

METROPOLITAN POLLUTION AND THE IMPACT ON LABOUR EFFICIENCY

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Abstract

The present environment around metropolitan areas because of industries, transport, etc. brings a tremendous amount of pollution that impacts the health of population but also the efficiency of its labour capacity.

Key words: Pollution, labour efficiency, health, metropolitan areas

INTRODUCTION

The paper presents estimation of the health losses and specially labour efficiency from metropolitan air pollution.

The methodology developed by US EPA and adjusted for Eastern European transition countries was applied for health risk assessment. PM_{2.5} more than P.M 10 was identified as the major source of human health risk, based on experience from the USA and EU studies. In the absence of reliable computed concentrations of PM_{2.5}, the study was based on monitoring data of total suspended particle (TSP) emissions. Additional cases of mortality and morbidity were calculated based on reporting data on TSP concentration that was recalculated into PM_{2.5}.

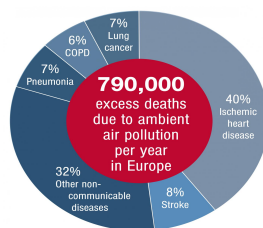


Fig. 1 Estimation of the health losses
source: medicalxpress.com.

Then the concentration–response function was applied to estimate individual risk. Next, individual risk was applied to the population exposed to the concentration reported for each metropolitan areas. For each metropolitan areas it has to considered individual data on baseline mortality and morbidity, population structure, labour efficiency, etc..

In total, air pollution related mortality represents about 6 percent of total mortality around the world.

Since applied method is sensitive to the primary data uncertainties we conducted sensitivity analysis applying Monte-Carlo method. Economic damage related to mortality risk was estimated at about 4 percent of GDP. There was no relevant WTP study in therefore we applied the benefit-transfer method in order to estimate VSL, since mortality attributed to air pollution is major component of health losses (about 94 percent). In order to compare and aggregate mortality and morbidity risks we recalculated them in DALY. Then morbidity represents about 30 percent of total air pollution health load. Data on baseline morbidity is less reliable than data on baseline mortality; therefore the morbidity risk estimates are more uncertain than mortality estimates. It is likely that morbidity risk is underestimated. Regardless of uncertainties mentioned above and some problems with reported data we can conclude that the mortality risk attributed to air pollution is significant. Therefore, costs of air pollution are sizable and in the nearest future may offset the economic growth.

Recovery of the economy based on restoration of polluting industries may lead to stagnation since mortality and morbidity risks not only puts burden on the economy, but also reduce labor force.

MATERIAL AND METHOD

Fossil fuel combustion is the main source of PM_{2.5} AND PM₁₀ pollutants. Unfortunately, levels of PM₁₀ or PM_{2.5} are not monitored on a reliable basis in many countries. Hence any assessment of the impacts of the particles has to be based on what is reliably monitored, which is total suspended particles or TSP, and the link between TSP and PM₁₀ or PM_{2.5}.

In this context it is worth noting that huge amount of effort goes into monitoring a large number of pollutants (more than 35) in the FSU countries. The purpose of undertaking such extensive monitoring was to set emissions standards for individual emitters so that actual concentrations of 1 PM₁₀ is particulate matter less than 10 microns in diameter; similarly PM_{2.5} refers to matter less than 2.5 microns in diameter. Small particles, however, are also formed from chemical interactions of SO₂ and other pollutants with ozone. Hence emissions of these pollutants are also important contributors to health impacts. 3 pollutants did not exceed certain health determined maximum allowable concentrations (MACs).

In practice, however, the MACs were not substantiated by practical methods and techniques of air pollution control. It was impossible to attain the desired accuracy of analytical control, to use adequate instrumentation, numerical estimation methods, unit emissions, technological standards,

emission control requirements. Neither was there was adequate financing or professional staffing. The improvements in these areas are very slow.

A large number of epidemiological studies provide evidence that exposure to air pollution is associated with increased morbidity and mortality (WHO, 2004). The most affected are respiratory and cardiovascular systems. The mechanisms “may involve decrements in pulmonary function, effects on heart rate variability and inflammatory response”. Also, there is an evidence of carcinogenicity of some components of urban air pollution. Both acute and chronic biological responses are affected by air pollution, since acute responses exacerbate the severity of chronic diseases. Epidemiologic literature proposes to use Cox proportional hazards model for the long term health risk estimation. Basically, they have the following form:

$$yC = - [y e C] pop B * (-\beta * \Delta - 1) * (1)$$

where:

yC is incremental number of cases of negative health outcome (morbidity or mortality);

ΔC is the change in mean population-weighted annual concentration of criteria pollutant²;

β is concentration-response coefficient;

yB is baseline level of the health outcome;

² PM pollution could be used as an indicator of pollution mix.

⁴ pop is exposed population to which it is appropriate to apply β (the same as in the epi studies, where β was estimated).

For small changes in the annual mean criteria pollutant concentration, it is appropriate to use a linear relationship between incremental health outcome and change in annual mean criteria pollutant concentration:

$$yC = C y pop B \beta * \Delta * * (2)$$

Then, β is concentration-response coefficient that reflects change in health outcome per unit of pollution (slope of concentration-response function).

Air pollution and mortality

For PM_{2.5} pollution, β values were developed for all cause mortality, cardiopulmonary mortality, and lung cancer mortality (Pope et al., 2002) Then β is the per cent change in health outcome per unit of pollution (i.e. the slope of concentration-response function).

It is appropriate to use β from epidemiological studies, when pollution in the focus area is in the range observed in the study used for the estimation. For example, WHO recommends to apply Pope coefficients for PM_{2.5} pollution in the range of 7.5-50 $\mu\text{g}/\text{m}^3$ PM_{2.5}. Beyond 50 $\mu\text{g}/\text{m}^3$ the β value is set at zero.

Experts agree that based on the current status of worldwide research, the risk ratios, or concentration response coefficients from Pope et al (2002) are likely to be the best available evidence for the mortality effects of ambient particulate pollution (PM 2.5). This study provided a global estimate of the health effects of environmental risk factors including health risk from environmental pollution. It was the American Cancer Society study within the framework of

Cancer Prevention II prospective study of risk factors for mortality, where 1.2 million Americans from 50 metropolitan areas 30 and older were involved. This study concentrated on long-term exposure to air pollution from fine particulates (PM2.5) that are the most harmful for human health and include sulfates and nitrates. Long-term pollution is more important than short-term, because it include the effects of long-term exposure that can not be captured by a short-term study. The participants were observed for about 16 years. The study controlled for age, sex, weight, height, smoking, alcohol use, occupational exposure, diet, education, marital status, etc. As a result the study came up with the list of concentration-response coefficients, which identify additional risk of non-accidental death, cardio-pulmonary and lung-cancer mortality.

If our goal is to assess total health risk caused by air pollution, one should take into account the difference between observed mortality and baseline mortality. From formula (1) above, y_B should be derived for the baseline situation if we would like to have y_B associated with the ΔC ambient concentration levels (of PM2.5, for example). If y is defined by the equation (2) (choosing a linear specification over the relevant range of C):

$$C B y = \beta * \Delta C * y \quad (3)$$

The baseline y_B however, is not directly observed, and is given by:

$$B C y = y - y_0 \quad (4)$$

where y_0 is the observed or recorded number of all cause non accidental or cardiopulmonary and lung cancer deaths. Substituting equation (4) in equation (3) provides the following solution for

y_B :

$$*() * / \{ 1 * () \} 0 y C y C C = \beta \Delta + \beta \Delta \quad (5)$$

We have applied Pope's all cause non accidental mortality coefficient $\beta = 0.004$ per $1 \mu\text{g}/\text{m}^3$ of PM2.5. If PM2.5 concentration is above $50 \mu\text{g}/\text{m}^3$, the value was set at $50 \mu\text{g}/\text{m}^3$. Since the Pope estimates apply only to persons over the age of 30, this share had to be estimated.

Air pollution related morbidity

Although available information on mortality is quite reliable, morbidity information is not. Therefore, we had to apply the method proposed by Ostro (1994) to estimate respiratory hospital admissions,

emergency room visits, restricted activity days, lower respiratory illness in children and respiratory symptoms. For chronic bronchitis we applied the approach from Abbey et al (1995). Method from Ostro (1994) doesn't require baseline morbidity. Thus it is applicable even with poor primary data about background morbidity indicators. Abbey's approach requires a baseline chronic bronchitis morbidity. Official data on chronic bronchitis were provided by the Ministry of Public Health of Ukraine. Both studies Ostro (1994) and Abbey (1995) link exposure to PM10 air pollution with additional morbidity end-points. For air pollution related cases of chronic bronchitis we applied the formula similar to (5), where y_c is additional number of chronic bronchitis and y_0 is observed number of cases for Euro B region. For other morbidity end-points we applied the following formula, as in Ostro (1994):

$$Y_c = \beta * C, \quad (6)$$

Where C is observed PM10 concentration and β is concentration-response coefficient.

The burden of health impacts is converted to monetary terms by valuing mortality and morbidity. Valuation is based on robust willingness to pay studies that quantify the value of human health risk reduction. These valuation studies have not been done in any other FSU country. Therefore the only method to apply for valuation is a benefit transfer approach. The physical estimates of mortality and morbidity can be converted in monetary values under certain assumptions. 3 Studies on the valuation of health effects of outdoor air pollution outside the OECD countries are rare. Recent work along this lines, using some benefit transfer has been undertaken in China (Eliason and Lee, 2003), in Russia (Bobilev, 2002) and Peru (Larsen, 2005).

CONCLUSIONS

This paper tried to show that metropolitan areas have more considerable health and mortality costs in human and monetary terms associated with air pollution. At a conservative estimate these costs amount to 4.6 million excess deaths lost annually. ***In monetary terms, we estimate the costs at around 5 trillion \$ from 88.00 trillion\$ the total world GDP or 18.5% of it almost double the economic annual average growth o 9.5%***

Studies in the EU of similar costs, but using much more detailed data and a more sophisticated modeling of the dispersion of air pollution and the creation of particles, comes up with air pollution costs from similar items in the range of 2 percent (Markandya and Tamborra, 2005). At the same time, the level of effort devoted to addressing it is much lower. Public and private sector spending on investment in air pollution control is very small (World Bank, 2003). Studies like these provide a useful guide to where efforts

should be made to reduce air emissions (the focus needs to be on particulate pollution control in certain cities we have identified), and how the air pollution problem compares with other sources of morbidity and mortality (it is more serious, for example, than most social causes of death and more serious than TB). This is not something that is generally appreciated or acted upon.

The paper also demonstrates how the analysis can be done using limited and uncertain information. Therefore, estimates presented in the paper were complemented by sensitivity analysis. Limited data on air pollution is not enough to develop a detailed action plan for environmental costs burden alleviation, however, it is a good way to draw attention to environmental problems ignored by now. Thus environmental degradation may soon become a significant barrier for economic growth and can not be ignored by policy makers.

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