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Transport Research Support

TRANSPORT EMISSIONS AND SAVINGS IN HEALTH COSTS

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Transport Research Support

The Transport Research Support program is a joint World Bank/ DFID initiative focusing on emerging issues in the transport sector. Its goal is to generate knowledge in high priority areas of the transport sector and to disseminate to practitioners and decision-makers in developing countries.

Abbreviations

Abbreviations	
BoD	Burden of disease
BTA	Benefit Transfer Approach
COI	Cost-of-Illness
DALY	Disability-Adjusted-Life-Years
EBD	Environmental burden of disease
TSP	Total Suspended Articles
VOLY	Value of a life year
VSL/VOSL	Value of statistical life
WTP	Willingness-to-Pay
YLD	Years lived with disability
YOLL	Years of life lost due to premature mortality or chronic exposure

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EXECUTIVE SUMMARY

This is the final paper of the project on *Transport emissions and savings in health costs* carried out by Ecorys for the World Bank. The report provides an initial overview of the research results and outlines the next steps.

The objective of the study is: "*The project will lead to an estimation approach of how to transfer results on the health impacts of transport into other contexts. The project will identify the fundamental determinants of the transport service - health costs nexus. It will establish an estimation equation relating fundamentals to health costs in different contexts of urban economy and geography.*"

The study focuses on the relation between transport emissions and air quality and resulting health impacts in an urban environment. Although the study will include an assessment of the analytical tools and software that can be applied in project analysis, no new tools and software will be developed in this study.

The scope is limited to health implications of transport emissions and omits other effects such as agricultural and forestry losses, as well as soiling and erosion of buildings and construction. The paper should be read as a step towards creating a tool for ex-ante assessments of health effects associated with transport emissions. It should also be noted that the paper suffers from all uncertainties and methodological challenges connoted with valuation and quantification of air pollutions and transport emissions on health.

1 Introduction

1.1 Background of the study

One of the main themes of the Daytona conference on performance measurement of transport projects and policies was the inclusion of environmental and social co-benefits in defining the outcomes of transport interventions.

In FY11 a flagship report on "Comprehensive Assessment of Transport Policies and Projects" will be produced focusing on the inclusion of emission reduction and savings in health costs as one outcome of transport policies.

The extension will translate the conference results into an ex ante evaluation instrument to account for the co-benefits of transport initiatives.

1.2 Objectives and scope

Against this background, the World Bank has approached Ecorys to make an inventory on the impacts of a reduction in transport emissions on savings in health costs.

The Tor states the following objective: "*The project will lead to an estimation approach of how to transfer results on the health impacts of transport into other contexts. The project will identify the fundamental determinants of the transport service - health costs nexus. It will establish an estimation equation relating fundamentals to health costs in different contexts of urban economy and geography.*"

The paper aims to provide the basis for an estimation equation and will focus on the relation between transport emissions and air quality in an urban environment. This is directly related to the fact that most health impacts are related to local air quality levels.

Obviously a complete assessment of the health impacts of air pollution in a specific situation would entail extensive modeling exercises, which is beyond the scope of this paper. The aim of the paper is to create an understanding of the factors that play a role in the causal relation between transport emissions and health effects and provides approximations from existing studies that can be used to assess these health impacts and related costs.

The paper focuses on the translation of air pollution levels into health impacts and health costs. It does not deal with the assessment of air pollution levels itself.

The overall structure of the paper follows the two key steps and elaborate on the their inherent challenges: (1) identify and measure

the health effects of air pollution, (2) to estimate the costs of the health effects.

1.3 Structure of the paper

The paper is divided into four chapters:

- **Introduction** – outlining the goals and background to the project;
- **The impacts of air pollution from traffic on health** – reviews the literature of which transport emissions and air pollutant that are damaging to health. It also sketch the most common diseases associated with air pollution. Finally it talks about the transferability of research carried out in Europe and the US to other parts of the world;
- **The valuation of health impacts** – reviews the literature on how to value the impacts on health associated with air pollution;
- **Synthesis** – brings the paper together and presents the conclusions and moves towards answering the research objective.

2 THE IMPACT OF AIR POLLUTION FROM TRANSPORT ON HEALTH

Exposure to air pollution from transport in the urban environment is believed to have significant negative impacts on human health. Particulate matter (PM) appears to be particularly damaging to health[1] but also Nitrogen Oxides (NO_x) and Sulphur Dioxides (SO_2) have been positively and significantly associated with all-cause mortality.[2] [3] [4]

Transport is the main culprit behind urban air pollution. Combustion of (fossil) fuels releases emissions in the forms of solid particles, liquid droplets, or gases and pollution separated in primary and secondary pollutants. Primary air pollutants are directly emitted (e.g. SO_2, CO_2), whereas secondary pollutants are formed in the air when primary pollutants react or interact (e.g. ground level ozone). Figure 1 (see next page) provides a short overview of the main pollutants.

The impact of air pollution on individuals is determined by type of pollutant, concentration in the air, presence of other pollutants, and length of exposure time.[5] There are also a number of *confounding (or situational) factors* such as age, gender, geographical position and habits. The probability of being exposed to air pollution, for example, is heavily dependent on where the individual is situated. Social-Economic Status (SES), for example, play a role in impact of air pollution on health even in highly developed cities.[6] Table 1 provides a few examples of confounding factors and their variations:

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[1] Mills, N. Et al (2008) *Adverse cardiovascular effects of air pollution*. Nature Clinical Practice: Cardiovascular Medicine.

[2] Stieb, D., S. Judek, R. Burnett (2002) *Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season*. J. Air & Waste Manage. Assoc. 52:470-484

[3] Burnett, R., J. Brook, T. Dann, C. Delocla, O. Philips, S. Cakmak, R. Vincent, M. S. Goldberg, D. Krewski (2002) *Association between pariculate and gas phase components of urban air pollution and daily mortality in eight Canadian cities*. Inhalation Toxicology, 2000, Vol. 12, No. s4 : Pages 15-39

[4] Kunzli, N. Et al (2005) *Ambient Air Pollution and Atherosclerosis in Los Angeles*. Environ Health Perspect. 2005 February; 113(2): 201–206.

[5] Mishra, V. (2003) *Health effects of air pollution*. Background paper for Population-Environment Research Network (PERN) Cyberseminar.

[6] Forastiere, F. Et al (2006) *Socioeconomic status, particulate air pollution, and daily mortality: Differential exposure or differential susceptibility.* American Journal of Industrial Medicine Special Issue: Ethical Considerations and Future Challenges In Occupational and Environmental Health Volume 50, Issue 3, pages 208–216, March 2007

TABLE 1: CONFOUNDING (SITUATIONAL) FACTORS: A FEW EXAMPLES

Factors	Variations
Developed vs. developing world	Air pollution in most urban areas is a cause of concern. However, in developed countries air pollution is addressed by setting limit values which restrain air pollution to a certain extent. On the other hand in many developing nations, regulatory limit values are absent, thus leading to increasingly high levels of air pollution, especially in cities and countries that face high economic growth.
Urban vs. rural	In cities the concentrations of air pollution tend to be higher due to higher traffic densities and related emissions. In developing countries, where fossil fuels are used for cooking, also indoor pollution is a significant source of increased mortality.
Individual resilience	Poor, malnourished, very young, old people, as well as people with pre-existing conditions are particularly susceptible to air pollution.

In the developed world many governments regulate air pollution and introduce limit values which has reduced the emissions. Nevertheless, a survey on Europe concluded that air pollution contributes to a 6 % increase in mortality of which half can be attributed to traffic along with 25,000 new cases of chronic bronchitis in adults, 290,000 episodes of bronchitis in children, more than 0,5 million asthma attacks; and more than 16 million person days of restricted activities.7 In the developing world, and in particular in regions such as South East Asia, fast growing economies rapidly cause an increase in air pollution. The WHO estimated that urban air pollution contributed to 800,000 deaths annually and costs 4.6 million lost-life years in 2002. The burden of disease is heavily tilted towards Asian countries where approximately two thirds of the costs occur.8 This causes an imperative to investigate the policy options for reducing the emissions in these countries and assess the health impacts of these options. Yet, the great majority of studies and research have been made in Europe and the US. Can these results, and in particular the Dose Response Functions simply be transferred to other parts of the world?

[7] N Künzli, R Kaiser, S Medina, M Studnicka, O Chanel, P Filliger, M Herry, F Horak Jr, V Puybonnieux-Texier, P Quénel, J Schneider, R Seethaler, J-C Vergnaud, H Sommer (2000) *Public-health impact of outdoor and traffic-related air pollution: a European assessment*. The Lancet • Vol 356 • September 2, 2000

[8] HEI (2004) *Health Effects of Outdoor Air Pollution in Developing Countries of Asia*. Health Effects Institute, Special Report 15. April 2004

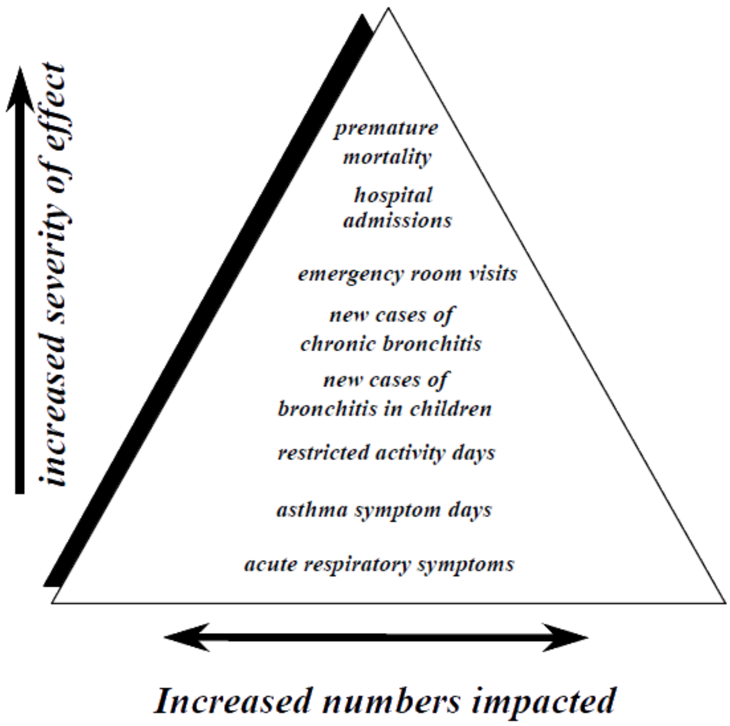
FIGURE 1: OVERVIEW POLLUTANTS

Most damaging pollutants from transport to human health (Source: WHO)	
Particulate Matter (PM)	Ozone (O3)
PM consists of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air. The particles are identified according to their aerodynamic diameter, as either PM10 (particles with an aerodynamic diameter smaller than 10 µm) or PM2.5 (aerodynamic diameter smaller than 2.5 µm). The latter are more dangerous since, when inhaled, they may reach the peripheral regions of the bronchioles, and interfere with gas exchange inside the lungs. Chronic exposure to particles contributes to the risk of developing cardiovascular and respiratory diseases, as well as of lung cancer.	Ozone at ground level is one of the major constituents of photochemical smog. It is formed by the reaction with sunlight (photochemical reaction) of pollutants such as nitrogen oxides (NOx) from vehicle and industry emissions and volatile organic compounds (VOCs) emitted by vehicles, solvents and industry. Excessive ozone in the air can have a marked effect on human health. It can cause breathing problems, trigger asthma, reduce lung function and cause lung diseases.
NOx	SO2
Epidemiological studies have shown that symptoms of bronchitis in asthmatic children increase in association with long-term exposure to NO2. Reduced lung function growth is also linked to NO2 at concentrations currently measured (or observed) in cities of Europe and North America. NO2 is also the main source of nitrate aerosols, which form an important fraction of PM2.5 and, in the presence of ultraviolet light, of ozone.	SO2 can affect the respiratory system and the functions of the lungs, and causes irritation of the eyes. Inflammation of the respiratory tract causes coughing, mucus secretion, aggravation of asthma and chronic bronchitis and makes people more prone to infections of the respiratory tract. Hospital admissions for cardiac disease and mortality increase on days with higher SO2 levels. When SO2 combines with water, it forms sulfuric acid; this is the main component of acid rain which is a cause of deforestation.

2.1 WHAT CONDITIONS ARE INDUCED AND/OR EXACERBATED BY TRANSPORT-RELATED AIR POLLUTION

The health effects of air pollution can be depicted in the "health pyramid". Many of the health effects related to air pollution are of limited severity. However, these effects, comprising diseases such as asthma and acute respiratory symptoms affect a large part of the population, and lead to a reduced quality of life. On the other hand a number of more severe impacts are observed, which do have a higher health impact but affect less people.

FIGURE 2: HEALTH EFFECTS PYRAMID



Source: Health Canada, 2004

Four groups of major health effects/diseases can be distinguished:

- cardiovascular diseases (angina, myocardial infection, hear failure, coronary heart disease)[9];
- respiratory diseases (bronchitis, acute respiratory illness, increased asthma; and. reduced lung functions);
- Lung cancer; and,
- Other effects (fertility problems, pregnancy) [10]

2.1.1 CARDIOVASCULAR EFFECTS

There is substantial evidence that supports a relationship between vehicle emissions levels (in particular PM) and cardiovascular disease. [11] In a study on PM levels influence on postmenopausal women in the US, a significant relationship between concentration levels and cardiovascular events, and cardiovascular related deaths.[12] In a similar

[9] Mills, N. Et al (2008) Adverse cardiovascular effects of air pollution. Nature Clinical Practice: Cardiovascular Medicine.

[10] World Bank (2003*) Health Impacts of Outdoor Air Pollution*. Briefing note part of the South Asia program on urban air quality management. South Asian Urban Air Quality Management Briefing, no. 11.

[11] See: Mills, N. Et al (2008) Adverse cardiovascular effects of air pollution. Nature Clinical Practice: Cardiovascular Medicine. AND Arden Pope III, C. and D. W. Dockery (2006*) Health Effects of Fine Particulate Air Pollution: Lines that Connect*. J. Air & Waste Manage. Assoc. 56:709–742

[12] Miller, K. A. (2007) Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women. The new england journal of medicine. Feb 1, 2007, Vol 356. No. 5.

vein, cardiovascular and stroke mortality rates have been associated with both ambient pollution at place of residence as well as residential proximity to traffic. Depending on the method of measurement, the increased risk can be substantial.[13]

Recent studies consider non-fatal cardiovascular outcomes like acute myocardial infarction (AMI) and have found an association with exposure to vehicle emissions, particularly as a result of long-term exposure to $PM_{2.5}$ and/or close residential proximity to busy roads. Short-term exposures have also been shown to be associated with ischemic effects and induce AMI.[14] A case-crossover study of 772 individuals in Boston found that elevated concentrations of $PM_{2.5}$ were associated with an increased risk of AMI within a few hours and one day following exposure.[15] Another study of 12,865 individuals in Utah found a similar effect for both AMI and unstable angina, and that this effect was worse for patients with underlying coronary artery diseases.[16] The specific toxicants most commonly associated with these effects are PMs, although there is also evidence of an adverse influence of CO and SO_2.[17] Increased levels of CO and NO_2 have also been implicated in increased incidence of emergency department visits for stroke.[18] It has been suggested that it is the strong association between air pollution and ischemic heart disease that drives the cardiopulmonary association with air pollution.[19]

2.1.2 Respiratory diseases

Research on respiratory diseases, make up for the lion's part of studies on the relation between transport emissions and health. The adverse effects on respiratory systems range from acute symptoms such as coughing and wheezing, to more chronic conditions such as asthma and chronic obstructive pulmonary disease (COPD), which includes chronic bronchitis and emphysema.

Many studies on the effect of vehicle emissions and respiratory health consider short term changes in exposure and daily symptoms in the study population, particularly in exacerbating symptoms in asthmatics as well as inducing asthma in otherwise healthy individuals. The

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[13] Jerret, M. et al. (2005) Spatial Analysis of Air Pollution and Mortality in Los Angeles. Epidemiology: November 2005 - Volume 16 - Issue 6 - pp 727-736

[14] Peters A, von Klot S, Heier M. et al (2004) *Exposure to traffic and the onset of myocardial infarction*. N Engl J Med 2004. 3511721–1730.1730

[15] Peters A. et al (2001) *Increased Particulate Air Pollution and the Triggering of Myocardial Infarction*. American Heart Association

[16] Arden Pope III, C. and D. W. Dockery (2006) Health Effects of Fine Particulate Air Pollution: Lines that Connect. J. Air & Waste Manage. Assoc. 56:709–742

[17] Fung et al (2005) Association between Air Pollution and Multiple Respiratory Hospitalizations among the Elderly in Vancouver, Canada. 2006, Vol. 18, No. 13 , Pages 1005-1011

[18] Villeneuve et al. (2005) Associations between outdoor air pollution and emergency department visits for stroke in Edmonton, Canada. European Journal of Epidemiology

[19] Jerret, M. et al. (2005) Spatial Analysis of Air Pollution and Mortality in Los Angeles. Epidemiology: November 2005 - Volume 16 - Issue 6 - pp 727-736

Children's Health Study in southern California found that asthma and wheeze were strongly associated with residential proximity to a major road[20], a finding that is consistent with many other studies of children.[21] Interestingly, similar effects have been found in populations of infants and very young children[22], as well as adolescents.

[20] McConnell et al. (2006) *Traffic, Susceptibility, and Childhood Asthma.* Environ Health Perspect: May 2006 - Volume 114 - Issue 5 - pp 766-772

[21] T.J. Oyana and P.A. Rivers (2005) *Geographic Variations of Childhood Asthma Hospitalization and Outpatient Visits and Proximity to Ambient Pollution Sources at A U.S.-Canada Border Crossing*, International Journal of Health Geographies, Volume 4 – Issue 14

[22] Ryan PH, LeMasters G, Biagini J, Bernstein D, Grinshpun SA, Shukla R, et al. 2005. *Is it traffic type, volume, or distance?*

Wheezing in infants living near truck and bus traffic. J Allergy Clin Immunol 116(2):279–284.

Another respiratory effect that has been associated with exposure to vehicle emissions is reduced lung function. While the magnitude of the effect reported is often small, there is consistency in these findings. Most studies investigate the effects in children; however, of particular interest is a study of exposure to NO_2 in healthy university students in Korea.[23] Exposure levels were found to be significantly associated with proximity of residence to main roads, and this exposure was associated with a reduction in lung function.

2.1.3 CANCER

There is an increasing body of literature that suggests that vehicle emissions are also associated with the development of cancer, particularly lung cancer, although other types have been implicated. A large recently published study in Europe of 4000 individuals studied the relationship between lung cancer and vehicle-related pollution.[24] Exposure to air pollution was measured as proximity of residence to heavy traffic roads. Additionally, exposure to NO_2, PM_{10}, and SO_2 was assessed from monitoring stations. The findings from this study indicate that residence in close proximity to heavy-traffic roads, or exposure to NO_2 increases the risk of lung cancer. This is consistent with studies conducted in Oslo and Stockholm that found a similar relationship between increased risk of lung cancer and levels of traffic-related NO_2. This effect has also been demonstrated in studies of fine PM and SO_2 and exposure to diesel exhaust.

The effect of vehicle emissions on childhood cancers, particularly leukemia, is also of concern. While the research is this area is somewhat limited, there is some indication that vehicle emissions are associated with an increased risk of childhood cancer as indicated by residential proximity to busy streets.[25]

Information on the relationship between vehicle emissions and other types of cancers is sparse. However, one recent study suggests that early life exposure to traffic emissions (which include PAHs) may be associated with breast cancer in women.[26] Specifically, higher exposure to traffic-related emissions at menarche was associated with pre-menopausal breast cancer, while emissions exposure at the time of a woman's first childbirth was associated with postmenopausal

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23 Hong et al. (2005) *Exposure to air pollution and pulmonary function in university students*. International Archives of Occupational and Environmental Health. Volume 78 – Issue 2 – pp. 132-138

24 Vineis P, Hoek G, Krzyzanowski M, et al. (2006) *Air pollution risk of lung cancer in a prospective study inEurope*. International Journal of Cancer - volume 119 - pp. 169–174

25 Pearson RL, Wachtel H, Ebi KL. (2000) *Distance weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers*. J Air Waste Manag Assoc - vol. 50 – issue 2 - pp. 175-180

26 Nie et al. (2007) *Exposure to traffic emissions throughout life and risk of breast cancer: the Western New York Exposures and Breast Cancer (WEB) study*. Cancer, Causes and Control - Volume 18 – issue – pp. 947-55

breast cancer. Lastly, a study in Finland of individuals exposed to diesel and gasoline exhaust occupationally found an association between ovarian cancer and diesel exhaust.[27]

Other health effects

There is evidence that suggests that exposure to traffic pollutants affects fertility in men. An Italian study evaluated sperm quality in men employed at highway tollgates.[28] Total mortality, forward progression, functional tests, and sperm kinetics were significantly lower in tollgate employees versus controls. In particular, NO and lead were implicated as toxins with adverse effects.[29]

There is also emerging evidence that vehicle-related emissions are associated with an increased risk of adverse pregnancy outcomes. Several studies have reported an association with low birth weight in infants and maternal exposure to emissions during pregnancy.[30] It has also been suggested that there is an association with preterm births and intrauterine growth retardation, but these studies are less consistent. Finally, there have been a few studies which suggest an increased risk in these infants of sudden infant death syndrome and birth defects like congenital heart defects but further research is needed to confirm these findings.[31]

Summary of impact on health

There is clearly a broad range of health conditions which can be attributed to long and short term exposure to air pollution. Moreover, the health effects lead to socio-economic unwanted situations when people are forced to stay home from school or remain absent from work. To sum up, the WHO makes the following overview of health effects of air pollution:

TABLE 1: LIST OF LONG AND SHORT-TERM EFFECTS.

Effects from ***short-term*** exposure
• Daily mortality
• Respiratory and cardiovascular hospital admissions
• Respiratory and cardiovascular emergency department visits
• Respiratory and cardiovascular primary care visits

27 Guo et al. (2004) *Risk of esophageal, ovarian, testicular, kidney and bladder cancers and leukemia among finnish workers exposed to diesel or gasoline engine exhaust.* International Journal of Cancer. Volume 111 – issue 2 – pp. 286 - 292

28 De la Rosa et al. (2003) *Traffic pollutants affect fertility in men.* Human Reproduction – volume 18 – issue 5 – pp. 1055-1061

29 Idid.

30 Bell et al. (2010) *Prenatal Exposure to Fine Particulate Matter and Birth Weight Variations by Particulate Constituents and Sources.* Epidemiology – volume 21 – issue 6 – pp. 884-891

31 Dales et al. (2004) *Air Pollution and Sudden Infant Death Syndrome.* American Academic Pediatrics – volume 113 – issue 6 – pp. 628 - 631

- Use of respiratory and cardiovascular medications
- Days of restricted activity
- Work absenteeism
- School absenteeism
- Acute symptoms (wheezing, coughing, phlegm production, respiratory infections)
- Physiological changes (e.g. lung function)

Effects from *long-term* exposure

- Mortality due to cardiovascular and respiratory disease
- Chronic respiratory disease incidence and prevalence (asthma, COPD, chronic pathological changes)
- Chronic changes in physiologic functions
- Lung cancer
- Chronic cardiovascular disease
- Intrauterine growth restriction (low birth weight at term, intrauterine growth retardation, small for gestational age)

Source: WHO, 2005

The negative impacts of air pollution and transport emissions on health are clear. To provide sound policy advice however, we need to understand which emissions that causes adverse health impacts, and at what dose. The next sections continue the discussion on what emissions leads to what disease, and at what dosage.

2.2 How to calculate the health impacts of air pollution?

That there is a link between air pollution from traffic and adverse health impact is clear, however, the exact causal chain is yet to be defined. The impacts of *individual* pollutants in *local contexts* on specific health conditions are only partly unveiled and epidemiological studies reveal that ***effects observed from air pollution often cannot be attributed to a single but rather a mix of pollutants***. Fine PM and ozone, for example, increase the risk of mortality and respiratory ailments, whereas nitrogen dioxides, ozone and PM increase the risk of allergic reactions. [32] The reason for taking this more cautious approach, however, seems to be linked methodological, measurement and data challenges to attribute a specific pollutant to a specific disease. [33] Moreover, due to correlations between pollutants, to calculate the impacts of emissions pollutant-by-pollutant would probably lead to large overestimations.[34] Nevertheless, most appraisal methods assume some linearity between a single pollutant and a health effect.

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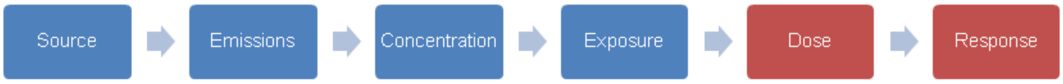
[32] Krzyzanowski, M. et al (2005) *Health effects of transport-related air pollution*. WHO Regional office for Europe,

[33] European Commission (2005) *ExternE - Externalities of Energy Methodology 2005 Update*. EUR21951

[34] N Künzli, R Kaiser, S Medina, M Studnicka, O Chanel, P Filliger, M Herry, F Horak Jr, V Puybonnieux-Texier, P Quénel, J Schneider, R Seethaler, J-C Vergnaud, H Sommer (2000) Public-health impact of outdoor and traffic-related air pollution: a European assessment. The Lancet • Vol 356 • September 2, 2000

Even if the causal chain link in terms of *dose-response*[35] between air pollution and health is not always unilateral and fully quantifiable[36], the "environmental pathway" of emission looks as follows[37]:



Road traffic **emissions** from both combustion and friction are generally considered the main **source** of air pollution in gaseous state and PM in different shapes and composition.[38] The emissions lead to increased **concentrations** of dangerous pollutants such as PM. Concentration levels from transport emissions are strongly influenced by the dispersion patterns of gaseous (partly related to physical conditions of a location) and ambient air quality levels. The risk of negative impact on health depends to a large extent on the length and intensity of **exposure** to the pollutant which leads to a **dose** entering the individual human body. The **response** to the dose largely depends on several factors such as type of pollutant, individual susceptibility and habits. Finally the overall health impacts are obviously related to the number of people affected, which will be higher in an urban environment than in rural areas.

2.2.1 THE HEALTH-AIR POLLUTION EQUATION

The aim with a general equation is to show what health effects one unit increase of a pollutant. At the most general level, three factors are involved: (1) the population exposed, (2) the pollutant to be investigated, and (3) the Dose Response function.

- The population needs to be established due to differences in susceptibility and durance of exposure. The some groups of people are particularly susceptible to air pollution such as elderly, asthmatic and small children. The second large factor with regard to population is exposure time.

- Pollutants have different impacts on health. PM is expected to be the most influential but also ozone, NOx and SO2 are damaging to health.

[35] Dose is defined as the concentration that reaches the target. See: World Bank (2003*) The Science of Health Impacts of Particulate Matter*. Briefing note part of the South Asia program on urban air quality management. South Asian Urban Air Quality Management Briefing, no. 13

[36] Mishra, V. (2003) *Health effects of air pollution*. Background paper for Population-Environment Research Network (PERN) Cyberseminar.

[37] Concentrations and exposure is separated since high concentrations does not indicate high exposure. High exposure is only possible if the person stays in an area with high concentration and the health effects are dependent of on the characteristics of the dose. See: WHO (2005) *Air quality guidelines: Global update 2005*. WHO Regional office for Europe, Denmark.

[38] Krzyzanowski, M. et al (2005) Health effects of transport-related air pollution. WHO Regional office for Europe,

- To assess the impact of increased or decreased emission's levels on morbidity (sickness) and mortality (death), policy analysts use regression analysis to calculate the coefficient for change. These coefficients are commonly known as Dose Response Functions (DRF) (also called Concentration Response Functions (CRF), or impact factors). Essentially, the DRFs determines the augmented health risk in % which follows one unit of increase in emissions . DRFs are derived from epidemiological studies and are defined in as linear or normal[39] and ***are central to quantify damages to human health from air pollution***. Without entering into the nitty-gritty of calculating DRFs, it is safe to say that much uncertainty remains. DRFs should optimally be put into a local context and weighted against a number of confounding factors. Moreover, questions still remains whether to use linear or non-linear functions. In a linear model, the response to a dose increase in a linear pattern from point zero. In a non-linear model, a threshold is introduced for where a lower level of air pollution does not result in negative health effects.

To formalise the equation we rely on Ostro[40] who developed the following relationship to estimate health impacts:

$$dH_i = b_i * POP_i * dA$$

dH_i	=	change in population risk of health effect i;
b_i	=	slope from the dose-response curve for health impact i;
POP_i	=	population at risk of health effect i;
dA	=	change in ambient air pollutant under consideration.

To estimate the economic value of the health impacts, valuation techniques normally based on Willingness To Pay (WTP) or Cost of Illness (COI) are used and adds the V_i to the equation. The function of social value then looks like:

$$dT = \sum V_i dH_i$$

This is further elaborated in the next chapter.

Furthermore, when breaking down the numbers one should distinguish between morbidity and mortality.[41] Derived from the original formula, the mortality function looks like follows:

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39 Judek, S. and D. Stieb (2004) *AQBAT - Estimating Health Impacts for Changes in Canada's Air Quality*. Health Canada (http://www.bc.lung.ca/mediaroom/news_releases/documents/AQBATEstimatingHealthImpactsforChangesinCanadasAirQuality.pdf)

40 Ostro, B. (1994). *Estimating Health Effects of Air Pollutants: A Methodology with an Application to Jakarta*. Policy Research Working Paper 1301. World Bank, Washington, D.C.

$$\Delta Mortality = b * \Delta PM_{10} * 0{,}01 * crude\ mortality\ rate * POP$$

Where *b* signifies the mortality coefficient. Mortality coefficients for PM_{10} has been suggested by Ostro, to add a rough sensitivity analysis in the transfer of DRFs. He suggests three levels: low, medium and high scenarios with corresponding coefficients.[42]

TABLE 2: LIST OF COEFFICIENTS FOR LOWER, CENTRAL AND UPPER BOUNDS.

Lower coefficient	Central coefficient	Upper coefficient
0.062	0.096	0.13

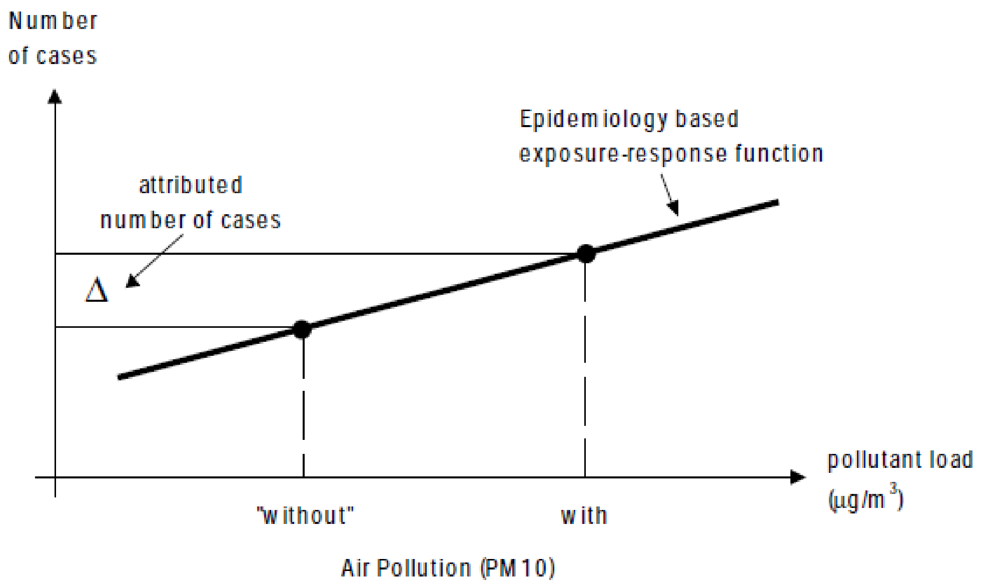
For effects on morbidity due to changes in the air pollutant concentrations the formula looks like follows:

$$\Delta Morbidity = c_i * \Delta PM_{10} * POP$$

Where c_i signifies the morbidity coefficient

In the original function, the b_i signifies the DRF.

FIGURE 3: RELATION BETWEEN AIR POLLUTION EXPOSURE AND CASES OF DISEASE.



With regards to non-linearity, recent studies indicate that there appears to be no threshold for when air pollution begin to have negative health effects. Studies have shown that negative effects have been observed even when air quality has been in line with or better than national or regional limit values.[43] In a study on asthma

[41] The morbidity and mortality formulas are adopted from: Quah, E. and Boon, T. L. (2003) *The economic cost of particulate air pollution on health in Singapore*. Journal of Asian Economics 14 (2003) 73–90.

[42] Yaduma, N., M. Kortelainen, A. Wossink (2011) *The Mortality and Economic Costs of Particulate Air Pollution in Developing Countries: A Nigerian Investigation*. Paper presented at European Association of Environmental and Resource Economists 18th Annual Conference 29 June - 2 July 2011, Rome

[43] Mills, N. Et al (2008) Adverse cardiovascular effects of air pollution. Nature Clinical Practice: Cardiovascular Medicine.

exacerbation on children in Singapore, for example, the authors established a positive correlation between increase in air pollution emergency room (ER) visits, despite the pollution levels being below WHO guidelines. For atmospheric SO_2 levels, an additional 2.9 ER visits for every 20 μg/m^3 increase was observed on days when levels were above 68 μg/m^3. And, for total suspended particles (TSP) an increase of 5.80 ER visits for every 20 μg/m^3 increase in daily atmospheric levels was observed on days with levels above 73 μg/m^3.[44] Yet, it cannot be ruled out that some pollutants have lower thresholds where health effects are negligible.

2.2.2 Local conditions in the developing world

The problem with transferring results obtained in the developed world directly to the developed world has been discussed extensively over the last decades. Researchers are frequently hindered by the time and resources needed to carry out larger cohort or time-series studies in developing countries to obtain context specific dose-response functions. Moreover, in many developing countries there are large problems in data-availability in terms of health care, demography, and pollutions level.

Already in 1998, the WB noted the three main challenges in applying results from developed countries to developing countries: (1) to use epidemiological studies for PMs in combination with measurements for the country in questions, (2) to use a disease-specific mortality profile, i.e. people die from different things in different countries, in some countries cardiovascular diseases for example, are more common than in others, and (3) to note the age patterns in the area investigated.[45] In all three cases however data availability is often scarce and collection of primary data too expensive. Fourteen years later, the three challenges remain highly relevant.

In policy analysis, when original data collection is deemed unfeasible due to resource constraints, it is common to use a Benefit Transfer Approach (BTA). It is normally deployed in environmental and health economics and employs estimates from one or more previous studies to predict the benefits, health and economic, from pollution mitigation at a different point in space, time, or both.[46] In this sense, the DRF functions developed in a European and US contexts could be adjusted to the local conditions and confounding factors. However, even if air pollution manifests itself similarly in many aspects globally; the developing world differs from Europe and North America in the nature of its air pollution, the conditions and magnitude of exposures,

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44 Chew, F.T, et al. (1999) *Association of ambient air-pollution levels with acute asthma exacerbation among children in Singapore.* Allergy. 1999 Apr;54(4):320-9.

45 World Bank (1998) The Effects of Pollution on Health:The Economic Toll. *Pollution Prevention and Abatement Handbook*, WORLD BANK GROUP, July 1998.

46 Yaduma, N., M. Kortelainen, A. Wossink (2011) *The Mortality and Economic Costs of Particulate Air Pollution in Developing Countries: A Nigerian Investigation*. Paper presented at European Association of Environmental and Resource Economists 18th Annual Conference 29 June - 2 July 2011, Rome

and confounding factors, including the level of health care.[47] Hence, the transfer of impact factors should still be approached with caution. The following story provides a telling example: "*cardiovascular and respiratory diseases have been reported to account for a quarter of non-trauma deaths in Delhi, compared to half in the United States. If the cardiopulmonary-specific CR function from a recent study were transferred to Delhi, all-cause mortality would increase by 1.5 percent when $PM_{2.5}$ exposure is increased by 10 µg/m^3, compared to a 4 percent increase if the all-cause CR function from the same study were applied*".[48]

In earlier reports on air pollution in developing countries, the WB has concluded that in absence of better data and local studies, the impact factors of Europe and the US represents best available data.[49] A similar conclusion is drawn by the European HEATCO project which concludes that: "*project related emissions should be calculated using national emission factors; if such factors are not available, emission factors from international sources can be applied, taking into account national vehicle fleet compositions as far as possible.*" [50]

In conclusion, to carry out large epidemiological studies in developing countries to retrieve locally substantiated DRFs, is in most cases impossible. Firstly, the levels of air pollution are not known, secondly, the clinical data is not available. Therefore, the use of BTA is preferable to establish DRFs to calculate the health benefits of emissions reductions.

2.2.3 Which DRFs to use

To determine the DRF is crucial in order to monetise the health costs in a transport or infrastructure project. The challenge is to determine the size of the DRF that applies to the local context, in particular in developing countries. The most reliable way of determining a DRF is to conduct epidemiological studies in a selected area. To investigate both acute and chronic effects there needs to be sufficient cross-sectional data for individuals and cover at least a ten year period.[51]

In Europe and the US, DRFs are well researched and already used extensively. In Europe, the HEATCO project (2006) concluded that

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47 HEI (2004) *Health Effects of Outdoor Air Pollution in Developing Countries of Asia*. Health Effects Institute, Special Report 15. April 2004

48 World Bank (2003) *Health Impacts of Outdoor Air Pollution*. South Asia Urban Air Quality Management Briefing Note No. 11. Briefing developed under the ESMAP programme.

49 World Bank (2003) *Health Impacts of Outdoor Air Pollution*. South Asia Urban Air Quality Management Briefing Note No. 11. Briefing developed under the ESMAP programme.

50 HEATCO (2006) *Developing Harmonised European Approaches for Transport Costing and Project.* Assessment (HEATCO). Deliverable D5: Proposal for Harmonised Guidelines

51 Yaduma, N., M. Kortelainen, A. Wossink (2011) *The Mortality and Economic Costs of Particulate Air Pollution in Developing Countries: A Nigerian Investigation*. Paper presented at European Association of Environmental and Resource Economists 18th Annual Conference 29 June - 2 July 2011, Rome

impacts from emitting 1000 tonnes of any pollutants demonstrates the large variety in impacts in lost life expectancy across European Union member states. On PMs the emissions on ground levels are between 5 - 10 times higher depending demographic differences and urbanization. Other aspects such as chemical composition and wind dispersion play integral roles. Moreover, with regards to PM_{10} the European project Air Pollution on Health: European Approach (APHEA) calculated that a 50 µg/m^3 increase in concentration levels would result in 2.1% increase in total mortality in Western European cities.[52] In the US, the dose-response function of elevated PM has been studied extensively. Arden Pope III et al covered all approximately 500,000 adults in all metropolitan areas of the US and concluded that each 10 - µg/m^3 increase in $PM_{2.5}$ is associated with all-cause, cardiopulmonary and lunch cancer mortality with 4%, 6% and 8% respectively.[53]

In Canada, Health Canada[54] employs the Air Quality Benefits Assessment Tool (AQBAT) which of course also use Concentration Response Functions (CRFs) to assess the impacts of air pollutions. The health outcome triggers are changes in the ambient concentration of NO_X, Ozone, SO_2, and $PM_{2.5}$ by census division provided by the ReFSoRT model and adjusted for exposure times and seasonality. The changes in concentration are then used to calculate changes in the 12 health endpoints for four different ambient air quality concentrations.

TABLE 3: HEALTH ENDPOINTS FROM THE AQBAT STUDY.

Health Endpoints	Contributing Pollutants and Averaging Times
Acute Exposure Mortality	24-hr NO_X, 1- hr O_3, 24-hr SO_2
Acute Respiratory Symptom Days	1-hr O_3 (May-Sep), 24-hr $PM_{2.5}$
Adult Chronic Bronchitis Cases	24-hr $PM_{2.5}$
Asthma Symptom Days	1-hr O_3 (May-Sep), 24-hr $PM_{2.5}$
Cardiac Emergency Room Visits	24-hr $PM_{2.5}$
Child Acute Bronchitis Episodes	24-hr $PM_{2.5}$
Chronic Exposure Mortality	24-hr $PM_{2.5}$
Minor Restricted Activity Days	1-hr O_3 (May-Sep)
Respiratory Emergency Room Visits	1-hr O_3 (May-Sep), 24-hr $PM_{2.5}$
Cardiac Hospital Admissions	24-hr $PM_{2.5}$
Respiratory Hospital Admissions	1-hr O_3 (May-Sep), 24-hr $PM_{2.5}$
Restricted Activity Days	24-hr $PM_{2.5}$

Source: Health Canada

[52] Katsouyanni, K., Zmirou, D., Spix, C., Sunyer, J., Schouten, J. P., Ponka, A., Anderson, H. R., Le Moullec, Y., Wojtyniak, B., Vigotti, M. A., & Bacharova, L. (1996) *Short-term effects of air pollution on health: a European approach using epidemiological time-series data*. Paper presented at the Second Colloquium on Particulate Air Pollution and Health, Park City, UT, USA.

[53] Arden Pope III, C. Et al (2002) *Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution*. Journal of American Medical Association, 2002;287(9):1132-1141

[54] http://www.hc-sc.gc.ca/

To assess the health effects in developing countries, we have to use BTA as described in an earlier section. Previous studies on health effects in developing countries have largely relied on DRFs developed by Ostro[55] (see for example Qua and Boon (2003) on a case in Singapore, and Zhou and Tol (2005) in Tianjin, China). On behalf of the WB, in 1994 Ostro developed DRFs based on analyses from the UK, the US and the US EPA's in particular. The author then adopted a BTA and applied the functions on Jakarta in Indonesia. Through a detailed literature review Ostro established the following overview of DRFs:

TABLE 4: OSTRO (1994) LIST OF DOSE-RESPONSE FUNCTIONS.

	Pollutant unit				
Outcome[56]	**PM_{10}** (10 µg/m^3)	**SO_2** (10 µg/m^3)	**Ozone** (pphm)	**Lead** (1 µg/m^3)	**NO_2** (pphm)
Premature mortality (% change)	0.96	0.48			
Premature mortality/100,000	6.72				
RHA/100,000	12.0		7.70		
ERV/100,000	235.4				
RAD/person	0.575				
LRI/child	0.016				
Asthma symptoms/asthmatic	0.326		0.68		
Respiratory symptoms/persons	1.83		0.55		
Chronic bronchitis/100,000	61.2				
MRAD/person			0.34		
Respiratory symptoms/1,000 children		0.18			
Respiratory symptoms/adult		0.10			0.10
Eye irritation/person			0.266		
Hypertension/100,000 adult					
Coronary disease/100,000 adult males				34.0	
Premature mortality/100,000 adult males				35.0	
IQ decrement (100,000) children				97.500	

Source: Ostro (1994) Estimating Health Effects of Air Pollutants: A Methodology with an Application to Jakarta. Policy Research Working Paper 1301. World Bank, Washington, D.C

Since the Ostro overview from 1994, there has been a series of studies which elaborates on the DRFs. In 2008 the WHO European Regional offices carried out a review of the methods used for economic valuation of transport-related health effects. It concludes that PM2.5 and black smoke indeed are the best indicators for measuring health effects from transport related emissions. Moreover, where PM2.5

[55] Ostro, B. (1994). *Estimating Health Effects of Air Pollutants: A Methodology with an Application to Jakarta*. Policy Research Working Paper 1301. World Bank, Washington, D.C.

[56] RHA = Respiratory hospital admissions; ERV = Emergency room visits; RAD = Restricted activity days; LRI = lower respiratory illness; MRAD = Minor restricted activity days; PPHM = Parts per hundred million

data is not available, the study suggest to use conversions rates from PM10. Finally, in some instances the use of other indicators such as NOx could be pertinent.[57]

The WHO makes an update of Ostro's PM overview and tries to include background rates, i.e. the rate which shows how often something happens in function of a particular disease. When these background rates are missing, WHO has used an impact function, which is a combination of DRFs and background rates. In essence, the impact functions are expressed as number of (new) cases or events per unit (such as 100.000) per unit of pollution per unit of time (day or year). [58]

The table on the next page provides the conclusions from WHO's literature overview.

57 WHO (2008) Economic valuation of transport related health-effects.

58 WHO (2008) Economic valuation of transport related health-effects.

Table 5 : Summary of suggested health end-points, age group and pollutant (in order of proposed priority if more than one health end-point) and suggested relative risk estimates (and 95% confidence intervals (CI)) for a 10 µg/m3 increase in pollutant

Health end-point	Age group	Pollutant	Relative risk estimate / impact function (95% CI)	Sources
Mortality				
Mortality from long-term exposure				
All causes	>30 years	$PM_{2.5}$	1.06 (1.02–1.10)	Pope et al. (2002)
	>30 years	PM_{10}	1.04 (1.03–1.06)	Künzli et al. (2000)
	Infants (1–12 months)	PM_{10}	1.04 (1.02–1.07)	Woodruff et al. (1997)
Cardiopulmonary	>30 years	$PM_{2.5}$	1.08 (1.02–1.14)	Pope et al. (2002); WHO Regional Office for Europe (2006)
Mortality from short-term exposure				
All cause	All ages	PM_{10}	1.006 (1.004–1.008)	Anderson et al. (2004); WHO Regional Office for Europe (2006)
	All ages	Black smoke	1.006 (1.004–1.008)	Anderson et al. (2004)
Cardiovascular	All ages	PM_{10}	1.009 (1.005–1.13)	Anderson et al. (2004); WHO Regional Office for Europe (2006)
	All ages	Black smoke	1.004 (1.002–1.007)	Anderson et al. (2004)
Respiratory	All ages	PM_{10}	1.013 (1.005–1.20)	Anderson et al. (2004); WHO Regional Office for Europe (2006)
	All ages	Black smoke	1.006 (0.998–1.015)	Anderson et al. (2004)
Morbidity				
Hospital admissions				
Respiratory (annual rate)	All ages	PM_{10}	7.03 (3.38–10.30) per 100 000 population[a]	Hurley et al. (2005)
Cardiac (annual rate)	All ages	PM_{10}	4.34 (2.17–6.51) per 100 000 population[b]	Hurley et al. (2005)
Lower respiratory symptoms[c] *including cough*[d] (annual increase)	5–14 years	PM_{10}	1.86 (0.92–2.77) extra symptom-days per year and child[e]	Hurley et al. (2005)
	Adults	PM_{10}	1.30 (0.15–2.43) extra symptom-days per adult with chronic respiratory symptoms (about 30% of the adult population)[f]	Hurley et al. (2005)
Chronic bronchitis[c] (new cases/year)	>27 years	PM_{10}	26.5 (1.9–54.1) per 100 000 population[g]	Hurley et al. (2005)
Restricted activity day[c]	15–64 years	$PM_{2.5}$	902 (792–1013) per 1000 population[h]	Ostro (2006)
Working-loss day[c]	15–64 years	$PM_{2.5}$	207 (176–238) per 1000 population[i]	Hurley et al. (2005)

Source: WHO

When one compares the WHO table with the DRFs used by Ostro it is clear that there are some discrepancies between diseases. For chronic bronchitis, for example, the DRF in Ostro is 61.2 in 100,000 for PM10. The WHO study, on the other hand, quotes Hurley et al (2005) and provides a range between 1.9 – 54.1 with a mean of 26.5. This is considerably lower than Ostro's (central estimate of) 61.5. However, the DRF for WHO study can be lower for several reasons; new evidence has emerged; it focuses on Europe; the parameters for exposed population is limited to above 27 years old; if it indicates new or only exacerbate diseases; etc. In addition, it should be noted that also Ostro recommends using a bandwith taking account of uncertainties in assessing the health impacts.

2.2.4 Recommended values for impacts

The impacts of air pollution on health are increasingly understood. Through epidemiological studies, reliable results on which pollution causes what disease can be achieved. When a full epidemiological study is unfeasible (mainly due to resource and time constraints), Benefit Transfer Approaches can be used to approximate the impacts. The main issues in doing this are:

- **Which health effects (endpoints) are included?** If overall mortality and chronic bronchitis are chosen, for example, there are a range of diseases which are excluded, and thereby the cost of air pollution from traffic runs the risk of being underestimated.
- **Which pollutants are included?** In general PM and black smoke are seen as the best indicators available. However several health effects are related to other emissions such as ozone and NOx. The choice of pollutasnts will depend to a large extent on the available data.
- **What is the time-frame of the exposure?** Short-term effects are generally better understood than long-term effects. Long-term effects are more complex and are potentially far more negative than short-term effects.
- **Which age groups are included?** For many DRFs the exposed populations which are included in the calculations start at above 25 to 30, and sometimes small children. The choice of age groups is also related to the type of health effects that are included in the analysis.

Despite uncertainties, three recommendations and central determinants of health impacts from air pollution can be distilled from the chapter.

First, **the key pollutant to investigate is PM**. To focus on one pollutant simplifies the data analysis and collection of data. Moreover, if one assumes a linear relationship between level of PM and health impacts (which precludes the idea of a threshold) then one could also argue that base-line levels of PM is not needed in the case study.

Second, it is clear that BTA has its drawbacks but the approach to use DRFs from developed countries seems to be widely accepted. The few studies which have conducted investigations on dose-response concentration in developing countries come very close to those derived in the developed countries. [59] Therefore we recommend to **use Ostro's (1994) DRFs when calculating the health impacts from pollution from traffic** (see page 22).

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[59] Yaduma, N., M. Kortelainen, A. Wossink (2011) *The Mortality and Economic Costs of Particulate Air Pollution in Developing Countries: A Nigerian Investigation*. Paper presented at European Association of Environmental and Resource Economists 18th Annual Conference 29 June - 2 July 2011, Rome

Finally, it is essential to understand **the age of the population at risk**. For overall mortality, the function is slightly easier, however, with many diseases, such as chronic bronchitis, there is an age window or level where the body is more susceptible for pollution. Therefore, one needs to understand the age distribution of the people exposed to the pollutant.

3 The cost of air pollution from transport on health

As shown in chapter 2, we use epidemiological data regarding the relation between air pollution and morbidity and premature mortality. The number of cases of morbidity and/or premature mortality attributed to air pollution is determined for each of the health endpoints separately, using specific exposure-response functions. The same operations can be carried out for the theoretical situation in which there is for instance no road traffic-related air pollution or policy-induced reduction of road traffic-related air pollution. The difference between the results of these two calculations corresponds to the cases of morbidity and premature mortality due to road traffic-related air pollution. The morbidity and mortality costs arising from reduced levels of road traffic-related air pollution are then evaluated for each health endpoint separately by multiplication of the number of cases with the respective cost estimates (willingness-to-pay factors for the reduction of the different health risks).

WHO[60] argues that the economic valuation involves three important steps: (i) establish average levels of air pollution, (ii) relate these levels to mortality and morbidity statistics of respiratory and cardiovascular diseases, and (iii) apply unit economic values. The basic equation of deriving the total economic cost (TEC) of air pollution for each outcome variable is:

TEC = change in ambient concentration x exposure-response coefficient x Population at risk x unit economic value[61]

Both in developed as well as developing countries, the mortality costs are predominant: about 70-75% of the total societal costs are made up by mortality costs[62;63].

3.1 Exposure-based methods

The environmental burden of disease (EBD) quantifies the amount of disease caused by environmental risks and exposure. Disease burden can be expressed in deaths, incidence or in Disability-Adjusted Life Years (DALY). The latter measure combines the burden due to death and disability in a single index.

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60 WHO (2004) *Public Health Monitoring of the Metro Manila Air Quality Improvement Sector Development Program*. Main report, March 2004.

61 WHO (2004) *Public Health Monitoring of the Metro Manila Air Quality Improvement Sector Development Program*. Main report, March 2004.

62 Bell, M.L., et al., (2006), The avoidable health effects of air pollution in three Latin American cities: Santiago, Sao Paulo, and Mexico City. Environmental Research 100 (2006) 431–440.

63 Sommer, H. et al., (2000). ECONOMIC EVALUATION OF HEALTH IMPACTS DUE TO ROAD TRAFFIC-RELATED AIR POLLUTION. An impact assessment project of Austria, France and Switzerland. OECD.

Using such an index permits the comparison of the burden due to various environmental risk factors with other risk factors or diseases. The environmental burden of disease is based on an exposure approach, supported by a comprehensive analysis of the evidence for the given health risks. Exposure-response relationships for a given risk factor are obtained from epidemiological studies and the derived attributable fractions are then applied to disease burden, expressed in deaths or DALYs, associated with the risk factor.[64]

3.2 Economic valuation

Medical costs or societal costs associated with a disease differ for those who survive a disease and those who die of it. For purposes of the cost analysis, survivors are defined as those people who are diagnosed with a disease, but do not die of it. Non-survivors are those who die of the disease at any point after diagnosis. Separate cost estimates for survivors and non-survivors are often provided. Health valuation experts have debated much over the preference for using the Value of a Statistical Life (VSL) or the Value of a Life-Year (VOLY) to quantify mortality costs. When the VSL or VOLY is used for non-survivors, their medical costs have already been incorporated into the cost estimate.

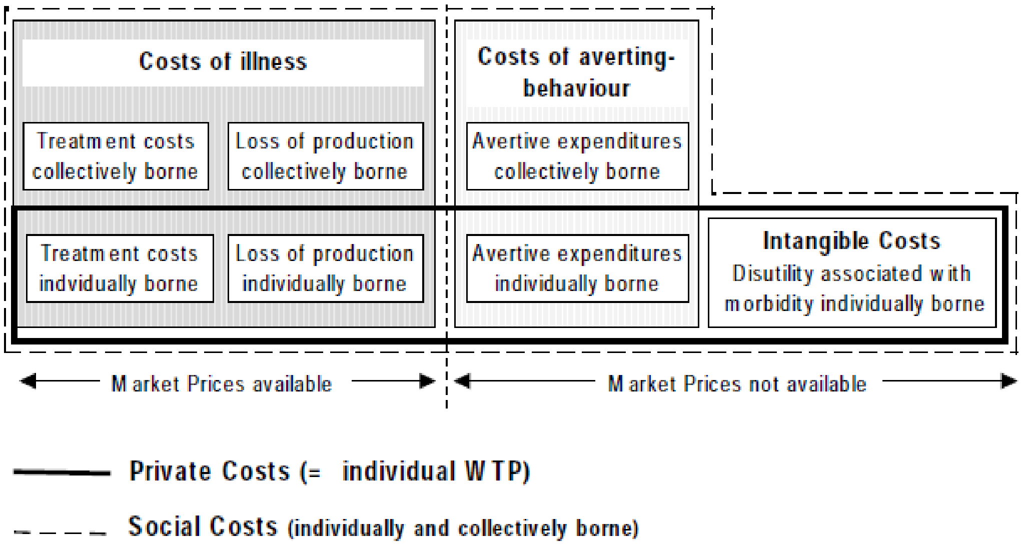
The Cost-of-illness (COI) is an estimate of the incremental *direct* medical costs associated with medical diagnosis, treatment, and follow-up care. This includes various cost elements, such as physician visits, hospitalization, and pharmaceuticals. However, when calculating the value of human health benefits, the ideal approach would be to estimate the value of these improvements in health to everyone affected by an illness e.g., the patient, family, friends, community.[65]

Economists use willingness-to-pay (WTP) techniques to estimate both *direct and indirect* costs. For example, the cost of an ambulance used to transport a person to the hospital is a direct medical cost, while child care and housekeeping expenses required due to illness are non-medical direct costs. In addition to the direct costs, there are opportunity costs (the value of productive and leisure time lost) to the patient and possibly to others.

[64] WHO (2003) *Introduction and methods. Assessing the environmental burden of disease at national and local levels. Environmental Burden of Disease Series*, No. 1, Geneva

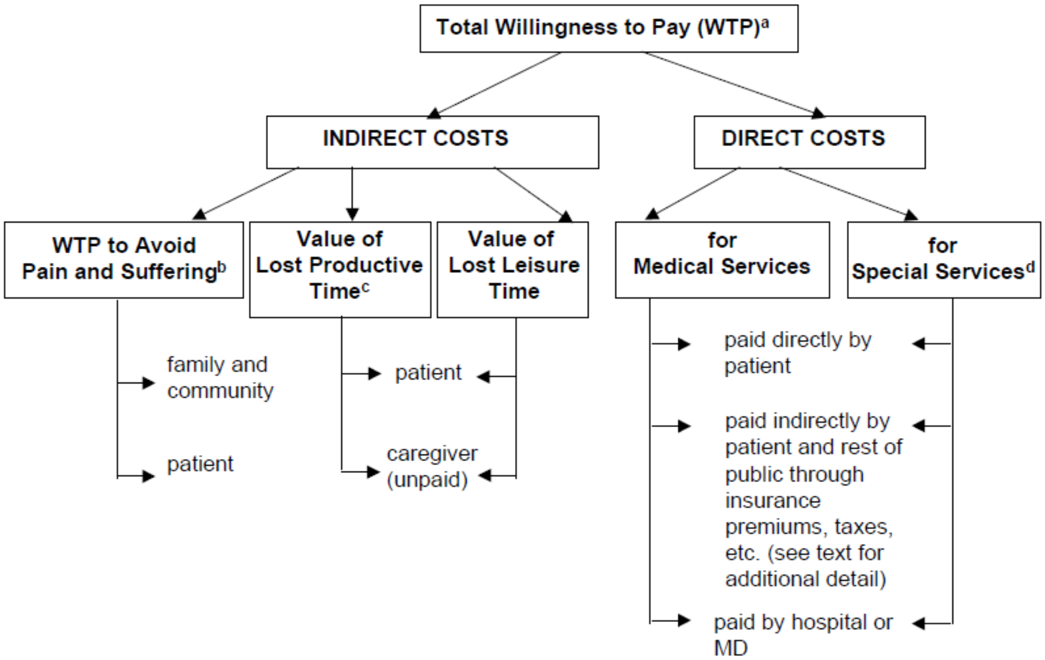
[65] EPA (2010) Cost-of-Illness Handbook. US Environmental Protection Agency.

FIGURE 4: FIGURE 4 WILLINGNESS TO PAY OVERVIEW I



WTP is a measure of value based on the premise, central to economic theory, that the value of a good (or a reduction in health risks) is simply what it is worth to those who consume it or benefit from it. Rather than summing the WTPs for a given risk reduction over all those who enjoy the risk reduction, however, it is often easier to think in terms of the value of an adverse health effect avoided. The total value of an avoided illness is what the otherwise-afflicted individual would be willing to pay to avoid it plus what others would be willing to pay for him or her to avoid it. The sum of these WTPs is the total value of the avoided case of illness, referred to here as total WTP.

FIGURE 5: WILLINGNESS TO PAY OVERVIEW II.



As an alternative to estimating WTP, the direct medical costs of treating diseases provides a lower-bound estimate of the benefits of reducing exposure to harmful pollutants.

3.3 COUNTRY-SPECIFIC OR UNIFORM VALUES FOR WTP OR COI

The literature on WTP based, air pollution related, mortality and morbidity costs is very rare in developing countries and most available studies refer to the US or European context. When using the same (average) value in countries with different per capita incomes would imply a misallocation of resources: the country with low per capita income would invest too much money in air pollution control and this money would be taken from other, more beneficial investments. In the country with high per capita income not enough would be invested in air pollution control. In other words: in a richer country the WTP for a defined risk reduction is higher than in a poorer country as the marginal utility gained by spending this amount for something else is lower. Therefore, both countries would reduce their welfare if they used the same marginal amount for risk reduction.

A simple GDP-per-capita or GNI-per-capita (purchasing power parity) based 'scaling' approach could be used as a benefit transfer technique to transfer WTP values for mortality and morbidity risk reduction from developed to developing countries or cities. However, such a procedure contains a number of drawbacks; the most obvious is the implicit assumption that preferences for health are similar between the country of interest and the U.S./EU and are determined largely by income (which ignores the potential importance of

cultural or other factors in influencing these preferences). This procedure also assumes that the income elasticity of WTP for improved health is equal to 1.0. A number of international valuation studies, however, give reason to question the appropriateness of this assumption[66]. Results from a Bangkok, Thailand study[67] shows that the WTP for avoiding a respiratory illness day actually exceeds what would be predicted following a simple national income adjustment, suggesting that health may be viewed as a basic necessity and ''that those with lower incomes may be willing to pay a higher share of that income to protect their health''[68]. This would suggest an income elasticity below 1. Many other studies indicate contradictory results showing elasticities as high as over 2.0.

[66] Yaduma (2011). The Mortality and Economic Costs of Particulate Air Pollution in Developing Countries: A Nigerian Investigation. Presented at European Association of Environmental and Resource Economists, Rome.

[67] Chestnut, L. G., Ostro, B. D., & Vichit-Vadakan, N. (1997). Transferability of air pollution control health benefits estimates from the United States to developing countries: evidence from the Bangkok study. American Journal of Agricultural Economics, 79, 1630–1635.

[68] V. Brajer et al. / Journal of Asian Economics 17 (2006) 85–102

3.4 Benefit transfer approaches

Virtually all health and economic cost studies conducted in developing countries have employed the benefit transfer method in estimating health effects (mortality and morbidity). The following equation is then applied:

$$WTPdeveloping\ country = WTP_{US\ or\ EU} * \left(\frac{GDP/cap_{developing\ country}}{GDP/cap_{US\ or\ EU}}\right)^{\beta}$$

[1]

Where β is the elasticity of WTP (for a marginal reduction in mortality or morbidity risk) with respect to income. Most empirical studies[69] conducted in both the developed and developing world estimated β to lie between 0.46 and 2.3. With reference to countries with very low GDP per-capita, the choice of β is of prime importance because of the large income disparity between these country and the US or EU. A low income elasticity – say 1.0 – will lead to a higher WTP estimate for a developing country than a high elasticity – say 2.0 . In order to cover the uncertainty in the choice of this elasticity, Robinson and Hammitt (2009)[70] suggest the use of three estimates – lower, central and higher respectively given by 1.0, 1.5 and 2.0 – in health risk analyses conducted in developing countries.

Based on average WTP values for 20 countries as listed in Table 3, we have analyzed the relationship between WTP for fatal mortality risk reductions and income level (GNI/capita ppp).

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69 Bellavance, F; Dionne, G. and Lebeau, M. (2009). "The Value of Statistical Life: A Meta-analysis with Mixed Effects Regression Model." Health and Economics, 28: 444-464.

70 Robinson, L. and Hammitt, J. (2009). The Value of Reducing Air Pollution Risks in Sub-Saharan Africa. A final report prepared for the World Bank Sub-Saharan African Refinery Study. Available on http://www.regulatory-analysis.com/robinson-hammitt-air-pollution-africa.pdf

TABLE 6: TABLE 6: SUMMARY OF WTP FOR FATAL RISK REDUCTIONS BY COUNTRY OF ORIGIN, YADUMA (2011).

Country	Number of Values	Mean Value	Standard Deviation
Australia	2	11,356,098	9,106,156
Austria	2	5,366,079	818,462
Canada	8	8,179,265	6,679,756
China	2	62,620	27,339
Chile	2	469,513	227,606
Denmark	1	14,158,299	–
France	1	14,717,858	–
India	3	295,059	121,672
Japan	1	11,356,098	–
Hong Kong	1	1,990,244	–
Malaysia	1	594,939	–
Mexico	1	313,774	–
New Zealand	3	2,150,047	544,144
Poland	1	2,720,982	–
South Korea	3	1,188,353	470,877
Sweden	4	4,306,819	2,530,969
Switzerland	2	12,208,692	4,930,996
Thailand	3	1,123,365	744,184
United Kingdom	9	6,713,427	7,001,068
United States	33	7,660,518	6,396,053
Total	83	6,203,952	6,142,585

In Figure 4, the relationship between income and WTP for fatal risk reductions, or in other words the VSL, is plotted based on 20 developing and developed countries. The trend line shows an exponential relationship, suggesting an income elasticity larger than 1.0.

FIGURE 6: RELATIONSHIP BETWEEN INCOME LEVEL AND WTP FOR FATAL RISK REDUCTIONS IN 20 COUNTRIES

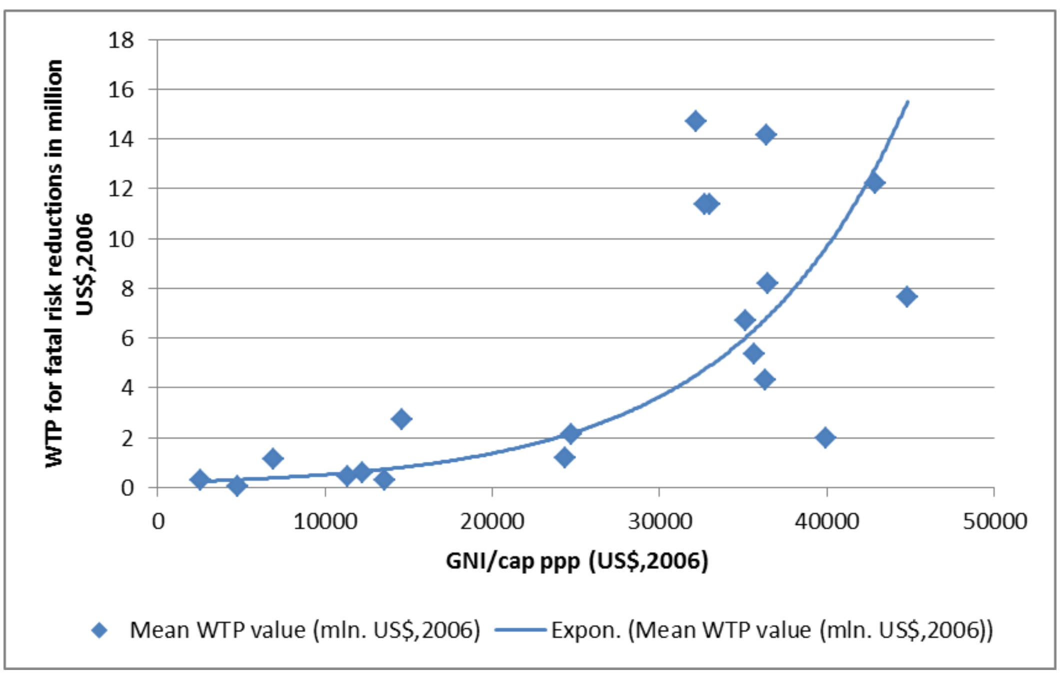
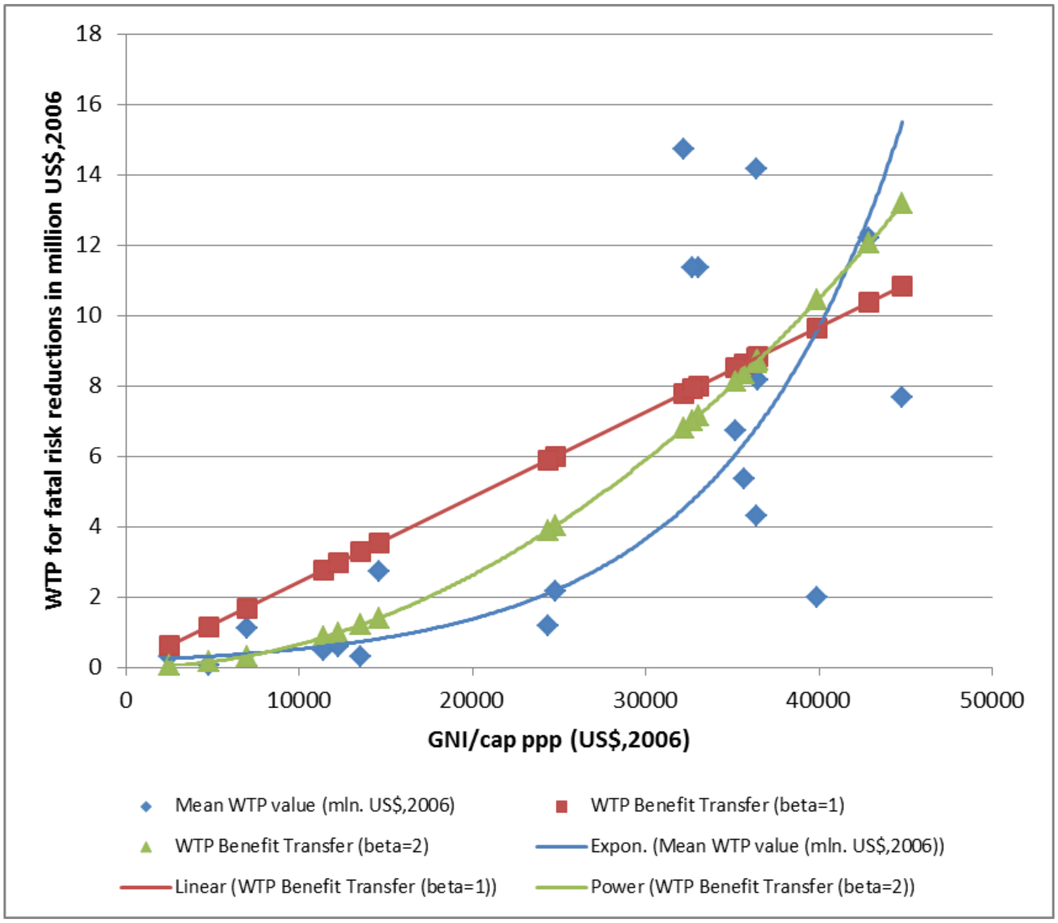


Figure 5 adds two other trend lines in addition to Figure 4. The red line shows the benefit transfer technique following formula [1] with an income elasticity of 1.0. The green line shows the benefit transfer technique following formula [1] with an income elasticity of 2.0. For the WTP in developed countries we used the average of the 10 countries with an income level above 30,000 US$ per capita.

FIGURE 7: TWO BENEFIT TRANSFER TECHNIQUES COMPARED FOR WTP FOR FATAL RISK REDUCTIONS



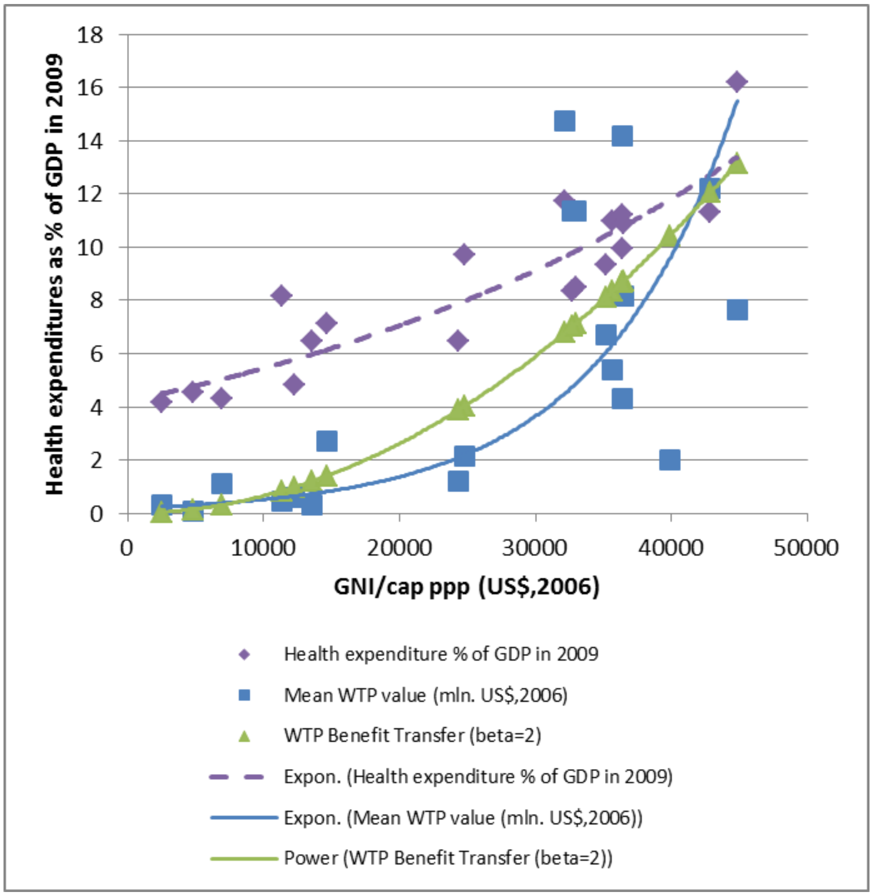
Source: Analysis Ecorys.

Furthermore, we have used the relationship between health expenditure[71] (as % of total GDP) and income to explore similarities and validate the relationship for the benefit transfer technique. Figure 6 shows that the income elasticity of health expenditure is larger than 1.0. The US for example have the highest per capita income and also by far the largest health expenditure of more than 16%

[71] Total health expenditure is the sum of public and private health expenditure. It covers the provision of health services (preventive and curative), family planning activities, nutrition activities, and emergency aid designated for health but does not include provision of water and sanitation (World Bank databank, 2011).

of GDP. Although the relationship does not support an income elasticity as high as 2.0, we note that health expenditures are only direct costs and no WTP values including items for which no market prices are available.

FIGURE 8: RELATIONSHIP BETWEEN HEALTH EXPENDITURE AND INCOME



Source: Analysis Ecorys.

Based on this analysis we propose to use an income elasticity of 1.50 as a lower bound (resulting in higher WTP values for developing countries), an elasticity of 1.75 as a central estimate, and 2.00 as an upper bound (resulting in lower WTP values for developing countries).

3.5 INSTRUMENTS AND TOOLS FOR VALUATION

In **Canada**, monetary valuations of health outcomes are calculated in AQBAT for each of the included Census Divisions based on health Endpoint Valuations (EPV) that assign monetary value to the specific health endpoints. In AQBAT, two endpoint valuations relate to either mortality or morbidity outcomes: for mortality, the value of a statistical life (VSL) is used, which is a measure of people's willingness to accept different levels of risk, and for morbidity, the combined value of lost wages, cost of treatment, averting expenditures and

pain and suffering related to morbidity outcomes. Annex B shows the monetary values for several health endpoints in Canada.

In **the US**, the environmental protection agency (EPA) recommends that the central estimate of $7.4 million (US$2006), updated to the year of the analysis, be used in all benefits analyses that seek to quantify mortality risk reduction benefits regardless of the age, income, or other population characteristics of the affected population. This approach was vetted and endorsed by the Agency when the 2000 Guidelines for Preparing Economic Analyses were drafted[72]. It remains EPA's default guidance for valuing mortality risk changes although the Agency has considered and presented alternatives. Other guidelines in the US are included in the Cost-of-Illness Handbook by EPA.

In the **EU** there have been many studies and working groups looking into the internalization of external costs and the valuation of health effects. Examples are UNITE, CAFE CBA for the Clean Air for Europe program, the EU research project ExternE / NewExt, and the HEATCO and IMPACT studies (see Annex A for summary values).

3.6 Recommended values for health risks

As WTP to reduce the risk of experiencing an illness or fatal risk is the preferred measure of value for mortality and morbidity effects, methods used to estimate WTP vary in the extent to which they capture the four components of WTP:

- "Averting costs" to reduce the risk of illness;
- "Mitigating costs" for treatments such as medical care and medication;
- Indirect costs such as lost time from paid work, maintaining a home, and pursuing leisure activities;
- Less easily measured but equally real costs of discomfort, anxiety, pain, and suffering.

The following table summarises the state-of-the-art WTP values for the most important air pollution-related health endpoints. The values are based on multiple studies in Europe and North America. These studies are generally considered to be most reliable as a basis for applying the benefit transfer technique to arrive at values for developing countries. We have focused on studies which capture as many components of WTP as possible. As a reference income for these recommended values we have used the GNI per capita ppp

[72] US EPA (2010). Guidelines for preparing economic analyses.

(purchasing power parity) value for high income countries (HIC) being 37,000 US$ (2010)[73].

[73] World Bank (2011). GNI per capita based on purchasing power parity (PPP). PPP GNI is gross national income (GNI) converted to international dollars using purchasing power parity rates. An international dollar has the same purchasing power over GNI as a U.S. dollar has in the United States. GNI is the sum of value added by all resident producers plus any product taxes (less subsidies) not included in the valuation of output plus net receipts of primary income (compensation of employees and property income) from abroad. Data are in current international dollars.

TABLE 7 RECOMMENDED VALUES (IN US$, 2010) FOR EVALUATION OF HEALTH RISKS IN HIGH-INCOME COUNTRIES. :

Health Endpoint	Type of Value	Low	Central	High
Mortality:	*US$, 2010*			
Acute Exposure Mortality or Chronic Exposure Mortality	VSL / WTP	5,700,000	7,600,000	9,500,000
Value of a Life-Year (VOLY)	VOLY / WTP	150,000	200,000	250,000
Morbidity:	*US$, 2010*			
Adult Chronic Bronchitis cases	WTP per case	262,500	350,000	437,500
Child Acute Bronchitis Episodes	WTP per case	291	388	484
Acute Respiratory Symptom Days	WTP per day	30	40	50
Asthma Symptom Days	WTP per day	37	49	61
Cardiac ER visits / hospital admissions	WTP per case	3,309	4,412	5,515
Respiratory ER visits / hospital admissions	WTP per case	2,184	2,912	3,640
Restricted Activity Days (RAD)	WTP per day	104	138	173
Minor Restricted Activity Days (MRAD)	WTP per day	34	45	57

4 GUIDELINES FOR CALCULATING THE HEALTH EFFECTS OF AIR POLLUTION FROM TRAFFIC

Air pollution from transport clearly has negative health impacts. To determine the size of the impacts and their causal chain however is not without difficulties. There a two main steps that require attention for addressing the costs for air pollution from traffic on human health. First, what health impacts are to be distinguished, and second, what do health costs can be attributed to these health impacts.

In sections 4.2 and 4.3 we present an approach to assess health impacts and related costs in developing countries based on the assumption that data limitations will exist and incorporating the most important factors to transfer findings from literature to local situations in developing countries. This includes the most import transfer factors being: population affected[74] , age distribution of affected population, the crude mortality rate[75] and the income level of the country (required to transfer health costs).

Obviously reality will be more complex than is represented by the above factors alone and any estimate will be surrounded by a number of inherent

[74] This includes obviously factors such as population density in relation to air pollution concentration indices.

[75] Which can be seen as a proxy for the baseline health condition in a country

uncertainties. Some of these can be addressed by including more air pollutants or health impacts in the equation, or by carrying out additional studies or using more advanced air pollution modelling, whereas other causal relations still have not been studied enough to establish any firm scientific basis for a quantitative estimation.

4.1 Uncertainties in assessing health impacts

Obviously reality will be more complex than is represented by the above factors alone and any estimate will be surrounded by a number of inherent uncertainties. Some of these can be addressed by including more air pollutants or health impacts in the equation, or by carrying out additional studies or using more advanced air pollution modelling, whereas other causal relations still have not been studied enough to establish any firm scientific basis for a quantitative estimation.

For example in assessing the population that is exposed to air pollution from traffic in an optimal situation one would take account of the proximity to the source[76], other pollution sources, time-activity patterns of the population affected, levels of indoor air pollution, etcetera. Also in an ideal situation one would apply a further differentiation of the population exposed by socio-economic class or income level[77].

As indicated earlier also uncertainties exist which can not be solved without further scientific research such as the shape of the DRFs in situations with extremely high pollution concentrations.[78] The DRFs currently used are derived from US and European studies and BTA often assume a linear transfer in the impacts. The high levels of pollution in many, mainly Asia, cities however could potentially have non-linear effects on health. To get an adequate answer to this issue large long-terms studies would be needed and DFRs under local conditions would need to be developed.

Also specific condition in developing countries which may impact on the health effects have not been studied sufficiently to draw hard conclusions. This includes for example issues like the general health system and access to health, nutritional patterns, and extreme poverty.[79]

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76 For example within a range of 300-500 m from a highway the highest exposure is created.

77 HEI Panel on the Health Effects of Traffic-Related Air Pollution (2010) *Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects*. HEI Special Report 17

78 HEI International Scientific Oversight Committee (2010) *Outdoor Air Pollution and Health in the Developing Countries of Asia: A Comprehensive Review*. Special Report 18. Health Effects Institute, Boston, MA. AND HEI (2004) *Health Effects of Outdoor Air Pollution in Developing Countries of Asia*. Health Effects Institute, Special Report 15. April 2004

79 HEI (2004) *Health Effects of Outdoor Air Pollution in Developing Countries of Asia*. Health Effects Institute, Special Report 15. April 2004

Yet, another area which is still surrounded by uncertainty, both in developing and developed countries, are the long-term impacts on health from air-pollution. This would require cohort studies with a long-term perspective. A recent literature review, for example, concluded that there are no available long-term cohort studies for Asia.[80]

Hence BTA methods to transfer health impacts in situations where researchers are bound by time and budgetary constraints can only be seen as crude estimation methods.

In the estimation of the costs related to the health effects similar uncertainties exist and more robust results would require extensive resources and time. The transfer of Willingness to Pay (WTP) estimates from developed countries to developing countries creates a relatively straightforward approach but uncertainties remain regarding the elasticities that are used as other issues such as culture, awareness of risks, life expectancy, etc, are not taken into account. Furthermore, there might be hidden 'mitigating costs' for treatments such as medical care and medication, and indirect costs such as lost time from paid work, maintaining a home, and pursuing leisure activities. This is not only valid for developing countries but also for a large number of developed countries.

4.2 Introducing a four-step approach to assess health impacts

Within the limitations that are posed by uncertainties in assessing health impacts of transport emissions in general and regarding the transfer of findings of developed countries to developing countries in particular, we present a step by step approach to valuate the economic effects of air pollution from transport on health. The approach builds on the approach as was formulated by Ostro ($dT = \sum V_i dH_i$.)[81]

4.2.1 Step 1: Scoping choices

First one needs to make a number of choices regarding the scope of assessing health impacts. This includes (1) establish which health impacts (endpoints) to look at, i.e. establish your *i*, (for example, asthma, bronchitis, and/or lung cancer); (2) chose which emission to include (preferably use a central indicator pollutant such as PM); and, (3) in function of the health endpoint and pollutants, establish which DRF to use (BTA as a minimum approach and raw data collection/epidemiological studies if resources are large).

Recommendations:

- Use PM as indicator pollutant

[80] HEI (2004) *Health Effects of Outdoor Air Pollution in Developing Countries of Asia*. Health Effects Institute, Special Report 15. April 2004

[81] Ostro (1994) *Estimating Health Effects of Air Pollutants: A Methodology with an Application to Jakarta*. Policy Research Working Paper 1301. World Bank, Washington, D.C

- Use DRFs from Ostro (1994, see table 5)

4.2.2 Step 2: Population exposed

Given air pollution concentration levels the number of people that is exposed is determined. Apart from the number of people exposed age and age distribution in an important factor (although this is also dependent on the health endpoint that is included in the scope). In this step also the crude mortality rate is collected (required for calculating the impact on mortality, and serving at the same time as a proxy for the baseline health condition of the population).

Recommendation:

- Determine population that is exposed (absolute number)
- Determine the age distribution of the population
- Establish the crude mortality rate

4.2.3 Step 3: Calculate health impacts

In the third step, the available data are used to calculate the health impacts using the formula given by Ostro (1994): dT = ∑VidHi

Recommendation:

- Calculation of health impacts based on Ostro

4.2.4 Step 4: Monetise health impacts

Finally, the health effects are monetised based on the (state-of-the-art) WTP ranges as provided in table 8.

Recommendations

- Use state-of-the-art WTP values

4.3 Example of the four-step approach in practice

To illustrate the above approach, we present a calculation sample for Beijing, China. During the Olympics the city became infamous for high air pollution levels and is a good example for results in air pollution and related health problems in south east Asian cities.

4.3.1 Step1: Scoping choices

In step one, we have chosen to look at PM_{10}, total mortality and acute chronic bronchitis. The selection guides us in determining which DRFs to use.

Air pollutants

PM_{10}

Health endpoints

Mortality

Morbidity

Acute Chronic Bronchitis

Choice of DRFs

According to Ostro (1994, table 5) and the use of benefit transfer approach, the DRFs are as follows:

- Premature mortality/100.000 = b_i = 6.72 or if calculated in increase in %

Lower coefficient	Central coefficient	Upper coefficient
0.062	0.096	0.13

- Chronic bronchitis/100.000 = b_i =61.2

Calculated per 10 μg/m^3 increase (or decrease).

4.3.2 Step 2: Population exposed

In step two we have determined the data from our case-study. For Beijing many of the values are readily available which facilitated data collection immensely. To set up a policy scenario we assume that Beijing adopts measures to lower the emissions of PM_{10} from current levels to 70 μg/m^3 (midpoint between current level and WHO guidelines value). It is important to note, that we don't consider it necessary to understand the baseline case if one only wants to make a rough estimation of how much a policy measure that lowers PM_{10} with x number of units, would save in terms of costs.

The following data is therefore required:

- Population in Beijing: 19,612,368[82]
- PM_{10} level in Beijing: 121 μg/m^3 annual mean[83]
- WHO Guideline value PM_{10} annual mean: 20 μg/m^3
- Goal of policy intervention: reduce PM_{10} concentrations 70 μg/m^3.
- Crude mortality rate: 0.0051

4.3.3 Step 3: Calculate health effects

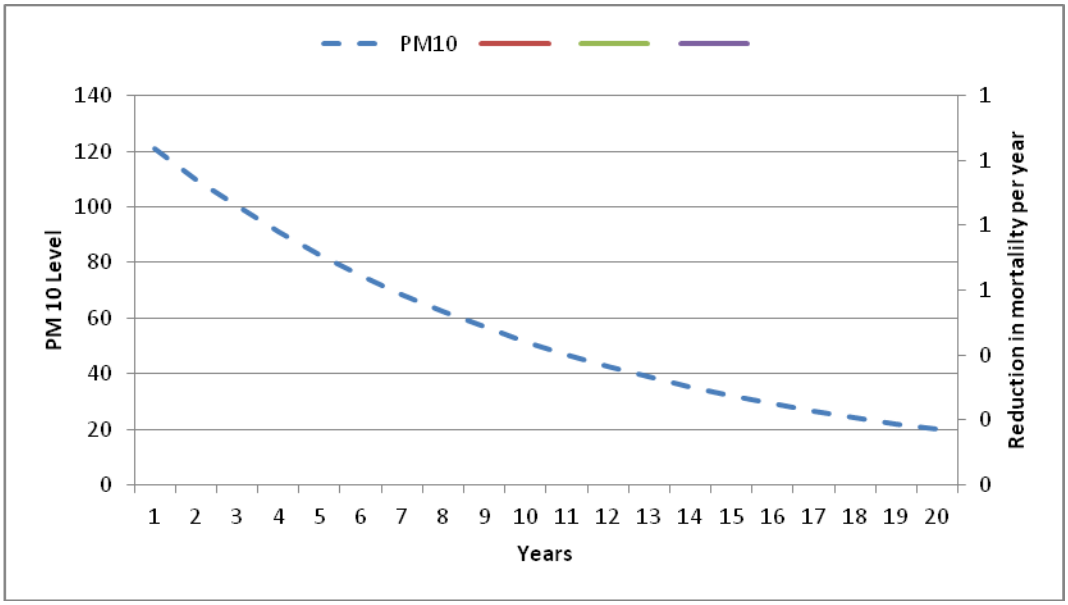
In step three, we try to calculate the health effects using the formulas spelled out above. For mortality, the function

[82] National Bureau of Statistics of China (2010) Communiqué of the National Bureau of Statistics of People's Republic of China on Major Figures of the 2010 Population Census[1] (No. 2). (http://www.stats.gov.cn/english/newsandcomingevents/t20110429_402722516.htm)

[83] WHO (2011) Database: outdoor air pollution in cities. Data for 2009 (http://www.who.int/phe/health_topics/outdoorair/databases/en/)

$\Delta Mortality = ci * \Delta PM10 * POP * 0.01 * crude\ mortality\ rate$, is used. If one assumes three different scenarios with a lower, central and upper coefficient the graphs looks something like follows:

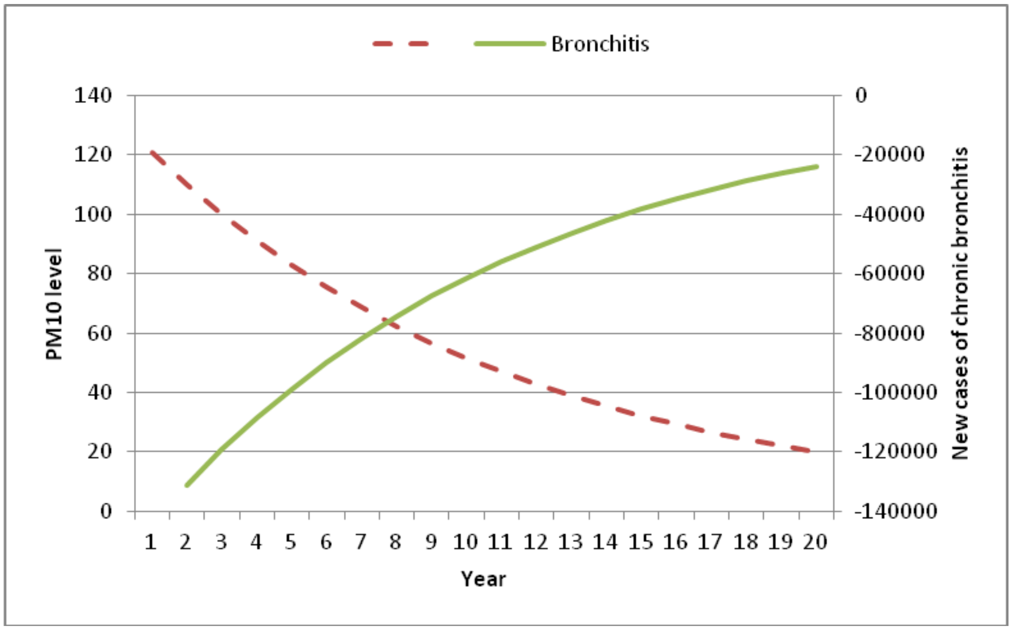
FIGURE 9: THE HEALTH EFFECTS OF REDUCING PM10 TO WHO GUIDELINE VALUES ON MORTALITY



The figure shows the relationships between decreased PM_{10} level from today's 121 µg/m^3 in Beijing to below WHO standards of 20 µg/m^3 in 20 years. For example, if the policy goal is to reduce PM_{10} levels to 70 µg/m^3 then a reduction of almost 300 in a low scenario, 400 in a medium case scenario and 600 in a high case scenario, i.e. 300, 400 and 600 saved lives per year due to lowered PM_{10} levels depending on the coefficient are reached. This amounts to the cumulative effects of 4,300; 6,800; and 9,000 over seven years.

For morbidity the formula $\Delta Morbidity = ci * \Delta PM10 * POP$ is used. It shows the relationships between the occurrence of new cases of chronic bronchitis as a function of the PM_{10} levels. Therefore, if the policy goal is to reduce PM_{10} levels to 70 µg/m^3 the number of new cases of chronic bronchitis each year slumps to around 85,000 per year. This amount to a cumulative effect of roughly 650.000 less cases over 7 – 8 years.

FIGURE 10: THE HEALTH EFFECTS OF REDUCING PM10 TO WHO GUIDELINE VALUES ON CHRONIC BRONCHITIS



4.3.4 STEP 4: MONETISE THE HEALTH EFFECTS

Finally, in step 4 the health impacts are transferred into health cost savings. In order to monetise the health effects as determined in steps 1-3, we apply formula [1] as follows:

$$WTP_{China} = WTP_{HIC} * \left(\frac{\frac{GNI}{cap}_{China}}{\frac{GNI}{cap}_{HIC}} \right)^{\beta}$$

The WTP (in US$, 2010) in China for a **fatal risk** reduction (per case of mortality) can be determined as follows:

Lower estimate:

$$WTP_{China} = 5{,}700{,}000 * \left(\frac{7{,}570}{37{,}000} \right)^{2.00} = 239{,}000$$

Central estimate:

$$WTP_{China} = 7{,}600{,}000 * \left(\frac{7{,}570}{37{,}000} \right)^{1.75} = 473{,}000$$

Upper estimate:

$$WTP_{China} = 9{,}500{,}000 * \left(\frac{7{,}570}{37{,}000} \right)^{1.50} = 879{,}000$$

The same approach can be used to quantify the morbidity health endpoint **Adult Chronic Bronchitis** WTP per case (in US$, 2010) in China:

Lower estimate:

$$WTP_{China} = 262{,}500 * \left(\frac{7{,}570}{37{,}000}\right)^{2.00} = 11{,}000$$

Central estimate:

$$WTP_{China} = 350{,}000 * \left(\frac{7{,}570}{37{,}000}\right)^{1.75} = 22{,}000$$

Upper estimate:

$$WTP_{China} = 437{,}500 * \left(\frac{7{,}570}{37{,}000}\right)^{1.50} = 40{,}000$$

4.3.5 Total health benefit from transport policy measure in Beijing

In conclusion, the total health benefits from transport policy measures can be calculated in several different ways. The brief case study on Beijing shows one approach. We concluded that under the assumptions made, the mortality rates reduced to 300, 400 and 600 emissions related deaths per year due. To calculate the saved health effects we use the cumulative number in reduced mortality. If these are matched with the lower, central and upper estimated in the monetisation of each health end-point the following figures appears:

- Lower estimate: 4,300 * 239,000 = $1 billion
- Central estimate: 6,800 * 473,000 = $3.2 billion
- High estimate: 9,000 * 879,000 = $7.9 billion

For chronic bronchitis, the following calculation is made:

- Lower estimate: 650,000 * 11,000 = $7.15 billion
- Medium estimate: 650,000 * 22,000 = $14.3 billion
- High estimate: 650,000 * 40,000 = $26 billion

ANNEXES

ANNEX A: HEALTH VALUATION IN EUROPE

Table 0.1: Proposed UNITE VSL values by country compared to mandatory country values (€ , 1998).

Country	*Official values in use* [A)] *million €*	***UNITE VOSL million €***	*(Official-UNITE)/Official %*
Austria	*1.52*	**1.68**	10%
Belgium	*0.40*	**1.67**	312%
Denmark(B)	0.52[*)]	**1.79**	244%
Finland	0.89[*)]	**1.54**	73%
France	*0.62*	**1.49**	141%
Germany	*0.87*	**1.62**	87%
Greece (B)	*0.14*	**1.00**	588%
Ireland	*1.04*	**1.63**	57%
Italy	*n.a.*	**1.51**	-
Luxembourg	*n.a.*	**2.64**	-
Netherlands	*0.12*	**1.70**	1269%
Norway (B)	*1.49*	**1.93**	29%
Portugal	*0.04*	**1.12**	2896%
Spain	*0.07*	**1.21**	1625%
Sweden (B)	1.48[*)]	**1.53**	4%
Switzerland (B)	*n.a.*	**1.91**	-
United Kingdom (B)	1.53[*)]	**1.52**	1%
Hungary (B)	*n.a.*	**0.74**	-
Estonia (B)	*n.a.*	**0.65**	-

Note: A) Based on Nellthorp, Mackie and Bristow (1998). HICPs for Eurozone has been used to adjust price level to 1998.
*) Latest available values from Tervonen (1999) has been used (For Sweden SIKA(2000)). Corresponding EUNET values are; DK 0.79; FIN 1.33; N n.a. ; S 1.80; UK 1.11.
B) Not in Eurozone, Exchange rate of 24 November 2000 used.

Source: UNITE(2001). UNIfication of accounts and marginal costs for Transport Efficiency. Deliverable 5. Valuation Conventions for UNITE.

Table 0.2: Monetary values for acute and chronic mortality (€, 2000).

Age group	Impact: Human health	Value (EURO 2000)
Total	Acute YOLL	75,000
Total	Chronic YOLL	50,000

Source: NewExt (2004). New Elements for the Assessment of External Costs from Energy Technologies.

Table 0.3: Health valuation data for the CAFE CBA (€, 2000).

Mortality	Based on median values		Based on mean values
Infant mortality	€ 1,500,000/death		€ 4,000,000/death
Value of statistical life	€ 980,000/death		€ 2,000,000/death
Value of a life year	€ 52,000/year		€ 120,000/year
Morbidity	**Low**	**Central**	**High**
Chronic bronchitis	€ 120,000/case	€ 190,000/case	€ 250,000/case
Respiratory, cardiac hospital admission		€ 2,000/admission	
Consultations with primary care physicians		€ 53/consultation	
Restricted activity day (day when person needs to stay in bed)		€ 130/day	
Restricted activity day (adjusted)		€ 83/day	
Minor restricted activity day		€ 38/day	
Use of respiratory medication		€ 1/day	
Symptom days		€ 38/day	

Source: AEA Technology (2005)[84].

[84] Methodology for the Cost-benefit analysis for Clean Air for Europe (CAFE). Volume 2: Health Impact Assessment.

Table 0.4: Cost factors for road transport emissions per ton of pollutant emitted in EU-27 (2002 Euros)

Pollutant emitted	NO_x	NMVOC	SO_2	$PM_{2.5}$	
Effective pollutant	O_3, Nitrates, Crops	O_3	Sulphates, Acid deposition, Crops	Primary $PM_{2.5}$	
Local environment				urban	outside built-up areas
Austria	4,300	600	3,900	450,000	73,000
Belgium	2,700	1,100	5,400	440,000	95,000
Cyprus**	500	1,100	500	230,000	20,000
Czech Republic	3,200	1,100	4,100	170,000	61,000
Denmark	1,800	800	1,900	520,000	54,000
Estonia	1,400	500	1,200	100,000	23,000
Finland	900	200	600	400,000	33,000
France	4,600	800	4,300	430,000	83,000
Germany	3,100	1,100	4,500	430,000	80,000
Greece	2,200	600	1,400	210,000	34,000
Hungary	5,000	800	4,100	150,000	54,000
Ireland	2,000	400	1,600	510,000	50,000
Italy	3,200	1,600	3,500	370,000	70,000
Latvia	1,800	500	1,400	80,000	22,000
Lithuania	2,600	500	1,800	90,000	28,000
Luxemburg	4,800	1,400	4,900	590,000	96,000
Malta (O_3 estimated)	500	1,100	500	170,000	16,000
Netherlands	2,600	1,000	5,000	470,000	88,000
Poland	3,000	800	3,500	130,000	53,000
Portugal	2,800	1,000	1,900	210,000	37,000
Slovakia	4,600	1,100	3,800	110,000	49,000
Slovenia	4,400	700	4,000	220,000	55,000
Spain	2,700	500	2,100	280,000	41,000
Sweden	1,300	300	1,000	440,000	40,000
Switzerland	4,500	600	3,900	640,000	86,000
United Kingdom	1,600	700	2,900	450,000	67,000

Notes: Cost categories included are: human health, crop losses, material damages.
* Values are applicable to all emissions at ground level (e.g. diesel locomotives).
** Estimated values as Cyprus outside of modelling domain.

Source: HEATCO (2006)

Table 0.5: Monetary values (European average) used for economic valuation (2002 Euros at factor costs)

Impact	€$_{2002}$ per unit
Human health, effects in respective units	
Acute mortality – Tears of life lost due to acute exposure	60,500
Chronic mortality – Years of life lost (YOLL) due to chronic exposure	40,300
New cases of chronic bronchitis	153,000
Hospital admissions (respiratory and attributable emergency cardiac)	1,900
Restricted activity days	76
Minor restricted activity days: cough days: symptom days (lower respiratory symptoms including cough): days of lower respiratory symptoms (excluding cough): days of lower respiratory symptoms, including cough, in children in the general population, i.e. extra symptoms days	31
Days of bronchodilator usage	1.0

Source: IMPACT (2008)[85].

[85] HEATCO (2006) Developing Harmonised European Approaches for Transport Costing and Project Assessment (HEATCO). Deliverable D5: Proposal for Harmonised Guidelines

Annex B: Health valuation in North America

Table 0.6: AQBAT health valuation in Canada.

Health Endpoint	Type of Value	Low	Central	High
Acute Exposure Mortality or Chronic Exposure Mortality	VSL / Wage Risk	$3,050,000	$4,050,000	$5,050,000
Acute Respiratory Symptom Days	WTP	-	14	-
Adult Chronic	WTP	175,000	266,000	465,000
Asthma Symptom Days	WTP	7	28	120
Cardiac Emergency Room Visits	WTP	-	4,400	-
Child Acute Bronchitis Episodes	WTP	150	310	460
Minor Restricted Activity Days	WTP	-	22	-
Respiratory Emergency Room Visits	WTP	-	2,000	-
Restricted Activity Days	WTP	-	48	-

Source: MARBEK(2007). Evaluation of Total Cost of Air Pollution Due to Transportation in Canada.

*Values are in Canadian $ converted to the year 2000.

Table 0.7: VSL estimates for the US (mean values in millions of 2006 US$)

Study	Method	Value of Statistical Life
Kniesner and Leeth (1991 - US)	Labor Market	$0.85
Smith and Gilbert (1984)	Labor Market	$0.97
Dillingham (1985)	Labor Market	$1.34
Butler (1983)	Labor Market	$1.58
Miller and Guria (1991)	Contingent Valuation	$1.82
Moore and Viscusi (1988)	Labor Market	$3.64
Viscusi, Magat, and Huber (1991)	Contingent Valuation	$4.01
Marin and Psacharopoulos (1982)	Labor Market	$4.13
Gegax et al. (1985)	Contingent Valuation	$4.86
Kniesner and Leeth (1991 - Australia)	Labor Market	$4.86
Gerking, de Haan, and Schulze (1988)	Contingent Valuation	$4.98
Cousineau, Lecroix, and Girard (1988)	Labor Market	$5.34
Jones-Lee (1989)	Contingent Valuation	$5.59
Dillingham (1985)	Labor Market	$5.71
Viscusi (1978)	Labor Market	$6.07
R.S. Smith (1976)	Labor Market	$6.80
V.K. Smith (1983)	Labor Market	$6.92
Olson (1981)	Labor Market	$7.65
Viscusi (1981)	Labor Market	$9.60
R.S. Smith (1974)	Labor Market	$10.57
Moore and Viscusi (1988)	Labor Market	$10.69
Kniesner and Leeth (1991 - Japan)	Labor Market	$11.18
Herzog and Schlottman (1987)	Labor Market	$13.36
Leigh and Folsom (1984)	Labor Market	$14.21
Leigh (1987)	Labor Market	$15.31
Garen (1988)	Labor Market	$19.80

Derived from U.S. EPA (1997a) and Viscusi (1992). Updated to 2006$ with GDP deflator.

Source: US EPA (2010). Guidelines for preparing economic analyses.

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