergency department visits and outpatient visits.
- Lai et al. (2013b) estimated annual numbers (per million people) of excess deaths from all natural causes and hospital admissions from cardiorespiratory causes attributable to SO₂, NOₓ, O₃ and PM₁₀ from marine emissions in the Pearl River Delta.
- Zhang et al. (2008) calculated the health effects of pollution caused by PM₁₀ in 111 Chinese cities in 2004, but the PubMed abstract does not indicate what effects were quantified.
- Pan et al. (2007) assessed the health benefits of different energy scenarios in Beijing for PM₁₀ and for SO₂ and in addition to mortality considered respiratory and cardiovascular hospital admissions, outpatient visits to internal medicine and paediatrics departments, total emergency room visits and asthma attacks but the PubMed abstract does not link each pollutant to specific effects.

India

There are very few abstracts in PubMed for studies of the health effects of air pollution in India that have been undertaken over the last decade. Patankar and Trevidi (2011) evaluated the health impacts of air pollution in Mumbai based on an analysis of the local relationship between air pollution and morbidity impacts. CRFs were developed for PM₁₀ and NO₂ and a range of health impacts, including symptoms such as cough, breathlessness, wheezing and cold, and illnesses such as allergic rhinitis and chronic obstructive pulmonary disease (COPD). No extra information was found during a wider search with Google.

Working conclusion

This limited review of HIA practice in OECD countries (other than USA and countries of the EU 28) suggests that many such studies have been undertaken and (understandably and correctly), there has been a strong focus on air pollution and mortality. In terms of morbidity, the pollutant-health outcomes considered have been similar to those studied here, with quantification in terms of NO₂ and SO₂ (as well as PM and ozone) in some locations. A mixture of international and local CRFs has been used. There is, however, no clustering of additional health outcomes or pollutant-health combinations that need be considered for inclusion in the core recommendations of the present report.
2.8 Pollutant-health combinations not included in the proposed core set

Combinations included both by USEPA and by HRAPIE

- **Acute bronchitis in children**: This was discussed briefly above.
- **Asthma exacerbations in the asthmatic population in relation to PM**: Various aspects of exacerbations, among them respiratory symptoms and bronchodilator usage, were included in the earlier EU CAFE project (Hurley et al., 2005). Results did not show a health impact that had any significant effect on the “bottom line” of benefits, after quantification and monetisation. Asthma is more prevalent in Western Europe than in many other areas of the world, and so major effects elsewhere are unlikely. Severe exacerbations will show as and be included in respiratory hospital admissions; others as Restricted Activity Days.

Combinations included by US EPA but not by HRAPIE or GBD

- **Emergency room visits (for asthma) in relation to PM and to ozone**: These reflect the particular health-care arrangements in the USA whereby people present at emergency room departments of hospitals. Some lead to admissions, which are included separately. Others do not. The arrangements for emergency care that does not require immediate hospitalisation varies by country; as in CAFE (Hurley et al., 2005), emergency room visits were considered to be too specific to the North American experience for generalisation.
- **Non-fatal heart attacks (myocardial infarction) in relation to PM**: This is an outcome which deserves closer consideration; it was not considered specifically by HRAPIE (WHO, 2013b) or the earlier European CAFE project (2005), or by GBD (2013).
- **School loss days in relation to ozone**: This could be considered in tandem with Work Loss Days and (Minor) Restricted Activity Days. HRAPIE proposed that the results for Minor RADs and ozone (and for RADs and PM) be applied at all ages, even though the underlying CRFs and available baseline background rates were available for more limited ages only, and so days of restricted activity in children are already included, at least to some extent.

Other combinations for which there is some evidence of effect of air pollution

There are many other pollutant-health combinations where there is at least some evidence of an effect of air pollution on health. This is not surprising. The effects, on mortality and on a wide range of morbidity outcomes, of short-term exposure to (of daily variations in) PM and Ozone are well accepted as causal; REVIIHAAP (WHO 2013a, Questions C1 and C2) concluded that there is a causal role for NO\textsubscript{2} also. These effects occur not only on days of pollution episodes but also on “ordinary” pollution days. The more serious effects are generally understood as outdoor air pollution working in combination with other factors to “trigger” earlier death in people with pre-existing serious cardio-respiratory disease. It is to be expected then that outdoor air pollution works with other factors to trigger a wider range of other health and health-related outcomes also.

US EPA (2011) lists some health outcomes possibly related to PM or to ozone respectively. Some are a consequence of short-term exposure (daily variations), others of longer-term exposures. (The role of longer-term exposure in the development of chronic disease is considered further in the next sub-section.)

- **PM**: Sub-chronic bronchitis, low birth weight, pulmonary function, chronic respiratory disease other than bronchitis, morphological changes, altered host defence mechanisms, cancer, non-asthma respiratory emergency room visits and UVB exposure;
- **Ozone**: Cardiovascular emergency room visits, asthma attacks, respiratory symptoms, chronic respiratory damage, increased responsiveness to stimuli, inflammation in the lung, premature aging of the lungs, acute inflammation and respiratory cell damage, increased susceptibility to respiratory infection, non-asthma respiratory emergency room visits, UVB exposure.
These health outcomes were not included in quantification of the benefits of pollution reduction following the US *Clean Air Act* because there is no consensus on causality; or causality has been established but empirical data are not available to allow calculation of benefits of the pollution reduction.

The categorisation of unquantified health effects is not exhaustive, partly because as the evidence-base continues to expand, additional pollutant-health combinations get suggested. For example, Perez (2014) includes other health outcomes. Her focus is principally on the early signs of damage from long-term exposure; this is considered next, below.

***Likely under-estimation of morbidity from chronic (long-term) exposure***

The effect of long-term exposure on mortality risks is now generally considered as causal also, certainly with PM$_{2.5}$; to a lesser degree of certainty with ozone and with NO$_2$ as well (REVIHAAP; WHO 2013a). Long-term exposure to air pollution affects the risk of death in combination with other risk factors, as does acute exposure. It works in combination with other factors, as one stressor among many, to initiate or to accelerate the processes of chronic cardio-respiratory disease and lung cancer which eventually lead to earlier death (see e.g. COMEAP, 2010). Accepting that long-term exposure to outdoor air pollution does indeed increase the risk of earlier death, it is to be expected that there are relationships also between long-term exposure to outdoor air pollution and various chronic diseases and that these relationships exist in reality *whether or not there currently are epidemiological studies that show them*.

In practice, the effects of long-term exposure on morbidity as expressed in major HIAs include only a possible effect of PM on prevalence of chronic bronchitis – see above. The mortality evidence, however, suggests that, for longer-term exposure, a much wider range of pollutant-health outcome pairs is implicated. Briefly, for ozone and for PM, the situation is as follows.

**PM**

PM$_{2.5}$ is the most relevant metric for representing the effect of long-term exposure to PM on mortality in adults (COMEAP, 2009) and it is the metric used by both HRAPIE and US EPA. Many health impact analyses, including in Europe (CAFE, HRAPIE), use all-cause mortality but do so with awareness that only some causes of death are known to be affected. Others, e.g. Global Burden of Disease, use specific causes of death. The exact grouping of causes varies from study to study but typically includes:

1. Cardiovascular causes, notably heart attack and stroke;
2. Non-malignant respiratory causes, notably chronic obstructive pulmonary disease (COPD); and
3. Lung cancer.

The implication of the present discussion is that there is chronic morbidity attributable to long-term exposure to PM and currently not quantified in major HIAs and CBAs, because of lack of direct evidence. Perez (2014) reports that “Strong evidence exist now for long-term exposure to PMs and cardiovascular diseases. Of special relevance are studies on an association with PM$_{2.5}$ and various markers of atherosclerosis such as thickness of the intima-media, coronary artery calcification, or pulse pressure. This pathophysiological pathway is supported by short-term epidemiological and panel studies showing variations in cardiovascular biomarkers of inflammation such as C-reactive protein or fibrinogen that are linked to subsequent cardiovascular diseases.” It is difficult, however, to see how the studies of detailed mechanisms can be used for benefits analysis, other than to support the inclusion of more easily observable clinical effects, in this case effects on risks of mortality. Perez also notes that “Other outcomes have more recently been related to PMs including diabetes [and] neurological development in children and disorders in adults... Association with birth outcomes including low birth weight, preterm birth and small for gestational age at birth have also been reported.”
Insofar as the chronic disease is fatal, it needs to be determined to what extent the loss in monetary terms is already incorporated into the valuation of mortality. But it is assumed here that not all of it is; and not all of the attributable chronic disease and associated disability is fatal; so some under-estimation is to be expected.

Ozone

Only effects of short-term exposure ("daily variations") are quantified in major HIAs in Europe and USA. There is some evidence (discussed e.g. in REVIHAAP, WHO 2013a; HRAPIE, WHO 2013b) that long-term exposure increases the risk of mortality in adults (respiratory mortality, Jerrett et al., 2009; cardio-respiratory mortality, Smith et al., 2009). HRAPIE recommended that relevant function for respiratory mortality be applied as Group B pollutant-outcome pairing for which there is uncertainty in the data used for quantification of effects. They noted the estimated impact of long-term exposure would include at least some of the acute mortality attributed to \( O_3 \). US EPA did not include an increase in respiratory mortality associated with long-term exposure to summertime ozone in its evaluation of the *Clean Air Act* (2011) but did include it in its more recent regulatory impact assessment of ozone (US EPA, 2014).

If the effect can be considered real, then the absence of any associated combinations linking long-term exposure to \( O_3 \) and health can be another likely under-estimation of morbidity effects. Again, Perez (2014) is informative: “In the last decade, several studies evaluated the chronic effect of ozone and chronic respiratory health and found evidence with lung function, asthma admission, and increase IgE in adult asthmatics. Some studies reported effects of long-term ozone exposure and onset of asthma in children. In recent years, the evidence for association with birth outcome has increased, and there are preliminary findings of ozone being related to cognitive decline in adults.” Although major recent HIAs such as HRAPIE (WHO, 2013b) did not attempt to quantify these effects, it is important that evidence insufficient for quantification is not ignored.

### 2.9 Summary and working conclusions

This review has recommended a common core set of pollutant-health (morbidity) combinations, for application in OECD countries, China and India, that meets the criteria of being unbiased (i.e. not systematically under-estimating or over-estimating the effects), credible (i.e. based on recognised expert reviews) and implementable (i.e. for which the necessary input data, including concentration-response functions and background rates of morbidity) are available or can be estimated. But it has also highlighted difficulties in their application. In total, the review has identified five pollutant-health pairs for consideration. These are, with commentary on their application:

1. Respiratory Hospital Admissions (RHA) and Cardiovascular Hospital Admissions (CVHA) in relation to PM and to ozone. While strongly based in evidence, experience from HIAs in Europe and the USA is that these, when quantified and monetised, make little difference to the “bottom line” of aggregated monetised benefits.
2. Restricted Activity Days (RADs) and associated Work Loss Days (WLDs) in relation to PM and/or ozone. These are widely used in HIAs internationally and when applied they suggest a noticeable effect on aggregate monetised benefits – small relative to mortality but one of the higher morbidity effects. However, they rest on a narrow evidence-base, from a series of studies in California, USA, in the 1980s. Additionally, the health outcomes are strongly socio-culturally determined and there may be difficulty in obtaining credible background rates. These various difficulties point to major uncertainties about transferability.
3. Chronic Bronchitis in adults in relation to PM only. This has been a long-standing pollutant-health combination quantified in HIAs in USA, Europe and elsewhere. There are studies, both in
USA and Europe, from which concentration-response functions can be derived; and when applied in HIAs, they give monetised results which typically are amongst the most influential of morbidity impacts. However, both in Europe and in the USA, recent expert review has questioned the overall evidence-base relating air pollution to prevalence and incidence of chronic bronchitis in adults, concluding that the case for causality is not as strongly established as had previously been thought. Consequently, in Europe this pathway is not included among those that can be quantified with greater confidence; and it is not part of the primary analysis in the most recent regulatory impact assessments of US EPA.

The two remaining pathways:

4. Acute Bronchitis in children aged 6-12 or 6-18 years, defined as “bronchitis in the past 12 months” (Hoek et al., 2012) based on responses to symptoms questionnaires; and
5. Acute Lower Respiratory Illness (ALRI) in children aged <5 years relate to children only, and as such may be expected not to have a major influence on final monetised results, compared with the monetised impacts on mortality.

In summary, this “bottom-up” approach to estimating the morbidity benefits of reducing outdoor air pollution does not inspire great confidence, but it is not easy to see how it can be improved significantly at the present time. Nevertheless, it is likely to be better than not quantifying these effects, which is equivalent to ignoring them.

There is a further possibility, that is, to do as is sometimes done, and to estimate the benefits of pollution reduction on morbidity as some percentage of the mortality benefits. This recognises that in current HIA, the effect on mortality of long-term exposure characterised as PM$_{2.5}$ dominates the monetised benefits. This is true even if the analysis, including monetisation, is based on attributable years of life lost; it is even more so if the analysis, including monetisation, is based on attributable deaths.

Recent epidemiology and its expert review for purposes of HIA have the effect of increasing rather than decreasing the relative importance of mortality impacts. This is because of a growing movement to quantify the effects on mortality of long-term exposure to pollutants other than PM$_{2.5}$. The possibility that long-term exposure to ozone, especially summer-time ozone, also increases the risk of premature mortality in adults is now seriously considered in HIAs in Europe and in the USA (WHO, 2013a, b; US EPA, 2014), though results suggest an effect smaller than that of PM$_{2.5}$. Of greater potential influence are recent developments in the understanding of the role of NO$_2$, where REVIHAAP (WHO, 2013a) concluded that the associations between NO$_2$ and mortality are likely to at least in part reflect a causal role of NO$_2$ that is additional to (even if not fully separable from) that attributed to PM$_{2.5}$ and ozone. On that basis, HRAPIE (WHO, 2013b) recommended that the mortality effects of long-term exposure to NO$_2$ be quantified for policy development in Europe, though with various caveats, e.g. recognition that this is not among the more certain of quantified pathways; quantification above 20 μg/m$^3$ annual average NO$_2$ only; adjustment for possibly some double-counting with effects of PM$_{2.5}$. Nevertheless, quantification is likely to lead to high estimates of impact especially if lower figures than 20 μg/m$^3$ annual average NO$_2$ are used.

These developments are likely to underpin the importance of mortality effects, especially those related to long-term exposure, within the overall benefits assessment, and correspondingly to downplay the aggregate effects of morbidity. This adds to the attraction of upscaling mortality effects by some proportion, if this can be justified by a combined assessment of health and monetary valuation issues.

Though strictly outside of the scope of this study, the present report re-considers this issue after monetary valuation aspects of a “bottom-up” approach have been considered.
3. Current partial or comprehensive estimates of the cost of morbidity from air pollution

3.1 The social cost components of air-pollution induced health impacts

The Damage Function Approach (DFA) is the general approach currently used to assess the economic value of health impact (European Commission, 1999). In this approach, the change in health outcomes as a result of a change in air pollution is quantified by estimating the change in the incidence of individual health end-points across the population. The resulting numbers of people suffering these health conditions as a result of the change in air pollution is then multiplied by the unit value estimated for each end-point. The unit value is comprised of three broad components: “resource cost”, “opportunity cost” and “disutility cost”. However, in applications made to date, these components have either been inconsistently measured or incompletely compiled (Hunt and Ferguson, 2010).

This section specifies the cost components so that future applications can become more standardised and complete. The following paragraphs present a coherent conceptual basis for the identification of morbidity cost components that explicitly considers the extent to which there may be overlap between components, and how such overlap can be treated in future empirical exercises.

In order to delineate the individual components of the economic valuation of morbidity health end-points resulting from pollution, a simple model outlined in Freeman (2003) is presented. The model examines the potential determinants of the willingness-to-pay (WTP) to avoid a given health outcome resulting from exposure to environmental pollution. It should be noted that in theory, willingness-to-accept (WTA) a compensating payment equates to the WTP; in practice, WTA tend to be larger since the income constraint is less binding. For that reason – and the comparative lack of empirical WTA studies – this paper focuses on the WTP approach.

In this model, health is measured by the number of sick days, \( s \), in any period of time, which ignores the severity of the illness and differences in the symptoms experienced. An example of an air-borne pollutant is particulate matter (PM) that can result in respiratory illnesses, amongst other health conditions.

Everything else being equal, \( s \) is determined by the level of exposure to pollutants, i.e. the dose, \( d \), which is dependent on the concentration of the pollutant, \( c \). At the same time, the risk of \( d \) is assumed to be a function of averting activities taken to reduce exposure to pollution. Averting activities include, for example, staying inside on days of high pollution or moving to live in a less polluting neighbourhood.

Additionally, the individuals can choose mitigating activities and treatments, \( b \), to reduce the health effects of a given level of exposure to pollutants. Examples of mitigating activities, \( b \), include visiting a doctor or taking medicines to reduce a symptom.

It can be shown formally (see Annex 2) that individuals can maximise their welfare by implementing \( a \) and \( b \) – i.e. averting and mitigating activities – to the point where the additional welfare from reductions in sick days, \( s \), equates to the loss in welfare from lost consumption associated with the costs of \( a \) and \( b \). This is equivalent to saying that the WTP for reduced pollution is the reduction in the costs of \( a \) and \( b \). Note that some OECD countries, such as Norway, prefer to use a top-down approach that contrasts with the bottom-up, damage function approach in giving most weight to the aggregate financial resource costs observed.

In this context, the assumption of “everything else being equal” ensures that other costs incurred in maintaining an individual’s health – such diet and exercise – are excluded from the valuation.

This Annex replicates the description of the Freeman model in Hunt and Ferguson (2010).
cost of achieving the ideal level of health given a variation in pollution levels. Thus, the WTP for a reduction in pollution is given by the marginal cost of reducing the number of sick days associated with the reduced pollution.

Note that the effect of pollution concentration, \( (c) \), on welfare consists of two components: the direct loss of utility associated with the illness (i.e. the pain & suffering) and the opportunity cost of the time lost due to the illness, (i.e. the cost of absenteeism) valued at the wage rate.

The model outlined in the previous paragraph and described in Annex 2 can be explained in more intuitive terms. The individual is attempting to minimise the cost to herself of managing the risk of an air pollution-induced health impact – the costs consisting of those incurred in avoiding exposure to air pollution, those incurred in treating the health impact, the loss of wages due to illness, and the pain and suffering\(^{23}\) associated with the illness. These four forms of costs together constitute the WTP to avoid the health impact, as illustrated in Box 1.

### Box 1. Constituent components of health impact economic values

<table>
<thead>
<tr>
<th>Cost Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resource costs</td>
<td><strong>Avertive expenditures</strong>, including, e.g., relocation to area of lower air pollution, staying inside, etc. <strong>Mitigating expenditures</strong>, including the direct medical and non-medical costs associated with treatment for the health impact (i.e. all the expenses the individual faces when visiting a doctor, ambulance, buying medicines and other treatments, plus any related non-medical cost, such as the cost of childcare and housekeeping due to the impossibility of the affected person being able to do so).</td>
</tr>
<tr>
<td>Opportunity costs</td>
<td>Costs related to loss of productivity and/or leisure time due to the health impact</td>
</tr>
<tr>
<td>Disutility costs</td>
<td>Pain, suffering, discomfort and anxiety linked to the illness</td>
</tr>
<tr>
<td></td>
<td><strong>Equals</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Economic value of avoiding the health impact</strong>(^{a})</td>
</tr>
</tbody>
</table>

\(^{a}\) This economic valuation is also known as the social welfare cost.

A critical conclusion to be drawn from the conceptual basis described in previous paragraphs is that the individual is able to find – and achieves – the welfare-maximising balance – or equilibrium – between the different cost components. The corollary of this is that the costs that are observed, or estimated through asking people directly, are assumed to be those that exist in this equilibrium. In practical valuation exercises there is therefore some skill required in order to ensure that these component measures do not overlap and so result in double-counting.

Table 5 below summarises where such overlaps might occur. In this table, *Original cost* indicates the cost component intended for measurement, whilst *Secondary cost* indicates components with which it may potentially overlap. Thus, a measure of disutility (top row in Original cost) may overlap with part or all of productivity, averters and medical costs. For example, a questionnaire that asks an individual to state her WTP to avoid the disutility cost component needs to be designed sufficiently carefully so that she does not include financial as well as non-financial concerns in her assessment of her loss of welfare. Similarly, a measure of the avertive expenditure associated with moving house that could be attributed to reducing air pollution expenditure would have to be separable from the disutility and productivity cost components in order to avoid double-counting. Furthermore, assuming that a treatment is effective, incurring the medical costs of treatment is likely to reduce both the disutility and productivity costs associated with the health condition. Indeed, this last example illustrates the point made in the previous paragraph that there is likely to be a balance between the different components. It therefore emphasises that the cost estimates need to be

\(^{23}\) Note that this includes welfare effects on others, such as close relatives.
consistent with this balance; simply summing individual components estimated independently of each other is likely to produce an inaccurate unit value, though the direction of bias depends on how the individual components are specified.

It should be emphasised that the total welfare – or social cost – is the measure of the opportunity cost of the resources diverted across society by the morbidity condition, including non-market costs. Thus, in aggregate, who bears the individual cost components is not differentiated. It is also worth highlighting that whilst the approach adopted in this paper assumes that the costs are captured in directly impacted markets, in instances where these costs result in significant effects on related markets or government finances, an economy-wide modelling approach would be needed to capture all the social welfare costs.

<table>
<thead>
<tr>
<th>Table 3. Checklist of potential over-lapping cost components</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td><strong>Original cost</strong></td>
</tr>
<tr>
<td>Disutility</td>
</tr>
<tr>
<td>Productivity costs</td>
</tr>
<tr>
<td>Averting costs</td>
</tr>
<tr>
<td>Medical costs</td>
</tr>
</tbody>
</table>

One solution to this issue of potential double-counting – particularly in instances where survey-based methods are adopted – is to design the questionnaire so that all cost components are explicitly included and considered by the respondent. The respondent then deliberately states a WTP for all components, thereby removing any ambiguity in the exercise.

An example of this practice is the study by Chestnut et al. (2006), in which the survey population had direct experience with the illness episode that caused an individual to be hospitalised, either as a result of a serious acute illness or an aggravation of a chronic illness. The authors included a series of COI questions that focused on the respondents’ most recent hospitalisation, whilst the WTP questions referred to preventing or shortening a hypothetical future hospitalisation. An advantage of this approach is that it removes the sampling effort that would otherwise be necessary to obtain individual COI measures as well removing the need for the additional scenario-definition required to estimate WTP specifically for the disutility costs of morbidity effects alone. In this context, it is interesting to observe that the practice pursued in a number of North American studies – as exemplified by the Chestnut et al. (2006) study – of asking respondents for COI as well as disutility WTP, contrasts with the majority of European studies that ask respondents only for the disutility WTP. One explanation for this difference is the possibility that North American respondents often bear more of the costs of illness directly themselves and so, when giving survey answers, are more likely to consider these costs.

4. Survey of economic unit values related to air pollution-related morbidity

4.1 Method

This section constitutes an application of the findings of the previous sections. A list of unit values for the health end-points specified for quantification is presented. The health end-points considered are:

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24. It is noted that recognition of the non-market elements of social costs in informing decision-making is a relatively recent practice that is not without its critics. Notably, there is a scepticism regarding the robustness of such estimates – particularly when a survey-based valuation method is used. The counter-argument – which is supported here – is that the sophistication of the studies, and the growing convergence of their results gives sufficient confidence that their findings are robust and should be included in what would otherwise be an incomplete measure of social costs.
• Chronic bronchitis – unit value per new case;
• Hospital admissions (Respiratory & Cardiovascular) – unit value per new case;
• Work-loss days – unit value per day;
• Restricted activity days – unit value per day;
• Acute lower respiratory infections in children aged less than 5 years – unit value per new case;
• Acute bronchitis in children – unit value per new case.

It is important to emphasise that the unit values presented here attempt to capture the value to society of avoiding the health end-points. Thus, consistent with the values needed to undertake social cost-benefit analysis, these values measure the effect on social welfare, in monetary terms. The component costs that constitute each unit value are derived from the peer-reviewed literature, as far as possible, supplemented by data from the “grey” literature. The literature search has been conducted by using a variety of search terms within a number of established databases and search engines. Key search terms included: “costs”, “health end-point”, “value”, “valuation”, “air quality”, “environmental health”, etc. in multiple combinations. Searches were made in databases including:

• ScienceDirect (www.sciencedirect.com/science),
• IngentaConnect (www.ingentaconnect.com),
• Wiley InterScience (www3.interscience.wiley.com/cgi-bin/home),
• the EconLit database,
• the EVRI database (www.evri.ca/) and;
• Google Scholar (http://scholar.google.co.uk/).

In order to derive each component cost estimate, a process of selection comparable with that reported in OECD (2012) for deriving mortality risk unit values was adopted. The selection process comprises the following:

• An assessment of the quantity of original WTP studies allows us to identify whether there is a narrow or wide body of evidence and therefore whether it is possible to establish whether the findings of one or more studies can be corroborated easily. Since the number of studies is rather limited, no study is excluded for being too old. However, it is likely that more recent studies, particularly in the peer-reviewed academic literature, reflect methodological state-of-the-art practice and so deserve greater weighting. In the database tables, as much information as is relevant in the subsequent screening and evaluation process is presented. This information includes details of the study type, date and location, method and unit values. Studies reported in both the academic and grey literature are considered in the first instance.

• The transferability criterion relates primarily to the geographical location of the original empirical study; as far as is possible, studies that produce values that have been derived in OECD or key partner countries are being used, since these are the countries of most relevance for this study. Given the sparseness and disparate nature of the evidence-base, insufficient data exists to undertake a full meta-analysis comparable to that in OECD (2012). However, the guidelines on benefit transfer outlined in that report are adopted.

In order to be consistent with the practice identified for valuation of mortality risk in OECD (2012), the unit value estimates are comprised of a central value, with low and high sensitivity values. As with the practice in OECD (2014), these unit values can then be adjusted by PPP for each OECD country and key partner countries. The price year is 2010.
As highlighted above, the *quality* criterion is related to the temporal aspect of the transferability criterion since it is likely that if a study has been undertaken more than a few years ago, the methodology may now be out-dated. Additionally, the criteria identified in OECD (2012) regarding sample size is being followed – the logic being that a larger sample size is likely to produce more robust results.

For each health end-point considered, a summary of the selected studies is presented in database tables. On the basis of the reported findings, it has been attempted – through an informal meta-analysis – to derive a unit value for each cost component to be used generically in subsequent OECD analyses of air quality regulation. For these health end-points, the limited number of studies – together with their differences in methods – suggested that the benefits of a formal meta-analysis would be limited. However, as more studies are undertaken, it is envisaged that using formal meta-analysis in the way that Vassanadumrongdee et al. (2004) have done for other morbidity end-points will become possible. The database tables can be used by analysts within individual countries to orientate themselves towards the possibilities for benefit transfer from studies that have been undertaken in their country or region.

The process of deriving unit values varies between end-point, depending on the data that is available. The unit values are derived either through an averaging of the data and/or a selection of individual studies that can be understood as representative. This latter method has been used either when the study has been undertaken in an OECD country or when the study is identified as being superior in quality. This process is inevitably reliant on the authors’ reading of the available studies and their judgement as to the quality of the studies. The unit value ranges that presented here are therefore somewhat subjective, and should be recognised to be indicative only.

### 4.2 Chronic bronchitis

Six primary research studies have been identified as providing unit value estimates for chronic bronchitis, cf. Table 4. They comprise two studies in the US, three studies in Europe, and one study in China. One study estimates costs of illness (COI), alone, whilst the other five studies estimate the disutility component. Four out of these five studies exclude the COI component; only Viscusi et al. (1991) may have allowed such resource costs to be considered by the survey respondents.

The one study that gives an estimate of COI for chronic bronchitis, (Leu et al., 1986), presents a single estimate of USD 7 292. (Note that all values presented in the text part of the discussion of chronic bronchitis and subsequent health end-points are expressed in US dollars, in 2010 prices). A review of the health economics literature confirms the fact that chronic bronchitis is not considered independently of chronic obstructive pulmonary disease (COPD) – indeed, it is understood to be a part of that disease group. In this context, it is interesting to find that a more recent survey, (Wouters, 2003), of cost data on COPD for six OECD countries estimates the COI per case to range from USD 1 700 to USD 7 800, corroborating the Leu et al. estimate. The Wouters study finds that medical costs range from 17% to 95% of total COI – depending on the individual country – the remainder being productivity costs borne by society. It is suggested that a mid-point of USD 4 750, with a range of USD 1 700 – USD 7 800, be adopted to represent the COI components in OECD analysis.

A review of the studies that estimate the disutility component shows that the severity of the chronic bronchitis illness valued varies considerably depending on the individual study. The two US studies, (Viscusi et al., 1991; and Krupnick & Cropper, 1992), which date from 1991-92, share the same survey instrument and utilise a definition of chronic bronchitis that is at the more severe end of the spectrum. Consequently, the range of estimates that they produce – USD 800 000 to USD 2 100 000 – is higher than the estimates produced in the other studies that use less severe definitions. Thus, the other two OECD studies produce central estimates of USD 40 000 and USD 85 000 for Switzerland and six European
countries, respectively, whilst the study undertaken in China provides a range of USD 650 to USD 1 330. These values for China are judged to be appropriate for use in China-specific analyses since they reflect relative income levels but are not considered further for OECD countries.

Subsequent work to transfer the results from the US studies to a policy context has resulted in an adjustment to account for the high severity. Thus, Bloyd et al. (1996) identify a 58% reduction to the results in order to derive values for a more moderate severity. The resulting range of values is then USD 336 000 to USD 882 000, with a mid-point value of around USD 600 000. Nevertheless, these remain an order of magnitude higher than the European values, and though part of this difference may be explained by the fact that the severity remains lower in the European studies, it seems unlikely that this accounts for all of the difference. Whilst the US studies are older, it is unclear whether both the risk-income and risk-risk methodologies are notably dated; indeed, these studies are peer-reviewed whilst the study of Maca et al. (2011) – though having a large sample size – has not been peer-reviewed. Although this valuation data suggests that it might be sensible to suggest alternative unit values – depending on the severity of the chronic bronchitis, the epidemiological evidence does not currently allow one to make this distinction.

In order to derive values that can be used in OECD-country analysis – in the absence of a single study or consensus within the values – the approach used here is to adopt average or mid-point values. In this instance, mid-point values are USD 330 000 with a range defined by the studies’ values, i.e. USD 40 000 to USD 882 000. A further line of evidence is provided by the studies that provide estimates of the risk-risk ratios between chronic bronchitis and mortality risk (value of statistical life, VSL). Whilst the US studies, using severe bronchitis, had ratios of 1:3, the study of Hammitt and Zhou (2006) in China found a range of 1:5 to 1:10. Further, a study that only estimated such ratios for a range of health outcomes (Dzielgielewska et al., 2005) found ratios of 1:10 and 1:11. Applying a mid-point ratio of 1:7 to the base VSL estimate presented in OECD (2012) of USD 3 000 000 therefore gives a range of values of USD 270 000 to USD 600 000 with a central estimate of USD 430 000. These estimates support the mid-point values identified in the WTP studies. It is therefore suggested that the values of USD 330 000 (USD 40 000 to USD 882 000) from these studies that directly estimate absolute values are adopted to represent the disutility component until better evidence becomes available.
### Table 4. Summary of original valuation studies: Chronic bronchitis

<table>
<thead>
<tr>
<th>Study/date/location; pollution type; methodology type; peer-reviewed or not</th>
<th>Value per new case (mean/median; range). Original currency year; USD&lt;sub&gt;2010&lt;/sub&gt;</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary valuation studies – North America</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viscusi et al. (1991); United States; Contingent valuation – Willingness-to-pay Peer-reviewed</td>
<td>Chronic bronchitis: USD&lt;sub&gt;1991&lt;/sub&gt;: 457 000 – 960 000 Median values for alternative risk-risk and risk-money trade-offs. USD&lt;sub&gt;2010&lt;/sub&gt;: 877 440 – 1 843 200.</td>
<td>WTP Disutility: 389 respondents. Survey did not mention other cost components though these might have been considered by respondent. 13 dimensions of CB described (see Annex 3); focused on a severe definition of CB.</td>
</tr>
<tr>
<td>Krupnick &amp; Cropper (1992); United States; Contingent valuation – Willingness-to-pay Peer-reviewed</td>
<td>Chronic: USD&lt;sub&gt;1991&lt;/sub&gt;: 460 000 – 1 060 000 Median values for alternative risk-risk trade-offs USD&lt;sub&gt;2010&lt;/sub&gt;: 883 200 – 2 035 200.</td>
<td>WTP Disutility; used Viscusi questionnaire to derive WTP from respondents familiar with illness (see Annex 3). Respondents were asked whether loss of income was consideration but explicitly asked respondents to exclude resource costs in questionnaire.</td>
</tr>
<tr>
<td><strong>Primary valuation studies – EU</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Priez and Jeanrenaud (1999); Switzerland; Contingent valuation – Willingness-to-pay Peer-reviewed</td>
<td>CHF&lt;sub&gt;1999&lt;/sub&gt;: 38 500 USD&lt;sub&gt;2010&lt;/sub&gt;: 40 598</td>
<td>WTP Disutility. Defined as: daily presence of mucus-producing cough during three months each year, for at least the last two successive years. Detailed description of health consequences given to respondent but not in paper. Less severe than Viscusi et al. Explicitly excluded resource costs in questionnaire. 757 respondents. Valued as private good.</td>
</tr>
<tr>
<td>Leu et al. (1986); Germany Cost of illness, Not peer-reviewed</td>
<td>DEM&lt;sub&gt;1984&lt;/sub&gt;: 8 000 USD&lt;sub&gt;2010&lt;/sub&gt;: 7 292</td>
<td>COI: direct (medical expenses) and indirect (production losses) costs were for one statistical case of chronic bronchitis. Quoted in Priez and Jeanrenaud (1999).</td>
</tr>
<tr>
<td>Wouters (2003); US, Canada, France, Italy, Netherlands, Spain, U.K. Cost of illness, Peer-reviewed</td>
<td>USD&lt;sub&gt;2010&lt;/sub&gt;: United States: 6 692; Canada: 4 762; France: 2 337; Italy: 2 183; Netherlands: 1 588; Spain: 7 760; United Kingdom: 4 147.</td>
<td>Survey of 3265 patients and 905 physicians. Costs include healthcare resource utilisation (including inpatient hospitalisations, emergency room visits, contacts with healthcare professionals, treatment and laboratory investigations), and lost productivity.</td>
</tr>
<tr>
<td>Dzielgiewska et al. (2005) Poland; Contingent valuation – Willingness-to-pay for improvement in air quality</td>
<td>Survey respondents asked for percentage of total rather than absolute value.</td>
<td>Survey asked respondents to offer WTP for air quality improvements of 25% and 50%, and proportion for each component; Bronchitis 10% and 11%, respectively. Description given in the survey: Bronchitis causes difficulties breathing, persistent cough and phlegm, and chest pain. It usually forces people to stay in bed, often in a hospital for a few months.</td>
</tr>
</tbody>
</table>
### Primary valuation studies – China

<table>
<thead>
<tr>
<th>Study</th>
<th>Description</th>
<th>Year</th>
<th>Value (USD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>2010</td>
<td>655 – 1,330</td>
</tr>
</tbody>
</table>

Risk-money & risk-risk (with mortality) trade-offs. Small percentage reductions in lifetime risks. 3,732 respondents. Median values recommended. COI components acknowledged in survey but not accounted for in unit values. Valued two symptoms, “coughing (with phlegm) and wheezing regularly,” and “living with an uncomfortable shortness of breath for the rest of one’s life,” i.e. less severe than Viscusi et al.
4.3 Hospital admissions (Respiratory and Cardiovascular)

Table 5 presents the unit values identified in sixteen studies that monetised the costs of illness and/or the disutility components of the hospital admissions health end-point. Six of these studies originate from North America, whilst five are from Europe and two from Brazil. There is one study identified for China and India, and one for Australia. Within the group of North American studies, one study – Stieb (2002) – synthesises the results from two Canadian studies – Anis et al. (2000) and Johnson et al. (2000) – that derive values for costs of illness and disutility, respectively. Three primary studies estimate disutility values alone whilst eight studies estimate costs of illness alone – five studies estimate both components.

The costs of illness are dominated by hospital treatment costs. For example, Chestnut et al. (2006), in their study in California, USA, estimate the ratio of treatment costs to productivity costs to be 3:1. In Europe, AEA Technology (2005) finds a ratio of 1.5:1, whilst Ortiz et al. (2011), in Brazil, find a ratio of 7:1. The UK Government guidance (Defra 2007) suggests that since hospital admissions from air pollution are likely to be borne in general by the retired population, productivity losses will be negligible. The guidance therefore does not consider these losses quantitatively.

Unusually, in the valuation of health impacts due to air pollution, hospital treatment costs also dominate the willingness-to-pay values associated with the disutility component of the overall welfare loss. For example, Ortiz et al. (2011) find a treatment cost: disutility ratio of approximately 12:1, whilst AEA Technology (2005) find a ratio of 3:1 in Europe, and Chestnut et al. (2006) find ratios of 9:1 – 12:1.

Hospital treatment costs vary across the studies, according to region. Thus, whilst the values found in Chestnut et al. (2006), Apelberg et al. (2003) and US EPA (2014) for the US are in ranges of USD 20,000 to USD 40,000 (respiratory) and USD 27,000 to USD 32,000 (cardiovascular), the range in Europe is USD 1,500 to USD 2,600, (AEA Technology, 2005; and Defra, 2007), the ranges in Brazil are USD 1,000 to USD 2,000 (respiratory) and USD 1,000 to USD 10,000 (cardiovascular) (Seroa da Motta et al., 2000; and Ortiz et al., 2011), and USD 545 to USD 670 (respiratory) and USD 1,400 (cardiovascular) in China and India. The range for Canada is USD 3,800 (respiratory) to USD 4,800 (cardiovascular). Generally, cardiovascular costs for a hospital admission case are marginally greater than those for respiratory admissions.
Table 5. Summary of original valuation studies: Hospital admissions
Respiratory hospital admissions (RHA) and Cardiovascular hospital admissions (CHA)

<table>
<thead>
<tr>
<th>Study/date/location; pollution type; Methodology type; Peer-reviewed or not</th>
<th>Value per new case (mean/median; range). Original currency year; USD2010</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary valuation studies – North America</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson et al. (2000); Canada; Primary – Choice Experiment Peer-reviewed.</td>
<td>1 day episode: CAD1997: 487 (352 – 644, 95% confidence interval) Total 1 day RHA = USD2010: 614 5 day episode: CAD1997: 799 (566 – 1 111, 95% confidence interval) Total 5 day RHA = USD2010: 1 007 10 day episode: CAD1997: 971 (668 – 1 375, 95% confidence interval) Total 10 day RHA = USD2010: 1 223</td>
<td>Respiratory &amp; cardiovascular hospital admissions. Definition: You are in hospital and need help caring for yourself (feeding, bathing, dressing, toilet). Sample size: 399. WTP for disutility component. Excludes COI components.</td>
</tr>
<tr>
<td>Anis et al. (2000); Canada Cost of illness Peer-reviewed.</td>
<td>CAD1997: 508 (Respiratory); 3 163 (Cardiovascular) USD2010: 650 (Respiratory); 4 049 (Cardiovascular)</td>
<td>Survey of patients costs – average. Direct cost of cardiorespiratory disease episodes including hospital utilization, physicians visits, concomitant medication use, equipment and out-of-pocket expenses. Opportunity costs and disutility excluded.</td>
</tr>
<tr>
<td>Chestnut et al. (2006); California, USA Willingness-to-pay + Cost of illness Peer-reviewed.</td>
<td>COI: USD2002: Respiratory 23 070 (chronic); 31 970 (acute); Cardiovascular: 36 342 COI: USD2010: Respiratory 27 918 (chronic); 38 716 (acute); Cardiovascular: USD 44 010 Disutility: USD2002: 1 600 (1 day hospitalisation); 2 100 (5 day hospitalisation); 2 700 (10 day hospitalisation) Disutility: USD2010: 1 938 (1 day hospitalisation); 2 543 (5 day hospitalisation); 2 370 (10 day hospitalisation).</td>
<td>Survey of patients who have been hospitalised for respiratory or cardiovascular illness, i.e. not representative of population. Survey includes both own and social COI plus disutility. Survey size: 394. COI treatment: opportunity cost ratio = 3:1. Opportunity costs include subsequent recovery time at home.</td>
</tr>
<tr>
<td>US EPA (2014); USA Not peer-reviewed.</td>
<td>Respiratory: USD2010: 40 500 (treatment costs) + 2 500 (4.5 days in-hospital productivity) = USD2010: 43 000 Cardiovascular: USD2010: 32 000 (treatment costs) + 5 000 (6 days in-hospital productivity) = USD2010: 37 000</td>
<td>Costs of illness: Hospital treatment costs plus productivity costs. Productivity costs (16 days respiratory; 33 days cardiovascular) valued at USD 150 per day. Derived from Agency for Healthcare Research and Quality (AHRQ) database: HCUPnet, Healthcare Cost and Utilization Project. Rockville, MD.</td>
</tr>
</tbody>
</table>
### Primary valuation studies – EU

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Methodology</th>
<th>Valuation Units</th>
<th>Values (2002/2010)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chilton et al. (2004); UK; General air pollution</td>
<td></td>
<td>Primary – Contingent valuation – Annual willingness-to-pay to avoid Not peer-reviewed.</td>
<td>GBP$<em>{2002}$: 1 560 (1 270 – 1 850) USD$</em>{2010}$: 3 089 (2 515 – 3 663)</td>
<td></td>
<td>Definition: Respiratory hospital admission. This would be most likely to benefit people in their 70's and 80's who have some kind of lung disease, or younger people with asthma or other chest conditions. By reducing the number of bad air days, such people would be less likely to develop attacks of breathing difficulties which require admission to hospital. Sample size: 517. Excludes Cost of illness components.</td>
</tr>
<tr>
<td>Ready et al. (2004); 5 European Countries; Meta-analysis – Contingent valuation – Willingness-to-pay Peer-reviewed.</td>
<td></td>
<td></td>
<td>GBP$<em>{1998}$ (PPP-adjusted): Pooled: 306 (standard error: 13.41); Netherlands: 283; Norway: 301; Portugal: 300; Spain: 426; United Kingdom: 164 USD$</em>{2010}$: Pooled: 594</td>
<td></td>
<td>Definition: Admission to a hospital for treatment of respiratory distress. Symptoms include persistent phlegmy cough, with occasional coughing fits, gasping breath, fever, headache and tiredness. Patient stays in the hospital receiving treatment for three days, followed by 5 days home in bed. Disutility component. Other components excluded. Total sample size 1264; Netherlands: 176; Norway: 203; United Kingdom: 269; Portugal: 235; and Spain: 381. Private good</td>
</tr>
<tr>
<td>Otterstrom et al. (1998) Helsinki, Finland. Contingent valuation – Willingness-to-pay + Cost of illness Not peer-reviewed.</td>
<td></td>
<td></td>
<td>FIM$<em>{1996}$: 10 494 per hospital day USD$</em>{2010}$: 7 870 per hospital day</td>
<td></td>
<td>Disutility (survey) + Treatment and opportunity costs; component unit values not identified. Survey size: approx. 600. Symptoms not specified</td>
</tr>
<tr>
<td>AEA Technology (2005); Europe; PM, NO$_2$ and O$_3$ Clean Air For Europe Cost-Benefit Analysis (CAFE CBA). Peer-reviewed.</td>
<td></td>
<td>Productivity loss: EUR$<em>{2003}$: 704 Treatment costs: EUR$</em>{2003}$: 969 Productivity loss: USD$<em>{2010}$: 1 070 Treatment costs: USD$</em>{2010}$: 1 473</td>
<td></td>
<td>Costs of illness: productivity loss for 8 days; costs of hospitalisation for three days. Source of costs not given. Added to WTP derived from Ready et al. (2004).</td>
<td></td>
</tr>
<tr>
<td>Defra (2007)</td>
<td></td>
<td>RHA: GBP$<em>{1997}$: 1 390 CHA: GBP$</em>{1997}$: 1 485 RHA: USD$<em>{2010}$: 2 502 CHA: USD$</em>{2010}$: 2 673</td>
<td></td>
<td>Disutility + Costs of illness: costs of hospitalisation 8 days (RHA), 9 days (CHA); GBP 181 per day (RHA), GBP 161 per day (CHA). No productivity costs. Added to disutility WTP derived from Ready et al. (2004) (low end) and Chilton et al. (2004) (high end).</td>
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</table>

### Primary valuation studies – China and India

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Methodology</th>
<th>Valuation Units</th>
<th>Values (2002/2010)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zhang et al. (2007); China; PM Primary – CV WTP + COI Peer-reviewed.</td>
<td></td>
<td></td>
<td>RHA: USD$<em>{2004}$: 582 CHA: USD$</em>{2004}$: 1 184 RHA: USD$<em>{2010}$: 669 CHA: USD$</em>{2010}$: 1 362</td>
<td></td>
<td>Hospital admissions (respiratory and cardiovascular); Costs of illness = treatment costs plus productivity costs (lost wages).</td>
</tr>
</tbody>
</table>
### Primary valuation studies – Other countries

<table>
<thead>
<tr>
<th>Study Source</th>
<th>Country</th>
<th>Methodology</th>
<th>Year</th>
<th>Unit</th>
<th>Estimate (USD)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seroa da Motta et al. (2000); Brazil</td>
<td>Various impacts from transport</td>
<td>Primary – Willingness-to-pay</td>
<td>1997; 2010</td>
<td>Respiratory: 1 944 – 1 986; Cardiovascular: 3 182 – 7 337</td>
<td>COI. Observed health expenditures provided by the public health system database (DATASUS).</td>
<td></td>
</tr>
<tr>
<td>DEC (NSW) (2005)</td>
<td>Not peer-reviewed</td>
<td></td>
<td></td>
<td>Respiratory: 3 880 – 4 660; Cardiovascular: 7 000 – 8 400</td>
<td>Costs of illness. No detail given on constituent parts.</td>
<td></td>
</tr>
</tbody>
</table>
Since the cost estimates are expressed in purchasing power parities, these differences cannot be explained by geographical variations in the cost of living. Though the studies do not disaggregate these costs in sufficient detail to be certain, the differences are likely to be a combination of the length of stay entailed in the case of a hospital admission, the precise cost components included in each estimate, and the accounting conventions adopted. Moreover, since the lengths of stay are broadly comparable, (generally 5-10 days), it is ventured here that the US estimates – which are notably higher than all the other estimates – are a product of the way in which capital and operational costs are attributed to hospital admissions. Nevertheless, since it is not possible to be certain of reasons for the differences in treatment costs, it is suggested that – as far as is practicable – the ranges identified in the previous paragraph should be used in OECD analyses, differentiating between respiratory and cardiovascular cases. Alternatively, in the case of analyses where common values are needed, a more crude approach would be to use a wider range for OECD countries of USD 1 500 to USD 40 000, with a broadly median mid-point of USD 5 000.

As noted above, productivity costs firstly vary according to whether it is assumed that hospital admissions are judged likely to affect the working population, and how many productive working days are estimated to be lost. The range varies therefore from zero days in the UK, to 3-4 days in Canada, 8 days in the EU, and 16 (respiratory) and 33 (cardiovascular) days in the United States. As with treatment costs, it is therefore suggested that these ranges be used in OECD analyses, differentiating between respiratory and cardiovascular cases, where possible. A crude alternative would be to use the full range identified here, (0-33 days), with a mid-point of 16 days. These day estimates should be multiplied by the work loss day unit values presented in this report.

The wide differences identified in the previous paragraphs in the unit values for the treatment cost and productivity cost components of hospital admissions also exist for the disutility cost component. Thus, Ortiz identifies willingness-to-pay values of USD 127 (respiratory) and USD 140 (cardiovascular) for 10 days hospitalization in Brazil whilst Chilton et al. (2004) derive a range of USD 2 500 to USD 3 700. The two North American studies – Chestnut et al. (2006) in the United States and Johnson et al. (2000) in Canada – produce values disaggregated by the length of stay in hospital: for one day the range is USD 600 (Canada) to USD 1 900 (United States); for five days the range is USD 1 000 (Canada) to USD 2 500 (United States); and for ten days the range is USD 1 200 (Canada) to USD 3 300 (United States). The study that pools the results from five EU countries – Ready et al. (2004) – produces a central value of USD 600 per case, and assumes three days of hospitalisation.

These differences in disutility unit values in part result from the length of time spent in hospital. It is also likely that the description of the severity of the symptoms has a role in explaining the spread of values. For example, whilst the Ready et al. (2004) describes the symptoms as, “persistent phlegmy cough, with occasional coughing fits, gasping breath, fever, headache and tiredness”, Johnson et al. (2000) describe the symptoms as being, “in hospital and need help caring for yourself (feeding, bathing, dressing, toilet)”. The more severe symptoms described by Johnson et al. help to explain the fact that their unit value for one day in hospital is USD 614 whilst Ready et al. derives a unit value of USD 600 for three days in hospital. Further, familiarity to the condition may also explain why the results of Chestnut et al. are higher again than Johnson et al.; respondents in Chestnut et al. are patients who have been hospitalised, whilst those in Johnson et al. are selected randomly from the general population in Toronto, Canada. Finally, the outliers in these values appear to be the estimates from Chilton et al., which are significantly higher. It is notable that this study did not value the endpoint separately but as part of a bundle of end-points; though it is not clear how this methodological difference would influence the unit value. Perhaps more importantly, this study has seemingly not yet been subject to an academic peer-review process.

It is suggested that the peer-reviewed unit values for disutility be used on a country and regional disaggregated basis, as far as is practicable, and disaggregated by length of stay if possible. Where such disaggregation is not possible, for OECD countries, a range of USD 600 to USD 3 300 may be adopted, with a mid-point of USD 2 000. For non-OECD countries, such as China and India, these values should be scaled on a GDP per capita basis, as is done for mortality risk unit values (OECD, 2012).
4.4 Work loss days

In Table 6 below, ten studies are identified that include valuation of work loss days. Of these ten studies, three are from North America; four are from Europe, with one study emanating from China, India and Thailand, respectively. Seven studies consider work loss days in the air pollution context whilst three studies from the UK report on surveys made of the costs to employers of absenteeism, irrespective of cause.

All but one study – Chestnut et al. (1997) – value work loss days in relation to the lost output that results from days not worked. In other words, in these studies only the productivity component of the total willingness-to-pay is valued. The Chestnut et al. study, using a survey of the general population, is therefore unique in estimating the willingness-to-pay to avoid a work loss day. In the survey, this is defined as avoiding “symptoms with loss of ability to work or conduct other basic activities.” Chestnut et al. note that “the responses indicated that these kinds of respiratory symptom days bother people more because of how they feel on those days and their reduced ability to undertake normal activities than because of concern about direct financial impacts such as medical expense or lost income”. Consequently, we can assume that the estimates – discussed below – of the value of avoided work loss days represent a lower bound on the total WTP.

There is a key assumption that is adopted in valuing loss in productivity. This assumption states that the value to society of productivity loss should be measured as the market value of lost output since this equals the market wage in a competitive labour market. The method most commonly used to estimate the value of lost productivity as a result of air pollution is the human capital approach (HCA) – most often derived from the median or mean wage rate across the potentially affected population. It should be noted, though, that the academic literature does not agree as to the most appropriate approach. For example, the friction cost approach – contrary to the human capital approach – assumes that the affected person would be replaced in the labour force (or at least a replacement would be sought) at some point in time and so is a more flexible measure of the value of productivity. The friction cost approach includes costs such as hiring and training new workers in the overall valuation. On balance, the HCA is likely to provide a higher estimate of the value of productivity change relative to the friction cost approach and the assumption of no replacement has led some to suggest that the HCA may over-estimate this cost component and hence overall productivity loss (Koopmanschap et al., 1995).

HCA makes additional assumptions about the labour market. For example, at the margin in a competitive market, it is assumed that the marginal revenue of a unit of labour is equal to the marginal cost. Marginal revenue represents the value of the marginal production of each unit of labour. However, marginal revenue is difficult to measure at the micro-level given lack of available data. HCA seeks to construct a value of the marginal cost of labour with which to value productivity losses, based on the pre-tax wage rate. However, it should be noted that the observed market wage rates may not equal marginal revenue of a unit of labour for a number of reasons: for example, if a job involves team production, or if there is time-sensitivity to outputs, the actual productivity loss may be greater than that measured by the wage rate (Zhang et al., 2011).

These caveats notwithstanding, in practice, the average market wage rate is typically used as a proxy for the marginal cost of labour, given its relative ease of measurement. Thus, the current US EPA guidance (US EPA, 2014) adopts a rate of USD 150 per day, based on the median wage level across the United States. EU air quality assessments (AEA Technology, 2005, and subsequent) also use the median wage – estimated from a survey of employers in the UK – though indirect costs associated with absenteeism, such as those that relate to lower customer satisfaction and poorer quality of products or services leading to a loss of future business. However, this component is only included in sensitivity analyses since the authors give a low level of confidence to this value because of a relatively low survey response rate for the question from which the value is derived. It is also unclear as to whether this represents an opportunity cost or whether competing companies may benefit, thereby representing an economic transfer only.

Note that in a perfectly competitive market the cost to society of this loss will equate to the cost borne by the individual affected since the individual will lose the wage that equates to the value of the lost output.
Table 6. Summary of original valuation studies: Work loss days

<table>
<thead>
<tr>
<th>Study/date/location/currency/price year; pollution type; methodology</th>
<th>Work loss day: Unit values per day</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Valuation Studies – North America</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stieb et al. (2002); Canada. Peer-reviewed. Lost productivity.</td>
<td>CAD\textsubscript{1997}: 120. USD\textsubscript{2010}: 151</td>
<td>Mean daily wage for Canada in 1997. Derived from govt. statistics.</td>
</tr>
<tr>
<td><strong>Primary Valuation Studies – Europe</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AEA Technology (2005); Europe; €2000; PM, NO\textsubscript{2} and O\textsubscript{3} Clean Air For Europe Cost-Benefit Analysis (CAFE CBA). Peer-reviewed.</td>
<td>EUR\textsubscript{2000}: 85 – direct; central estimate. EUR\textsubscript{2000}: 253 – direct &amp; indirect (sensitivity); USD\textsubscript{2010}: 137 – direct; central estimate. USD\textsubscript{2010}: 407 – direct &amp; indirect (sensitivity)</td>
<td>Productivity cost. Median. Derived from CBI (1998) survey of costs of absenteeism to employers.</td>
</tr>
<tr>
<td>CBI (2011); UK, Non-air quality. Not peer-reviewed.</td>
<td>GBP\textsubscript{2010}: 117. USD\textsubscript{2010}: 193</td>
<td>Productivity cost. Median. Includes salary cost &amp; replacement costs. Survey of costs of absenteeism to private sector employers.</td>
</tr>
<tr>
<td>DWP (2013) UK, Non-air quality. Not peer-reviewed.</td>
<td>GBP\textsubscript{2010}: 73 (wage); 94 (wage + taxes + contribution to fixed costs) USD\textsubscript{2010}: 120 (wage); 154 (wage + taxes + contribution to fixed costs)</td>
<td>Mean wage, lower quartile.</td>
</tr>
<tr>
<td><strong>Primary Valuation Studies – China and India</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Primary Valuation Studies – Other Countries</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DEC (NSW) (2005); New South Wales, Australia</td>
<td>AUD\textsubscript{2002}: 190 – 228. USD\textsubscript{2010}: 342 – 410</td>
<td>Productivity cost. Mean daily wage rate – national.</td>
</tr>
<tr>
<td>Chestnut et al. (1997); Bangkok, Thailand. Air pollution. Peer-reviewed. Primary – CV – WTP</td>
<td>USD\textsubscript{1996}: 63 Mean; 24 Median. USD\textsubscript{2010}: 88 Mean; 33 Median</td>
<td>Disutility of work loss day: symptoms with loss of ability to work or conduct other basic activities. Estimated by survey. Sample size: 141.</td>
</tr>
</tbody>
</table>
The suggestion here for OECD analysis is to follow current empirical practice and adopt the mean wage rate for the population likely to be considered in a given air quality assessment. Sub-national or industry-specific data may often be available from statistical publications or other sources, and the Li et al. study in Hebei, China, listed in Table 6, is an example of this. However, a default is to adopt national data. OECD country data is presented in Table 7 below. Additionally, China has an average wage of USD 33 whilst India has an average wage of USD 15 (International Labour Organisation database, (USD_{2010} PPP). Given the possibility – highlighted by EU Commission practice – that there may be indirect costs, it is suggested that these data should be viewed as conservative and that where robust data on indirect costs exist, these should be added to the direct.

Table 7. Mean daily wages

<table>
<thead>
<tr>
<th>Country</th>
<th>USD_{2010}, PPP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>183</td>
</tr>
<tr>
<td>Austria</td>
<td>164</td>
</tr>
<tr>
<td>Belgium</td>
<td>168</td>
</tr>
<tr>
<td>Canada</td>
<td>158</td>
</tr>
<tr>
<td>Chile</td>
<td>49</td>
</tr>
<tr>
<td>Czech Republic</td>
<td>74</td>
</tr>
<tr>
<td>Denmark</td>
<td>177</td>
</tr>
<tr>
<td>Estonia</td>
<td>67</td>
</tr>
<tr>
<td>Finland</td>
<td>142</td>
</tr>
<tr>
<td>France</td>
<td>142</td>
</tr>
<tr>
<td>Germany</td>
<td>151</td>
</tr>
<tr>
<td>Greece</td>
<td>107</td>
</tr>
<tr>
<td>Hungary</td>
<td>76</td>
</tr>
<tr>
<td>Iceland</td>
<td>118</td>
</tr>
<tr>
<td>Ireland</td>
<td>186</td>
</tr>
<tr>
<td>Israel</td>
<td>102</td>
</tr>
<tr>
<td>Italy</td>
<td>127</td>
</tr>
<tr>
<td>Japan</td>
<td>125</td>
</tr>
<tr>
<td>Korea</td>
<td>122</td>
</tr>
<tr>
<td>Luxembourg</td>
<td>198</td>
</tr>
<tr>
<td>Mexico</td>
<td>44</td>
</tr>
<tr>
<td>Netherlands</td>
<td>173</td>
</tr>
<tr>
<td>New Zealand</td>
<td>111</td>
</tr>
<tr>
<td>Norway</td>
<td>168</td>
</tr>
<tr>
<td>Poland</td>
<td>81</td>
</tr>
<tr>
<td>Portugal</td>
<td>87</td>
</tr>
<tr>
<td>Slovak Republic</td>
<td>74</td>
</tr>
<tr>
<td>Slovenia</td>
<td>119</td>
</tr>
<tr>
<td>Spain</td>
<td>131</td>
</tr>
<tr>
<td>Sweden</td>
<td>140</td>
</tr>
<tr>
<td>Switzerland</td>
<td>187</td>
</tr>
<tr>
<td>Turkey</td>
<td>84</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>151</td>
</tr>
<tr>
<td>United States</td>
<td>198</td>
</tr>
</tbody>
</table>


4.5 Restricted activity days and minor restricted activity days

Table 8 identifies ten studies that present values for either restricted activity days (RAD) or minor restricted activity days (MRAD). Three of these studies – Johnson et al. (2000) in Canada, Berger et al. (1987) in the United States, and Ready et al. (2004) for five countries in Europe – conduct primary valuation studies of the disutility component of the total willingness-to-pay for a RAD. The other seven studies use the results of these primary studies, in combination with cost of illness data, or – in the case of the study in India by Srivastava and Kumar (2002) – rely on cost of illness data alone.

The assumptions relating to the definition of costs of illness are generally not stated in a clear enough way to identify what these estimates consist of. Nevertheless, in the case of three studies – Berger et al. (1987), Stieb
et al. (2002) and AEA Technology (2005) – these details are given. In the case of Berger et al., COI equates to about USD 24, comprising both medical costs and productivity costs, and Stieb et al. also uses a percentage of productivity lost to generate a value of around USD 12; whilst in AEA Technology, it is assumed that the severity of the RAD is such that the work day is lost entirely, valued at USD 115.

Table 8 shows that the definitions of the RAD and MRAD end-points used in the primary studies are not consistent with each other. The methods used to derive the WTP disutility values are also quite different. For example, whilst the Berger et al. and Ready et al. studies use a contingent valuation approach to allow respondents to give a WTP directly, Johnson et al. adopt a choice experiment approach that relies on deriving an implicit WTP from the trade-offs respondents make between different sets of attributes, including cost. Whilst it is not clear whether – and in what way – alternative techniques influence WTP results, it is likely that the combination of methodological and definitional differences account for the range of values for the disutility component. These values start at USD 29 in the Johnson et al. study for Canada, through a central value of USD 62 in Europe from Ready et al. to USD 153 in the US from Berger et al.

In the absence of an established definition of a RAD across world regions, it is important that the values currently used in individual countries and regions continue to be used in geographically disaggregated analyses. However, where an international, cross-regional analysis is to be undertaken, the apparent lack of commonality across the studies suggests that a range of values should be used that incorporate the values above. This appears to be the approach adopted by DEC in New South Wales, Australia who has a range of USD 44 to USD 298. It is not clear which studies are used to generate this range. However, combining the results presented above for COI and disutility components gives a similar range of USD 41 to USD 268. A mid-point of USD 170 within this range might then be used as a central value for RADs in OECD countries, adjusted in both these countries and non-OECD countries in the same way as mortality values are treated in OECD (2012). MRADs are not assumed to have COI component, whilst the two studies that provide estimates of the disutility component – Apelberg et al. (2003), based on an adjustment to Berger et al. for the United States, and Ready et al. (2003) for Europe – produce a range of USD 53 to USD 70, with a central value of USD 62.
Table 8. Summary of original valuation studies: Restricted activity days
Restricted activity days (RADs) and Minor restricted activity days (MRADs)

<table>
<thead>
<tr>
<th>Study/date/location/pollution type; methodology</th>
<th>Restricted activity days: Unit values per day</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Stieb et al. (2002); Canada. Transfer – Contingent valuation (Stated preferences) + Cost of illness + Opportunity cost Peer-reviewed. | RAD: CAD$_{1997}$: 25 + 23. Total = 48 (13-82, 95% confidence interval)  
RAD: USD$_{2010}$: 60 (16-103) | Lost productivity plus pain, suffering and avertive expenditures out of hospital.  
Lost productivity (work loss days) assumed to comprise 21% of restricted activity days – applied this factor in estimating lost productivity costs associated with restricted activity days.  
Combines findings of Johnson et al. (2000) below with own calculations for productivity costs. |
| Johnson et al. (2000); Canada; Primary – Choice Experiment Peer-reviewed. | RAD: CAD$_{1997}$: 23 - 1 day episode  
RAD: USD$_{2010}$: 29 - 1 day episode | Definition: You can go to work, go to school, do housework, but you have some physical limitations (trouble bending, stooping or doing vigorous activities) and cannot participate in social or recreational activities because of this health condition. 399: sample size. WTP for disutility component. Excludes COI components. |
MRAD: USD$_{2010}$: 70 | Median WTP estimate to avoid one MRAD from Berger, et al. (1987). Distribution is assumed to be triangular with a minimum of USD 22 and a maximum of USD 83. Range is based on assumption that value should exceed WTP for a single mild symptom (the highest estimate for a single symptom--for eye irritation--is USD 16.00) and be less than that for a WLD. The triangular distribution acknowledges that the actual value is likely to be closer to the point estimate than either extreme. |
RAD: USD$_{2010}$: 177 | Disutility WTP: among those who experienced coughing spells in the previous year, for avoiding one extra day of cough with certainty is USD$_{1985}$: 76.  
Costs of illness: calculated as expenditures on doctor visits and medicine net of insurance reimbursements plus lost earnings. USD$_{1985}$: 12. |
### Primary valuation studies – EU

<table>
<thead>
<tr>
<th>Study</th>
<th>RAD/USD2010: 32 (standard error 5);</th>
<th>MRAD/USD2010: 27 (standard error 2);</th>
<th>RAD Definition: Three days with flu-like symptoms including persistent phlegmy cough with occasional coughing fits, fever, headache and tiredness. Symptoms are serious enough that patient must stay home in bed for the three days. RAD values presented here divide study values by three to give per day estimate.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ready et al. (2004); 5 European Countries; Meta-analysis – Contingent valuation – Willingness-to-pay Peer-reviewed.</td>
<td>Netherlands: 23; Norway: 40; Portugal: 29; Spain: 37; United Kingdom: 27</td>
<td>Netherlands: 28; Norway: 36; Portugal: 28; Spain: 39; United Kingdom: 20</td>
<td>MRAD Definition: One day with persistent phlegmy cough, some tightness in the chest, and some breathing difficulties. Patient cannot engage in strenuous activity, but can work and do ordinary daily activities. Disutility component. Other components excluded. Total sample size 1264; Netherlands: 176; Norway: 203; United Kingdom: 269; Portugal: 235; and Spain: 381. Private good.</td>
</tr>
</tbody>
</table>

#### RAD: GBP, PPP-adjusted: Pooled: 32 (standard error 5);

- Netherlands: 23
- Norway: 40
- Portugal: 29
- Spain: 37
- United Kingdom: 27

#### USD2010: Pooled: 62

- Netherlands: 23
- Norway: 40
- Portugal: 29
- Spain: 37
- United Kingdom: 27

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>AEA Technology (2005); Europe; PM, NO₂ and O₃ Clean Air For Europe Cost-Benefit Analysis (CAFÉ CBA). Peer-reviewed.</td>
<td>RAD = USD2010: 177</td>
<td>EUR2000: 85</td>
<td>Costs of illness: productivity loss only.</td>
</tr>
</tbody>
</table>

#### Productivity loss:

- EUR2000: 85
- USD2010: 115

### Valuation Studies – China and India

<table>
<thead>
<tr>
<th>Study</th>
<th>RHA: INR2001: 37.5</th>
<th>RHA: USD2010: 1.4</th>
<th>Costs of illness: Productivity loss only.</th>
</tr>
</thead>
</table>

#### Transfer values from US EPA (1999), derived from Berger et al. (1997). |

|-------------------------------|----------------|----------------|Transfer values from US EPA (1999), derived from Berger et al. (1997). |

#### RAD/person/year = USD2000: 847

#### RAD/person/year = USD2010: 1,071

#### Note: unit not comparable since number of days/year not specified. Methodology not described in detail. |

### Valuation Studies – Other countries

|-------------------------------|-------------------------|-------------------------|Results from benefit transfer of existing studies. |
4.6 Acute lower respiratory infections and acute bronchitis in children

Table 9 below identifies nine studies that derive unit values relating to acute lower respiratory infections (ALRI) and acute bronchitis in children. The studies are all from different countries, apart from that by Dickie and Hubbell (2004) which derives an alternative set of results from the same set of observations as Dickie and Messman (2004). Since the latter study is peer reviewed, the focus is on the results of this study. Thus, out of eight independent studies, four are primary stated preference studies that estimate the WTP to avoid disutility. These four studies also incorporate COI components into the survey questions.

It should be noted, however, that whilst – as described above – the epidemiological definition for ALRI is given by Mehta et al. (2013), the economic valuation research community has not attempted to derive estimates for these end-points. Instead, the table reflects a wide range of alternative health end-points, ranging from a mother’s WTP to avoid a day’s cold symptoms, (Liu et al., 2000, in Taiwan), to the avoidance of 24 days of severe symptoms, i.e. cases that involve high discomfort and activity restriction (Dickie & Messman, 2004). The former study derives a value of USD 72 per day whilst the latter derives a value of USD 511 per case.

Perhaps as a result of methodological or cultural differences, however, there does not seem to be a simple correlation between severity and WTP. Thus, the central value from the Braun Kohlova and Scasny (2006, not peer-reviewed) study for a case of mild bronchitis in Czech Republic is USD 55 – less than for a day of cold in Taiwan. More reassuringly, the value derived by Ortiz et al. (2011) for a visit to a hospital out-patient’s department is USD 175. As far as is possible, it is advised to match the unit value with the epidemiological definition most in line with that used in the valuation study.

In order to generate indicative numbers in economic assessments that adopt the ALRI and acute bronchitis end-points it is nevertheless possible to use the range of estimates presented in the table below. Given that the definitions of end-points map best to the valuation studies that derive values for relatively severe symptoms and conditions, it seems sensible to adopt results from e.g. the Dickie and Messman study where 24 symptom days are avoided – USD 464 per case (with a range of USD 301 to USD 511, depending on severity) for both health end-points. This range overlaps with that of USD 386 to USD 1 369 adopted in the DEC study for New South Wales, Australia. However, since the DEC study does not give details of the provenance of these values, it is judged here that it is preferable to adopt the range given in the peer-reviewed Dickie and Messman study.

26. Mehta et al. (2013) describe ALRI as “including pneumonia and bronchiolitis of bacterial and viral origin”; as being “nearly always diagnosed clinically, based on severe respiratory symptoms” and as being “characterized by acute-onset cough or difficulty in breathing with fast breathing for age”; severe ALRI was defined as “acute cough or difficulty in breathing in drawing of the lower chest wall necessitating hospital admission”.

27. See Appendix 1 of Gehring et al. (2006) for alternative definitions used by different countries. The most common is “a child has suffered from a respiratory disease in the last 12 months.”
### Table 9. Summary of original valuation studies: Acute lower respiratory infections

Acute lower respiratory infections & acute bronchitis in children

<table>
<thead>
<tr>
<th>Study/ date/ Location/ Pollution type; Methodology</th>
<th>Acute Lower Respiratory Infections - Unit values per new case (mean/median; range). Original currency year; USD 2010</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Valuation studies – North America</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Dickie & Hubbel (2004); Hattiesburg, Mississippi, US. Note: Supplementary analysis to Dickie & Messman (2004). CV Not peer-reviewed. | \( \text{USD}_{2004} \): 161-409 per case \((\text{non-asthmatics})\)  
\( \text{USD}_{2004} \): 91-297 per case \((\text{asthmatics})\)  
\( \text{USD}_{2010} \): 186-472 per case \((\text{non-asthmatics})\)  
\( \text{USD}_{2010} \): 105-343 per case \((\text{asthmatics})\) | Disutility WTP + COI not separated. Four symptoms (cough with phlegm, shortness of breath with wheezing, chest pain on deep inspiration, and/or fever with muscle pain and fatigue) of short \((\text{two days or one week})\) duration. Sample size: 295 |
| Dickie & Messman (2004); Hattiesburg, Mississippi, US. CV Peer-reviewed. | \( \text{USD}_{2000} \): 160 per case \((1 \text{ symptom day avoided})\) \((128 – 217)\)  
\( \text{USD}_{2000} \): 367 per case \((24 \text{ symptom days avoided})\) \((238 – 404)\)  
\( \text{USD}_{2010} \): 202 per case \((1 \text{ symptom day avoided})\) \((162 – 275)\)  
\( \text{USD}_{2010} \): 464 per case \((24 \text{ symptom days avoided})\) \((301 – 511)\) | Disutility to avoid respiratory symptoms. WTP + COI not separated. Main result is typical symptoms whilst those in brackets illustrate mild to severe. "mild" cases involve low discomfort and activity restriction, while "severe" cases involve high discomfort and activity restriction. Sample size: 415 |
| **Valuation studies – EU**                        |                                                                                                 |          |
| Braun Kohiova & Scasny (2006); Czech Republic. Not peer-reviewed. | EUR\(_{2005}\): 38 per case of mild bronchitis.  
USD\(_{2010}\): 55 per case of mild bronchitis. | Mild bronchitis. Disutility WTP + COI not separated. Symptoms: phlegmy cough; breathing difficulties; slight fever; headache and tiredness. Child must stay at home in bed for 5 days. Medicines: light analgesics. Mother or another relative stays at home with child. Sample size: 415 |
| **Valuation studies – China and India**          |                                                                                                 |          |
Child bronchitis: USD\(_{2010}\): 1.5 | Costs of Illness: no details given. |
| Zhang et al. (2007); China Peer-reviewed.        | \( \text{USD}_{2007} \): 13 per Out-patient visit for paediatrician  
\( \text{USD}_{2010} \): 14 per Out-patient visit for paediatrician | Costs of Illness: derived from public statistics \((\text{in Chinese})\). No details given. Outpatient visit for paediatrician. i.e. emergency room visit. |
| Kan & Chen (2004) Shanghai, China Peer-reviewed. | \( \text{USD}_{2001} \): 13 per Out-patient visit for paediatrician  
\( \text{USD}_{2010} \): 16 per Out-patient visit for paediatrician | Costs of Illness: no details given. Outpatient visit for paediatrician. i.e. emergency room visit. |
| **Valuation Studies – Other Countries**           |                                                                                                 |          |
| Liu et al. (2000); Taiwan CV. Peer-reviewed.     | \( \text{USD}_{2000} \): 57 per day – cold  
\( \text{USD}_{2010} \): 72 per day – cold | Disutility + COI. Not separated. Mother’s WTP for child to avoid one day of cold symptoms. Respondent asked for WTP to avoid re-occurrence of cold previously experienced. Reminded of losses including: \((1)\) increased medical expenditures, \((2)\) spending time visiting the doctor or hospital, \((3)\) poor working performance, \((4)\) missing leisure time and daily activities. |
Child bronchitis: USD\(_{2010}\): 386 – 1 369 | Results from benefit transfer of existing studies. |
| Ortiz et al. (2011); Brazil; Primary – CV; WTP + COI +OC Peer-reviewed. | Emergency Room Visit.  
EUR\(_{2007} \): 91 per visit to hospital \((\text{child 0-5 years of age})\)  
USD\(_{2010} \): 175 per visit to hospital \((\text{child 0-5 years of age})\) | Emergency Room Visit. Disutility \((\text{survey})\) + Treatment + opportunity costs. 400 sample size. Respiratory symptoms not specified. Air quality context: private good. |
5. Concluding comments

The analysis in Sections 1 and 2 led to identification of a set of health end-points that may be used in OECD analyses of the impacts of air quality on human morbidity. Six health end-points are suggested as important to include in such analyses – their importance based on factors such as the strength of the epidemiological evidence and likely dominance in accounting for the bulk of welfare impacts, at least in OECD countries. The six health end-points are:

1. Cases of chronic bronchitis;
2. Hospital admission cases resulting from respiratory or cardiovascular symptoms;
3. Work loss days;
4. Restricted activity days and minor restricted activity days
5. Acute lower respiratory infections in children aged less than 5 years
6. Acute bronchitis in children

As noted earlier, the strategy adopted in the present report is to recommend a core set of pollutant-health pathways and recommend that these be used, or at least be seriously considered for use, more widely. This is in line with another “principle”, that puts emphasis on the weight of evidence internationally rather than the lack of evidence (or indeed occasionally contrary evidence) locally. It does not, however, avoid the need for future quantification exercises to think through the issues afresh. Other authors, closer to the exact circumstances of particular countries, will now or in the future be better placed than the present ones to judge whether the inclusion of a specific pollutant-health pathway is in fact likely to improve the overall estimate of health impacts; or if indeed the barriers to transferability of relationships, and other information gaps, are so large that quantification cannot be supported.

Section 4, the evidence relating to the economic valuation of (avoiding) these six health end-points has been collated and reviewed. It was found that the international experience in valuing these end-points is very limited – the few studies that have been identified frequently using different definitions of the same end-points. This restricts the depth of comparison that can be made. However, the unit values for each end-point appear to be of broadly similar magnitude, at least. On this basis – and emphasising that the uncertainties preclude more than indicative assessment – it is suggested that the ranges presented in Table 10 could be considered to be adopted in OECD country analyses. Alternatively, analysts may prefer to evaluate the individual studies themselves and – for example – select values for transfer that best reflect their specific decision contexts. In order to be consistent with the approach adopted in mortality risk valuation it is recommended that adjustments to country-specific contexts are made for exchange rates, inflation and income growth in the same way. This is described in Box 2.2: Calculating country-specific VSLs: Adjustment factors and illustrative example in OECD (2014, page 54).

<table>
<thead>
<tr>
<th>Health end-point</th>
<th>Central unit value</th>
<th>Range (lower – higher)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases of chronic bronchitis</td>
<td>334 750</td>
<td>41 700 – 889 800</td>
</tr>
<tr>
<td>Hospital admission cases</td>
<td>2 000</td>
<td>600 – 3 300</td>
</tr>
<tr>
<td>Work loss days</td>
<td>Country-specific</td>
<td>Country-specific</td>
</tr>
<tr>
<td>Restricted activity days &amp; Minor restricted activity days</td>
<td>RAD: 170</td>
<td>RAD: 41 – 268</td>
</tr>
<tr>
<td></td>
<td>MRAD: 62</td>
<td>MRAD: 53 – 70</td>
</tr>
<tr>
<td>Acute lower respiratory infections in children aged &lt; 5 years</td>
<td>464</td>
<td>301 – 511</td>
</tr>
<tr>
<td>Acute bronchitis in children</td>
<td>464</td>
<td>301 – 511</td>
</tr>
</tbody>
</table>

Given the uncertainty in the international transferability of many of the health end-points, and more generally in CRFs for air pollution-related morbidity other than hospital admissions, there is a risk that any finalised set of CRFs for which reasonable certainty exists would severely under-estimate total effects. There is also a risk that the uncertainties in transferring CRFs developed in Europe and North America to very different populations and circumstances elsewhere could greatly reduce the usefulness of any quantification.
based on these CRFs for decision-making. In contrast to morbidity, background rates of mortality are well defined in most countries and the CRFs linking air pollution to mortality are well established and apparently transferable between different geographical regions.

It would seem logical to anticipate that there is a relationship between total air pollution-related morbidity and air pollution-related mortality and that that relationship would be reasonably similar in different populations exposed to broadly similar pollutants generated by transport and other combustion sources.

A pragmatic approach to calculating air pollution-related morbidity effects might therefore be to assume that they are a (near) constant fraction of the total health impact. If the relationship between morbidity and mortality can be established for areas of the world where there is reasonable certainty about CRFs and background rates, then it might be possible to use that relationship to estimate the total scale of morbidity effects where CRFs and background rates are unknown. It would still, of course, be necessary to determine whether such a relationship would hold in economic terms; nevertheless it would be useful to understand if such an approach is at all feasible in public health terms.

There is information for both the US and the EU about the calculated mortality and morbidity burdens associated with air pollution. In the US EPA (2011) analysis of the benefits and costs of the Clean Air Act from 1990 to 2020, the economic valuation of morbidity effects for the 2010 scenario is 4% of that of mortality, with the mortality impacts (and hence the analysis as a whole) dominated by the effects on mortality in adults aged 30 years or more of long term exposure to PM$_{2.5}$ (Table 11).

Table 11. The health impact assessment and cost-benefit analysis of the US Clean Air Act

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Pollutant</th>
<th>Incidence</th>
<th>2000</th>
<th>2010</th>
<th>2020</th>
<th>Valuation, millions USD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2000</td>
</tr>
<tr>
<td>Mortality – adults 30 and older</td>
<td>PM</td>
<td>110 000</td>
<td>160 000</td>
<td>230 000</td>
<td>710 000</td>
<td>1 200 000</td>
</tr>
<tr>
<td>Mortality – infants</td>
<td>PM</td>
<td>160</td>
<td>230</td>
<td>280</td>
<td>1 300</td>
<td>1 900</td>
</tr>
<tr>
<td>Mortality – all ages</td>
<td>Ozone</td>
<td>1 400</td>
<td>4 300</td>
<td>7 100</td>
<td>10 000</td>
<td>33 000</td>
</tr>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic Bronchitis</td>
<td>PM</td>
<td>34 000</td>
<td>54 000</td>
<td>75 000</td>
<td>14 000</td>
<td>24 000</td>
</tr>
<tr>
<td>Non-fatal myocardial infarction</td>
<td>PM</td>
<td>79 000</td>
<td>130 000</td>
<td>200 000</td>
<td>8 100</td>
<td>14 000</td>
</tr>
<tr>
<td>Hospital admissions, respiratory</td>
<td>PM, ozone</td>
<td>20 000</td>
<td>41 000</td>
<td>66 000</td>
<td>290</td>
<td>640</td>
</tr>
<tr>
<td>Hospital admissions, cardiovascular</td>
<td>PM</td>
<td>25 000</td>
<td>45 000</td>
<td>69 000</td>
<td>760</td>
<td>1 300</td>
</tr>
<tr>
<td>Emergency room visits, respiratory</td>
<td>PM, ozone</td>
<td>58 000</td>
<td>86 000</td>
<td>120 000</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td>Acute Bronchitis</td>
<td>PM</td>
<td>96 000</td>
<td>130 000</td>
<td>180 000</td>
<td>42</td>
<td>61</td>
</tr>
<tr>
<td>Lower respiratory symptoms</td>
<td>PM</td>
<td>1 200 000</td>
<td>1 700 000</td>
<td>2 300 000</td>
<td>22</td>
<td>30</td>
</tr>
<tr>
<td>Upper respiratory symptoms</td>
<td>PM</td>
<td>980 000</td>
<td>1 400 000</td>
<td>2 000 000</td>
<td>30</td>
<td>42</td>
</tr>
<tr>
<td>Asthma exacerbation</td>
<td>PM</td>
<td>1 200 000</td>
<td>1 700 000</td>
<td>2 400 000</td>
<td>61</td>
<td>90</td>
</tr>
<tr>
<td>Minor restricted activity days</td>
<td>PM, ozone</td>
<td>49 000 000</td>
<td>84 000 000</td>
<td>110 000 000</td>
<td>2 900</td>
<td>4 900</td>
</tr>
<tr>
<td>Work loss days</td>
<td>PM</td>
<td>8 000 000</td>
<td>13 000 000</td>
<td>17 000 000</td>
<td>1 300</td>
<td>2 000</td>
</tr>
<tr>
<td>School loss days</td>
<td>Ozone</td>
<td>1 200 000</td>
<td>3 200 000</td>
<td>5 400 000</td>
<td>110</td>
<td>290</td>
</tr>
<tr>
<td>Outdoor worker productivity</td>
<td>Ozone</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>30</td>
</tr>
</tbody>
</table>

Note: All incidence and valuation results are rounded to two significant figures. All estimates are annual estimates for individual target years of the analysis. Mortality valuation estimates reflect a delay in mortality incidence from the time of the exposure change in the target year, reflecting application of a 20-year-distributed cessation lag and a 5% discount rate.


Table 12 summarises results from the implementation by Holland (2014) of the HRAPIE CRFs as part of the recent cost-benefit analysis for the EU’s new clean air policy. At first sight these give an implied value of morbidity impacts is about 37–38% of the mortality impact, or 27% to 28% of the total impact. Note, however, that the valuation of adult mortality was based on value of a life year (VOLY), with median value selected from a range of studies. As a result, the results are not directly comparable with those of the United States, where valuation is in terms of values of a statistical life (VSL). In fact, both approaches were used in Europe and the range of monetised benefits in the EU study reflects the different results that they give. Using median VOLY estimates of monetary value gives lowest mortality results and so gives rise to the highest proportion of morbidity to total benefits. If instead the highest value of total benefits from the EU assessment is used, i.e. EUR 198 billion and EUR 207 billion for 2025 and 2030 respectively (bottom row of Table 4.2), then the
morbidity benefits as a proportion of total benefits is <10% and so is much more comparable to that found in USEPA (2011) where valuation of mortality was based on VSL.

Table 12. The cost-benefit analysis of the European Commission’s clean air policy
Benefits of moving from CLE to the MTFR scenario, EUR million per year, EU 28

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>CLE – MTFR, 2025</th>
<th>CLE – MTFR, 2030</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Particulate matter</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic mortality (all ages), median VOLY</td>
<td>42 605</td>
<td>41 623</td>
</tr>
<tr>
<td>Infant mortality (0-1 year), median VSL</td>
<td>198</td>
<td>185</td>
</tr>
<tr>
<td>Morbidity</td>
<td>16 187</td>
<td>16 388</td>
</tr>
<tr>
<td><strong>Ozone</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute mortality (all ages), median VOLY</td>
<td>161</td>
<td>160</td>
</tr>
<tr>
<td>Morbidity</td>
<td>595</td>
<td>599</td>
</tr>
<tr>
<td><strong>Total health benefits</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality only, median VOLY, median VSL for infant mortality</td>
<td>42 424</td>
<td>41 968</td>
</tr>
<tr>
<td>Mortality and morbidity, median VOLY, median VSL for infant mortality</td>
<td>57 996</td>
<td>57 759</td>
</tr>
<tr>
<td><strong>Range</strong></td>
<td>57 966 – 198 377</td>
<td>57 759 – 207 054</td>
</tr>
</tbody>
</table>

Note: Summary results from Holland (2014) from the cost-benefit analysis for the European Commission’s clean air policy, using results from HRAPIE (WHO, 2013b). Results show differences between two scenarios: baseline current legislation (CLE) and maximum technically feasible reduction (MTFR) at two time-points. For VOLY and VSL, see the text above.

Source: Holland (2014).

On preliminary investigation the ratio of mortality to morbidity effects in EU and US evaluations is very different (when stated in monetary terms) suggesting that this approach would require a more substantial quantity of development work than can be accommodated within the scope of this project. Somewhat more detailed investigation suggests that, as anticipated, the apparent differences are strongly influenced by different approaches to valuation of mortality in HIA work for the US Government and for the EU; the differences are much less when EU mortality valuations are based on VSL rather than VOLYs and, under some valuations, morbidity costs become <10% of the mortality ones.

There is therefore a case for maintaining OECD’s previous proposal (OECD, 2014) that morbidity costs can be estimated approximately by applying a 10% mark-up on the costs of mortality, where mortality costs have been based on VSL methods. Given that the evidence points to under-estimation of morbidity impacts in quantitative HIA, it may be that morbidity costs could be assumed to be somewhat higher, at 10% to 15% of mortality costs (mortality being valued using VSL methods); the high end reflecting an assumption that morbidity effects are currently likely to be under-estimated. On the other hand, there are indications that also mortality costs could be under-estimated in recent assessments, not least as they have not included mortalities caused by NO\textsubscript{2}. This can argue for maintaining a 10% mark-up.

28. On the other hand, there are indications that also mortality costs could be under-estimated in recent assessments, not least as they have not included mortalities caused by NO\textsubscript{2}. This can argue for maintaining a 10% mark-up.
REFERENCES


ANNEX 1. SUPPLEMENTARY MATERIAL FOR SECTION 1

Table A1.1 Pollutant-health combinations recommended for use in the European Commission’s cost-benefit analysis of policy options in the CAFÉ project

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Health outcome</th>
<th>Applicability</th>
<th>Reliability/independent contribution to effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{10}$ – long term</td>
<td>New cases of chronic bronchitis in adults</td>
<td>Adults aged 27+ without chronic bronchitis</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$ – short term</td>
<td>Cardiovascular hospital admissions</td>
<td>All age</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$ – short term</td>
<td>Respiratory hospital admissions</td>
<td>All age</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$ – short term</td>
<td>Primary care consultations asthma</td>
<td>0-14 years 15-64 years 65+ years</td>
<td>Sensitivity analysis</td>
</tr>
<tr>
<td>PM$_{2.5}$ – short term</td>
<td>Primary care consultations upper respiratory symptoms – not allergic rhinitis</td>
<td>0-14 years 15-64 years 65+ years</td>
<td>Sensitivity analysis</td>
</tr>
<tr>
<td>PM$_{2.5}$ – short term</td>
<td>RADs</td>
<td>18-64 years All ages</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{2.5}$ – short term</td>
<td>WLD</td>
<td>15-64 (all)</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{2.5}$ – short term</td>
<td>Minor RADs (MRADs)</td>
<td>18-64 years All ages</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$ – short term</td>
<td>Medication use in children with asthma</td>
<td>5-14 years</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$ – short term</td>
<td>Medication use in adults with asthma</td>
<td>Adults aged 20+ years</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$</td>
<td>Acute lower respiratory symptoms in children – symptom days</td>
<td>Children aged 5-14</td>
<td>Main analysis</td>
</tr>
<tr>
<td>PM$_{10}$</td>
<td>Acute respiratory symptoms in children – symptom days</td>
<td>Adults</td>
<td>Main analysis</td>
</tr>
<tr>
<td>O$_3$ – short term</td>
<td>Respiratory hospital admissions</td>
<td>Adults aged 65+</td>
<td>Main analysis</td>
</tr>
<tr>
<td>O$_3$ – short term</td>
<td>Primary care consultations - allergic rhinitis</td>
<td>0-14 years 15-64 years</td>
<td>Sensitivity analysis</td>
</tr>
<tr>
<td>O$_3$ – short term daily 8 hour max</td>
<td>MRADs</td>
<td>18-64 years All ages</td>
<td>Main analysis</td>
</tr>
<tr>
<td>O$_3$ – short term daily 8 hour mean</td>
<td>Medication use in children with asthma</td>
<td>5-14 years</td>
<td>Sensitivity analysis</td>
</tr>
<tr>
<td>O$_3$ – short term daily 8 hour mean</td>
<td>Medication use in adults with asthma</td>
<td>Adults aged 20+ years</td>
<td>Main analysis</td>
</tr>
<tr>
<td>O$_3$ – daily 8 hour</td>
<td>Acute respiratory symptoms – cough - lower respiratory symptoms</td>
<td>Children 5-14 years</td>
<td>Main analysis</td>
</tr>
<tr>
<td>O$_3$</td>
<td>Acute lower respiratory symptoms – symptom days</td>
<td>Adults</td>
<td>Sensitivity analysis</td>
</tr>
</tbody>
</table>

Table A1.1 shows the pollutant-health pairs recommended and used for assessment of air pollution and morbidity in the European CAFÉ project (Hurley et al., 2005). Quantification was for PM and O$_3$ only. For a number of health end-points where country specific baseline rates were not available, the baseline rates for quantification were derived from the source study for the CRF. These CRFs are indicated in italics in the Table above.
ANNEX 2. ECONOMIC VALUATION OF MORBIDITY HEALTH END-POINTS

Individual components

In the model summarised in Freeman (2003), health is measured by the number of sick days, \( s \), in any period of time, which ignores the severity of the illness and differences in the symptoms experienced. Among other determinants of the health status, the level of exposure to pollutants or the dose of the contaminant, \( d \), depends on the concentration of the pollutant, \( c \), and the amount of the averting activities, \( a \), undertaken to reduce the exposure to pollution. Additionally, the individuals can choose mitigating activities and treatments, \( b \), to reduce the health effects of a given level of exposure to pollutants. Examples of mitigating activities, \( b \), include visiting a doctor or taking medicines to reduce a symptom, while examples of averting activities, \( a \), include staying indoors in days of high levels of pollution.

The health production function of an individual can be formalised as follows:

\[
s = s(c, a, b), \quad \text{with } \frac{\partial s}{\partial c} > 0 \text{ and } \frac{\partial s}{\partial a} \cdot \frac{\partial s}{\partial b} < 0 \quad (1)
\]

Individuals maximise their utility function, \( u \), subject to their budget constraint. Utility depends on the consumption of a numeraire good, \( X \), normalised with a price of 1, leisure \( f \) and health. Formally:

\[
\begin{align*}
\text{max } & \quad u = u(X, f, s) \\
\text{subject to } & \quad I + p_w(T - f - s) = X + (p_a * a) + (p_b * b) \\
\end{align*}
\]

where:
- \( I \) non-labour income;
- \( p_w \) wage rate;
- \( T \) total time available;
- \( p_a \) price of averting activities;
- \( p_b \) price of mitigating activities;

and \( \frac{\partial u}{\partial X}, \frac{\partial u}{\partial f} > 0 \) and \( \frac{\partial u}{\partial s} < 0 \)

1. First-order conditions for a maximum include: \( \frac{\partial u}{\partial X} = \lambda; \quad \frac{\partial u}{\partial f} = \lambda * p_w; \) and

\[
\lambda * \frac{p_b}{\partial s/\partial b} = \frac{\partial u}{\partial s} - \lambda * p_w = \lambda * \frac{p_a}{\partial s/\partial a}, \quad \text{where } (\lambda) \text{ is the marginal utility of income.}
\]

Pollution can affect utility through health, aesthetic amenities and odour, but in this simple model, pollution affects utility only through health. In this case, WTP for reduced pollution is the reduction in the cost of achieving the optimal level of health given a variation in pollution levels. The marginal willingness-to-pay (MWTP) for a reduction in pollution, \( (w_c) \), is given by the marginal cost of reducing the number of pollution.
sick days associated with the reduced pollution. It is obtained by differentiating the indirect utility function $v(I, p_a, p_b, p_c)$ and solving for ($w_c$):

$$w_c = \frac{dI}{dc} = -\frac{\partial v / \partial c}{\partial v / \partial I} = -\frac{\partial v / \partial c}{\lambda} \quad (3)$$

However, the effect of pollution concentration, ($c$), on utility consists of two components, the direct loss of utility associated with the illness and the opportunity cost of the time lost due to the illness, valued at the wage rate:

$$\frac{\partial v}{\partial c} = \left( \frac{c u_s}{c_s} - \frac{c u_p}{c_p} \right) - \left( \lambda * p_a * \frac{c s}{c} \right) = \left[ \frac{c u s}{c_s} - \left( \lambda * p_a \right) \right] \frac{c s}{c} = \left[ \lambda * \frac{p_b}{c s / c b} \right] \frac{c s}{c} \quad (4)$$

Substituting equation (4) into equation (3) and using the implicit function rule we obtain the expressions for MWTP, or ($w_c$):

$$w_c = -p_a \frac{\partial c u / \partial c}{\partial c s / \partial a} = p_a \frac{\partial c u / \partial c}{\partial c s / \partial a} = p_b \frac{\partial c b / \partial c}{\partial c s / \partial b}, \quad (5)$$

where $\frac{\partial c s / \partial c}{\partial c s / \partial a}$ and $\frac{\partial c s / \partial c}{\partial c s / \partial b}$ are the marginal rates of substitution between pollution and the other inputs in the production of health, which are equal at the margin in order to minimise the cost of producing health (or maximising utility). As can be seen in equation (5), MWTP can be calculated from the reductions in expenditures on either mitigating or averting behaviour measures taken to attain the original health status, ceteris paribus.

Freeman (2003) discusses the difficulties associated with the practical implementation of (5) and, alternatively, suggests another expression relating MWTP to observable costs of illness. The author suggests one initial step to obtain the demand functions for ($a$), $a(I, p_a, p_b, p_c)$, and ($b$), $b(I, p_a, p_b, p_c)$. These demand functions give the optimal quantities of ($a$) and ($b$) as functions of income, prices and pollution. By taking the total derivative of the health production function, one can estimate the effect of a change in pollution on illness:

$$w_c = \left( p_a \frac{d s}{d c} \right) + \left( p_b \frac{d b}{d c} \right) + \left( p_a \frac{d a}{d c} \right) - \left( \lambda \frac{c u s}{c s / \lambda} \frac{d s / c s}{d c} \right) \quad (6)$$

Equation (6) suggests that MWTP for reduced pollution is the sum of observable reductions in the economic value of reductions in sick-time and mitigating activities (the cost of illness), averting activities and the monetary equivalent of the disutility of illness. The marginal willingness-to-pay for an exogenous reduction in illness, ($w_c$), can be derived as a special case of (6) by supposing that averting behaviour, ($a$), is not possible, or is minimal, and that mitigation, ($b$), reduces sick days from its exogenous level, ($s*$),
according to \( s = f(s^*, b) = s^* - s(b) \). The analogue to equation (6) states that MWTP is the sum of the cost of illness (mitigation costs plus lost wages) and the monetary equivalent of the lost utility. Formally:

\[
W_{iw} = p_w + \left( \frac{\partial b^*}{\partial s} \right) - \left( \frac{\partial u}{\partial s} \right)^* \lambda 
\]  

(7)
ANNEX 3. DESCRIPTIONS OF CHRONIC BRONCHITIS

Viscusi et al. (1991) and Krupnick & Cropper (1992)

Viscusi, Magat and Huber (1991)
Respondents were asked to assume that the symptoms listed below were associated with chronic bronchitis. Respondents were faced with alternative levels of risk of suffering from chronic bronchitis and asked to trade them off against money amounts to avoid these risks.

Health Implications of Chronic Bronchitis
1. Living with an uncomfortable shortness of breath for the rest of your life
2. Being easily winded from climbing stairs
3. Coughing and wheezing regularly
4. Suffering more frequent deep chest infections and pneumonia
5. Having to limit your recreational activities to activities such as golf, cards, and reading
6. Experiencing periods of depression
7. Being unable to do the active, physical parts of your job
8. Being limited to a restricted diet
9. Having to visit your doctor regularly and to take several medications
10. Having to have your back mildly pounded to help remove fluids built up in your lungs
11. Having to be periodically hospitalized
12. Having to quit smoking
13. Having to wear a small, portable oxygen tank

Krupnick and Cropper (1992)

Description of Chronic Bronchitis Read to Version I Respondents
CHRONIC BRONCHITIS IS A SERIOUS AND, AT TIMES PAINFUL RESPIRATORY DISEASE

HOW YOU WOULD FEEL WITH CHRONIC BRONCHITIS
- you would feel an uncomfortable shortness of breath
- you would be easily winded from minor tasks
- you would cough and wheeze regularly
- you would suffer more frequent deep chest infections and pneumonia

CHRONIC BRONCHITIS WOULD RESTRICT YOUR LIFESTYLE
- you would have CHRONIC BRONCHITIS for the rest of your life
- you would have to limit your recreational activities to activities such as golf, card, and reading
- you would experience periods of depression because of these restrictions

YOUR SALARY AND MEDICAL EXPENSES WOULD BE COVERED
- you would continue to work, but would not be able to do those parts of your job involving active physical effort
- you would be compensated by the government’s Social Security Disability Program for any lost salary and wages
you would be compensated by your own medical insurance plan and/or Medicare and Medicaid for any significant medical expenses

HOW YOU WOULD BE TREATED FOR CHRONIC BRONCHITIS

- you would be trained to breathe more effectively
- you would be limited to a restricted diet
- you would visit your doctor regularly and take several medications
- you would require mild pounding of your back to help remove fluids that would build up in your lungs
- you would be periodically hospitalized
- if you smoked, you would be urged to quit smoking
- you would eventually have to use a small, portable oxygen tank when you leave home

(The questionnaire also included 2 pictures of man with portable oxygen tank)