

Support Vietnam EREA/MOIT to Conduct a Strategic Environmental Assessment (SEA) of the National Power Development Plan 8 in the Period 2021-2030 with Vision to 2050 (PDP8)

Assignment No GT#69/SEAO PDP8-VIE (Southeast Asia – Vietnam)

MODULE 4: ASSESSING THE IMPACT OF THERMAL POWER ON HUMAN HEALTH

Learning Goals

- To learn key concepts, in understanding air pollution and health issues in relation to thermal power plants.
- To strengthen skills to quantify, value, and assess air pollution impacts.

Module Outline

1. Overview and Key Terminologies
 - a) Cause and effect of air pollution from the thermal power plants
 - b) Emission (Technology and emission factors)
 - c) Atmospheric fate and chemistry (pollution dispersion and ambient air quality)
 - d) Exposure and health impact (mortality, morbidity)
2. Economics of air pollution and health impact
 - a) Value for mortality risk
3. Impact assessment tools and methodology
 - a) Zone of influence
 - b) Benefit transfer
4. Hands on case-study

1. OVERVIEW AND KEY CONCEPTS AND TERMINOLOGIES

Introduction and key approaches for impact assessment

Air pollution is associated with detrimental effects on human health, natural ecosystems and climate.

When evaluating the potential impact of PDP it is desirable to quantify impacts in a consistent manner.

Whilst this is difficult given the diversity of the impacts, approaches based on monetary valuation are the most common, and these have several advantages.

An important factor in any economic appraisal of air pollution is the cost of health impacts. Health risk valuation is highly controversial. Many people are uncomfortable with the idea of assigning values to the lives saved from policy interventions. Nonetheless, policymakers should still consider methodologies that have been developed for this exact purpose, despite the implication.

In reality, people are constantly trading off money and mortality risk in a variety of decisions on a daily basis (e.g., when deciding whether to pay extra for a safer vehicle or to accept a higher-paying but riskier job like cleaning skyscraper windows).

The health costs of air pollution are dominated by its effects on mortality, which in turn are dominated by the effects of airborne particulate matter (PM).

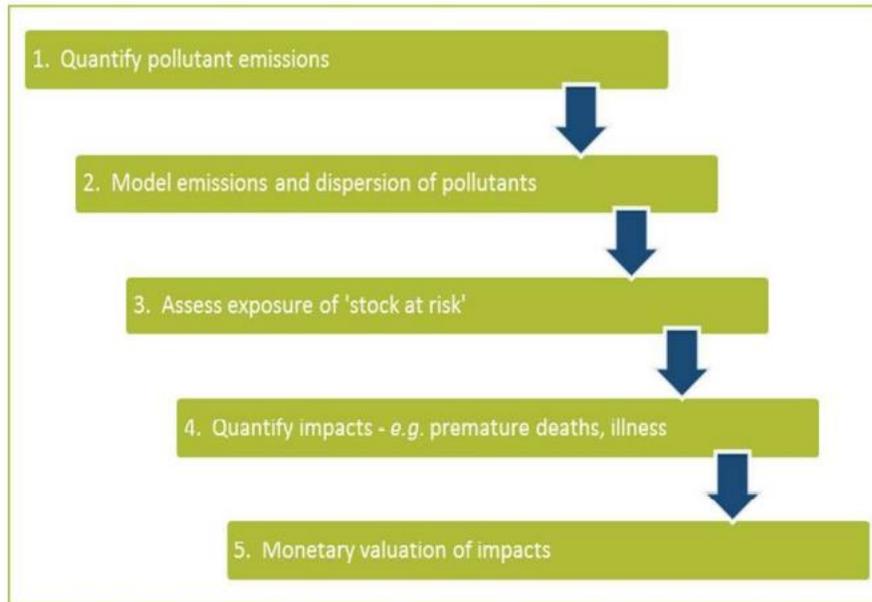
Ambient concentrations of PM are most defined in terms of two metrics: PM₁₀ and PM_{2.5}, the mass concentrations of particles with an aerodynamic diameter of less than 10 µm and 2.5 µm respectively.

In recent years various methods have emerged for quantifying and valuing the health effects (and other environmental effects) of air pollutants, including PM. Many of these methods have adopted a very similar approach and have even relied upon the same underlying health studies.

Two main approaches to valuing changes in air pollution:

- 'impact pathway' approach; and
- 'damage cost' approach.

Impact pathway approach: The detailed valuation of the health impacts of air pollution involves a 'bottom-up' calculation in which environmental benefits and costs are estimated by following the steps shown in Figure below. This approach was developed through a series of joint EU-US research projects in the 1990s.



In some circumstances a variant of the impact pathway approach may be applied. For example, when setting air quality standards, Steps 1 and 2 may be disregarded and changes in exposure to pollutant concentrations between current and future scenarios (the latter being based on the proposed standard) are used to quantify the impacts on health.

The overall impacts are calculated using the following general relationships:

$$\text{Impact} = \text{Concentration} \times \text{Stock at risk} \times \text{Response function}$$

$$\text{Cost} = \text{Impact} \times \text{Unit cost of impact}$$

The main steps in the calculation are discussed in more detail below:

Quantifying emissions:

The first step in the calculation involves the quantification of emissions, with disaggregated road-based or grid-based source apportionment. This requires a detailed emissions inventory.

Emission factors are widely used to estimate the amount of emissions because few data are required. Locally derived or site-specific emission factors could reduce error compared to the estimation by using default or referenced emission factors.

Emission factors of NO_x, SO₂ and PM emissions from thermal powerplants could be developed by dividing annual emissions of selected power plants by their related activity, as shown below.

$$\text{Locally derived emission factor} = \text{Annual emission} / \text{Related activity}$$

Where: Related activity is electricity generation expressed in terms of either mega-watthours (MWh) or heat input (GJ). Then the locally derived emission factors could be expressed in terms of mg of gaseous emission per unit of gross electricity produced (kg/MWh) and per unit of heat input (kg/GJ).

Modelling air pollution

The second step involves an analysis of pollutant dispersion and chemistry across different spatial scales. Importantly, this includes the consideration of both primary pollutants (e.g. SO₂, primary PM) and secondary pollutants (secondary PM such as sulfates, or secondary gaseous pollutants such as ozone), and the assessment of changes in pollutant concentrations. A large amount of information is required on baseline emissions and pollution concentrations, as these determine the formation of secondary pollutants.

Determining exposure

The third step is the quantification of the exposure of people, the environment (e.g. crops) and buildings that are affected by air pollution (i.e. linking pollution with the 'stock at risk' using, for example, population data).

Intake fractions are used to estimate how much pollution from stationary and mobile emissions sources is inhaled by exposed populations. Intake fractions depend on three main factors:

- The height at which emissions are released: The most important distinction is between emissions from tall smokestacks, such as at power plants, which are more likely to be dispersed without harm but are also transported considerable distances, and emissions released at ground level, such as from cars and residential heating, which tend to stay locally concentrated.
- The size of the population exposed to the pollution: For smokestack emissions, people living 2,000 kilometers or more from a plant can still intake some of the pollution (Zhou and others, 2006). Even if a plant were to be located away from an urban center, its emissions could still cause significant health damage elsewhere. Long-distance transportation of pollution also raises thorny issues about how one country should account for cross-border environmental damage when setting its own fuel taxes.
- Meteorological conditions (most notably wind speed and direction), topography (e.g., proximity to mountain barriers that may block pollution dispersion), and ambient ammonia concentrations (which catalyze atmospheric reactions of SO₂ and NO_x).

The Health Response to Air Pollutants In Europe Project (HRAPIE) coordinated by WHO-Europe for the EU Commission, is one of the most comprehensive scientific study undertaken to date on the health effects of air pollution bringing together a large number of senior experts from Europe and North America. HRAPIE provides response functions for exposure to three pollutants, fine particles (PM_{2.5} or PM₁₀), NO₂, and ozone. However, there is currently a debate as to how to apply the NO₂ recommendations, and reliable analysis of impacts for this pollutant is not yet possible. No account was taken by HRAPIE of effects of SO₂. The table below provides the summary of information from HRAPIE showing endpoints for health impact assessment, relative risk from a 10ug/ μ 3 change in exposure of different criteria pollutants:

EFFECT	POLLUTANT	EXPOSURE PERIOD	RELATIVE RISK FROM A 10 $\mu\text{g.m}^{-3}$ CHANGE IN EXPOSURE
All-cause mortality, age 30+	PM	Long	1.062
All-cause mortality	O ₃	Short	1.0029
Post -neonatal infant mortality	PM	Long	1.04
Respiratory hospital admissions	PM	Short	1.019
Respiratory hospital admissions	O ₃	Short	1.0044
Cardiovascular disease (CVD) hospital admissions	PM	Short	1.0091
Cardiovascular disease (CVD) hospital admissions	O ₃	Short	1.0089
Prevalence of bronchitis in children	PM	Long	1.08
Incidence of chronic bronchitis in adults	PM	Long	1.117
Restricted activity days	PM	Short	1.047
Work loss days	PM	Short	1.046
Asthma symptoms in asthmatic children	PM	Short	1.028
Minor restricted activity days	O ₃	Short	1.0154

Source: Health and Environment Alliance, 2016, Health Impacts of Coal-Fired Power Stations in the Western Balkans

Estimating Impacts

The fourth step involves the quantification of the impacts (health and non-health) of air pollution. The adverse health effects of ambient air pollution are divided into two categories:

- i) Morbidity, and
- ii) Mortality.

Morbidity effects may range from the relatively mild sub-clinical effects such as increased coughing, reduction in lung function or increased medication usage, through to seeking medical attention by a general practitioner, emergency department attendances and hospital admission.

Mortality is the most widely recommended health impact for use in studies quantifying the effects of air pollution. The assessment of mortality from chronic exposure is a critical issue because valuation approaches that look at long-term changes in air pollution associated with PDP will need a different approach to those approaches that estimate at short-term changes associated with specific policies. This requires analysis of costs and benefits over the longer term.

PM is known to be the most damaging pollutant to human health in terms of overall health costs, particularly in the longer term. Many studies have used PM₁₀ as an indicator of PM. However, there is increasing evidence that the adverse health effects - particularly mortality - are more closely associated with PM_{2.5}

A recent UK report states that PM2.5 is considered to be the best index of PM for quantitative assessments of the effects of policy and strategic planning interventions with long term impacts.

There are two methods of calculating the proportion of deaths attributable to a change in PM exposure.

The first method uses a 'static' concentration-response (C-R) function derived from epidemiological studies, in which:

$$\text{Attributable proportion} = \text{Annual death rate} \times \text{Study population size} \times \% \text{ increase in health effect per increase in exposure} \times \text{Change in exposure}$$

The second method is based on 'life tables'. This approach follows a stratified (by age) study population over time. It takes into account the probability of each age band dying, and compares a baseline scenario with a scenario in which the exposure changes (Hurley et al., 2005). The life-table method is based on a matrix defined simultaneously by the calendar years into the future and the age distribution of the study population. The effect of a specific exposure on health is given by the differences between the two matrices (between the exposure-changed scenario and the baseline). This estimation method expresses health impacts in terms of 'years of life lost' (YOLL) from air pollution.

2. ECONOMICS OF AIR POLLUTION AND HEALTH IMPACT

Monetary valuation of impacts

In the final step a monetary value is assigned to the impacts. Health impacts from changes in PM emissions are often monetized using unit costs for:

- Value of a statistical life (VSL),
- Value of a statistical life year (VOLY),
- Hospitalization for respiratory disease and
- Hospitalization for cardiovascular disease.

The single most important health endpoint in the valuation of PM health impacts is mortality, and specifically mortality from chronic exposure. This has dominated valuations in all studies to date. However, mortality from chronic exposure is also the most complex health endpoint to assess.

VSL: The monetary valuation of the VSL is often derived using the 'willingness to pay' (WTP) approach.

WTP approach surveys individuals about their willingness to pay to avoid a specific health effect. The VSL is defined as an aggregate measure of a community's WTP to reduce the risk of premature mortality. Once the number of deaths saved or lost due to changes in PM emissions is established (static method of attributable deaths), the VSL is applied to the number, producing the cost or benefit of the change.

VOLY: The other approach in the monetary valuation of premature mortality is the VOLY. The VOLY is usually calculated via an annualised equivalent of VSL estimates. The VOLY can then be applied to the YOLL to derive a cost due to changes in PM emissions.

In their report for the Australian Commonwealth Department of Environment, Water, Heritage and the Arts (DEWHA), Jalaludin et al. (2009) recommend that the use of the VOLY is preferable to the use of the VSL in monetising the air pollution effects on premature mortality, and should be used whenever feasible and practicable. The cost of hospital admissions and other morbidity outcomes are usually based on the average use of hospital or medicinal resources for a patient group.

Damage cost approach

Applying the impact pathway approach to every policy impact assessment is very resource intensive, and most likely prohibitively so. As a result, many countries have adopted simple 'look-up' tables to allow direct valuation based on emissions alone. These are frequently referred to as 'damage costs', and allocate dollar-per-tonne values to emissions.

Damage costs for a specific country or jurisdiction are usually generated via a full impact pathway approach utilising location-specific inputs and data. The level of detail used to generate damage costs varies. Some approaches involve the quantification of health impacts as well as monetary values, whereas others use disaggregated values that differentiate emissions according to the sector or location of emissions.

Damage costs provide a simple way to value changes in PM. They are estimates of the costs to society due to the impacts of changes in emissions. Damage costs assume an average impact on an average population affected by changes in air quality.

Data Limitation: Application of methodologies discussed above in Viet Nam requires

- Robust inventory
- Atmospheric modelling capacity.
- The availability of data on air quality and health (including health costs).
- Variability in data across provinces.
- Variability across urban and regional communities (e.g. in terms of air quality impacts and the benefits of emission reductions).

4. IMPACT ASSESSMENT TOOLS AND METHODOLOGY

Zone of influence

The health impacts are spatially discrete – they depend on distance from the power station and also on the existing levels of ambient air quality: in other words, the background levels of pollution from other sources. There will be differences between different parts of the country and different power stations. This depends on:

- The pattern of distribution of the emissions (the pollution plume);
- Population density and consequently the number of people at risk;
- Existing background pollution levels.

EIA reports from planned power stations and international experience are used to define three zones of risk of increased health impacts from air pollution from power stations:

Zone 1: up to 5 km from the power stations, where the risks of increased disease incidence is high, especially where existing ambient air quality is poor. These areas experience high levels of deposition of both gaseous pollutants and particulate matters;

Zone 2: between 5 and 10 km of the plants, the zone in which there is a medium risk of increased disease incidence. The remaining PM pollutants will be deposited in this zone along with further levels of gaseous pollutants;

Zone 3: between 10 and 30 km from the power stations: this is a low risk zone, with little or no PM deposition but some residual risk of impacts from gaseous pollutants.

Risks are increased, even in Zone 3, where background air quality is poor and/or where clusters of power stations with overlapping risk zones are found.

Benefit transfer approach

Given the time and data constraint, the comprehensive assessment based on pollutant dispersion, exposure of population (including vulnerable population), and incidence of health impact is not possible. It is therefore advised to undertake health impact assessment and valuation based on the benefit transfer approach.

Different countries have adopted different approaches for valuing the health impacts of PM. The most advanced and detailed studies have been those undertaken in Europe and the US, where independent scientific committees have provided advice on health quantification and valuation. These studies have examined major changes in air pollution standards, capturing the complexity associated with chronic health effects using the impact pathway approach. As an example, in the Clean Air for Europe (CAFE) programme damage costs have been applied to a range of sectoral and policy-specific contexts, whilst the United States Environmental Protection Agency (USEPA) has used damage costs (for secondary PM) when updating air quality standards for NO₂ and SO₂.

The main studies identified in the literature and considered in detail were:

European Union - CAFE programme. The objectives of the CAFE programme were to establish the capacity to assess the costs and benefits of air pollution policies, and to conduct a CBA of the effects of these policies. The impact pathway approach was used to value the health impacts of

air pollution (environmental endpoints such as crop damage were also assessed), although damage costs were also generated (AEA Technology Environment, 2005).

United Kingdom – Review of Air Quality Strategy. The UK has a long tradition of CBA for air pollution. The analysis of impacts and external costs has been taken forward by the Department of Health’s Committee on the Medical Effects of Air Pollutants (COMEAP) and the Interdepartmental Group on Costs and Benefits (IGCB). IGCB undertook an economic analysis of the UK Air Quality Strategy using an impact pathway approach. IGCB also generated damage costs by sector, with further disaggregation for transport-related emissions according to population density (Defra, 2007).

United States – National Air Quality Standards. The US has long adopted CBA for air quality regulations and impact assessment. The USEPA has significantly developed the cost-benefit method for air pollution as part of the Benefits and Costs of the Clean Air Act (Fann et al., 2009). The general benefits analysis framework used an impact pathway approach, using detailed air quality models. The USEPA did not publish PM damage costs.

Summary of International approaches

Aspect	CAFE	UK Air Quality Strategy Review	USEPA
General approach	Impact pathway and damage cost	Impact pathway and damage cost	Impact pathway (damage costs for SO ₂ and NO _x)
Pollutants considered	Primary and secondary	Primary and secondary	Primary and secondary
Emission inventory	Various	NAEI – 11 sectors including point source, agriculture and transport	USEP NEI - point, non-point, on-road, non-road, and event
Approach for air quality	Detailed models (RAINS)	Detailed national models (plus EMEP)	Detailed air quality models (CMAQ)
Population assumptions and inputs	Detailed population and life tables	Detailed population and life tables	Detailed population and life tables
Mortality - chronic analysis of PM	PM _{2.5} , 6% hazard rate, all equally casual, no lag between exposure and effect, annual pulse, using life tables	PM ₁₀ , 6% hazard rate, all equally casual, various lag effects, life tables (UK specific), annual pulse and sustained pollution changes	PM _{2.5} , 6% hazard rate, all equally casual, lag distribution
Morbidity	Infant mortality Chronic bronchitis Respiratory hospital admissions Cardiac hospital admissions Restricted activity days Respiratory medication use Lower respiratory symptom days	Respiratory and cardio-vascular hospital admissions only	Infant mortality Bronchitis: chronic and acute Hospital admissions: respiratory and cardiovascular Emergency room visits for asthma Non-fatal heart attacks (myocardial infarction) Lower and upper respiratory illness Minor restricted-activity days Work loss days Asthma exacerbations (asthmatic population) Respiratory symptoms (asthmatic population)
Application of health functions (% of baseline rates, values per population).	Various	Baseline rates	Baseline rates
Functions used for estimating health endpoints	Pope <i>et al.</i> (1995, 2002) for chronic effects	Pope <i>et al.</i> (1995, 2002) for chronic effects	Pope <i>et al.</i> (1995, 2002) for chronic effects
Valuation of health endpoints	VSL and VOLY	VOLY	VSL
Overall economic framework	Current prices, no uplift or discounting	Current prices, then uplift at 2% per year, followed by declining discount rate starting at 3.5%	Projected real income growth (split by endpoint)

The seminal work on **Getting Energy Prices Right** embraces the principle that fiscal instruments must be center stage in “correcting” the major environmental side effects of energy use is well established. The work aims to help put this principle into practice by setting out a practicable methodology and associated tools for determining the right price. The book provides estimates, data permitting, for 156 countries of the taxes on coal, natural gas, gasoline, and diesel needed to reflect environmental costs. Underpinning the policy recommendations is the notion that taxation (or tax-like instruments) can influence behavior; in much the same way that taxes on

cigarettes discourage their overuse, appropriate taxes can discourage overuse of environmentally harmful energy sources.

The Viet Nam Energy Outlook Report 2019 assessed impact of the future energy system on air pollution and health applying benefit transfer approach. The methodology for estimating the externalities is based on the Getting Energy Prices Right report.

Understanding “Getting Energy Price Right” Report

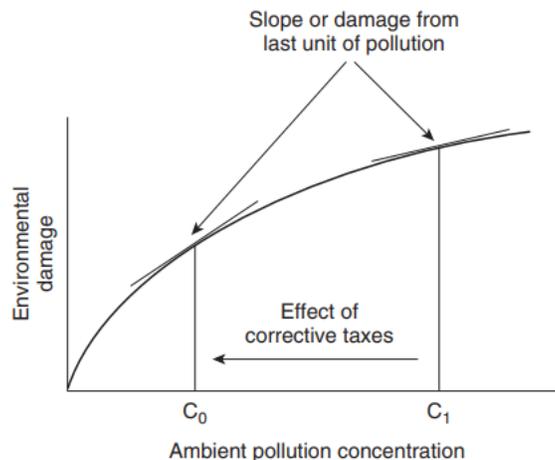
Methodology on Air Pollution Impact

The major problem from local air pollution is elevated mortality risks for exposed populations. For coal plant emissions, damage is assessed by first estimating how much pollution is inhaled by people in different countries based on combining data on power plant location with data indicating how many people live at different distance classifications from each plant (smokestack emissions can be transported over considerable distances). This pollution intake is then combined with baseline mortality rates for pollution-related illness and the latest evidence on the relationship between exposure and elevated risks, though substantial uncertainties surround this relationship. Health effects is then monetized, which is a contentious exercise, but is done for illustrative purposes using evidence on how people in different countries value the trade-off between money and risk from numerous studies analyzed in OECD (2012). Finally, damage is expressed per unit of energy content or fuel use using country-level data on emission rates. The same approach is used to measure air pollution damage from natural gas plants.

Shape of the Air Pollution Damage Function

Some evidence suggests that the relationship between environmental damage and ambient air pollution concentrations begins to flatten out at higher levels of pollution concentration (because people’s ability to take in more pollution becomes saturated). Therefore, the slope of the environmental damage function is flatter at the high pollution concentration C_1 in Figure 3.3.1 , compared with the slope at the lower concentration level C_0 . Thus, additional pollution emissions do less harm at concentration C_1 than at C_0 .

Figure: Shape of the Air Pollution Damage Function



This function might suggest that, given other factors, the corrective tax on fuel or emissions should be lower, paradoxically, in high-pollution countries. This possibility is ignored in this study, however. If corrective taxes of the scale typically estimated in this volume were to be introduced, emissions would fall dramatically, most likely lowering pollution concentrations below levels at which the damage curve might flatten out.

Measuring Pollution Damage from Fuel Use

Even though valuing climate change damage from carbon dioxide (CO₂) emissions is important considerations, here we discuss only measuring damage from the most important harm from local air pollution: human mortality risk. Climate change damage from CO₂ is dealt in a separate module.

Local Air Pollution Damage: Although local air pollution causes a variety of other harmful environmental effects, the central issue is premature human mortality. The pollution-mortality impacts from fuel combustion in this study is valued using the following steps:

- Determining how much pollution is inhaled by exposed populations, both in the country where emissions are released and, for emissions released from tall smokestacks, in countries to which pollution may be transported
- Assessing how this pollution exposure affects mortality risks, accounting for factors, such as the age and health of the population, that affect vulnerability to pollution-related illness
- Monetizing the health effects
- Expressing the resulting damage per unit of fuels.

The focus here is damage from an incremental amount of pollution rather than damage from the total amount of pollution because the incremental amount is relevant for setting efficient fuel taxes (same also applies for the PDP).

For a very limited number of countries, previous studies have estimated local air pollution damage, and major modeling efforts are ongoing at the global level. This report is the first attempt to provide an assessment of fossil fuel emissions damage across a broad range of developed and developing countries, using a consistent methodology. Although insofar as possible key country-specific factors determining environmental damage is captured, not all potentially significant factors, most notably cross-country differences in meteorological conditions affecting pollution formation, can feasibly be included.

Estimating Population Exposure to Pollution: The main cause of mortality risk from pollution considered in this report is particulate matter with diameter up to 2.5 micrometers (PM 2.5), which is small enough to permeate the lungs and bloodstream. PM 2.5 can be emitted directly as a primary pollutant from fuel combustion, but is also produced as a secondary pollutant from chemical reactions in the atmosphere involving primary pollutants, the most important of which is sulfur dioxide (SO₂), but also nitrogen oxides (NO_x).

“Intake fractions” is applied in this report. are used to estimate how much pollution from stationary and mobile emissions sources in different countries is inhaled by exposed populations. Specifically, these fractions, as used here, indicate grams of PM 2.5 inhaled per ton of primary PM 2.5, SO₂, and NO_x

For long-distance pollution, a strength of the approach used here is that it uses highly disaggregated data on population density (in different countries) up to 2,000 kilometers away from emissions sources. Therefore, the estimates of population exposure may be considerably more accurate than in other studies using much more spatially aggregated population data, or that only consider people living within shorter distances of the emissions source. A weakness is that the intake fraction approach cannot easily account for crosscountry differences in meteorological and related conditions, not least because emissions are transported across multiple climate zones and wind patterns. However, studies suggest that population exposure is usually, by far, the more important factor (Zhou and others, 2006).

Although intake fractions have been extensively estimated for emissions released at ground level for many different regions (mobile sources), estimates are much more limited for emissions released from tall smokestacks because of the complexities involved in modeling long-distance pollution transport.

The approach in this report uses a widely cited study by Zhou and others (2006), which follows a two-step statistical procedure. Using a sophisticated model of regional air quality, they start by simulating how emissions are transported to different regions, then map the result to data on regional population density, to estimate intake fractions for a variety of primary pollutants from 29 coal plants in China. Zhou and others (2006) use the California Puff (CALPUFF) air quality model, calibrated to Chinese data on regional emissions sources and pollution concentrations. This model is recommended by the US Environmental Protection Agency for estimating long-distance pollution transport (for documentation, see www.src.com/calpuff/calpuff1.htm).

From Pollution Exposure to Mortality Risk. The report applies two steps process to assess how additional pollution exposure increases mortality risk in different countries. The first step is to establish the baseline mortality rate for illnesses potentially aggravated by pollution. The second is to multiply these baseline mortality rates by estimates of the increased likelihood of mortality with extra pollution relative to mortality without extra pollution, and then aggregate over illnesses.

Much of the discussion relies on work by the World Health Organization's Global Burden of Disease project, which provides the most comprehensive assessment to date of mortality and loss of health from pollution-related and other diseases, injuries, and risk factors for all regions of the world.

Baseline mortality rates. The increased mortality risk from extra pollution inhaled by a population of given size will depend on the age and health of the population. Seniors, for example, are generally more susceptible to pollution-induced illnesses than younger adults.

The role of these factors can be summarized by calculating an age-weighted mortality rate for illnesses potentially worsened by pollution. The focus is on the four adult diseases—lung cancer, chronic obstructive pulmonary disease, ischemic heart disease (from reduced blood supply), and stroke—all of whose prevalence is increased when people intake pollution.

Annual mortality rates from these four illnesses were estimated for each country, taking into account the age structure of the population, as follows: Global Burden of Disease data provide mortality rates for the four diseases for 12 different age classifications at the regional level, with the world divided into 21 regions.

Pollution damage estimated in this volume is understated in the sense that premature deaths of those younger than 25, most notably from infant mortality, are excluded. One reason for omitting these deaths is that the valuation of mortality risk for infants is even more unsettled and contentious than that for adults.

Increased mortality from air pollution. A limited number of studies for the United States have estimated the relationship between pollution concentrations and increased mortality for pollution-related diseases—so-called concentration response functions. For example, Pope and others (2002) track the health status of a large cohort of adults in 61 U.S. cities over a long period to attribute health outcomes to PM 2.5 concentrations as opposed to other factors such as age, gender, income, dietary habits, smoking prevalence. They estimate that each 10 microgram/cubic meter increase in PM 2.5 concentrations increases annual mortality risks from all pollution-related illness in the United States by 6.0 percent. However, based on more recent evidence US EPA now assumes a 10 microgram/cubic meter increase in PM 2.5 concentrations raises all pollution-related mortality risks by 10.6 percent.

An important question is whether these findings—which are based on evidence for the United States, where PM 2.5 concentrations vary geographically by about 5–30 micrograms/cubic meter—apply to other regions.

Valuing Mortality Risks. Methodological approaches for valuing mortality risks—or more precisely, the value per premature death avoided—are discussed in this report, along with empirical evidence, and the possible implications for different countries.

Methodological approaches. Two distinct approaches are often used to assess people’s “willingness to pay” to reduce mortality risk. A third approach—generally less preferred by economists—based on valuing losses in human capital.

The “revealed preference” method uses observed market behavior to assess mortality risk values, most usually by inferring a person’s willingness to accept lower wages in return for a job with lower fatality risk (given other characteristics of jobs and workers). In contrast, the “stated preference” method relies on responses to questionnaires, most usually contingent-valuation studies in which people are asked direct questions about their money and risk trade-offs.

The human capital approach to valuing mortality risk does not (unlike willingness-to-pay approaches) measure people’s own valuation of these risks—instead it focuses on measuring productivity losses from premature mortality. Traditionally this approach has been applied to lost years of working-age life, with a person’s annual productivity proxied by market wages or per capita GDP, and productivity losses across future years discounted back to the present. However, the human capital approach may undervalue the full economic cost of premature mortality in several respects. For example, the value of lost nonwork time (i.e., time in retirement and leisure time while working age) is often excluded. And people’s valuation of pain and suffering before death are also excluded, as is grief to surviving family members. For these reasons, economists generally prefer willingness to-pay approaches.

The starting value for mortality risk valuation used in this analysis, and its extrapolation to other countries, is based on a widely peer reviewed study by OECD (2012). This extrapolation accounts for differences in per capita income across countries.

Starting value for mortality risk reduction. In OECD (2012), the central case recommendation is to value mortality risks in OECD countries as a group at \$3 million per life saved, in 2005 U.S. dollars. This amount (which is updated below) was obtained from an extensive statistical analysis using several hundred stated preference studies applied to environmental, health, and traffic risks in a variety of countries (mostly Canada, China, France, the United Kingdom, and the United States). Stated preference studies were used because they have been conducted in numerous countries, while revealed preference studies have mainly been confined to the United States (which has ample labor market data). Stated preference studies tend to produce lower valuations than revealed preference studies; therefore, pollution damage estimates might be understated here.

Income adjustment. The value for mortality risk per life for individual countries (denoted $V_{country}$) is extrapolated from that for the OECD as a whole (denoted V_{OECD}), using the following equation:

$$V_{country} = V_{OECD} \left(\frac{I_{country}}{I_{OECD}} \right)^{\epsilon}$$

In equation, $I_{country}$ and I_{OECD} denote real income per capita in a particular country and for the OECD, respectively. Relative per capita income is appropriately measured using purchasing power parity rather than market exchange rates because purchasing power parity, which takes the local price level into account, more accurately reflects people's ability to pay out of their income for local products or risk reductions. The income per capita figures are obtained from IMF (2013) and World Bank (2013).

The exponent ϵ in measures how mortality risk values vary with income; specifically, it is the percentage change in the mortality value per 1 percent change in real per capita income. Based on OECD (2012), the illustrative calculations in this analysis assume ϵ is 0.8. ²⁴ The \$3 million mortality value for the OECD is updated to 2010 for inflation (using the average consumer price index for the OECD) and real income (using equation (4.1) and the ratio of per capita income in the OECD in 2010 to that in 2005) to give $V_{OECD} = \$3.7$ million. This amount is then extrapolated to other countries, using equation and the countries' relative per capita incomes for 2010.

A tricky issue is how to value mortality risks for people across the border in other countries.

5. HANDS ON CASE-STUDY (PDP 8)

Applying benefit transfer approach to PDP 8

The estimated Vietnamese externality costs are as the following:

	NOX	SO2	PM2.5
Natural gas (2010 USD/t)	2,027	3,274	3,988
Coal (2010 USD/t)	4,060	5,823	7,243

These economic cost factors are based on the value of statistical life/mortality risk (denoted V) which is assumed to vary across economies following the relationship $V_1 = V_2 \cdot (I_1 / I_2)^{0.8}$, where I denotes the GDP per capita at PPP in two different economies 1 and 2.

The cost factors could be applied to calculate the economic cost. The formula could be used to extrapolate externality costs from 2010 to 2019 using report using the World Bank (GDP PPP) conversion factor for Vietnam, which will give the following values.

	NOX	SO2	PM2.5
Natural gas (2019 USD/t)	2,602	4,460	5,120
Coal (2019 USD/t)	5,213	7,476	9,300

These economic cost factors could be adopted to calculate impact costs per MW of future installed capacity once the emissions are estimated applying emission factors.

These original economic cost factors (2010\$) are based on premature human mortality, which is, by far, the most important category in damage assessments.

Converting 'dust' to PM2.5

The emission factor is only available for total dust. Hence conversion of emissions of 'dust' (often referred to as 'total suspended particulates', or TSP) to PM2.5, the fraction of 'dust' that is less than 2.5 micrometers in diameter is required. This conversion is necessary as the coarser fractions tend to deposit in the upper airways and do not penetrate deep into the lung. In making such estimates it is necessary to note that the fractionation of TSP is dependent on the fuel, the way that the fuel is processed, the abatement technologies in place and so on. The US Environmental Protection Agency (USEPA) provides a breakdown of emissions for different abatement technologies. Other sources are listed for comparison, though in none of these cases is it clear what abatement technology underpins the estimates.

URCE	ABATEMENT	PM2.5:TSP	PM10:TSP	PM2.5:PM10	EMISSION1
USEPA, 1998 sub-bituminous coal	Uncontrolled	6%	23%	26%	10A
	Multiple cyclones	3%	29%	10%	2A
	Scrubber	51%	71%	72%	0.6A
	Electrostatic precipitator (ESP)	29%	67%	43%	0.08A
	Baghouse	53%	92%	58%	0.02A
USEPA, 1998 anthracite coal	Uncontrolled	6%	23%	26%	10A
	Multiple cyclones	24%	55%	44%	2A
	Baghouse	32%	67%	48%	0.02A
USEPA, 1998 lignite	Uncontrolled	10%	35%	29%	6.6A
	Multiple cyclones	27%	67%	40%	1.3A
Huang et al, 2014 coal	Not stated	10%	26%	38%	
Huang et al, 2014 lignite		10%	35%	29%	
SCAQMB, 2006 coal		15%	40%	37.50%	
UK NAEI, 2015 coal				44%	

Source: Health and Environment Alliance, 2016, Health Impacts of Coal Fired Power Stations in the Western Balkans