

ENVIRONMENT DIRECTORATE

**APPROACHES AND ISSUES IN VALUING THE COSTS OF INACTION OF AIR POLLUTION ON HUMAN HEALTH - ENVIRONMENT WORKING PAPER No. 108**

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## ABSTRACT

This paper presents a review of existing approaches to estimate the costs of inaction, as well as the benefits of policy action, for air pollution. It focuses primarily on health impacts from air pollution. The paper presents the “impact pathway approach”, which includes various steps in the analysis of the costs of air pollution. These include quantifying emissions, calculating the concentrations of the pollutants, applying epidemiologic studies to calculate the physical health effects and applying valuation methods to calculate the economic costs of the health impacts.

The report also reviews applications of the impact pathway approach to applied economic studies that aim at calculating the macroeconomic costs of air pollution. It proposes possible approaches for including the feedbacks from the health impacts of air pollution in an applied economic framework. While ideally this requires serious modifications of the modelling frameworks and an improvement of the available empirical results, some impacts, such as changes in health expenditures and labour productivity, can easily be incorporated, following the literature on the economic costs of the health impacts of climate change.

**Keywords:** water use, water scarcity, economic growth, CGE model.

**JEL classification:** C68, O44, Q15, Q25.

## RESUMÉ

Ce document présente une revue des études existantes qui estiment les coûts de l'inaction, ainsi que les effets bénéfiques de l'action politique, relatif à la pollution de l'air. Il se concentre principalement sur les impacts de la pollution atmosphérique sur la santé. Le document présente l'approche qui se base sur une "analyse de voies d'impact" et ses différentes étapes. Il s'agit notamment de quantifier les émissions, de calculer des concentrations de polluants, d'appliquer les résultats des études épidémiologiques pour calculer les effets sur la santé physique et des méthodes d'évaluation pour calculer les coûts économiques des impacts sur la santé.

Le rapport passe également en revue les applications de l'analyse de voies d'impact dans les études économiques appliquées qui calculent les coûts macroéconomiques de la pollution de l'air. Il propose des stratégies possibles pour inclure une évaluation des impacts de la pollution de l'air sur la santé dans un modèle économique appliquée. Bien qu'idéalement cela nécessite des modifications importantes des cadres de modélisation et une plus grande disponibilité des résultats empiriques, certains impacts tels que les changements de dépenses de santé et de productivité du travail peuvent facilement être incorporés en se basant sur la littérature sur les coûts économiques des impacts sur la santé liés au changement climatique.

**Mots clés :** Utilisation de l'eau, pénurie d'eau, croissance économique, modèle EGC

**Classification JEL:** C68, O44, Q15, Q25

## **FOREWORD**

This report on “Air pollution and human health: approaches and issues when valuing the costs of inaction” has been prepared as a background for modelling air pollution impacts within the OECD CIRCLE project. The report provides a background for the various steps involved in the estimation of the health impacts related to air pollution, the economic valuation of the impacts and the state-of-the art approaches for modelling air pollution impacts in applied economic models. This report is mostly methodological in character, to provide a basis for a quantitative evaluation of the costs of inaction on air pollution in the CIRCLE project.

The paper has benefitted from comments received by the technical experts of CIRCLE project and by delegates of the Working Party on Integrating Environment and Economic Policies (WPIEEP) and of the Environment Policy Committee (EPOC). Comments and suggestions from Shardul Agrawala, Nils Axel Braathen, Rob Dellink, Franco Sassi, and Simon Upton of the OECD are gratefully acknowledged.

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## 1. Introduction

With an estimated annual toll of 7 million premature deaths, air pollution can be considered as the most serious environmental health risk (WHO, 2014a). The health risks of air pollution also go beyond premature death and affect health and life quality through increased incidence of illnesses and sickness. Further, air pollution also has consequences on the environment, affecting agricultural crop yields, biodiversity, land and water, and on human activities, with impacts on visibility, buildings and materials.

This report summarises existing approaches to estimate health-related costs of inaction of air pollution and the monetary value of improved health in presence of policies targeted to reduce air pollution. The report presents the standard economic approaches used to attribute an economic value to health impacts caused by air pollution. It then examines economic models have been used to study these costs for selected countries or at global level.

Much of the existing work on the health effects of air pollution is based on the so-called “impact pathway approach”, which is currently used by government agencies, including the US Environmental Protection Agency (EPA) and the European Commission (EC).<sup>1</sup> This approach involves a sequence of steps that require quantifying emissions from a specified area, inputting them into an atmospheric dispersion model to estimate concentrations at end locations, and applying epidemiologic concentration-response functions to predict the biophysical effects of improving or worsening air quality. The impacts of air pollution on human health considered usually include both mortality and morbidity, i.e. the increased incidence of illness. The approach then assigns a monetary value to the health impacts considered using techniques such the direct valuation of the willingness-to-pay (WTP) to reduce environmental risks. The overall cost of inaction of air pollution is then calculated as sum of the monetary values for all of the endpoints over a specified area or population.

In stating their WTP individuals will consider medical expenses incurred to mitigate symptoms, averting expenses (expenses in measures that can reduce or even completely remove the negative health impacts), income lost to illness, and the value of the disutility (discomfort, pain and suffering) associated with being ill. A range of potential biases may occur in studies estimating the WTP, such as those related to altruism (e.g. in evaluating the impacts on someone else’s health) or to background risk (i.e. due to the differences in individual valuations of the starting risk of e.g. dying prematurely). A full discussion of these biases, their explanations and assessment of their importance is beyond the scope of this report. Modellers or investigators in charge of valuation and assessment projects generally apply their judgement when selecting the WTP that should be applied to a particular situation. In many cases, they also apply robustness checks to show how sensitive the results are to their assumptions.

The impact pathway approach can be incorporated in economic models such as endogenous growth, partial and general equilibrium models in order to calculate the market costs of air pollution. These models are stylised representations of one or more economies, where the main economic sectors or agents are summarised into a representative utility or production function. The models are usually based on assumptions of national, local or global market equilibria. They are also often used also to calculate emissions, which depend on the projected growth and composition of the economy. The economic feedbacks of the health impacts caused by the emissions can also be calculated within such models.

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<sup>1</sup> In the air pollution literature, this approach is also referred to as “damage function approach”. Both term refer linking emissions to impacts and economic costs via a series of sequential steps.

Including environmental impacts into a macroeconomic setting allows for an evaluation of the sectoral and economy-wide costs of inaction. Hence, a large part of this report focuses on how the insights from the literature on estimating the impacts of air pollutants on human health at the micro level can be used in the setting of applied economic models. This is done comparing three main sets of studies that integrated health impacts of air pollution in applied economic models.

The first is a series of studies on the costs of health impacts of air quality prepared using the Massachusetts Institute of Technology (MIT)'s EPPA-HE model (Matus, 2005; Matus et al., 2008; 2011; Nam et al., 2009; Selin et al., 2009). These have different geographical coverage, including China, the US and the EU and one global assessment for ozone only (Selin et al., 2009). The second is the ExternE project and its follow up projects (ExternE, 1998; Rabl et al., 2014), which address the benefits of pollution control within the EU using the European Commission's GEM-E3 model. The third includes cost-benefit analyses of air pollution policies done for the European Commission (Clean Air for Europe project, 2005; Holland, 2014) using the GAINS model operated at the International Institute for Applied Systems Analysis (IIASA). Some reference will also be made to models that consider the feedbacks on the economy of health impacts related to climate change: the OECD's ENV-Linkages model, FEEM's ICES model and the World Bank's ENVISAGE model.

The main purpose of this comparison is to obtain an overview of existing methods to study the economic feedbacks of air pollution in applied economic models. The quantitative results of the studies are not relevant for this report. A direct comparison of such results would be too complicated given the heterogeneity of the models, of the geographical coverage and, more importantly, of the different scenarios considered. Rather, the comparison is undertaken to identify available methodologies, empirical results and modelling frameworks to determine recommendations for future research.

The remainder of this report is organized as follows. Section 2 introduces the impact pathway approach and highlights the main health endpoints to be considered. Section 3 provides an overview of the functions used to calculate the impacts of air pollution on health (linking pollution, concentrations or exposure levels with the health endpoints) and of the main approaches to attribute an economic value to morbidity and mortality impacts of air pollution. Section 4 reviews the existing approaches to study the economic feedbacks of air pollution as analysed in applied economic models. Finally, Section 5 offers caveats and recommendations for future research.

## **2. From emissions of air pollutants to impacts on human health**

To estimate the health-related costs of inaction of air pollution, many rely on the impact pathway approach. The first step in this approach is to compute emissions from major anthropogenic sources alternate scenarios (usually, a baseline scenario reflecting the current conditions and one or more reduced-emission scenarios). Air pollutants usually covered by epidemiologic investigations and economic assessments include particulate matter (PM) with diameter less than 2.5 or 10 micrometres (PM<sub>2.5</sub> and PM<sub>10</sub>, respectively), sulphur dioxide (SO<sub>2</sub>), ozone (O<sub>3</sub>), nitrogen oxides (NO<sub>x</sub>), carbon monoxide (CO) and lead (Pb).<sup>2</sup> Studies of the health impacts of heavy metals, benzene and other chlorinated solvents, dioxins, and other toxic substances are also available. Recent research has been conducted on specific components of particulate matter and other by-products of fossil fuel combustion (e.g., black carbon).

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<sup>2</sup> Volatile organic compounds (VOCs) and nitrogen oxides (NO<sub>x</sub>) are ozone precursors. At relatively high temperatures and under certain atmospheric conditions, they combine to form O<sub>3</sub>. VOCs are emitted by a variety of sources, including cars and certain manufacturing plants, and NO<sub>x</sub> are the by-products of fossil fuel combustion. However, ambient concentrations of VOCs are not routinely measured at air pollution monitors. Pollution monitors often gather observations on NO<sub>2</sub>. Ammonia (NH<sub>3</sub>) is not always considered a "criteria" or a toxic air pollutant, but it is a precursor to particulate matter, and as such often regulated.

Once emissions have been estimated, it is necessary to calculate concentrations of pollutants, as excessive concentrations are the main cause of impacts on human health. The concentrations are calculated using atmospheric concentration models. These models consider the emissions as well as geographic and climatic characteristics, as these influence the concentration levels. When calculating projections in the future, they also take into account climatic projections as changes in temperatures and precipitations can affect concentrations.

The concentrations are then used to assess the biophysical health effects of air pollution. This is no simple matter, due to the difficulties in obtaining reliable data and addressing estimation issues. The impacts of air pollution on health are usually assessed with dose-response, concentration-response or exposure-response functions. These functions link air pollution levels with health endpoints, after adjustments for plausible exposure and breathing rates (which depend on time spent outdoors, age and intensity of physical activity). Since it is difficult to measure actual exposure correctly, many integrated assessment models and epidemiologic studies rely on concentration-response functions, which link health endpoints directly with concentrations. Concentration-response functions are typically estimated by gathering data on the occurrence of the health endpoints of interests, and running regressions that relate them to air pollution, controlling for factors such as temperature, relative humidity, wind speed or season.

The EPPA-MIT model, IIASA's GAINS model and the ExternE project rely on a relatively narrow set of concentration-response functions that are however well established in the literature and widely used in policy assessments (see Rabl et al., 2014). The choice of the concentration-response functions influences the results of such studies. A higher geographical coverage of the studies will also help applications of the results in large-scale analysis as often these studies are specific to certain geographical areas or types of population (see Section 3.4). Further studies to fill the gaps in the literature would be useful as well as sensitivity analysis on the choice of the concentration-response functions used.

The health endpoints that are most commonly associated with air pollution are "acute" mortality (namely, deaths linked with spikes in air pollution), "chronic" mortality (either expressed as premature deaths or the loss of life expectancy due to long-term exposure to air pollution), chronic bronchitis, hospitalisations for respiratory and cardiovascular illnesses, infant mortality, and other effects on infants. In addition, analysts have also examined the limitations that illness cause on daily activities. Examples include the so-called "restricted activity days" and "work loss days".

Table 1 lists the health endpoints considered in the studies done with MIT's EPPA model, in the assessments done with IIASA's GAINS model, and those covered in the ExternE assessments. While this is an extensive list, it is not fully comprehensive. The studies do not consider the possibility that air pollution may cause subclinical symptoms that do not result in absences from work, but nevertheless decrease the productivity of labour. Further, they do not include the potential effects on new-borns and children that result in impaired neurodevelopmental and cognitive effects, which in turn affect the accumulation of human capital and result in lower earnings (as well as additional expenditures to remedy school absences and lower educational attainment). These effects are linked with lead, mercury and other heavy metals. Recent literature is also exploring new health endpoints which are not included in current studies, such as the effect of air pollution on pregnancy outcomes.

The key step in assessing the health impacts of air pollution is the estimation of the concentration-response function. Different types of studies can be conducted to do estimate this type of function. These include time series analysis, cross-sectional studies, prospective cohort studies, and clinical experiments, among others. Time series analyses have been conducted, for example, to study how daily acute mortality figures, hospital admissions, emergency room visits or doctor visits for specific illnesses are affected by daily fluctuations in pollution levels. These studies generally examine same-day relationships between pollution and the health endpoints controlling for other variables that affect mortality and morbidity.

**Table 1. Health endpoints examined in three key models/assessments**

	MIT	IIASA	ExternE
Mortality from chronic exposure (life years lost)		√	√
Mortality from chronic exposure (premature deaths)		√	
Acute mortality	√	√	√
Infant mortality		√	√
Respiratory hospital admissions	√	√	√
Cerebrovascular/cardiovascular hospital admissions	√	√	√
Chronic bronchitis adults	√	√	√
Chronic bronchitis children	√	√	
Chronic cough children	√		
Congestive heart failure	√		
Asthma attacks	√		√
Asthma symptoms, asthmatic children		√	
Cough	√		√
Lower respiratory symptoms (wheeze)	√		√
Ischaemic heart disease	√		
Medications/bronchodilator use			√
Work loss days		√	√
Restricted Activity Day	√	√	√
Minor Restricted Activity Day	√	√	√
Symptoms Days	√	√	√

Source: authors' comparison of Matus (2005), Matus et al. (2005, 2008, 2011), Nam et al. (2009), Selin et al. (2009); IIASA (2014); Rabl et al. (2014); ExternE (1996, 1998, 2000).

Daily mortality is of course an outcome of great interest, and one that has been studied in different world regions (mostly in the US, Canada and Europe). Table 2 summarises results on the factors influencing acute mortality due to PM<sub>10</sub> in Europe (Rabl et al., 2014). In empirical studies, pollution levels of previous days are sometimes considered to explain daily mortality. Today's pollution levels increase today's premature deaths while yesterday's pollution levels decrease them. The impact of pollution levels of even earlier days is instead very weak. This pattern suggests that there exists a pool of particularly susceptible persons. As underlined in Spix (1994), once that pool is wiped out by a pollution spike, the next day's mortality is necessarily lower.

Cross-sectional studies identify the effect of air pollution by gathering data in places with different pollution levels. In longitudinal studies it is possible to control for unobserved heterogeneity in mortality and illness rates. The National Health Interview Survey, for example, was used in Ostro and Rothschild (1989) to study the association between air pollution and minor restricted activity days in the US.

Prospective cohort studies follow individuals of different ages, occupations, baseline health status, etc. in different places for many years. They aim at estimating survival models where the duration of life is linked with demographics, individual characteristics and pollution levels. These studies have been used to estimate the relative risks of dying prematurely as well as changes in life expectancy due to different pollution levels (see Rabl et al., 2014). The data used in prospective cohort studies include birth and death certificates issued by local or national government bodies, health self-reports collected through surveys, hospital admissions and hospital discharge figures.

While most research consists of epidemiological studies of an observational nature, clinical experiments exist where volunteers are asked to breathe low doses of air pollution and various outcomes are measured. However, these studies suffer from a number of limitations. The concentration-response functions are often estimated at relatively low pollution levels. Hence, caution should be used when extrapolating results to higher pollution levels. Further, most of these studies do not take into consideration

behavioural changes in response to high air pollution levels that may reduce exposure (e.g. limiting certain outdoor activities). Pollution warnings and forecast programmes exist in many places, yet studies that explicitly focus on responses to such warnings, such as Neidell (2009), are rare.<sup>3</sup>

**Table 2. Variation of acute mortality due to PM<sub>10</sub> in Europe**

Summary of effects		
	Mortality (% increase per 10 µg/m <sup>3</sup> )	
		95% confidence interval
Average, Europe	0.60%	0.40-0.80%
City with low average NO <sub>2</sub>	0.19%	0.00-0.41%
City with high average NO <sub>2</sub>	0.80%	0.67-0.93%
Cold climate	0.29%	0.16-0.42%
Warm climate	0.82%	0.69-0.96%
City with low standardised mortality rate	0.80%	0.65-0.95%
City with high standardised mortality rate	0.43%	0.24-0.62%

Source: Rabl et al. (2014), based on Katsouyanni et al. (2001)

There are a number of difficulties in estimating concentration-response functions. First, due to data limitations, the majority of the studies impute the pollution concentrations measured at existing monitors to larger areas with different pollution levels. Pollution levels also vary during the day depending on commuting time. Second, there is a high correlation between different air pollutants, which makes it difficult to disentangle the contribution of each pollutant to the health endpoints. Finally, there are issues with the selection of residents used for the studies. If people with low socio-economic status live in highly polluted areas, the slope of the concentration-response functions may appear steeper if these persons also in poor health. Conversely, if people who suffer from asthma or are sensitive to pollution relocate to less heavily polluted areas, the concentration-response functions estimated from the more heavily polluted areas will be flatter, because the people that remain are less sensitive to pollution.

A number of approaches are possible to circumvent these problems. One is to seek a shock in pollution levels, due for example to plant closures or drops in economic activities, which is completely uncorrelated with existing conditions and population characteristics. This method was used in Dockery and Pope (1993) and Chay and Greenstone (2003) to examine infant mortality outcomes after a decline in pollution due to an economic recession. It was also used by Lavaine and Neidell (2014) to study birth weight changes in France as consequences of a strike that paralysed refineries and resulted in a dramatic drop in SO<sub>2</sub> emissions. Even in a country with universal health care where prenatal care is free, and birth weights are generally in the healthy range, the reduction in SO<sub>2</sub> level does lead to a significant improvement in the average birth weight.

The next possibility is simply to improve the accuracy with which ambient concentrations are imputed in the places where each affected persons are. Currie et al. (2009), for example, examine low birth weight outcomes having the exact address of the mothers, which allows them to improve the quality of the match with the nearest air quality monitors. Lleras-Muney (2010) instead uses data from military bases that are within a specified distance of a monitor. The author uses data from the medical records of military families over 1989-1995 to examine how child hospitalisations for respiratory illnesses are affected by pollution

<sup>3</sup> The Neidell study finds evidence consistent with the notion that people adjust their behaviours in response to the warnings, and uses attendance figures to evening- and night-time events (e.g., visits to the Griffith observatory or major-league baseball games) for comparison purposes. The study is based on the Los Angeles area. In some cities on the East Coast of the United States (e.g., Washington, DC), where the main air pollution problem is ozone in the summertime, health warnings are often issued on high-pollution days that also happen to be extremely hot and humid, so that it would be unpleasant for most people to spend time outdoors anyway. There are no studies that seek to identify the additional effect of the warning above and beyond the normal behaviour in response to high temperatures and humidity levels.

levels.<sup>4</sup> Using this type of data helps avoid the problem of selection and sorting by focusing on a sample of persons who have little choice over the place where they live. However, studies on such specific population types cannot easily be applied to other populations or regional areas. For example, access to health services in US military bases is excellent and thus not representative of larger populations.

Moretti and Neidell (2011) resort to instrumental variable techniques to address the issue of measurement error associated with exposure. They focus on pollution in Los Angeles, and their instrument is ship traffic to the Port of Los Angeles, which creates emissions causing in turn increased ambient pollution concentrations. They show that the slope of the concentration-response function is three times as steep when the instrumental variable approach is used, compared to regular ordinary least squares (OLS).<sup>5</sup>

Finally, Cameron and De Shazo (2009) deploy choice experiments where respondents were asked to choose between risk-reducing alternatives defined by a number of attributes. One of them was a reduction in the risk of dying. Another was the age at which the respondent would die. But telling someone that he or she will die at a specific age (a deterministic, no-uncertainty concept) is incompatible with the notion that there is a risk of dying at a given age and hence uncertainty as to what a person's lifetime will be. This construct is also less useful in calculating the costs of inaction.

As illustrated by comparing this sub-set of studies, the health effects of pollution will be grossly underestimated unless specific steps are taken to correct for the measurement error that results from using ambient concentration without incorporating behavioural responses that allow individuals to limit exposure to air pollution. Future research in this field should seek further data and techniques to overcome the estimation and measurement issues. One should also be careful with the spatial scale of the study or application, as local results are not easily generalised to a larger geographical scope (see Section 3.4 on benefit transfer).

### 3. From health impacts to monetary values

Different studies not only rely on different concentration-response functions, but also on different methods to evaluate the economic costs related to premature deaths, illness and other morbidity impacts. A summary of the monetised values used by the EPPA-MIT model, IIASA's GAINS model and the ExternE project is presented in Table 3. The differences in estimates across the studies depend on the regional areas covered, but also on the methodologies used.

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<sup>4</sup> While she puts a lot of efforts into avoiding selection and sorting issues, her data imply other difficulties. Most military bases are away from cities, and as such they have relatively low pollution levels and typically lack pollution monitoring stations.

<sup>5</sup> Mismeasured pollution levels create correlation between pollution and the error term in the regression model that relates health outcomes to pollution levels. Under these circumstances, OLS produce biased estimates of the slope of the concentration-response functions. This problem is addressed using instrumental variable estimation. The method of two-stage least squares (2SLS) is one such technique. Briefly, one looks for "instruments" for ambient concentrations of pollution – namely variables that are strongly correlated with them, but which do not cause health outcomes directly and are thus independent of the econometric error term in the concentration-response function. In the Moretti and Neidell paper, these instruments are ship traffic (expressed in tons per day) and its interaction with the distance from various neighbourhoods in the Los Angeles area. In the so-called "first stage", one regresses ambient concentration at a particular monitor on weather and time controls, plus the instruments. A prediction is made for ambient concentration levels based on these regressors and the regression coefficients from this "first stage." In the second stage, one fits the concentration-response function, but actual ambient concentrations are replaced with the *predicted* levels from the first stage.

Table 3. Summary of unit values for health endpoints from selected models and projects

Health Endpoint	MIT			IIASA			ExternE		
	Cost (USD <sub>2000</sub> )	Cost (€ <sub>2014</sub> )	Pollutants	Cost (€ <sub>2005</sub> )	Cost (€ <sub>2014</sub> )	Pollutants	Unit Cost (€ <sub>2000</sub> )	Unit Cost (€ <sub>2014</sub> )	Pollutants
Restricted Activity Day	106	118	PM <sub>10</sub> , Nitrates	92	111	PM <sub>2.5</sub>	130	175	PM <sub>2.5</sub>
Respiratory Hospital Admissions	11 115	12 363	PM <sub>10</sub> , Nitrates, SO <sub>2</sub> , O <sub>3</sub>	2 220	2 688	all	2 000	2 687	PM <sub>10</sub> , O <sub>3</sub>
Cerebrovascular/Cardiovascular Hospital Admissions	11 115	12 363	PM <sub>10</sub> , Nitrates	2 220	2 688	O <sub>3</sub> , PM <sub>2.5</sub>	2 000	2 687	PM <sub>10</sub>
Symptoms Days	11	12	O <sub>3</sub>				38	51	
Chronic Bronchitis Adults (per case)	148 296	164 944	PM <sub>10</sub> , Nitrates	53 600	64 894	PM <sub>2.5</sub>	200 000	268 740	PM <sub>10</sub>
Chronic Bronchitis Children (per case)	318	354	PM <sub>10</sub> , Nitrates	588	712	PM <sub>2.5</sub> /NO <sub>2</sub>			
Chronic Cough Children	318	354	PM <sub>10</sub> , Nitrates						
Congestive Heart Failure	11 115	12 363	CO, Nitrates, PM <sub>2.5</sub>						
Asthma attacks	52	58	O <sub>3</sub>	42	50.9	PM <sub>2.5</sub>	139	187	
Cough	318	354	PM <sub>10</sub> , Nitrates				38	51	O <sub>3</sub>
Lower Respiratory Symptoms (wheeze)	11	12	PM <sub>10</sub> , Nitrates				38	51	PM <sub>10</sub>
Ischaemic Heart Disease	11 115	12 363	PM <sub>10</sub> , Nitrates, CO						
Minor Restricted Activity Day	11	12	PM <sub>10</sub> , O <sub>3</sub>	44	53.3	O <sub>3</sub>	38	51	PM <sub>2.5</sub>
Emergency Room Visit	315	350					670	900	
Acute Mortality	30 225	33 618	PM <sub>10</sub> , Nitrates, SO <sub>2</sub> , O <sub>3</sub>	57 700 / 138 700	69 858 / 167 924	all	60 000	80 622	O <sub>3</sub> /SOMO <sub>35</sub>
Infant Mortality				1.6 - 3.3 mln	1.9-4 mln	PM <sub>2.5</sub>	3 mln	4 mln	PM <sub>10</sub>
Work loss days				130	157.4	PM <sub>2.5</sub>	295	396	PM <sub>2.5</sub>
Medications/bronchodilator use							1	1	PM <sub>10</sub>
Mortality from chronic exposure (life years lost)				57 700 / 133 000	69 857 / 161 023	all	40 000	53 748	PM <sub>2.5</sub>
Mortality from chronic exposure (premature deaths)				1.1 - 2.2 mln	1.3 - 2.7 mln	all			

Source: Comparison of Matus (2005), Matus et al. (2005, 2008, 2011), Nam et al. (2009), Selin et al. (2009); IIASA (2014); Rabl et al. (2014); ExternE (1996, 1998, 2000).

### 3.1 Metrics for valuing mortality impacts

#### *The Value of a Statistical Life*

One of the most widespread methods to evaluate the economic costs of premature deaths is the use of the Value of a Statistical Life (VSL).<sup>6</sup> The VSL is the willingness-to-pay (WTP) for a marginal reduction in the risk of dying, and is therefore defined as the rate at which the people are prepared to trade off income for a reduction in the risk of dying prematurely ( $R$ ):

$$(1) \quad VSL = \frac{\partial WTP}{\partial R} .$$

The VSL can be derived from a simple static, one-period framework, where individuals maximise the expected utility of consumption:

$$(2) \quad E(U) = (1 - R) \cdot U(y) + R \cdot V(y)$$

where,  $U(\ )$  and  $V(\ )$  denote the state-dependent utilities of consumption when alive and dead, respectively. It is straightforward to show that the VSL can be calculated as the ratio between the utility differential between the two states, and the expected marginal utility of income, given the risk of dying prematurely:

$$(3) \quad VSL = \frac{U(y) - V(y)}{(1 - R) \cdot U'(y) + R \cdot V'(y)} ,$$

As the utility of income can be safely assumed to be zero when dead, the expression for the VSL can be simplified to the ratio between and the change in utility for a given risk of dying prematurely:

$$(4) \quad VSL = \frac{U(y)}{(1 - R) \cdot U'(y)} .$$

In policy analyses, the VSL is regarded as a summary measure of the WTP for a mortality risk reduction, and a key input into the calculation of the benefits of policies that save lives, or the cost of inaction. For mortality, the costs of inaction are computed as the VSL multiplied by the expected number of lives lost attributed to air pollution.

The concept of VSL is generally deemed as the appropriate construct for policy analyses, when the identities of the people whose lives are saved by the policy are not known yet. As shown in the above mentioned example, in practice VSL is computed by first estimating WTP for a specified risk reduction  $\Delta R$ , and then by dividing WTP by  $\Delta R$ .

Several methodologies can be used to estimate the VSL. The “traditional” approach argues that the value of a life is the present value of the stream of income generated by a person over his or her remaining lifetime.<sup>7</sup> This approach has been criticised because it places a very low value or no value at all, on

<sup>6</sup> Definitions and some of the materials covered in this section can also be found in OECD (2012).

<sup>7</sup> This is the approach used in cost of illness studies.

individuals that are not gainfully employed, such as homemakers or retired persons, even if these people *are* willing to pay for a reduction in their own risk of dying.

The most widespread method to estimate VSL with revealed preferences analysis is through compensating wage differentials in labour markets. The rationale of compensating wage studies is that workers must be offered higher wages for them to accept jobs with greater risks of dying, and that employers are willing to do so to the extent that this is cheaper than installing safety equipment in the workplace. In a typical compensating wage study, the estimations relate the wage rate to the risk of premature death, while controlling for education and experience of the worker, as well as other job and worker characteristics that influence wages. Viscusi (1993) argues that the correct specification of the wage regression is:

$$(5) \quad w_i = \beta_0 + \mathbf{x}_i\boldsymbol{\beta}_1 + p_i\beta_2 + q_i\beta_3 + (q_i \times WC_i)\beta_4 + \varepsilon_i$$

where  $w$  is worker  $i$ 's wage rate (or its log transformation), and  $\mathbf{x}$  is a vector of individual, workplace and occupational characteristics, such as experience, education, age, gender, marital status, union status of the worker, industry dummies, occupation dummies, and geographical dummies. The variable  $p$  measures the risk of dying on the job, while  $q$  is the risk of non-fatal injuries. The  $\beta$ s are unknown coefficients to be estimated, and the VSL can be inferred from  $\beta_1$ . Viscusi (1993) recommends that equation (5) should include expected worker compensation in the event of a non-fatal workplace accident, i.e.,  $(WC \times q)$ , where  $WC$  is the level of workers compensation paid out to the worker if he experiences an accident at work.

Based on their survey of the US literature, Viscusi and Aldy (2003) recommend VSL figures of USD 5-9 million (2000 dollars). Estimates of the VSL based on compensating wage studies are available for several European countries, including the UK, where they usually range between USD 4 and USD 11 million,<sup>8</sup> and Switzerland (USD 6.5 – USD 9.5 million 2000 dollars; Baranzini and Ferro Luzzi, 2001).<sup>9</sup>

There are some shortcomings to this approach. Leigh (1995), Arabsheibani and Marin (2000, 2001), Black and Kniesner (2003), and Black et al. (2003) also suggest that estimates of the VSL from compensating wage studies are econometrically very fragile, for reasons that include poorly measured workplace risk, collinearity of risk estimates with industry dummies used to account for inter-industry wage differentials, endogeneity (an individual's level of risk may be determined simultaneously with his job, and hence with the wage), omitted regressors, and heterogeneous preferences for risk and income, and econometric identification issues. For example, Leigh (1995) shows that if the regression equation attempts to control for inter-industry wage differentials, the coefficient on fatal risks becomes insignificant, rendering the researcher unable to estimate the VSL; omitting the non-fatal risks instead results in inflated coefficients on the fatal risks, and hence higher VSL figures.

<sup>8</sup> In some cases, studies based on UK data have found the VSL to be much larger than the upper bound of this range. For example, Siebert and Wei (1994), Sandy and Elliott (1996), Arabsheibani and Marin (2000) and Sandy (2001) peg the VSL in the range between EUR 4.3 million and EUR 74.4 million (equal to USD 4.0 million to USD 68.5 million at the 2000 exchange rate). A meta-analysis by CSERGE (1999) generates a range of VSL figures between EUR 2.9 million and EUR 100 million, resulting in weighted average equal to EUR 6.5 million (all 2000 €, the corresponding dollar amounts being USD 2.7, USD 92.1 and USD 6.0 million).

<sup>9</sup> Using the 1995 Swiss Labour Force Survey (SLFS) and the 1994 Swiss Wage Structure Survey (SWSS), Baranzini and Ferro Luzzi peg the VSL implicit in the choices of Swiss workers in the range of CHF 10 to CHF 15 million (Swiss Francs, equivalent to USD 6.5 to USD 9.5 million 2000 US dollars). They find that the VSL depends on risk level, union coverage, and age.

Unlike earlier studies, which imputed risks using the average fatality rate in the worker's occupation or industry, Viscusi (2004) uses recently released occupational safety data and constructs fatality rates for industry-occupation cells, which should reduce measurement errors. Kniesner et al. (2006) offer a nice discussion of the many econometric difficulties involved in wage-risk studies, and propose an approach to filter out some of the measurement errors affecting workplace risks. Hintermann et al. (2010) use a panel dataset documenting wages and work conditions in the UK labour market in recent years, and find no evidence of wage compensation when heterogeneity of worker preferences and endogeneity of workplace risks are accounted for in the econometric analysis.

Clearly, when using the VSL estimated from labour market studies, one is implicitly assuming that the trade-offs between risk and income observed in labour markets can be applied in other contexts, such as environmental policies. However, there is no particular reason to believe that the VSL observed in labour markets should be the VSL used to estimate the mortality benefits of air pollution or other environmental health policies (or the costs of inaction). Different VSLs may be justified on the grounds that the beneficiaries of environmental policies (for example, the elderly and persons with chronic cardiovascular and respiratory illnesses) are different from the workers examined by most compensating wage studies. The nature of the risks is also different: workplace risks are voluntary, while the risks associated with environmental stresses are often involuntary, which could lead to different WTP, even if the magnitude of the risk reductions were the same.

Despite concerns about the econometric identification of compensating wage differentials and the appropriateness of transferring the VSL from labour markets to other safety and environmental policy context, U.S. government agencies do rely on VSL figures from wage-risk studies. For example, in its 2000 Guidelines for Economic Analyses, the US EPA relied on 21 compensating wage studies, out of a total of 26 studies, to produce a VSL figure (USD 6.1 million 2000 dollars). This figure was thus based on evidence from labour markets, but was used in subsequent environmental policy analyses.

An alternative revealed preferences approach to estimating the VSL is to relate the price of a product to the product's attributes, including its safety. Atkinson and Halvorsen (1990) analyse the price of cars as dependent on car attributes, such as the car size, fuel efficiency, luxury index, and the risk of dying in an accident when driving a specific to find the implicit marginal price of risk. Gayer et al. (2000, 2002) observe the prices of homes near Superfund sites (sites contaminated by uncontrolled hazardous waste where remedial investigation and clean-up activities are performed by or under order of the US EPA), and combine them with cancer risks predicted using pollutant concentrations, dilution and groundwater models, and resident exposure assumptions. Using regression analyses and assumptions about people's beliefs about risks, they disentangle the Value of a Statistical Case of Cancer (VSCC) implicit in the value of homes at different distances from the contaminated site. If the VSCC is divided by the probability of dying of cancer, conditional on contracting cancer in the first place, one obtains a VSL that is specific to the contaminated site policy context and the cancer cause of death.

Another alternative is the use of averting behaviour studies, which examine the time or money spent by an individual on averting behaviours or expenditures (i.e. activities that increase safety and reduce the risk of death). For example, Blomquist (1979) infers VSL values using data on the behaviour of drivers. Jenkins et al. (2001) examine the use of helmets when riding a bicycle. A meta-analysis by Ashenfelter and Greenstone (2004) instead infer the VSL from U.S. States' decisions about speed limits. Based on the fact that in 1987 the U.S. Federal Government allowed the States to raise the speed limit on their rural interstate roads, the authors argue that the States that adopted higher speed limit must have valued saved travel hours more than possible fatalities. A review of averting behaviour studies, suggests that the VSLs from these studies are typically smaller than those produced with other approaches (Blomquist, 2004).

Finally, it is possible to estimate the VSL using stated preference studies, which rely on what people *say* that they would do under well-specified hypothetical circumstances. Stated preference studies do not involve actual money transactions, and they are useful as an alternative (or a supplement to) to revealed preference studies when (i) it is not possible to implement the latter, (ii) there is no guarantee that people's perception of risks matches objective risks, and (iii) there is little or no variation in the risk to be valued.

Stated preference methods have indeed been used to place a value on mortality risk reductions and thus to infer the VSL. Researchers have generally used one of four main research methods: (i) contingent valuation, (ii) conjoint choice experiments, (iii) the chained approach first illustrated in Carthy et al. (1999), and (iv) risk-risk questions, paired with a valuation question (Viscusi et al., 1991). OECD (2012) presents a meta-analysis of VSL estimates obtained in various countries using stated preference methods to obtain a reference VSL value for OECD countries. Stated preference studies generally produce lower VSL figures than those obtained with labour market studies.

### *The Value of a Statistical Life Year*

Prospective cohort studies can be used to estimate the change in life expectancy associated with pollution exposure. The natural valuation metric when assessing the costs of inaction would therefore seem to be the willingness-to-pay for an extension in remaining life expectancy. When that life expectancy extension is measured in years, the metric is termed Value of a Statistical Life Year (VOLY).

Three things should be noted. First, as shown in equation (6), reducing the risk of dying at any age increases remaining life expectancy. However, the same increase in life expectancy can be obtained through a number of different reductions in the risk of dying at different ages, and if people have preferences for *when* the risk reductions take place, then there is no unique WTP for a specified extension in life expectancy. Moreover, Alberini and Scasny (2011) show that when people are reminded of the remaining life expectancy changes associated with a specified mortality risk reduction, their WTP for the mortality risk reduction is much lower than if the life expectancy information had been omitted.

Second, there are only very few studies that have specifically inquired about individual preferences for remaining life expectancy and the willingness-to-pay for an improvement in it. Johannesson et al. (1996), Morris and Hammit (2000), Chilton et al. (2004), and Desaigues et al. (2011) are exceptions, and critics have raised concerns about the credibility of some of these studies.<sup>10</sup>

<sup>10</sup>

Johannesson et al. (1996) and Morris and Hammit (2000) both value an extension to expected remaining lifetime that would be experienced at a future age (60, 70 or 75 years) and be paid for only once (this year), and examine risk reductions or remaining lifetime extensions that are four to ten times larger than those typically associated with air pollution exposures. For example, the respondents in the Morris and Hammit study were asked to consider a vaccine against pneumonia that would be administered to the respondent and paid for only once (this year), and would result in an extension in remaining lifetime of 11 (at age 60) or 5 months (at age 70). The corresponding risk reductions would be 0.2% – or 2 in 1000 – per year. Johannesson et al. report that the mean WTP for a one-year increase in remaining lifetime at age 75 is between EUR 400 and EUR 1500, depending on the statistical modelling of the responses, and that the corresponding VSL is in the range of USD 30 000 to USD 110 000. The Johannesson et al. results show an increasing WTP with age – though criticism has been levelled at this study on the basis of its elicitation method and small sample size. Morris and Hammit's estimates of median WTP imply that an extension of one year in remaining lifetime is worth between USD 698 and USD 492, and that VSL is USD 140 000 – USD 148 000. The Desaigues et al. study was conducted in 9 countries for a total of only 1463 respondents.

Third, the notion of VOLY is used in policy analyses in addition to or instead of that of VSL, but, depending on the age of the people whose lives are saved by the policy, can offer recommendations in conflict with those obtained by using VSL. Consider for example two alternative public programmes, and suppose that both save 100 lives. But suppose that with one, the lives saved are those of young adults, whereas the other saves the lives of the elderly. Then, as long as the VOLY is constant with respect to age, the policy that saves young adults, who have a longer life expectancy, would be concluded to offer greater benefits if the VOLY is used. By contrast, if the VSL is used, and a single figure is applied to people of all ages, the two policies would be concluded to provide the same benefits.

In the absence of direct estimates of the WTP for a life expectancy extension, researchers have derived the VOLY from estimates of the VSL. Specifically, assuming that a VOLY is constant over the rest of one's remaining lifetime, and letting  $T$  be the number of expected remaining life years, the VOLY and the VSL are related as follows:

$$(6) \quad VSL = \sum_{t=1}^T VOLY \cdot (1 + \delta)^{-t},$$

where  $\delta$  is an appropriate discount rate. In the ExternE project (1999), the VOLY in equation (6)<sup>11</sup> is further weighted by the conditional probability  $P$  of surviving an additional year, conditional to having survived until the present age  $a$ :

$$(7) \quad VSL = VOLY_r \cdot \sum_{i=a+1}^T P_i (1 + \delta)^{i-a-1}.$$

In ExternE (1999), the central estimate of VOLY for acute effects was EUR 0.12 million, whereas for chronic effect it was EUR 0.084 million, using a discount rate of 3%, derived from a VSL of EUR 3.14 million. Figures used in the Clean Air for Europe cost-benefit analysis are displayed in Table 4.

**Table 4. Values for use in the Clean Air Policy Package**

(Euros 2008)

	VSL	VOLY
Median	€1.09 million	€57 700
Mean	€2.22 million	€138 700

Source: Holland (2014).

### 3.2 A simple model for valuing morbidity impacts

This section presents a simple model for valuing morbidity linked with high air pollution exposure. The model is suited for minor as well as acute illnesses, and is an application of a household production function framework.<sup>12</sup> The model is based on a utility function, in which utility ( $U$ ) depends on aggregate consumption ( $X$ ), leisure ( $L$ ) and health, represented by the number of sick days ( $D$ ).

<sup>11</sup> In the original paper the formula says VOSL.

<sup>12</sup> In the household production function approach, an individual household derives utility from combining private and public goods (e.g., air quality, transportation infrastructure). Some of these private or public goods do not provide utility *per se*, but only via other goods or services used by the household. In the model described in this section, it is assumed that the household does not experience utility or disutility from pollution *per se*, but does experience disutility from the sick days that pollution causes. Another example is in the context of energy usage. Households do not demand electricity *per se*, but because they combine it with their capital stock (e.g., appliances and lighting fixtures in the home) to produce energy services (lighting, a warm home, cooked meals, fully charged cell phones, etc.).

$$(8) \quad U = U(X, L, D; \mathbf{Z}_U).$$

It can be safely assumed that utility increases with leisure and health but decreases with sick days. Utility also depends on each individual's characteristics ( $Z_U$ ), which represent preferences for income, leisure, and health. In the utility function (8), pollution does not influence utility directly, but has an indirect effect by triggering illness and causing sick days. The dose-response function summarises the relationship between pollution and health outcomes:

$$(9) \quad D = D(P, A; \mathbf{Z}_D).$$

Equation (9) shows that sick days depend on pollution ( $P$ ) and averting activities ( $A$ ), undertaken by the individual to reduce exposure to pollution.  $Z_D$  is a vector of individual characteristics, allowing for individual predisposing factors and baseline health. The individual chooses the levels of  $L$ ,  $X$ , and  $A$  to maximize utility subject to a budget constraint:

$$(10) \quad y + w \cdot [T - L - W(D(P, A))] = X + p_M \cdot M(D(P, A)) + p_A \cdot A.$$

Equation (10) posits that the individual must allocate his time between work and leisure and spend income on aggregate consumption, medical care  $M$ , which in turn depends in the number of sick days, and on the averting activity  $A$ . The prices per unit of  $M$  and  $A$  are equal to  $p_M$  and  $p_A$ , respectively. Work time lost to illness ( $W$ ), which can be expressed for example as the number of work days lost, is in the budget constraint because it reduces the work time available to the individual.

Based on this model, it is possible to evaluate the individual's WTP to reduce pollution. Higher pollution levels make individuals worse off because they increase the number of sick days. Thus, all else equal, individuals experience higher utility levels when pollution is lower. The WTP is defined as the maximum amount of money that can be subtracted from the individual's income at the new, lower level of pollution to keep him or her at the same utility level as with the initial levels of pollution and income. Formally, WTP satisfies:

$$(11) \quad V(y - WTP, w, p_M, p_A, P_1) = V(y, w, p_M, p_A, P_0)$$

where  $V(\cdot)$  is the indirect utility function and  $P_0$  and  $P_1$  are the initial and final levels of pollution with  $P_0 > P_1$  so that environmental quality improves. Harrington and Portney (1987) show that WTP for a small change in pollution can be decomposed into:

$$(12) \quad WTP = w \frac{dW}{dP} + p_M \frac{dM}{dP} + p_A \frac{dA^*}{dP} - \frac{U_D}{\lambda} \cdot \frac{dD}{dP}$$

where  $A^*$  is the demand function for  $A$ , and  $dA^*/dP$  gives the optimal adjustment of  $A$  to a change in pollution;  $\lambda$  reflects the marginal utility of income. Equation (12) indicates that the WTP for a small reduction in pollution includes four components: (i) marginal lost earnings, (ii) marginal medical expenditures, (iii) the marginal cost of the averting activity, and (iv) the disutility of illness, which reflects all negative impacts caused by the illness, such as pain and suffering, but also possible disruption of normal daily life. This last element if converted into dollars through dividing by the marginal utility of income.

Equation (12) can be rearranged as follows:

$$(13) \quad WTP = \frac{dD}{dP} \left[ w \cdot \frac{dW}{dD} + p_M \frac{dM}{dD} + p_A \frac{dA^*}{dD} - \frac{U_D}{\lambda} \right],$$

showing that the WTP can be equivalently expressed as depending from the dose-response function ( $dD/dP$ ), which links a marginal change in pollution ( $dP$ ) to marginal changes in the diseases ( $dD$ ), and to the marginal values of the illness, which includes the sum of marginal lost earnings, medical expenditures and averting activities, minus the monetary value associated with the disutility of illness. These are same four main components as in Equation (12).

Equation (13) has important implications for assessing the cost of inaction with respect to air pollution. It provides support for the damage function approach, namely for assessing dose- or concentration-response functions and for combining them with WTP to avoid illness. It also supplies a framework to estimate WTP with the cost of illness approach, which only considers the first two components in the equation (lost earnings and medical expenditures). This approach is used extensively in policy analyses and in various assessment efforts. The US EPA, for example, has issued a handbook with cost-of-illness figures for a variety of health endpoint linked with pollution (US EPA, 2007). The ExternE project and its sequels have deployed the cost-of-illness to monetise a variety of health endpoints, including chronic bronchitis.

Cost-of-illness calculations are relatively easy to do. They have been compiled by government agencies for a variety of illnesses, and do not require conducting surveys of individuals. However, they only provide a lower bound for the true WTP to avoid the illness, and as such they understate the true costs of inaction. Chestnut et al. (1996), Dickie and Gerking (1996), and Alberini and Krupnick (2002) compared full WTP for angina pectoris, doctor visits, and minor respiratory illnesses, respectively, with the cost of those illnesses as reported by survey respondents in the US and Taiwan, and concluded that the full WTP is two to four times larger than the cost of illness alone.

Bartik (1988) focuses on the link between illness and averting activity, namely  $p_A \frac{dA^*}{dD}$  in Equation (13). However, their approach is based on stringent assumptions about households. Averting costs calculations are conducted by tallying the expenditures associated for example with purchasing and running air filters or purchasing bottled water when the domestic water supply is unsafe. These simple calculations – much like the cost-of-illness approach – underestimate the true WTP to avoid the illness.

The only way of fully capturing non-market values is to conduct a stated preference study, i.e., surveys where individuals are asked to report their WTP to reduce pollution or avoid the illness associated with pollution exposures. This can be done with methods such as contingent valuation and discrete choice experiments.

The model described by equations (8)-(10) contains a number of important assumptions, including the assumption that the individual does not derive disutility directly from pollution, but only indirectly because pollution causes sick days. An implication of this assumption is the decomposition of total WTP into the components shown in equations (12) and (13), and the fact that the value of illness does not change with the nature of the pollutant. The individual is also assumed to have flexible work hours, and while pollution can force the individual to adjust his labour supply, it does not otherwise influence productivity and the wage rate. These assumptions may be reasonable when the individual is self-employed, when the effects of pollution on the labour market are sufficiently small and localised, and when individuals are not exposed to pollution at their workplace.

### *Effects on labour productivity*

The household production model presented above can be amended to accommodate for the situation when individuals are required to work a fixed number hours, and for sick leave, the cost of which is generally absorbed by the employer and/or taxpayers. There are also indirect impacts of these labour productivity effects, for instance of industrial production and hence on competitiveness, which can have consequences for economic growth. While the empirical literature is best suited to quantify the direct impacts, applied economic models with a general equilibrium framework are the ideal framework to study the economy-wide costs, including the indirect consequences.

Assuming that air pollution causes no other impact on labour and labour productivity other than the loss of work days, estimates of the cost of absenteeism are available from ExternE and related research projects. A study by the Confederation of British Industry (CBI, 1998), which surveyed representatives of the manufacturing, service, and public sector, estimates the median direct cost to business per employee-day absence to be EUR 85. The mean cost is EUR 114. The survey respondents were asked to estimate the salary costs of absent employees, the costs of replacement staff, and lost service and production time. The study also provided an estimate of the indirect cost of absence, due to lower customer satisfaction and poorer quality of products and services. The mean indirect cost is EUR 168, but this figure should be used with caution due to the low response rate for this particular question. Adjustments of these figures to the mean earnings of workers in different EU countries have resulted in total figure of EUR 295 per employee absence day (Rabl et al., 2014). In projects like ExternE and its sequels, pollution affects productivity only as people get sick and miss work. No direct or other indirect effects are covered.

The effects of air pollution on labour supply have been observed in various areas. Hanna and Oliva (2011) examine an exogenous variation in air pollution due to the closure of a large refinery in Mexico City to understand how air pollution impacts labour supply. The closure led to an eight percent decline in pollution in the surrounding neighbourhoods. They found that a one percent increase in sulphur dioxide results in a 0.61% decrease in the hours worked. The effects do not appear to be driven by labour demand shocks, nor differential migration, as a result of the closure in the areas near the refinery, and so Hanna and Oliva conclude that it is correct to ascribe the changes in labour supply to changes in air pollution levels.

One important, but understudied, issue is whether air pollution affects not only hours worked, but also has a direct impact on the productivity of labour. This might be the case if, for example, pollution causes subclinical symptoms that do not result in loss of work time, but reduce productivity at work. There are a few studies that address this issue. Zivin et al. (2011) measure the number of hours worked and labour productivity of farm workers in Southern California. Chang et al. (2014) study the effect of outdoor air pollution on the productivity of indoor workers at a pear-packing factory in Northern California. They find that an increase in fine particulate matter (PM<sub>2.5</sub>) outdoors leads to a statistically and economically significant decrease in packing speeds inside the factory. Specifically, a 10-unit change in PM<sub>2.5</sub> decreases worker productivity by 6%. They find that pollutants that do not travel indoors, like ozone, have little effect and that PM<sub>2.5</sub> has little effect on hours worked or decision to work. They also note that PM<sub>2.5</sub> begins to affect productivity at levels well below current US air quality standards.

### *3.3 Issues in valuing mortality and morbidity impacts*

In both theory and practice, obtaining credible estimates of the economic value of the health impacts of air pollution for policy analyses or cost of inaction studies is complicated by a number of factors, such as the age of the people affected, co-morbidity (i.e., the presence of chronic illnesses that affect the baseline risk of dying and may themselves be associated with pain and discomfort), and the futurity of risk reductions considerations. This section discusses some of these issues. For a more comprehensive discussion, see OECD (2006).

### Age and background risk

Some observers suggest that WTP should be lowered when a policy reduces risks for older persons or persons in compromised health, because they have a higher background risk and a shorter life expectancy.<sup>13</sup> This issue is particularly relevant in estimating the VSL. To analyse this issue, it is possible to use multi-period models, such as the life cycle model. In this model, an individual at age  $j$  receives expected utility  $V_j$  over the remainder of his lifetime:

$$(14) \quad V_j = \sum_{t=j}^T q_{j,t} (1 + \rho)^{j-t} U_t(C_t),$$

Where  $V_j$  is the present value of the utility  $U$  of consumption  $C$  in each period,  $U_t(C_t)$ , times the probability that the individual survives to that period,  $q_{j,t}$ , discounted to the present value at the subjective rate of time preference  $\rho$ . The present value of utility is evaluated from present to the maximum lifetime  $T$ . The specific expression of the budget constraint of the individual depends on the assumptions made on opportunities for borrowing and lending. If, for example, it is assumed that the individual can borrow and lend at the riskless rate  $r$ , but never be a net borrower, and that the individual's wealth constraint is binding only at  $T$ , the VSL at age  $j$  is equal to:

$$(15) \quad VSL_j = (1 - D_j)^{-1} \sum_{t=j+1}^T q_{j,t} (1 + \rho)^{j-t} \frac{U_t(C_t)}{U'_t(C_t)},$$

where  $D_j$  is the probability of dying at age  $j$ .<sup>14</sup>

If the term  $\frac{U_t(C_t)}{U'_t(C_t)}$  is constant with respect to age, then it can be brought outside of the summation in

equation (15), implying that WTP is proportional to the discounted remaining life years. If, in addition, the discount rate is zero, then WTP for a reduction in the risk of dying is indeed strictly proportional to remaining life years. Hence, adjusting VSL for age or other factors affecting remaining life expectancy relies on two restrictive assumptions: (i) that the utility divided by marginal utility does not vary with age, and (ii) that the discount rate is zero. There is no particular reason to believe that these assumptions should be true in practice. For example, if the marginal utility of consumption increases with age, then it is no longer appropriate to assume that WTP is proportional to remaining life years.

Because the risk of dying because of pollution exposure is greater for persons with chronic cardiovascular and respiratory illnesses, it is important to verify whether the WTP depends on a person's health status. In equation (15), persons with chronic illnesses would be argued to have a higher  $D$  (the probability of dying in their  $j$ -th year of age), and lower probabilities of surviving to future ages. However, it is not clear how the remaining terms in (15) depend on health status, implying that theory does not offer predictions about the effect of impaired health on the VSL.

<sup>13</sup> In the transportation safety and environmental policy contexts, it has been noted that deaths occur disproportionately in certain age groups. For example, the majority of the people dying in road traffic accidents are young males, whereas epidemiological evidence from the US (Pope et al., 1995) indicates that the over 75% of the lives saved by the Clean Air Act are those of persons 65 years old and older. Likewise, heat waves have been linked with increased premature mortality among the elderly (see, for example, Basu and Samet, 2002; Currero et al., 2002; Ebi et al., 2004; Median-Ramon et al., 2006; Median-Ramon and Schwartz, 2007). This has led to the question whether the VSL should be adjusted for age.

<sup>14</sup> VSL at age  $j$  is defined as the willingness-to-pay for a marginal change in  $D_j$ , the probability of dying at age  $j$ .

In a theoretical exercise, Shepherd and Zeckhauser (1982) assume that the utility function is of the form  $C^\beta$ , and show that for plausible values of  $\beta$ , WTP for a risk reduction has an inverted-U shape that peaks when the individual is in his 50ies. Jones-Lee (1989) and Johannesson et al. (1997) find empirical support for this notion. Both studies report that WTP for a risk reduction of a specified size is at its highest when the individual is approximately 50 years old. WTP is lower among younger and older individuals, which is consistent with the quadratic relationship predicted by Shepherd and Zeckhauser.

Empirical studies (reviewed in Krupnick, 2007) suggest the WTP for any given risk reduction declines by a relatively modest amount only among the very elderly, and Alberini and Chiabai (2007) report that persons surveyed in Italy about cardiovascular mortality risks are willing to pay more if they are in poor health. This finding is in sharp contrast with the practice of some government agencies (e.g., in the US, the Food and Drug Administration) and in medical decision-making of relying on quality-adjusted life years (QALY), whereby survival with a chronic illness would be deemed as less desirable than survival in perfect health.

Viscusi and Aldy (2007) estimate the relationship between VSL and age using labour market data, finding that older workers have greater risk vulnerability and face flatter wage-risk gradients than younger workers, and that the relationship between VSL and age follows an inverted-U shape. Their estimates of the VSL, based on US labour markets, range from USD 6.4 million for younger workers to a peak of USD 9 million for workers aged 35-44, and then decline with age, to the point that it is USD 3.8 million for the oldest workers in the sample (persons aged 55-62).

### *Latency*

Using stated preference methods, it is possible to ask people to report their WTP for a risk reduction that starts in the future. Economic theory holds that if actuarially fair annuities are available, WTP for a risk reduction occurring at age  $a+t$  is equal to:

$$(16) \quad WTP_{a,a+t} = \pi_{a,a+t} \cdot (1 + \rho)^{-t} \cdot WTP_{a+t,a+t},$$

Where  $WTP_{a+t,a+t}$  is the WTP at age  $a+t$  for a risk reduction beginning at age  $a+t$ , and  $\pi_{a,a+t}$  is the probability of surviving from age  $a$  to age  $a+t$  (Cropper and Sussman, 1990). Since  $\pi_{a,a+t}$  and  $(1+\rho)^{-t}$  are less than one, WTP for a future risk reduction should be less than the WTP for an immediate risk reduction of the same size if the person were  $(a+t)$  years old. This assumes that a person is willing to pay for an immediate risk reduction at age  $(a+t)$  no less than what he or she is willing to pay for the same risk reduction, to be incurred immediately, at age  $a$ .

Only a few studies have sought to elicit WTP for latent risk change. They include Johannesson et al. (1996), Krupnick et al. (2002), Alberini et al. (2006), Alberini and Chiabai (2007b), Alberini et al., (2007), Tsuge et al. (2005), and Alberini and Scasny (2013). These studies estimate the discount rate applied by individuals to future health risks reductions to range between zero and 27%.

### *Cohort and country effects*

Preferences on the WTP may change over time due to changes in income or in the WTP for reducing the risk of ill health or premature death. This issue is particularly relevant for the valuation of issues like air pollution, as emissions and pollution scenarios span over decades. While this issue also applies to morbidity, cohort and country effects are discussed with respect to the evaluation of mortality through the VSL.

The present value of the benefits of saving lives over long periods of time can be calculated as:

$$(17) \quad \sum_{t=1}^T VSL_t \cdot L_t \cdot (1 + \delta_t)^{-t},$$

where  $\delta$  is an appropriate discount rate,  $L$  is number of lives saved, and the VSL is allowed to change over time. There are at least four reasons why VSL might change over time. First, as people become wealthier with economic growth, it seems reasonable to believe that their WTP for a reduction in the risk of death may increase.

Second, preferences can change over time, and the rate at which people are willing to trade off income for risk reductions may change with economic growth. Costa and Kahn (2004) estimate compensating wage studies for different years in the US. Using Census micro-data and fatality risk figures from the Bureau of Labour Statistics for 1940, 1950, 1960, and 1980, they conclude that the quantity of safety and the compensating differential have increased over time. Liu et al. (1997) estimate VSL and its relationship to income using data on workplace fatality risk and wages over 16 years in a rapidly developing economy, Taiwan. They also find that VSL increases with economic growth. The estimated income elasticity of VSL is between 2 and 3. This income elasticity should be interpreted as that associated with economic growth, and should be applied when computing the mortality benefits of policy packages that produce mortality risk reductions over a very long horizon.<sup>15</sup>

Third, the VSL may reflect the different composition by age in the population, and different prevalence of chronic illnesses. Finally, the discount rate used may change over the time horizon. This would be the case if, for example, people did not use standard constant exponential discounting, exhibiting instead, for example, hyperbolic discounting.

If people behave differently in short-run and long-run trade-offs, and if they are more impatient in the short-run decisions than in the long-run decisions, discounting may need to be captured through non-linear functions (Lowenstein and Prelec, 1992; Harvey, 1994; Laibson, 1997). Different reasons are mentioned in economic literature to explain why people might rationally choose hyperbolic discounting. They may prefer sure results, their preferences could change, or they may have an urgent need such as hunger or paying rent. In addition, they may simply be unable to distinguish between period  $t$  and period  $t+I$ , when  $t$  is a long time from now (see discussion on latency).

Finally, considering assessments and models that cover multiple countries, raises the question of whether a different VSL should be used for each country. Economic theory and empirical studies suggest that wealthier persons – and hence, wealthier countries – should hold greater WTP amounts for the same risk reduction, which would imply higher VSLs. See Section 3.4 for further discussion on adapting VSL values for different countries.

### ***Infant mortality, low birth weights, and other infant and child outcomes***

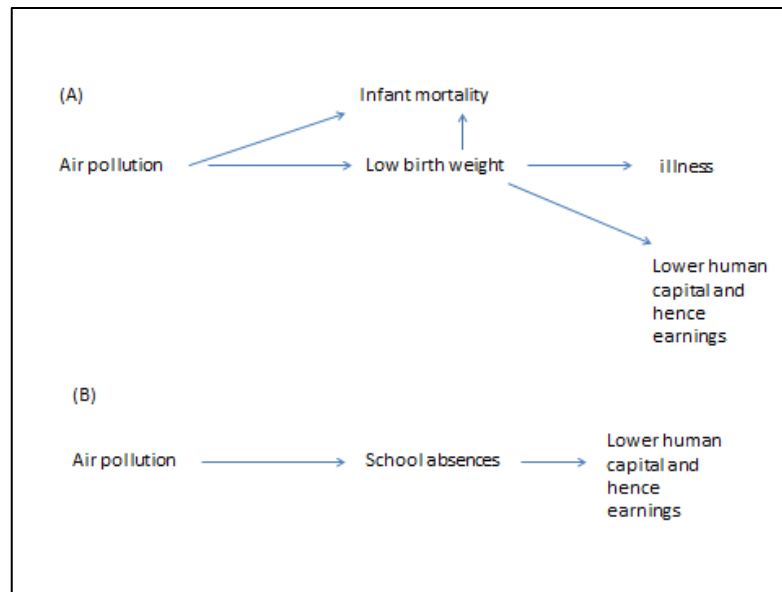
One of the most debated issues in evaluating health impacts is attributing monetary values to impacts on infants and children. The models described in the previous sections are not adapted to situations where pollution interferes with the accumulation of human capital, and thus affects future labour supply and the wage rate. Figure 1 presents two possible pathways for pollution to consider such effects. The pathway in panel (A) shows that air pollution may cause i) infant mortality, or ii) low birth weight, which in turn may cause infant mortality and illness, and has been linked with lower IQ, school absences, worse educational

<sup>15</sup> This “income elasticity” is expected to be different from that estimated within any given study by exploiting the cross variation in income of the individuals or households in the sample. In general, the income elasticity of VSL describes how WTP for a unit reduction in mortality risk depends on income.

attainments, and lower earnings (see Black et al., 2007, for a survey of the literature on this link and for an original and convincing study based on Norwegian twins).

Panel (B) in Figure 1 focuses on school absences associated with air pollution, which impose costs on society (remedial programmes, future lower wages due to worse educational attainment, lost wages and reduced productivity for parents) (Currie et al., 2009).

**Figure 1. Pathways of effects of pollution on infant health endpoint and human capital formation**



Source: Authors' elaboration of the literature (Black et al., 2007; Currie et al., 2009).

Regarding the air pollution-infant mortality link, the ExterneE project, the MIT-EPPA model and the IIASA model have used estimates the exposure-response function estimated in Woodruff et al. (1997), even though Lipfert et al. (2000) found that the association was not robust when maternal characteristics were controlled for. Woodruff et al. (2008) later presented stronger evidence based on US counties for 1999-2002, and found evidence of an association between PM<sub>10</sub> levels and respiratory deaths (and between ozone and sudden infant death syndrome). Chay and Greenstone (2003) present evidence of effects using a quasi-experiment approach that relies on the fluctuation in pollution over a relatively short period due to an economic recession.<sup>16</sup> Currie et al. (2009) address issues of pollution mismeasurement by taking advantage of precise information about the place of residence of mothers, control for unobservables using maternal characteristics and fixed effects, and find evidence of causal effects of carbon monoxide on infant mortality.

It is not clear what metric should be used to monetise infant mortality effects. Rabl et al. (2014) are generally proponents of using the VOLY to monetise the mortality benefits of air pollution reductions (or

<sup>16</sup> Chay and Greenstone (2003) estimate the impact of total suspended particulates (TSPs) on infant mortality by utilising air pollution reduction variations produced by the 1981-1982 recession. They find that a 1- $\mu\text{g}/\text{m}^3$  reduction in TSPs is associated with 4 to 7 fewer infant deaths per 100,000 live births at the county level, which is an elasticity of 0.35. They suggest that foetal exposure is a potential pathophysiologic mechanism based on mortality reduction effects being driven by a reduction in deaths within the first month after birth. They found significant effects of TSPs reductions on deaths within 24 hours of birth and on infant birth weight.

the mortality costs of inaction with respect to poor air quality), but in the case of infant mortality, they conclude that it is preferable to use the VSL because i) there are no estimates of life expectancy loss associated with infant mortality (especially if the infant is frail), and ii) there are no estimates of the VOLY for children.

Credible estimates of the VSL for infants are not available either, although studies (see OECD, 2010, for a discussion) have elicited child VSL using a variety of approaches (ranging from stated preference studies to consumer purchases of safety equipment, such as bicycle helmets) and with mixed results. Specifically, some have found child VSL to be greater than adult VSL (see Alberini and Scasny, 2011), others have found it to be lower, and others yet have concluded that there are no statistically significant differences between the two.

A number of studies have linked air pollution with low birth weight and/or duration of gestation. These studies (summarised in Currie et al., 2009) have generally been of an observational nature. Quasi experiments (e.g., Lavaine and Neidell, 2014) or better efforts to control for unobservables (Currie et al., 2009) have, however, generally confirmed the existence of an effect of sulphur dioxide, particulate matter, and carbon monoxide on birth weight and gestation time.

One important question is how to attach a monetary value to low birth weight outcomes. One approach (recently deployed in Scasny and Zverinova, 2014) is to ask prospective parents how much they would be willing to pay to reduce the risk of a low birth weight new-born. However, altruism may lead to over-estimates when valuing someone else's life. Alternatively, using a cost-of-illness approach, it is possible to quantify the medical expenses associated with treating illnesses and health problems attributable to a low birth weight, the additional education expenditures associated with addressing poor attendance and frequent absences from school due to illnesses attributable to a low birth weight, and the lower earnings associated with worse educational attainment.

Using the estimated elasticity between birth weight and earnings of 0.1 from Black et al. (2007), the average earnings of full-time workers in 2003, and the total number of births in 2003, Currie et al. (2009) estimate that a decline in carbon monoxide concentrations of the magnitude observed in Texas from 1989 to 2003 resulted in a nationwide increase in earnings of USD 720 million for the 2003 birth cohort. This calculation assumes participation in the labour force for 30 years and a discount rate of 6%, and almost certainly understates the true increase in earnings, since future earnings of this cohort are likely to be higher than current earnings. This approach is similar to that used to quantify the damage costs of lead specifically due to its neurotoxicity and effect on IQ (see Rabl et al., 2014).

### ***Emerging pollutants and dreaded health endpoints***

There is an increasing availability of new information on the impacts of air pollution on health. Epidemiologic research has recently focused on specific components of air pollution or by-products of fossil fuel combustion (e.g., black carbon), but the valuation procedures presented in this document ignore the specific chemical, because they focus on the health endpoint itself. Future research might wish to explore whether the specific source of health risks results in different WTP figure to reduce those health risks.

A further issue is the treatment of cancer death. When assessing the mortality risks associated with air pollution exposure, researchers have investigated whether the VSL in the context of cancer death differs from that arising in respect of other causes of death. This is because psychometric research suggests that people's perceptions of risks, and thus the WTP to reduce them, may be affected by other dimensions of risks (included how dreaded a risk is) than the sheer probabilities of the adverse event. Empirical research in this area has produced mixed results (see Alberini and Scasny, 2013 and OECD, 2012), and government

agencies follow different guidelines. For example, the US Environmental Protection Agency uses a single VSL figure, without distinguishing for the cause of death, whereas DG-Environment applies a 50% cancer premium with respect to the central value.

### 3.4 Benefit transfer

Ideally, concentration-response functions and economic values for all health endpoints would be available for all countries and sub-regions as needed by different studies. In reality however, the geographical coverage of empirical studies is often limited. To address this issue, the practice of benefit transfer is used to adapt estimates in specific certain geographical area to other areas, time periods, populations, pollutants and pollutant levels.

Although some studies have been conducted in cities in developing countries (which generally have high air pollution levels), the bulk of the concentration-response functions comes from studies in the United States and Europe. Summary measures of the slopes of these functions are generally adopted in applied economic models, and predictions based on them are usually applied to other areas, even though the main causes of deaths in the population and pollution levels may be different.

Value benefit transfer procedures (discussed at length in Ready and Navrud, 2004, and OECD, 2012) are generally conducted in three different ways. The first is to simply apply an existing WTP without further adjustment. The second is to adjust for income:

$$(18) \quad WTP_B = WTP_A \cdot \left( \frac{y_B}{y_A} \right)^\alpha$$

where subscript *A* denotes the study site, subscript *B* the policy site (i.e., the one where the WTP to avoid a given health endpoint needs to be computed), *y* is income, and  $\alpha$  is the income elasticity of WTP. If  $\alpha$  is 1, then the adjustment is simply proportional to income. Other values of the income elasticity have been attempted and are usually less than one.

The third and last procedure is to estimate a WTP function, where the WTP is related to characteristics of the health endpoint to be valued and of the population or sample from which the WTP information has been gathered. Using the estimated coefficients, a prediction is made for area or population *B* based on plugging in the characteristics of the health endpoint and population at site *B*.

One does, however, wonder whether transfers produce credible results, as they assume similarities in preferences for health risk reductions. Ready et al. (2004), for example, examined the reliability of benefit transfers between countries in Europe. They used a standardised contingent valuation survey questionnaire in five different European countries to measure the benefits of specific health impacts related to air and water quality. They found that consistent inter-country differences in willingness-to-pay to avoid ill health episodes could not be explained by measurable differences in individual characteristics. International transfer of unit values resulted in an average transfer error of 38%. Accounting for measurable differences among countries in health status, income and other demographic measures, either through ad hoc adjustments to the transferred values or through value function transfer, did not improve transfer performance either.

## 4. Applied economic models used for policy analysis and estimation of damages of pollution

This section presents selected modelling frameworks used to quantify the damages of air pollution (or the benefits of reducing air pollution). More specifically this section considers two broad classes of

modelling approaches. One class is based on the integrated assessment of economic growth and environmental impacts. The other is based on general equilibrium theory. The former class of modelling frameworks focuses on IIASA's cost-benefit analysis of the EU Clean Air Package, which relies on the GAINS model. For the latter class this report analyses the Mayeres and Van Regemorter (2008)'s adaptation of the GEM-E3 model and MIT's EPPA-HE model. Table 5 summarises the features of three main modelling approaches used to study the costs of air pollution. As part of the second class of models, three further models (ENV-Linkages, ICES and ENVISAGE) are also considered as they include health impacts related to climate change.

Comparing the approaches taken by these models also permits a comparison of the advantages of undertaking different modelling frameworks and in particular to compare GAINS, which is an integrated assessment model (IAM) with a state-of-the-art dispersion model, with computable general equilibrium (CGE) models.

This selection of models also highlights a fundamental difference between the two approaches. While the first approach considers air pollution and associated market effects from the point of view of the affected individual, the second approach takes the point of view of the society as a whole. This has the immediate consequence that while the first approach is more suitable to capture finer physical, chemical, medical or epidemiologic features of air pollution, the second approach can better account for economic and sectoral impacts. Thus, in a sense, the two approaches can give complementary information.<sup>17</sup>

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Another feature that partial and general equilibrium models share and set them apart from the sequential approach is, quite trivially, that the results they yield satisfy the condition that the economy under scrutiny is in an a state of equilibrium, and thus they generate equilibrium values for prices and quantities sold and purchased. In other words, they assume well-functioning markets. Some valuation methods in the sequential approach also implicitly assume the existence of a market (hedonic pricing and travel cost methods, for instance) but others do not, and for some of them, the *raison d'être* is precisely the absence of a market (contingent valuation methods, for instance).

Table 5. Summary of the features of alternate models to estimate the damages of air pollution

	<b>GAINS</b>	<b>GEM-E3 (as in Mayeres &amp; Van Regemorter, 2008)</b>	<b>EPPA – HE</b>
<b>Model type</b>	- Integrated assessment model - Damage function approach	CGE model	CGE model
<b>Regions covered</b>	Global (health impacts in European Union)	Global (health impacts in European Union)	Global (health impacts in Europe, China and US and global for O <sub>3</sub> )
<b>Pollutants</b>	- Air pollutants emissions: PM <sub>2.5</sub> , PM <sub>10</sub> , O <sub>3</sub> , SOMO35, SO <sub>2</sub> , NO <sub>x</sub> ; no heavy metals, arsenic, benzene, mercury, or lead - GHG emissions	- Air pollutants emissions: NO <sub>x</sub> , SO <sub>2</sub> , VOC (as co-pollutants of CO <sub>2</sub> emissions) - GHG emissions	- Air pollutants emissions: O <sub>3</sub> (Selin et al., 2009); O <sub>3</sub> and PM <sub>10</sub> (Nam et al., 2009); O <sub>3</sub> , CO, NO <sub>2</sub> SO <sub>2</sub> , PM <sub>2.5</sub> and PM <sub>10</sub> (Yang et al., 2005) - GHG emissions
<b>Concentration levels and spatial resolution</b>	Very high resolution	High resolution (through FASST model).	Very high resolution. Maps are also adjusted time wise, based on population trends.
<b>Regional application of concentration-response functions</b>	The same function is applied to all regions but there is a distinction among different baselines.	The same function is applied to all regions but with different baseline life tables, like in ExternE	Distinction of functions by region using detailed raster maps for population weighted concentrations (1°x1° resolution).
<b>Health endpoints</b>	- Restricted activity days - Respiratory hospital admissions - Cerebrovascular/Cardiovascular Hospital Admissions - Chronic Bronchitis (adult) Bronchitis (children) - Minor restricted activity day - Acute mortality - Infant mortality	- Restricted activity days - Respiratory hospital admissions - Cerebrovascular/Cardiovascular Hospital Admissions - Chronic Bronchitis (adult) - Asthma attacks - Cough - Lower respiratory symptoms (wheeze) - Minor restricted activity day - Emergency room visit - Acute mortality	- Hospital admissions - Emergency room visits for respiratory illness - General practitioner visits: asthma, lower respiratory symptoms - Respiratory symptoms in asthmatics: adults and children - Respiratory medication use - adults and children - Restricted activity day - Cough day symptom work loss day - Minor restricted activity day - Chronic bronchitis - Chronic mortality

	<ul style="list-style-type: none"> <li>- Work loss day</li> <li>- Asthma symptoms</li> <li>- Chronic mortality</li> </ul>	<ul style="list-style-type: none"> <li>- Infant mortality</li> <li>- Work loss day</li> <li>- Medications/bronchodilator use</li> <li>- Chronic mortality</li> </ul>	<ul style="list-style-type: none"> <li>- Acute mortality</li> </ul>
<b>Mortality</b>	Included	Included applying ExternE concentration-response functions	Included differentiating acute mortality (valued at 0.5years lost) from chronic mortality (valued through cumulative exposure and the age-cohort characterization of the population, which allows tracking changes in deaths through time).
<b>Mortality due to ozone</b>	Acute mortality only	Acute mortality only	Acute and chronic mortality
<b>Mortality due to PM</b>	Acute and chronic mortality (PM <sub>2.5</sub> )	Acute and chronic mortality (both PM <sub>2.5</sub> and PM <sub>10</sub> )	Acute and chronic mortality (both PM <sub>2.5</sub> and PM <sub>10</sub> )
<b>Valuation of mortality</b>	VSL and VOLYs	VSL/VOLY figures from ExternE or the conventional approach based on loss of output	Output and leisure lost
<b>Age cohorts</b>	Yes	N/A	Yes
<b>Role of age cohorts</b>	Mortality is affected by age cohorts.	Mortality is not affected by age cohorts.	The model includes a cohort structure, which allows differentiating between chronic and acute mortality impacts. There is an age-dependent probability of death due to chronic exposure to pollutant that becomes larger as age increase, and is significantly lower but not zero, before the age of 30. Acute mortality is assumed to imply a loss of 0.5 years for the workforce in the current period, that is “a flow accounting of less than a period” and as such it should not cover infant mortality.
<b>Valuation of infant mortality</b>	VSL	N/A	Accounted through the cohort structure, but it does not get any specific separate treatment.
<b>Low birth weight</b>	N/A	N/A	N/A
<b>Medical costs</b>	Included	Included	Included as households use health services (i.e., hospital care and physician services) from the service sector.

<b>Lost work days</b>	Included	Included	Included as, besides purchasing health services from the public and private health sector, household labour is drawn from labour and leisure, reducing the amount available for other uses.
<b>Restricted activity days</b>	Included	Included	Yes, see above. Lost work time, thus both lost and restricted activity days, are considered as a reduction in work force (in monetary equivalents). In Yang et al. (2005) each RAD is valued at \$ 106 (2000 USD) based on Holland et al. (1998).
<b>Loss of leisure</b>	No	Pollution reduces time available to work, but no attempt is made to value the reduction of leisure separately from loss of work time	Yes. Pollution effects work time as well as leisure and the separate effects are quantified. Workers' leisure is valued at their wage rate, taking into account that the latter is age dependent and peaks at 50-54 years; leisure is also used to account for the children and the elderly, whose time losses are valued at 1/3 and 2/3 of the wage rate, respectively.
<b>Valuation of disutility from illness</b>	Included	Captured by the WTP-based figures from ExternE	Partly captured, through the loss of leisure.
<b>Effects on labour productivity labour or labour supply (i.e. hours worked)</b>	N/A	Productivity of labour does not depend on pollution but pollution affects the labour supply and the labour demand and as a result should be reflected in the equilibrium wage rate.	No, only labour supply (and availability of leisure)
<b>Effects on size of the labour force</b>	N/A	Accounted, through mortality effects.	Accounted, through effects of acute and chronic mortality through time.
<b>Adjusting unit values for regional income</b>	N/A	N/A	Yes, since the model has a geographical disaggregation at the national level.

#### ***4.1 Integrated assessment framework***

The main modelling example of integrated assessment for air pollution impacts is the **GAINS model**. In IASA (2014), the GAINS model is applied to perform a cost-benefit analysis of the final policy scenarios in the EU Clean Air Package.

GAINS estimates emissions, mitigation potentials, and costs for SO<sub>2</sub>, NO<sub>x</sub>, CO, PM<sub>2.5</sub>, PM<sub>10</sub>, NH<sub>3</sub>, and VOC pollutants as well as the six greenhouse gases specified in the Kyoto protocol. Emissions estimates are pollutant-specific and based on scenarios about generation of electricity, energy demand, transportation, agriculture, manufacturing, etc. Emissions also depend on emission factors, removal efficiency of emission control measure, and the extent to which such measures are applied. This varies with the stringency of regulations about pollution control and different policy scenarios can be produced to reflect different stringency levels. The GAINS model further computes the costs associated with each possible pollution control measure. This feature makes the model appropriate for benefit-cost analyses.

Emissions are then entered into a dispersion model, which produces estimates of the concentrations of the various pollutants at a high level of resolution for Europe. Calculations are made for the formation of particulate matters and ground-level ozone.

Once ambient concentrations are quantified, the GAINS model applies WHO-reviewed concentration-response functions to calculate the health impacts. The health endpoints considered are restricted activity days, respiratory hospital admission, cerebrovascular and cardiovascular hospital admission, symptom days, chronic bronchitis in adults, bronchitis in children, minor restricted activity days, acute mortality, infant mortality, chronic mortality, work loss days and asthma symptoms. The physical health endpoints are first quantified for each given emissions scenario, and monetary values are attached to them. Unit values are adapted from the Externe project.<sup>18</sup> Ecosystem and vegetation impacts are also calculated.

Results from IASA (2014) suggest that the net benefits of reducing air pollution vary dramatically across countries and across scenarios. For example, current pollution control regulations lead to net benefits while there are net costs under the maximum technically feasible reduction scenario. Rabl et al. (2014) criticise the GAINS model in that the true extent of potential abatement is underestimated and costs are exaggerated. This biases the cost-benefit analyses against finding a positive net benefit under the most aggressive regulation scenarios.

#### ***4.2 General equilibrium framework***

In a CGE model, all the roles individuals play are covered, be they those of single individuals, households, or parts of a private or public institutions. This societal focus is by no means a feature unique to CGE models, but it is shared by other economic modelling approaches such as partial equilibrium models or input–output studies. However, while partial equilibrium models focus on a single sector and take as given what happens in the rest of the economy, general equilibrium models look simultaneously at the equilibrium of all the sectors and agents in the economy and thus allow tracking the effects of any given exogenous impact while they “trickle down” across the economy. These two features of CGE models

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<sup>18</sup> For example, the IASA (2014) study removes the cost of respiratory medication use because of its insignificant contribution to total damage. The cost of bronchitis in children is added, valued at EUR 588, or EUR 42 per day for 14 days. They reduce the value of chronic bronchitis in adults based on literature review found in the HEIMSTSA study (Hunt, 2008; Navrud, 2008). The remainder of the costs involved are from WHO databases and had been previously adopted in the Clean Air for Europe benefit-cost analysis (Watkiss et al., 2005). When unit values are very different across different countries, as is the case with chronic bronchitis with adults, the IASA report simply adopts the average of these values.

– the societal focus and the coverage of all economic sectors and agents – underline the need to use a CGE setting to account for some of the components of health impacts.

Consider for instance morbidity. The WTP to avoid morbidity effects due to pollution includes four main components: (i) lost earnings, (ii) medical expenditures, (iii) the cost of the averting activity, and (iv) the disutility of illness (see Section 3).

Regarding (i), CGE models are normally equipped to handle the loss of earnings due to illness associated with pollution exposure, the loss of output associated with premature mortality, and can in some cases accommodate lost leisure due to pollution exposures.<sup>19</sup> From a private point of view, the relevance of this component depends on whether the individuals whose WTP was elicited receive labour income irrespective of their health status. This can be very different for a self-employed person who earns only when is able to work, compared to an employee with a fixed monthly salary. From society's point of view, however, the inability of both individuals to work is a loss which affects the overall welfare through loss of output and the reduced availability of a primary factor of production, labour.

As to the second component – medical expenditures – these are usually included in WTP figures only in terms of the fraction of the costs the respondents anticipate they will have to pay directly in case of illness and it is not covered by insurance or public funds. From society's point of view, however, they imply a variation in the demand of the services provided by the health sector, and a variation in the related expenditures for the public sector or the private insurance sector, which can be larger than that directly borne by the individuals, depending on the rules of the health systems of the countries under scrutiny. Analogously, averting activities, such as spending more time at non-polluted locations or buying an air filtering system to filter out pollutants, have both a strictly private component and an economic impact on the rest of the society.

Finally, the non-monetary effects on welfare related to pain, discomfort and psychological distress related to being ill is typically captured by WTP elicitation, but usually cannot fit into a CGE setting. The non-monetary effects are usually hard to include in a CGE setting for a number of reasons. First, they cannot be accounted for in Socioeconomic Accounting Matrix (SAM), at least not in a straightforward and uncontroversial way. Moreover, the WTP may vary considerably in the weight and magnitude of these three components across the various endpoints. To illustrate, respiratory hospital admissions have an 85%-15% split between health care costs and labour/leisure lost while restricted activity days have no healthcare costs, according to Matus et al., 2008. Some ailments are much more dreaded than others, as is the case of cancer vs. heart attacks (Rabl et al., 2014).

This does not mean that all CGE models are naturally equipped to deal with such issues, despite being in principle the most suitable framework into which such impacts can be introduced. On the contrary, traditionally, even CGE models that deal with externalities have for a long time failed to include into their structure the effects from the environment back to the economy, or as they are often referred to, the *feedback effects*.<sup>20</sup> Much can be learnt from economic theory on modelling externalities (see Box 1).

In general, the degree of achievement of efforts in modelling environmental feedbacks in CGE models depends upon the realism of the assumptions made both in the health impacts assessment and in the design

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<sup>19</sup> This in theory also includes the time devoted to taking care of family members who have been fallen ill, a component usually not captured in health damage assessments in a CGE setting. The sole exception is the EPPA-HE framework which includes a household health production function and thus may in principle allow for these effects, although the data used for calibration of the various applications of the model do not appear to include them explicitly.

<sup>20</sup> See for instance Mayeres and Van Regemorter (2008).

of the CGE modelling framework. As a rule, some degree of approximation is unavoidable, particularly if the model was not designed for this purpose to start with. Compromises and simplifications are sometimes made in terms of the representation of health-relevant endpoints, the ability of the model to account for all relevant harmful emissions, the compatibility of the sector disaggregation in the CGE with the proper characterisation of the health service sector, the level of detail of labour and population dynamics, etc.

### Box 1. Lessons from economic theory

Lessons can be learnt from traditional approaches to deal with externalities in CGE models. This can be seen as an indirect and unavoidably imprecise way to deal with external costs, compared to including explicitly the health impacts into the equations of the model. A typical policy exercise in CGE model is to introduce or vary a tax on a given good or production factor and see how the economy reacts to the change, adjusting itself to a new equilibrium in which all sectors and agents have adapted to the new tax. This approach has been widely applied in externalities regulation studies in a CGE setting. In particular, if the new tax is levied on the polluting emissions causing the health impacts under scrutiny, then this approach amounts to force the model to take health-related external damages into account by means of a (quasi) Pigouvian tax.

Economic theory tells us that if the only distortion to the economy is a negative externality, optimality can be restored by imposing upon the polluter a tax equal to the marginal external damage of the optimal emission level. This is the Pigouvian tax. Despite being highly stylised, CGE models do allow for a level of realism beyond the theoretical construction on which the Pigouvian tax idea is based, and thus portray a second-best world with plenty of distortions (mainly due to existing taxes and subsidies). Thus a full internalisation à la Pigou is precluded to all CGE, but a partial internalisation is possible, and its precision depends on how well the polluting sector(s) and related emission(s) are portrayed in the CGE. If the model under scrutiny does not yield emissions of a given pollutant, the damages related to that pollutant should be included by enriching the structure of the model or approximated in terms of dollars per unit of output of the sectors that emit that pollutant, in order to compute the relevant tax. This requires significant computations outside the model and simplifying assumptions about the relationship between sector output and damages.

This approach does not actually change the structure and/or the parameters of the model to provide a richer depiction of health damages. The original model is unchanged, and the modelling exercise is limited to imposing an exogenous change in the taxes faced by the consumers. Once the economy has adjusted to that shock, there is no permanent alteration in the way the economy is portrayed.

### *The GEM-E3 model*

Mayeres and Van Regemorter (2008) amend the GEM-E3 CGE model to incorporate conventional air pollution. They are specifically interested in comparing the predictions of the standard model with those including air pollution feedbacks, namely after air pollution affects individuals' labour supply, medical expenditures incurred to mitigate illness associated with air pollution exposure, and consumption. Attention is restricted to the European Union. They assume that the representative consumer has utility function:

$$(19) \quad U = \alpha_1^0 \ln(C - \bar{C}) + \alpha_2^0 \ln(L - \bar{L}) + \alpha_3^0 \ln(H - \bar{H}) - \sum_{m=1}^M \alpha_{H,m}^0 A_m .$$

Utility is derived from consumption, leisure and health status above subsistence levels. Moreover, individuals experience disutility directly from the levels of ambient concentrations of air pollution. This is apparent from the last term in the right-hand side of equation (19), which for all practical purposes implies that the individual would benefit from lower levels of pollution even when dead. Equation (19), however, is not a state-dependent utility function, and so it is unclear how a WTP to reduce mortality risks can be derived from this model. Mayeres and van Regemorter (2008) admit that this utility function is better suited to represent illness associated with pollution exposure, but not mortality, which continues to be

“modelled ex post, except for the medical costs related to it”. This means that mortality risks are assumed not to affect the number of hours the consumer chooses to work, the medical expenditure associated with mitigating symptoms, and consumption.

The model further contains a consumer budget constraint, a production function, trade between countries, and allows for part of the medical costs to be absorbed by taxpayers. The model results in a consumption demand function, a leisure demand function, and a demand for medical care. These are affected by pollution levels directly (as is the case with the demand for medical care) and indirectly, via the effect that pollution has on disposable income (lower pollution levels free up income and may reduce one’s labour supply).

The marginal WTP for a small change in pollution levels is shown to be the sum of three components: i) the loss of work income due to illness, ii) the marginal change in medical expenditures to mitigate health symptoms, and iii) the value of the disutility of pollution *per se*. The authors interpret this last term as the disutility of mortality (excluding health costs) and non-health related impacts, due to the specific form of utility function in Equation (19).

The labour supply and demand are affected by pollution, but not the productivity of labour. Mayeres and van Regemorter (2008) run this amended and the original GEM-E3 model and to assess the effect of a carbon tax to reach the EU Kyoto protocol targets. The conventional air pollutants of interest are PM<sub>2.5</sub> and PM<sub>10</sub>, NO<sub>x</sub> and VOCs (which combined in certain proportions and under certain atmospheric conditions generate ozone), and SO<sub>2</sub> associated with burning fossil fuels. These emissions are thus co-pollutants with CO<sub>2</sub> emissions. An air dispersion module within GEM-E3 produces estimates of the concentrations at a fine level of geographic resolution, and concentration-response functions and WTP values from ExternE complete the exercise.

Mayeres and van Regemorter (2008) show that productivity losses to producer and medical costs borne by the government account for a small share of the total damages associated with air pollution (ranging from 0.1% to 14.3%, depending on the specific pollutant). Consumer-experienced damages account for 81% to 94% of the damages. Even within consumer-borne damages, loss of work or leisure time accounts for no more than 3.4% of all losses, medical expenditures 16% to 46.6% of the costs, and the remainder (the value of the disutility directly associated with pollution levels) from 46% to almost 90% of the total. This breakdown mirrors that from the ExternE project.

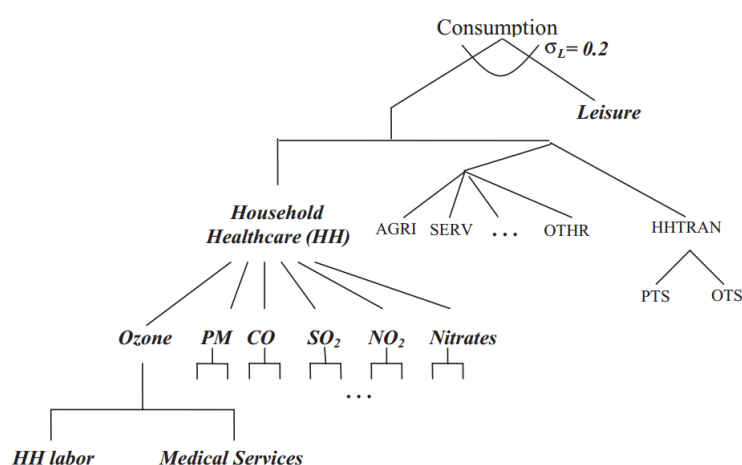
Several aspects of the Mayeres and van Regemorter (2008)’s work needs to be emphasised. First, the treatment of mortality is not reported in detail, so it is unclear whether mortality effects were calculated for different age cohorts or at an aggregate, all-age level. Second, the sensitivity of the model results with respect to the particular functional form assumed for the utility function is unclear. Third, it is assumed that a single value for all countries was imputed to each health endpoint considered, but the paper is silent about this aspect of the computations. Fortunately, the model is limited to the European Union, suggesting that discrepancies in values and baseline health risks across countries are likely to be relatively small.

### ***The EPPA-HE model***

EPPA-HE is another effort to modify a CGE modelling framework to explicitly account for health-related issues. It is based on EPPA (Paltsev et al., 2005), a multi-region, multi-sector, recursive dynamic CGE model. The health effect extension of the EPPA model, EPPA-HE has been used to evaluate health damages due to local air pollution in the US (Yang et al., 2005, Matus et al., 2008), China (Matus, 2005; Matus et al., 2011), in the EU (Nam et al., 2009) and at the global level for ozone emissions only (Selin et al., 2009).

Pollution impacts the economy through health costs (labour lost and medical expenditures) which feed back into the economy and ultimately into next periods' emissions.<sup>21</sup> Health is incorporated into the model by means of a household production function that includes, among its inputs, the provision of health services necessary to cope with illness, and a service sector that uses household labour as input and reduces the amount of it available for other activities (productive activities or leisure). This latter effect is supposed to capture also the non-market component of health cost. EPPA-HE includes WTP estimates in its dataset and the decomposition of the components of WTP to avoid health damages is consistent with EPPA-HE consumption structure: hospitalisation costs are treated as a demand for medical services, lost work time is treated as a reduction in the labour force, and damages beyond these market effects are captured as a loss of leisure. The model partially incorporates mortality-related disutility effects as the loss of a finite resource for the individual, i.e. time, which is evaluated at the wage rate and can be used to work or for leisure.<sup>22</sup> Figure 2 shows the details of the consumption structure in EPPA-HE and the role of local air pollution on consumers' welfare through the household production function.

**Figure 2. Household and consumption structure in the EPPA-HE model**



Source: Matus et al., 2005.

The practical question of how to allocate costs among the three components presents itself also in the EPPA-HE framework. Contrary to Mayeres and Van Regemorter (2008), the largest share goes to direct medical costs (50 to 85%), followed by loss of leisure (10 to 15%) and by loss of labour. This difference might be due to two factors: on one hand, Mayeres and Van Regemorter (2008) allow for a partial governmental subsidisation of medical expenditures; on the other hand, the baseline allocation of time between leisure and work is admittedly based on a range of arbitrary assumptions, to allow for non-observable components such as the (value of) the time of children and of the elderly.

The EPPA-HE framework focused on a feature of health damages often overlooked in economic evaluation: damages come from either acute or chronic impacts which generate damages through time according to very different dynamics. Given the right base data, a dynamic CGE is the ideal setting to

<sup>21</sup> Note that the same happens in GEM-E3, where emissions are endogenous and thus the equilibrium ones in each period reflect all impacts on the economy.

<sup>22</sup> As stated in Yang et al. (2005), health effects raise other issues as well, such as non-use value, and interdependency of welfare among individuals. Thus the non-use part of facing an increased risk of death, i.e. the dread of it, is not included.

capture this difference and hence characterise acute and chronic health impacts more realistically than through point estimates. This is an area in which they effectively manage to improve on Mayeres and Regemorter (2008), where acute and chronic conditions generated by air pollution were depicted in the same way. Avoiding this distinction, Nam et al. (2009) argue, leads to an inaccurate depiction of the flow of labour lost over time. By adding an age cohort module to the CGE structure of the EPPA model, Nam et al. (2009) are able to capture the different time paths of 29 health outcomes in the EU.

The model allows for an age-dependent probability of death due to chronic exposure to pollutant. This probability increases with age, and is significantly lower, but not zero, before the age of 30. Acute mortality is assumed to imply a loss of 0.5 years for the work force in the current period, which is “a flow accounting of less than a period”. Beside the evolution of damages through time, the model is also able to capture the cumulative effect of pollution both directly on the condition of aging individuals affected by pollution-related illness for years, and indirectly, as its cumulative economic burden bears on the potential growth and welfare of the economy through its effect on productivity, GDP and savings,<sup>23</sup> and therefore investments and future growth.

This approach is applied in the EPPA-HE model to evaluate the economic impact of current pollution levels against an hypothetical pollution-free counterfactual, or to assess the benefits in terms of reduced economy-wide damages from pollution control policies such as an hypothetical set of air quality control measures in China, the CAFE program in the EU, the Clean Air Act in the US, or to compare the costs of ozone pollution under scenarios with 2000 and 2050 ozone precursor and greenhouse gas emissions in the (SRES) A1B scenario.

The EPPA-HE approach is quite comprehensive and covers a good deal of the relevant issues. In particular, its cohort structure and the population-weighted concentrations add to its realism. However, the coverage is not complete: infant mortality does not receive a separate treatment, children are regarded as smaller and less relevant (in monetary terms) adults, and there is a very limited treatment of the disutility value of the illness and other non-market effects.

#### ***4.3 Lessons from general equilibrium models including climate change impacts***

Lessons can also be learnt from CGE models that include the economic feedbacks from climate change impacts and in particular from climate-related health impacts. This section focuses on three main modelling frameworks: ICES (Eboli et al., 2009), the ENVISAGE (Roson and Van der Mensbrugge, 2012) and ENV-Linkages (Dellink et al. 2014; OECD, 2015). The methodology used to account for climate change health impacts is roughly the same in the three models and has been originally developed within the ICES framework (Bosello et al., 2006), and then adapted to the two other modelling frameworks. The approach adopted in these models remains one of the few established methodologies to include health-related damages from anthropogenic emissions into a CGE and can be used as a starting point to introduce new features into the models.

These three models share a few fundamental characteristics, but each retains its elements of originality. They all are neo-classical multi-regional recursive dynamic computable general equilibrium models. In each world region, three categories of representative agents entertain economic relationships

<sup>23</sup>

In a cohort structure with cohorts that are allowed to change their magnitude through time and with health impacts differentiated by age, these impacts will have presumably different effects on productivity of labour (morbidity) and labour stock (mortality) through time. To capture that, the labour productivity parameters in the production function should be allowed to vary through time as well. As to savings, they are generally increasing during the active portion of an individual life and decreasing or negative after retirement.

within the region and abroad: productive firms, households and the public sector. The output structure can be more or less detailed according to what it is required by the policy issue under scrutiny; however, in general, given the focus on climate change, the energy sector is the one depicted in greater detail. All models obviously yield GHG emissions, mostly from the energy sector, but also from land use, land use change and forestry, with varying degree of accuracy across models.

The models differ in a number of ways. ICES has quite a rigid labour structure, with full employment and no mobility across or within regions, while ENV-Linkages allows for more flexibility in terms of unemployment and migration in the baseline (though not endogenously in response to policy changes) and thus, in principle, could accommodate a richer depiction of local air pollution health impacts on the productive sectors; ENVISAGE stands somewhat in between with full employment and no international migration in the standard setting, but allowing for an alternate setting with national labour market segmentation with migration from rural to urban activities.

The idea behind the modelling of health impacts in these CGE frameworks is that climate change will affect the conditions in which diseases develop and propagate, and there will be different impacts across diseases and regions of the world. The authors consider a number of diseases and health conditions likely to be affected by climate change – cardiovascular and respiratory diseases, tropical vector-borne diseases (malaria, schistosomiasis, dengue, diarrhoea) – in terms of morbidity and mortality. Vector-borne diseases such as malaria may spread beyond their current areas if the expected temperature increase under climate change creates the condition for infected *Anopheles* mosquitos to thrive outside their current territory. On the other hand, the incidence of some cold-related diseases on sensitive segments of the population, such as respiratory chronic conditions in the elderly, may improve if climate gets milder in regions currently experiencing harsh winters. Bosello et al. (2006) also assume that heat waves will strike exclusively in urban areas, increasing the occurrence of heat-related mortality through acute cardiovascular and respiratory conditions.

Related economic impacts take place through two main channels. First, changes in morbidity and mortality are translated into changes in the marginal productivity of labour, using different appropriate routes for different pathologies. This involves specific out-of-the-model computations to translate changes in chronic and acute mortality in the appropriate, exogenous parameter shifts. Second, the impact on the health services sector is modelled as a shift in factor-specific productivity of the relevant sector. Their argument is that the additional health expenditures (which are expressed in terms of GDP estimated outside the model and are used as an input into their simulations) can be interpreted as coming from a partial equilibrium analysis where the prices and quantities of all other sectors do not change. Thus they impose a shift in the parameters that can induce the desired shift in expenditures, keeping fixed the rest of the economy, and they accomplish this through the said change in productivity.

There is no specific health sector in the model, only a coarser disaggregation between market and non-market services, within which the health sector is included.<sup>24</sup> This approach can thus accommodate a number of climate change health impacts on the world economy, and goes a long way to translate a variety of heterogeneous inputs into a consistent set of simulations. However, there is no internal dynamics in labour as to employment rate and leisure and the impact of diseases thereon, and there is no direct channel to account for non-market health impacts. As to non-market impacts, Bosello et al. (2006) remark that their focus is intentionally on direct economic impacts only, and that, on the basis of the estimates collected in Tol (2002), “the immaterial effects of the risk of death outweigh the economic effects”.

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<sup>24</sup> The health sector is still bundled with other public sector activities even in the most recent version of the GTAP database, GTAP 8 ([www.gtap.agecon.purdue.edu/databases/v8/v8\\_sectors.asp](http://www.gtap.agecon.purdue.edu/databases/v8/v8_sectors.asp)).

## 5. Suggestions and recommendations

Based on the review of the various approaches used to compute the cost of inaction on air pollution, there are a number of suggestions and recommendations that can be drawn. Applied studies should rely on the latest and most reliable statistical and econometric analyses that estimate concentration-response functions linking pollution levels with physical health endpoints. Some efforts should be devoted to reconciling widely different estimates of the cost of certain illnesses (e.g., chronic bronchitis) across different countries (mostly the US and European countries) as they differ by one or two orders of magnitude.

Including the impacts of air pollution on health in CGE models requires a significant restructuring of the models along the lines of the EPPA-HE model. A less demanding, but necessarily partial, approach would be to mimic the approach used by other CGE models to assess the health impacts from climate change. This means translating health impacts into productivity losses, changes in labour supply, and changes in medical expenditures. Additional calculations and efforts are needed since air pollutant emissions and concentrations have local characteristics while the models are set up on a larger scale.

Adding each of the components of the monetised impacts of air pollution implies a fundamental alteration of the original framework. Necessary additional features would involve expanding the social accounting matrix and reworking the production and consumption trees to allow for a richer depiction of labour/leisure, of household activities and possibly the direct negative impact of pollution on household utility, followed by the necessary re-calibration of the model. Including age cohorts to allow for the difference in acute and chronic mortality would add to the complication, because it would imply allowing a time-varying production function and consequences on savings.

In the process of modelling local air pollution health impacts, one faces a trade-off between practical feasibility and the attainable level of refinement and accuracy. If feasibility concerns due to time and resources constraints are of outstanding relevance, it would then make sense to go for the more straightforward option that entails exploiting the common approach with ICES and ENVISAGE to account for climate-change-related health effects, extending such procedure to local air pollution. Given considerably more time and resources, a more refined option would be to go beyond this approach along the lines of the EPPA-HE model or that in Mayeres and Van Regemorter (2008), to introduce a richer utility function, a cohort system or a health production function into the modelling framework.

There are a number of difficulties that need to be taken into consideration. If loss of life expectancy is used to quantify chronic mortality, more credible estimates of the VOLY must be obtained than the ones currently in use. More and improved estimates for the unit values for certain morbidity health endpoints are also needed. In some cases estimates from one region are applied to others. In other cases, estimates are used from studies that used standardised questionnaires in different geographical areas, but executed the survey with different modalities, which makes the results difficult to compare.

There are also a number of shortcomings that would need substantial efforts to improve the existing empirical literature. For instance, the studies reviewed rely on the valuation of “chronic” and “acute” mortality associated with air pollution, taking available estimates of VSL and VOLYs generally without considering whether these figures should be adjusted for the cause of death or the income of the affected countries. The empirical studies that are at the origin of the values used can be of limited quality or rigour. This is especially important in the case of VOLY, which has been typically derived from VSL estimates under certain assumption.

The studies allow for pollution to result in sickness that affects the labour supply, in that workers have to reduce work hours. None of the studies allows for the possibility that pollution exposures might affect labour productivity directly, even if labour supply is not affected. Recent empirical studies have suggested

such effects exist. It would seem likely that pollution affects the productivity of labour in places with extremely high levels of air pollution, and this should be further investigated.

The five modelling approaches reviewed do not cover toxic air pollutants, such as heavy metals, that have the potential to affect cognitive skills and neurological development, and thus have long-term effects on schooling and lifetime earnings. It would seem that in certain geographical areas this might be a serious concern and might deserve a proper investigation. Infant mortality is accounted for in many of the models, but other effects that might in turn influence mortality or result in long-term loss of earnings, such as low birth weight, are not currently included.

While the complications and nuances discussed above are important to understand and consider, they should not imply that no analysis can be done until perfect data is available and techniques are developed to overcome all possible biases. Rather, these should be seen as a word of caution for interpreting the results of air pollution impact assessments. Researchers need to do their best to avoid pitfalls as much as possible, be open about assumptions made, and draw conclusions that acknowledge the uncertainty on the numerical results obtained. Despite the caveats and uncertainties, the available tools are strong enough to allow at least qualitative conclusions to be drawn.

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