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IMPACT OF AIRBORNE PARTICULATE MATTER ON HUMAN HEALTH: AN ASSESSMENT FRAMEWORK TO ESTIMATE EXPOSURE AND ADVERSE HEALTH EFFECTS IN POLAND

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Abstract: Fine particulate matter ($PM_{2.5}$) air pollution is one of the main environmental health problems in developed countries. According to modeling estimates the $PM_{2.5}$ concentrations in Poland are among the highest in Europe. In this article we focus on exposure assessment and estimation of adverse health effects due to $PM_{2.5}$ air pollution. This article consists of two parts. In the first part, we discuss the main methods used to estimate emission-exposure relationships and adverse health effects due to $PM_{2.5}$ air pollution. In the second part, we present an assessment framework for Poland. We illustrate this framework by estimating the premature deaths and change in life expectancy in Poland caused by anthropogenic, primary $PM_{2.5}$ emissions from different European countries, and, in proportion, the premature deaths in different European countries caused by primary $PM_{2.5}$ emissions from Poland. The $PM_{2.5}$ emissions were evaluated using the inventory of the European Monitoring and Evaluation Programme (EMEP). The emission-exposure relationships were based on the previously published study and the exposure-response functions for $PM_{2.5}$ air pollution were estimated in expert elicitation study performed for six European experts on air pollution health effects. Based on the assessment, the anthropogenic primary $PM_{2.5}$ from the whole of Europe is estimated to cause several thousands of premature deaths in Poland, annually. These premature deaths are both due to $PM_{2.5}$ emissions from Poland and transportation of $PM_{2.5}$ from other European countries, both of these in almost equal parts. The framework presented in this article will be developed in the near future to a full scale integrated assessment, that takes into account both gaseous and PM air pollution.

INTRODUCTION

The harmful impact of air pollution on human health has been noticed for centuries [43]. Hundreds of epidemiological studies in the 1990s and 2000s have indicated that the current air pollution levels are capable of harming public health [1]. In particular, the par-

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ticulate matter (PM), and especially the fine ($PM_{2.5}$) and ultrafine particles, have been associated with a number of adverse health effects [e.g. 53]. The assessment studies have estimated that the fine particulate matter causes annually over 800 000 premature deaths worldwide [9], and 350 000 in Europe alone [73]. Thus, PM air pollution is one of the major environmental health problems in both the developed and the developing world.

Substantial achievements have been made since the mid 20th century in abating the ambient air pollution. For example, the recent changes in legislation and the economical system in Eastern Europe have reduced PM precursors and primary PM emissions by approximately 45% in the 32 European Economic Area countries between the years 1990–2004 [13]. However, the European Economic Area report concluded that apart from the reduction in emissions, the ambient PM concentrations have not decreased since 1997 [13]. Thus, it seems that the abatement actions have not been sufficient or effective to protect human health in the ambient environment.

Assessment methods for PM air pollution have been developed and recommended by several organizations. For example, the global update of the World Health Organization (WHO) air quality guidelines in 2005 provided values for various air pollutants, including PM, and reviewed the assessment methods for the use of risk assessment and policy analysis [35, 75]. The exposure-response functions for PM air pollution, that describe the relationships between exposure and related health effects, have been defined and discussed, for example in the WHO report concerning burden of disease caused by outdoor air pollution [50], or the European Externalities of Energy (ExternE) project [18]. The ExternE methodology was further updated in 2007 in a joint exercise of several European cost-benefit analysis projects [67]. Also the development of European Regional Air Pollution Information and Simulation model (RAINS) ([8] in this issue) for the Clean Air for Europe (CAFE) program has involved a number of expert meetings and panels focusing on assessment methods [e.g. 70, 74]. In Poland the assessment methods have been discussed by Juda-Rezler [31].

The goal of this paper is twofold. Firstly, we address the basic problems and methods related to the assessment of the emission-exposure relationship and adverse health effect due to particulate matter in ambient air. Secondly, these methods are illustrated by estimating the health impacts of particulate matter air pollution caused by different European countries in Poland and vice versa. The assessment framework presented in this article will be updated in future to estimate the adverse health effects caused by both gaseous and PM air pollution in Poland. This article is partly using material from the PhD dissertation of the principal author [62].

METHODS FOR ESTIMATING EXPOSURE AND HEALTH EFFECTS FOR PM AIR POLLUTION

Definition of PM air pollution

The solid and liquid particles suspended in the air are commonly referred to as particulate matter (PM). PM can be emitted or formed from a number of primary sources and secondary processes; both the physical and chemical properties of PM can vary widely,

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in terms of the pollutant source, and the formation and transformation processes during the atmospheric transport. PM is commonly categorized based on the aerodynamic size of the particle. In a regulatory context, the two most commonly used categories are thoracic particulate matter with an aerodynamic diameter less than 10 μm (PM_{10}), and the fine particulate matter with a diameter less than 2.5 μm ($PM_{2.5}$). Other commonly used fractions are ultrafine particulate matter (UF or $PM_{0.1}$) and total suspended particulate matter (TSP).

The primary PM is emitted into air directly from sources, while secondary PM is formed in the atmosphere through physical and chemical processes, from precursor gases. The precursor gases include sulphur dioxide, nitrogen dioxide, ammonia, anthropogenic volatile organic compounds (VOC) and biogenic VOC [76]. Primary PM can be formed directly through mechanical grinding, or in various nucleation processes, and can grow by condensation of gaseous compounds on the particle surface [e.g. 16, 76]. During coagulation, the particles are attached to each other, thus decreasing in number and increasing in size. Clearly, due to the processes of condensation and coagulation, the PM inhaled by people has a different chemical composition, size and physical characteristics compared with the PM originally emitted into the atmosphere.

Integrated assessment of PM air pollution

The integrated assessments, and other assessment methods like risk assessments, cost-benefit analyses or environmental health impact assessments, are used to describe integrated procedures, where scientific information is systematically collated and synthesized to aid decision making. The integrated assessment process aims to cover all the relevant interactions between society and the environment. It is typically based on mathematical models. They provide quantitative estimates, like, e.g., the number of premature deaths due to air pollution emissions.

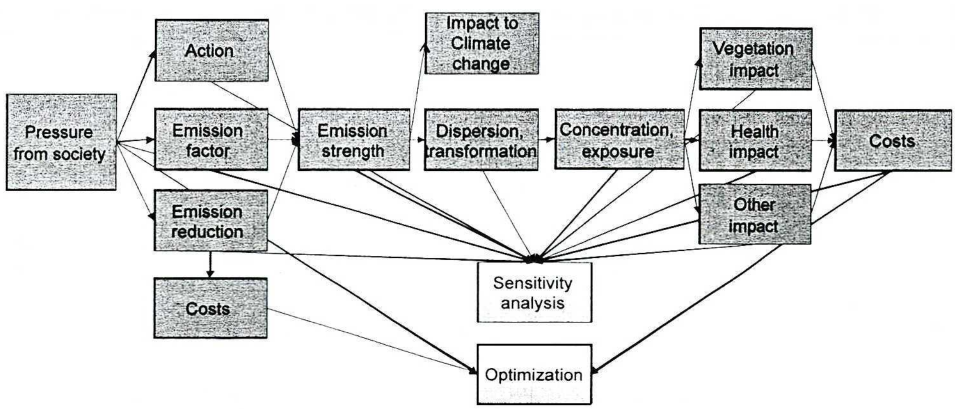

Fig. 1. A general integrated assessment framework for $PM_{2.5}$ air pollution; dark grey boxes present the causal chain of impacts and light grey boxes analyses performed within the framework; in this article, we focus on health impacts and on estimation of emission-exposure relationships for PM; figure based on Tainio *et al.* [62]

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A general integrated assessment framework for $PM_{2.5}$ air pollution is presented in Figure 1. The $PM_{2.5}$ air pollution is emitted from a number of source categories, the most important of which are in many cases traffic and energy production [76]. The $PM_{2.5}$ air pollution is dispersed through the ambient air and causes adverse health effects to humans, damages vegetation, and has other detrimental effects. The most comprehensive integrated assessment model for $PM_{2.5}$ air pollution in Europe is the Regional Air Pollution Information and Simulation (RAINS) model, developed by International Institute for Applied Systems Analysis (IIASA) (http://www.iiasa.ac.at/rains/) [8].

Assessing exposure to anthropogenic $PM_{2.5}$

Exposure can be defined as the contact of an individual to a concentration of a pollutant in the breathing zone during a specified time. Breathing zone is the volume, where people inhale the air. The $PM_{2.5}$ concentration in the breathing zone consists of particles from different emission sources that can originate from local or long-range transported distances. Since people spend most of their time indoors, also most of the PM are inhaled indoors. However, most of the integrated assessment studies use the ambient concentrations of $PM_{2.5}$ as a proxy of exposure, both outdoor and indoor. The $PM_{2.5}$ penetrates easily indoors through normal gas exchange between outdoor and indoor; outdoor and indoor concentrations are therefore in many cases close to each other. This simplification can nevertheless have a substantial impact on results. Various emission sources emit $PM_{2.5}$ of a varying particle size distribution. The size is a crucial factor in determining the extent of penetration of PM indoors. Moreover, although indoor $PM_{2.5}$ emission sources have only a minor impact on ambient concentrations, they have major impact on indoor concentrations and exposures.

The exposure due to specific $PM_{2.5}$ emission source categories (e.g. traffic, power plants, domestic combustion) can be estimated with a dispersion method or a receptor-analysis modeling method. Dispersion modeling methods use atmospheric models to estimate the transport, diffusion and scavenging of PM in ambient air after its release. For example, the van Zelm *et al.* [79] study used dispersion models to evaluate PM_{10} concentrations over Europe. Receptor-analysis methods are based on a set of PM measurements at a specified receptor location, combined with a statistical analysis using characteristic source tracer profiles. The location can be, e.g., a permanently located monitor in a city or a personal monitoring device. For example, exposure in the APHEA study was estimated based on $PM_{2.5}$ and PM_{10} measurements in a number of European cities [5].

Atmospheric dispersion models

Atmospheric dispersion models estimate the dispersion of pollutants in time and space. The atmospheric dispersion models require various sets of input data, such as, for example the locations and strengths of the emission sources, various meteorological datasets, and land-use and terrain data. The models subsequently evaluate the advection and diffusion of the pollutants, their chemical and physical transformation, and the removal of the air pollutants from the atmosphere (deposition). For a review of different modeling systems, the reader is referred to, e.g., the paper by Juda-Rezler [32] or Support Center for Regulatory Atmospheric Modeling of EPA (http://www.epa.gov/scram001/).

The effective spatial and temporal resolutions of the dispersion model depend on the resolutions of the input data (those of the emission data, meteorological fields, and other data), and on the computational grid. The spatial and temporal resolutions are crucial, when the exposure to different $PM_{2.5}$ emission source categories is to be evaluated. The dispersion modeling systems used in PM studies are often divided into urban and regional/continental (possibly also global) scale systems, based on spatial scale. The regional scale dispersion models predict long-range dispersion of the PM on the national or continental scale [e.g., 80]. Although such models can predict air pollutant concentrations far away from release locations (e.g., in a different country), the concentrations predicted nearing the vicinity of the emission sources (less than a few or a couple of tens of kilometers) is often underestimated, especially for low height emission sources. The dispersion models often assume that the emissions are distributed evenly inside any single emission grid cell, the size of which can characteristically be tens of kilometers in evaluations on a European scale. When sources have a high spatial correlation with the population, this underestimation of concentrations will also result in an underestimate of the population exposure.

The urban-scale dispersion models evaluate the dispersion of air pollutants in smaller geographical areas, such as one urban area, with a smaller grid size than the regional scale dispersion models. In this respect, urban-scale models can evaluate better the spatial variation over short distances. However, the large continental level integrated assessment involves sources in hundreds of cities and implementing an urban-scale dispersion model for all of these cities is currently not feasible. Moreover, the urban scale dispersion models alone are unable to predict PM concentrations due to long-range sources. Therefore many urban scale studies utilize a variety of strategies to incorporate the long-range transported PM into the model results. A good solution is to apply a multi-scale modeling system. For example, Stein *et al.* [61] and Gariazzo *et al.* [19] have combined the results of regional and urban scale models.

Dispersion models are the most common method to estimate exposure or emission-exposure relationships for various emission sources in assessment studies. For example, Levy and Spengler [41], Levy *et al.* [42] and Wyrwa [78] used dispersion models to estimate exposure and adverse health effects due to $PM_{2.5}$ emissions from power plants.

Receptor-analysis models

Receptor models rely on $PM_{2.5}$ measurements performed at a receptor location (e.g., an urban monitoring station). The source categories of measured PM can be traced by comparing the chemical properties of PM with information on emission source profiles using statistical source apportionment methods [25, 65]. The receptor approach has been used especially in epidemiological studies to compare the toxicity differences between different types of PM [e.g. 39, 44].

The advantage of receptor methods is that the $PM_{2.5}$ concentrations at the receptor location are known with sufficient accuracy. The main limitation is the possible misidentification of emission source categories in the source apportionment. The variation in results between different source apportionment methods was studied in U.S. in 2003 by comparing source apportionment methods between different research groups and between methods [25, 65]. The study concluded that the selection of the source apportionment method did not confer any significant uncertainty to the results [65]. With respect to the main source categories, emissions from traffic and burning vegetation had the greatest uncertainty. On the other hand, the methodological review of Grahame and Hidy [21] noted several disadvantages of the source apportionment method. Their main critique was that the source identification varies between the methods used and the location of emissions. Thus, with the receptor approach alone, it is difficult to draw conclusions on what and where emission sources or source categories should be abated. The reliability of the predictions of receptor analysis are also critically dependent on the quality and amount of the experimental data used.

The estimation of exposure in geographically extensive integrated assessment studies is impractical with receptor methods. The measurements of PM are conducted mainly in cities and the estimation of $PM_{2.5}$ concentrations is rarely done in rural areas. Also, applying source apportionment method so that it includes chemical analyses from hundreds of measurement stations is both time consuming and expensive. The receptor based exposure assessment fits best to a geographically limited area, in which there is a sufficiently densely spaced network of PM measurement stations.

Receptor methods have been used especially to estimate exposure to traffic related PM. Hutchinson and Pearson [27] used receptor method to estimate the health effects of traffic in the United Kingdom and Tainio *et al.* [64] to estimate the health effects of local buses in Helsinki Metropolitan Area, Finland.

The intake fraction concept

The dispersion models generate large amount of data that need to be summarized and incorporated into the integrated assessment model. The most common way is to estimate source-receptor relationships. The source-receptor relationship describes the change in the pollutant concentration (receptor) in relation to emission strength (source). The intake fraction (*iF*) concept [4] is an application of the source-receptor relationship. The *iF* is defined as an "*integrated incremental intake of a pollutant released from a source category and summed over all exposed individuals*" [4].

For $PM_{2.5}$, *iF* can be calculated from the following equation, when using outdoor concentration of $PM_{2.5}$ as a proxy of the population exposure:

$$iF = \frac{BR}{Q}\sum_i C_i \cdot Pop_i \tag{1}$$

where *iF* is the intake fraction; *BR* is the average breathing rate [$m^3 \cdot day^{-1} \cdot person^{-1}$]; Q is the emission strength [$g \cdot s^{-1}$]; C_i is the modeled concentration increase of $PM_{2.5}$ in a grid cell i [$g \cdot m^{-3}$]; and Pop_i is the population number in the grid cell i. A breathing rate of 20 $m^3 \cdot day^{-1} \cdot person^{-1}$ is generally used in $PM_{2.5}$ *iF* studies [e.g. 72] based on a past EPA recommendation [14]. The number of the grids cells (i) depends on the scale and the resolution of the assessment. Large integrated assessments may have hundreds of thousands of cells.

The exposure E (i.e. population weighted average concentration in the study area) to $PM_{2.5}$ can be calculated in the integrated assessment using equation:

$$E = \sum_i C_i \frac{Pop_i}{Pop} = \frac{Q \cdot iF}{Pop \cdot BR} \tag{2}$$

In $PM_{2.5}$ integrated assessments, the exposure, and *iF*, is usually estimated for annual average concentrations.

The *iF* concept has several benefits in integrated assessments [17]. First, the *iF* concept allows for the validation of results between exposure studies. The *iF*s for similar source categories should have fairly similar values; typical for outdoor air pollutants, like $PM_{2.5}$, between 10 per million to 0.1 per million [3]. Second, the *iF* allows for rapid adoption and use of *iF* estimates from previous studies. This enables comparison of health risks from a number of sources in early assessment and then concentrating further efforts on those sources, health effects, and uncertainties, which have a major impact on assessment results.

The *iF* concept has been used in a number of $PM_{2.5}$ exposure studies. For example, Levy *et al.* [40] illustrated the exposure to $PM_{2.5}$ and precursor gas emissions from individual power plants in the US using the *iF* concept. Zhou *et al.* [81] estimated *iF*s for power plants and Wang *et al.* [72] for industrial processes in China. Marshall and Behrentz [45] used *iF* to estimate the passengers' exposure to vehicle emission. Greco *et al.* [22] estimated spatial pattern of the *iF* of vehicle emissions in the city of Boston in the U.S.

Exposure-response function for $PM_{2.5}$

The exposure-response function describes the change in the background health effect caused by the change in the exposure level. $PM_{2.5}$ has been associated in epidemiology and toxicology with a number of adverse health effects [e.g. 53, 59]. The World Health Organization (WHO) concluded in 2003 that long-term exposure to $PM_{2.5}$ may reduce life-expectancy due to cardiopulmonary and lung cancer mortality [74]. In addition, $PM_{2.5}$ can evoke lower respiratory symptoms and reduced lung function in children, and cause chronic obstructive pulmonary disease (COPD) and impaired lung function in adults [74]. The mechanisms causing adverse health effects are incompletely understood, although several plausible mechanisms have been identified [53].

The exposure-response functions for PM are usually derived from epidemiological cohort studies that have studied correlations between $PM_{2.5}$ concentrations over a long time period (years) and health effects [e.g. 12, 52]. The integrated assessment studies that are based on exposure-response functions from these epidemiological cohort studies use typically annual $PM_{2.5}$ concentrations in their assessment. The integrated assessment on $PM_{2.5}$ has also focused on long term mortality impact because the major part of adverse health and economical impacts of PM are due to it [e.g. 15] in comparison to other adverse health effects (e.g. morbidity).

The long-term epidemiological cohort studies

A number of epidemiological studies have been undertaken to examine the effect of long-term exposure and mortality for $PM_{2.5}$ [53] for estimating the value of the relative risk (*RR*). Relative risk is calculated with equation:

$$RR = \frac{P_1}{P_0} \tag{3}$$

In this equation, P_1 is the probability of health effects among those that were exposed (in this case exposed to the defined dose of $PM_{2.5}$) and P_0 probability of health effect among those who were not exposed or were in a lower exposed population group. The main epidemiological cohort studies for $PM_{2.5}$ are co called Harvard Six Cities (HSC), American Cancer Society (ACS) and Dutch cohort studies. The main characteristics and results from these studies are described in Table 1.

Table 1. Comparison of different long-term epidemiological studies for $PM_{2.5}$, the results from different studies have been scaled to the same exposure level with Monte-Carlo methods (ACS = American Cancer Society, HSC = Harvard Six Cities, CI = confidence interval) Table copyright Tainio *et al.* [62]

Study	Percent change in all cause mortality per annual average 1 $\mu g \cdot m^{-3}$ change in $PM_{2.5}$ concentration (mean and 95% CI)	$PM_{2.5}$ concentration range in the study [$\mu g \cdot m^{-3}$] (min-max)	Number of people in the analyses
ACS [55]	0.64 (0.33–0.93)	9.0–33.5	295 223
ACS reanalysis [34]	0.68 (0.37–0.96)	9.0–33.5	295 223
ACS update [52]	0.58 (0.15–1.00)	5.0–30.0**	319 000
ACS Los Angeles [30]	2.17 (1.05–3.20)	6.0–30.0**	22 905
HSC [12]	1.25 (0.34–2.04)	11.0–29.6	8111
HSC reanalysis [34]	1.34 (0.42–2.13)	11.0–29.6	8111
HSC update [39]	1.50 (0.63–2.30)	10.2–29.0	8096
Dutch cohort [24]*	2.74 (-1.21–5.66)*	9.6–35.8*	4 492
Dutch cohort update [2]	0.58 (-0.36–1.45)	23.0–36.8	117 528

* the effect is for black smoke
** based on visual inspection of figures in the article

The implications from these epidemiological studies have been reviewed and discussed in tens of publications [e.g. 53, 67]. The exposure-response estimates differ substantially between the studies with the mean mortality increase due to 1 $\mu g \cdot m^{-3}$ $PM_{2.5}$ exposure varying from 0.58% to 2.74% (Tab. 1). Pope and Dockery [53] discussed two possible explanations for this phenomenon. First, as noticed in the reanalysis of HSC and ACS studies, education seems to modify the mortality impact so that those individuals with higher education have lower mortality risk [34]. The education level in ACS cohort is higher than in HSC cohort, so the lower mortality increase in ACS study in comparison to HSC could be partly due to differences in the level of education of the cohort population. Second, the exposure estimates differ significantly between studies. In general, studies that have used finer spatial resolution to relate people to air pollution levels (HSC, ACS Los Angeles, and Dutch cohort) tend to report higher mortality impacts.

The HSC, ACS and Dutch cohort studies have concentrated on the adult population. Several epidemiological studies have also examined the association between PM and mortality in infants (age less than one year old) [see e.g. reviews 20, 60, 66]. These reviews concluded that there are some evidence for an association between PM levels and different mortality outcomes but many methodological weaknesses may have modified the results.

Expert judgment studies

Expert judgment (elicitation of expert judgment) provides a method to assess and combine scientific information [10]. In an expert judgment study, several experts are formally asked to answer some particularly interesting questions (exposure-response function of $PM_{2.5}$ in this case). The experts then provide, based on their knowledge, the best guess and uncertainty intervals for their estimates. Two expert judgment studies have examined the relationship between $PM_{2.5}$ exposure and mortality impact [11, 28, 29, 57, 69].

The U.S. Environmental Protection Agency (EPA) has prepared a pilot and full study to characterize uncertainty in $PM_{2.5}$ exposure-response function for mortality [28, 29; 57].

The pilot study was performed with five experts from whom questions about both short-term and long-term mortality impacts due to $PM_{2.5}$ exposure were asked. The five experts estimated that 1 $\mu g \cdot m^{-3}$ change in $PM_{2.5}$ exposure would change median non-accidental mortality in U.S. from 0% to 0.7% [28]. The uncertainty was recognized as being high.

After the pilot study, the EPA performed an expert judgment study with twelve experts [57]. The study concentrated solely on long-term mortality and involved more detailed questions concerning the shape of the exposure-response function, confounding, threshold, and causality. In that study, the individual experts' median estimates for the change in non-accidental mortality due to 1 $\mu g \cdot m^{-3}$ change in $PM_{2.5}$ exposure varied from 0.4% to 2.0% [29]. In general, the experts in this study estimated a higher mortality response to $PM_{2.5}$ exposure than pilot study. This was explained as being due both to changes in the assessment protocol as well as new epidemiological evidence published after the pilot study (especially Jerrett *et al.* [30] and Laden *et al.* [38] studies). However, uncertainty was again recognized as being high.

The second expert judgment study was performed for six European air pollution experts [11, 69]. In this study, the experts provided quantitative estimates of mortality impacts of hypothetical short- and long-term changes in $PM_{2.5}$ concentrations in the U.S. and Europe, as well as of several other variables. The expert's estimates were then combined based on calibration of questions. The median change in mortality due to 1 $\mu g \cdot m^{-3}$ change in $PM_{2.5}$ exposure was 0.60% or 0.97% in U.S. and 0.62% or 0.98% in Europe, depending on the method of combining expert's answers [69]. In general, experts were considering the uncertainties to be much higher than those reported in epidemiological studies. The experts also estimated that exposure-response function for $PM_{2.5}$ is higher than that observed in cohort studies.

Toxicity differences

Ambient $PM_{2.5}$ is emitted from a number of sources, and it has different chemical and physical characteristics, depending on the source. It is assumed that these differences modify the toxicity of PM so that particles with different chemical composition or different physical characteristics (e.g. size, shape) have different toxicity.

The toxicity differences between different PM sources have been investigated in three time-series studies in U.S. [37, 44, 68]. Laden *et al.* [37] used the elemental composition of $PM_{2.5}$ to identify the sources of measured PM and then related the PM concentration to variation in daily mortality. They concluded that the sources from both traffic and coal combustion were associated with mortality while crustal sources were not important. Mar *et al.* [44] and Tsai *et al.* [68] used factor analysis and Poisson regression to estimate source-specific risk ratio for $PM_{2.5}$. Mar *et al.* [44] concluded that the combustion-related pollutants and secondary sulphate PM were associated with mortality. Tsai *et al.* [68] detected a statistically significant association to PM from oil burning, industry, sulphate PM and traffic. However, Grahame and Hidy [21] pointed out that the identification of long-range transported sources was dependent on the source-apportionment method and therefore might lead to biased estimates.

In Europe, toxicity differences between sources have been studied in the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study [51]. In the ULTRA study, a panel of elderly subjects was visiting biweekly a clinic where a number of health indicators were measured and recorded. Lanki *et al.* [39] compared the

$PM_{2.5}$ exposure to an ischemic marker in the electrocardiogram (ST-segment depression) in Helsinki, Finland. The $PM_{2.5}$ was apportioned to five source categories using absolute principal component analysis with multivariate linear regression based on both PM and gaseous air pollutant concentrations [71]. In the epidemiological analysis, the local traffic and long-range transported PM were associated with ST-segment depression [71]. In a recent article from the same study comparing data from three cities (Amsterdam, the Netherlands, Erfurt, Germany, and Helsinki, Finland,), the conclusion was that the traffic and long-range transported $PM_{2.5}$ were associated with health outcomes [23].

There are also epidemiological studies where a change in legislation or some other intervention has rapidly decreased the PM concentration in a specific location. A study in Dublin, Ireland, noticed a reduction in mortality after banning of the coal sale in the city area [7]. Another study compared the health effects and air pollution in Utah Valley, U.S., during a strike in a large steel mill and found that the all-cause mortality was correlated with PM_{10} concentrations [54].

The toxicity of different source categories was also addressed in the European elicitation study of expert judgment [11, 69]. As part of the study, experts were asked to give mortality impact estimates for the least and the most toxic components of PM mixture and to define those elements. All experts identified that combustion-related PM, especially from traffic, were more toxic than the average PM mixture and that secondary PM (sulphate, nitrate or both) and crustal material were less toxic than the average PM. The uncertainties were recognized to be high. The toxicity differences were also discussed in the review of New Energy Externalities Developments for Sustainability (NEEDS) project that developed exposure-response functions for PM and ozone [67]. The review concluded that current evidence is not strong enough for quantification of toxicity differences between PM properties or sources.

In the 2007 WHO workshop in Bonn, Germany [77], the evidence on exposure and toxicity differences of different PM sources was discussed. The conclusion was that the current scientific knowledge does not provide sufficient information to separate the toxicities of different PM sources from one another. However, it was acknowledged that the evidence is strong for major combustion sources.

Measures of public health

Several measures of public health have been developed to express the change in population health status due to exposure to stressors. For example, McAlearney *et al.* [47] reviewed 13 different health measures including life-expectancy, quality-adjusted life-years (QALY), disability-adjusted life-years (DALY), health-adjusted life-expectancy, and healthy days gained. The review did not include the most common measure, i.e. premature death. Integrated assessments use these measures of public health in order to express the change in population health status due to exposure to environmental stressors. The selection of the measure depends on the environmental stressor, availability of data, computer resources, and skill.

Premature death

The premature death (mortality) measures the change in mortality due to exposure to environmental stressor. Other terms for premature death are avoidable death [e.g. 33] and attributable cases [e.g. 36]. The mortality after the exposure M can be expressed as:

$$M = M_b(1+DRI) \tag{4}$$

where M_b is the baseline mortality and DRI is the death rate increase due to particulate matter concentration. Taking into account that DRI is small; the premature death due to $PM_{2.5}$ exposure can be also estimated with the equation:

$$M = M_b \cdot \exp(\mathrm{DRI}) = M_b \cdot \exp(\beta \cdot \Delta E) \tag{5}$$

with $DRI = \beta \cdot \Delta E$, where β is the exposure-response coefficient, ΔE change in $PM_{2.5}$ exposure. The β can be estimated from the risk ratio (RR) with the equation:

$$\beta = \frac{\ln RR}{\Delta E_r} \tag{6}$$

where RR is the risk ratio and the ΔE_r is the change in $PM_{2.5}$ concentration to which RR has been related. The premature death can be estimated for all mortality outcomes combined or separately for different mortality outcomes (e.g. lung cancer and cardiopulmonary mortality).

The premature death measure has been criticized [6, 56]. The authors argued that premature death is misleading because the measure does not provide any information on how premature is the actual death. Thus, it does not distinguish between a case where death is advanced by one day from the situation of one year, or one decade. Rabl [56] also concluded that the premature death is not meaningful because the number of deaths from different stressors would exceed the total observed mortality and because the number of people dying due to air pollution exposure cannot be measured.

Despite these criticisms, the premature death is widely used in integrated assessments because of its easy intelligibility and the availability of data. Other requirements in integrated assessment such as economical valuation also favor premature death, as discussed by the CAFE cost benefit analysis team [26].

Life expectancy

The life expectancy measure has been supported by most premature death critics [e.g. 56]. Life-expectancy is a statistical measure of the average life span of a population and it takes into account the age when adverse effects occur. For example, one infant death due to exposure to $PM_{2.5}$ leads to a reduction of almost 80 years of life, while a heart attack at the age of 50 will lead to a reduction of 30 years. The life-expectancy can be estimated with life tables that express the probability of surviving over the next age interval [48].

The life tables are based on hazard rates which describe the probability of an event during a given time interval. The hazard rate is estimated with the equation [48]:

$$H_b = \frac{m}{pop} \tag{7}$$

where m is a number of deaths in a time interval (e.g. one year) and pop is the number of population in the same time interval. Thus, $1 - H_b$ defines the probability to survive over the time interval. The hazard rates can be subdivided to, e.g., different mortality outcomes, or different sexes. The environmental stressors affect the life expectancy estimates by multiplying hazard rates with the relative risks due to a given exposure.

The most common life expectancy measure is the life expectancy at birth. It is estimated by calculating hazard rates based on population and mortality data from the birth year, assuming that the hazard rates remain constant over the lifespan of the population. More sophisticated methods take into account the change in hazard rates over the time, e.g. by adopting the mortality projections from WHO [46]. Conditional life expectancy can be estimated for different age groups or taking into account population age structure.

The estimation of life expectancy requires more time and data than the premature death measure. First, the life table requires information on both population and mortality statistics at a more detailed level than premature death measure (e.g. mortality divided into one year intervals). These statistics are readily available at the national level, for example from WHO and UN databases, but for smaller geographical areas (e.g. cities) the data may be inadequate. Second, the life table models require more computational efforts than the premature death measure, which may hamper their usefulness in decision support systems.

Adjusted health measures

Adjusted health measures (also known as weighted health indicators) measure the change in population health status by combining different health effects into one measure. The main benefit of adjusted health measure is the combination of mortality and morbidity effects. Two common adjusted health measures are the "quality adjusted life year" (QALY) and the "disability adjusted life year" (DALY) [47, 58].

The QALY measure combines the life expectancy and the quality of the life. The QALY defines the quality of life by using so-called quality of the life weight factors. These weight factors are based on individual's feeling of their quality of life and can have a value between 1 (full health) and 0 (death) [58]. A number of QALY's gained in one year is simply the quality factor, i.e.:

$$\mathrm{QALY} = Q \tag{8}$$

where Q is the quality weight based on the individual's health status. This equation can be combined with the life table calculations so that both life expectancy and the QALY are estimated for each time interval.

The DALY measure resembles QALY in many ways. The main difference between QALY and DALY is the interpretation of weighting factors. In QALY, the weighting factor is based on quality of life enjoyed by individuals, whereas the DALY weighting factor represents the loss of functioning caused by a disease [58]. The DALY weights are scaled from 1 (death) to 0 (no disability). The DALY weights are usually based on expert valuations while QALY weights are based on measurement sampled from the population [58]. The DALY measure have been developed and applied especially in the Global Burden of Disease study [49].

THE APPLICATION OF METHODS IN CASE OF POLAND

In this chapter, the methods presented in previous chapters will be applied by estimating premature deaths and change in life expectancy in Poland due to primary $PM_{2.5}$ emissions from Poland and elsewhere in Europe. Also estimates are computed of the premature deaths in Europe due to primary $PM_{2.5}$ emissions originated from Poland. These calculations are based on previously published data; we have not used any high-resolution emission or dispersion computations in the case of Poland (only those on a European scale). The assessment framework presented in the following paragraphs will be used in future to estimate the adverse health effects of both gaseous and PM air pollution by using high-resolution emission and dispersion computations.

The emission-exposure relationships for $PM_{2.5}$ air pollution

The emission-exposure relationships for the primary anthropogenic emissions of $PM_{2.5}$ for different European countries were adopted from Tainio *et al.* [63]. In that study, emission-exposure relationships for European anthropogenic primary $PM_{2.5}$ emissions were estimated and intake fractions were used to illustrate these relationships. Short description of the study is provided below.

The atmospheric dispersion of $PM_{2.5}$ originated from different European countries was evaluated using the dual-core Lagrangian-Eulerian regional and continental scale dispersion model SILAM (http://silam.fmi.fi), for the $PM_{2.5}$ emissions in 2000. The emissions of $PM_{2.5}$ were based on European Monitoring and Evaluation Programme (EMEP, http://www.emep.int/) data and the concentrations due to emissions were estimated with a horizontal resolution of approximately 30 km over the whole of Europe. The intake fractions were estimated by combining the concentrations with the population (using the Equation 1 of this article). The population data were prepared for each European country so that *iF*s could be estimated for population of each country. The matrix showing *iF*s that correspond to the emissions of various European countries exposing the populations in various European countries is presented in the additional file of Tainio *et al.* [63].

In Table 2 *iF*s are presented for primary anthropogenic $PM_{2.5}$ emissions from Poland based on Tainio *et al.* [63]. For example, the interpretation of *iF* equal to 0.18 per million in the case of Ukraine means that on the average for every gram of $PM_{2.5}$ emitted in Poland, 0.18 μg is inhaled in Ukraine. The average exposure of the populations (*Pop*) in different countries due to primary $PM_{2.5}$ emissions from Poland is also presented in Table 2. The values of *iF* and *Pop* required for the equation 2 are included in Table 2. For Br and Q the same values were used as in Tainio *et al.* [63], i.e. Br = 20 $m^3 \cdot day^{-1} \cdot person^{-1}$ and $Q = 5\ 500\ g \cdot s^{-1}$.

By using *iF*s, as those in Table 2, we can separately address the exposures that result from the emissions from individual countries to populations of different countries. With the same approach, we can divide exposure in one country to emissions from different countries. As well, the *iF* can be estimated for different source categories (traffic, power plants) [e.g. 63].

Premature deaths and the change in life expectancy

The premature deaths in different European countries caused by anthropogenic primary $PM_{2.5}$ emissions from Poland, evaluated using Equation 5, are presented in Figure 2. As expected, the major fraction of premature deaths due to Polish emissions occurs in Poland. The background non-accidental mortality statistics for different European countries was adopted from the World Health Organization (WHO) mortality database (http://www.who.int/healthinfo/morttables/en/). For the exposure-response function we assumed,

Table 2. The intake fractions (per million) for anthropogenic primary $PM_{2.5}$ emissions originated from Poland in 2000 (the intake fraction and population numbers are based on Tainio *et al.* [63], the population average exposure has been calculated in the present article, the countries have been ordered starting from highest *iF*)

Country	*iF* for primary $PM_{2.5}$ emissions from Poland (per million)	Population of the country (million)	Population average exposure [$\mu g \cdot m^{-3}$]
All countries	2.14	703.8	0.07
Poland	1.23	38.0	0.78
Ukraine	0.18	47.8	0.09
Germany	0.12	81.9	0.04
Russia	0.08	68.0	0.03
Czech Republic	0.07	10.2	0.16
Belarus	0.06	10.0	0.15
Romania	0.06	22.2	0.06
Slovakia	0.04	5.4	0.20
Hungary	0.04	10.2	0.09
Turkey	0.03	66.9	0.01
Italy	0.03	55.0	0.01
Lithuania	0.02	3.4	0.15
United Kingdom	0.02	57.7	0.01
Serbia	0.02	10.6	0.04
Sweden	0.02	8.5	0.04
Moldavia	0.01	4.3	0.08
Netherlands	0.01	15.8	0.02
Austria	0.01	8.0	0.04
Denmark	0.01	5.1	0.05
Bulgaria	0.01	8.0	0.03
France	0.01	57.8	0.00
Greece	0.01	10.1	0.02
Latvia	0.01	2.2	0.08
Belgium	0.01	10.3	0.01
Croatia	0.01	4.0	0.03
Bosnia	0.00	3.9	0.03
Finland	0.00	5.2	0.02
Albania	0.00	3.1	0.02
Norway	0.00	4.1	0.01
Slovenia	0.00	2.0	0.02
Estonia	0.00	1.3	0.03
Macedonia	0.00	2.0	0.02
Switzerland	0.00	7.2	0.00
Ireland	0.00	3.6	0.01
Spain	0.00	38.7	0.00
Luxemburg	0.00	0.4	0.01
Cyprus	0.00	0.6	0.01
Malta	0.00	0.3	0.01
Portugal	0.00	9.6	0.00

based on [69] that the change in non-accidental mortality due to 1 $\mu g \cdot m^{-3}$ change in $PM_{2.5}$ exposure is 0.98%. Differential toxicity (in terms of various emission source categories) was not taken into account in these calculations.

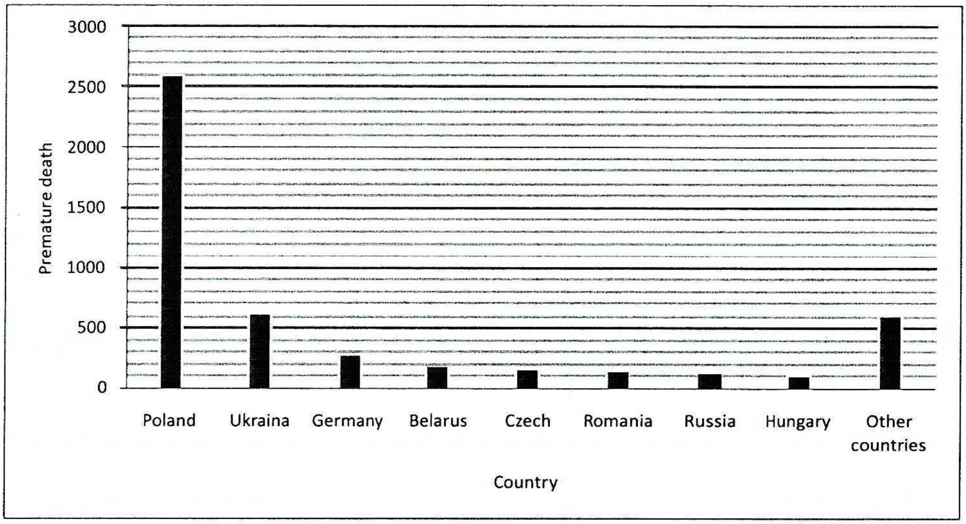

Fig. 2. The number of premature deaths in different European countries caused by primary anthropogenic $PM_{2.5}$ emissions originated from Poland

Premature death contributions in Poland are presented in Figure 3, due to primary anthropogenic emissions of $PM_{2.5}$ originated from various European countries. The primary anthropogenic $PM_{2.5}$ is estimated to cause several thousands of premature deaths in Poland in 2000. According to these computations, approximately half of all premature deaths in Poland are due to anthropogenic primary $PM_{2.5}$ emissions from Poland itself. The European Clean Air for Europe (CAFE) assessment has estimated that $PM_{2.5}$ exposures cause in 2000 approximately 33 000 premature deaths in Poland [73]. Clearly, the primary anthropogenic $PM_{2.5}$ exposure constitutes only a fraction of all health effects caused by the exposure to both primary and secondary anthropogenic $PM_{2.5}$.

The change in life expectancy in Poland due to anthropogenic primary $PM_{2.5}$ exposure in Poland was estimated with the life table model. The life table model is presented in Table 3. The hazard rates for different age intervals are based on WHO mortality database and to year 2000 mortality and population data. The mortality outcomes have been divided to accidental and to non-accidental mortality. The exposure for $PM_{2.5}$ is estimated to increase the non-accidental mortality. In the left-hand-side of Table 3, we show the life table based on the WHO data. In the right-hand-side of Table 3, we have enhanced the hazard rates due to non-accidental mortality by assuming that hazard rates would be lower, if people were not exposed to anthropogenic primary $PM_{2.5}$. The difference of these two life tables, 0.21 years (2.5 months), represents the loss of life expectancy due to anthropogenic primary $PM_{2.5}$ exposure in Poland.

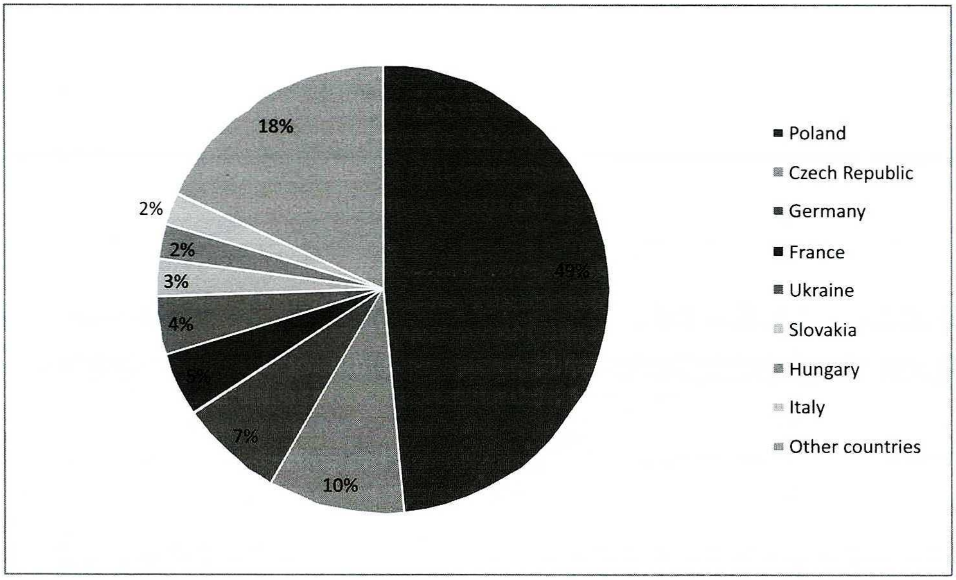

Fig. 3. The percentage contributions of various countries, caused by their emissions of primary anthropogenic $PM_{2.5}$, to premature deaths of the population in Poland; the total premature mortality is estimated to be approximately 5000; the contribution of Poland itself is 49%, that of Czech Republic is 10%, and the other countries have been listed clockwise

CONCLUSIONS

We have discussed and illustrated several methods that can be used to estimate adverse health effects caused by $PM_{2.5}$ air pollution. $PM_{2.5}$ is a major environmental problem in Poland and abatement actions are required to reduce the adverse health effects. We have first discussed methods to estimate emission-exposure relationships and adverse health effects due to $PM_{2.5}$ and then presented an assessment framework that can be used to estimate $PM_{2.5}$ induced adverse health effects in Poland. This framework will be used in future to develop an integrated assessment model for air pollution in Poland.

The approximate results obtained indicate that the anthropogenic primary emissions of $PM_{2.5}$ caused several thousands of premature deaths in Poland in 2000, and lowered the population life expectancy by approximately 2.5 months. The emissions from Poland are responsible for almost 50% of these premature deaths. Contributions from other countries depend on their primary emissions, emission categories (e.g., release heights) and on the prevailing wind directions and other meteorological conditions. For instance, Ukraine, the second largest emitter in Europe and a neighbor of Poland, contributes only 4% to the health impact in Poland regarding primary anthropogenic $PM_{2.5}$, and is only fifth on the contribution list, and Russia, the largest emitter in Europe, contributes less than 2%. The Czech Republic and Germany, with much smaller emissions, are the second and third on the contribution list. Emission of $PM_{2.5}$ from Poland affects mainly Poland itself, but then its close neighbors: Ukraine, Germany, Russia, Czech Republic, Belarus, and Romania. Also here the influence of prevailing West wind directions can be clearly noticed.

Table 3. The life expectancy estimates for Poland: the left-hand-side of table presents life expectancy in Poland including the adverse health effects caused by anthropogenic primary $PM_{2.5}$, the right-hand-side of table presents life expectancy without exposure to anthropogenic primary $PM_{2.5}$, life tables are for year 2000 and are based on WHO mortality database

	Life table with exposure to anthropogenic primary $PM_{2.5}$					Life table without exposure to anthropogenic primary $PM_{2.5}$			
Time interval (age)	Hazard rate, non-accidental mortality	Hazard rate, accidental mortality	Population at the beginning of time interval	Died during the time interval	Lives lived	Hazard rate, non-accidental mortality	Population at the beginning of time interval	Died during the time interval	Lives lived
0 to 4	0.0017	0.0001	378 348	3 461	1 883 086	0.0017	378 348	3 407	1 883 222
5 to 9	0.0001	0.0001	374 887	336	1 873 592	0.0001	374 941	333	1 873 871
10 to 14	0.0002	0.0001	374 550	507	1 871 484	0.0002	374 608	502	1 871 783
15 to 19	0.0004	0.0003	374 043	1 189	1 867 244	0.0003	374 106	1 178	1 867 583
20 to 24	0.0004	0.0003	372 854	1 327	1 860 954	0.0004	372 927	1 315	1 861 350
25 to 29	0.0007	0.0003	371 527	1 890	1 852 909	0.0007	371 613	1 869	1 853 390
30 to 34	0.0010	0.0003	369 637	2 487	1 841 967	0.0010	369 743	2 457	1 842 574
35 to 39	0.0022	0.0004	367 150	4 878	1 823 554	0.0022	367 286	4 813	1 824 399
40 to 44	0.0029	0.0005	362 272	6 148	1 795 989	0.0029	362 473	6 064	1 797 208
45 to 49	0.0058	0.0005	356 124	11 291	1 752 392	0.0057	356 410	11 129	1 754 226
50 to 54	0.0074	0.0005	344 833	13 650	1 690 039	0.0073	345 281	13 458	1 692 758
55 to 59	0.0137	0.0005	331 183	23 550	1 597 038	0.0135	331 822	23 221	1 601 059
60 to 64	0.0171	0.0005	307 633	27 103	1 470 406	0.0168	308 601	26 755	1 476 119
65 to 69	0.0306	0.0006	280 530	43 784	1 293 188	0.0301	281 847	43 280	1 301 034
70 to 74	0.0431	0.0009	236 746	52 056	1 053 588	0.0423	238 567	51 611	1 063 806
75 to 79	0.0929	0.0021	184 689	87 748	704 077	0.0913	186 956	87 396	716 289
80 to 84	0.0929	0.0021	96 941	46 058	369 562	0.0913	99 560	46 541	381 447
85 to 89	0.0929	0.0021	50 883	24 175	193 979	0.0913	53 019	24 785	203 133
90 to 94	0.0929	0.0021	26 708	12 689	101 817	0.0913	28 234	13 199	108 175
95 to 99	0.0929	0.0021	14 019	6 660	53 443	0.0913	15 036	7 029	57 607
Sum					26 950 308				27 031 032
				Life expectancy (years):	71.23			Life expectancy (years):	71.44

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WPŁYW PYŁÓW W POWIETRZU ATMOSFERYCZNYM NA ZDROWIE LUDZKIE: METODOLOGIA OCENY EKSPOZYCJI I SZKODLIWYCH SKUTKÓW ZDROWOTNYCH W POLSCE

Zanieczyszczenie powietrza drobnym pyłem ($PM_{2.5}$) jest jednym z głównych problemów zdrowotnych związanych ze środowiskiem. Wartości stężeń $PM_{2.5}$ w Polsce znajdują się wśród największych w Europie. W tej pracy skupiono się na ocenie ekspozycji ludzi na $PM_{2.5}$ oraz na oszacowaniu szkodliwych skutków tego zanieczyszczenia. Artykuł składa się z dwóch części. W pierwszej części przedstawiono podstawowe metody estymacji zależności ekspozycji od emisji i wyznaczania szkodliwych skutków spowodowanych zanieczyszczeniem powietrza drobnymi pyłami. W drugiej części przedstawiono zarys modelu zintegrowanego do oceny szkodliwości drobnych pyłów dla Polski. Jest on ilustrowany oszacowaniem liczby przedwczesnych zgonów i zmianą oczekiwanej długości życia w Polsce spowodowanymi antropogenną emisją pierwotnych drobnych pyłów w krajach europejskich oraz odwrotnie, liczbami przedwczesnych zgonów w krajach europejskich spowodowanych emisją pierwotnych drobnych pyłów w Polsce. Emisje $PM_{2.5}$ oceniono na podstawie inwentaryzacji dokonanej w ramach European Monitoring and Evaluation Programme (EMEP). Zależność ekspozycji od emisji oparto na wynikach wcześniej publikowanych badań, a odpowiedź na ekspozycję na zanieczyszczenia $PM_{2.5}$ oceniono na podstawie ocen zebranych od sześciu ekspertów europejskich zajmujących się zdrowotnymi skutkami zanieczyszczenia powietrza. Z przeprowadzonej oceny wynika, że antropogenna emisja pierwotnych drobnych pyłów w Europie powoduje w Polsce kilka tysięcy przedwczesnych zgonów rocznie. Są one wynikiem zarówno emisji w Polsce, jak i transportu pyłów z innych krajów europejskich, mniej więcej w równych częściach. Przedstawiona w artykule konstrukcja będzie rozwijana w celu uzyskania zintegrowanej oceny w pełnej skali, obejmującej zarówno zanieczyszczenia gazowe jak i pyły.