Raport Badawczy

Research Report

RB/2/2017

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Warszawa 2017

Burden of Disease due to multiple air pollutants emitted from urban sources in Warsaw, Poland

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Abstract

Air pollution is a significant public health issue all over the world, and especially in urban areas where local emissions can impact a large number of inhabitants. In this study, we quantify the health burden due to local air pollution for the Warsaw, Poland. From different air pollutants we considered particulate matter (PM), nitrogen oxides (NOx), sulfur dioxide (SO_2), benzo(a)pyrene (BaP) and heavy metals. The annual mean concentrations were predicted with CALMET/CALPUFF modeling system using year 2012 emission and meteorological data. The emission fields comprised high (power generation) and low (industry) point sources, mobile sources (transport) and area sources (housing). The exposure to these pollutants was estimated using population data with spatial resolution of 500 × 500 m^2. Changes in mortality and in disability-adjusted life-years (DALYs) were estimated with relative risk functions obtained from literature. The local emitted air pollution was predicted to cause approximately 1600 attributable deaths (95% confidence interval: 1100 to 2000) and 29 000 DALYs (95% CI: 21 000 to 36 000) per year. Most of the health effects (80%) were due to exposure to fine particulate matter (PM2.5). Mobile sources and area sources contributed 46% and 52% of total DALYs, respectively. When the inflow from outside was included, the burden nearly doubled to 51 000 DALYs (95% CI: 39 000 to 62 000 DALYs). These results indicate that local decisions can potentially improve air quality and reduce associated negative health effects, but the national-level policy is required for reducing the strong environmental impact of PM emissions.

Keywords: air pollution; exposure; mortality; disability-adjusted life years (DALY); health impact assessment

1. INTRODUCTION

The ambient air pollution causes one of the biggest environmental health challenges in many Global cities. According to World Health Organization (WHO)[1], air quality in the majority of urban agglomerations – especially in low- and middle-income countries – do not meet the respective air quality guidelines[2]. Air pollutants, and fine particulate matter ($PM_{2.5}$) in particular, are emitted into the atmosphere from many sources and cause a multitude of environmental and health effects. Some of the health effects (stroke, heart disease, lung cancer, chronic and acute respiratory diseases, including asthma) are mainly caused by fine fractions of particulate matter[3]. A high concentration of this type of air pollution is estimated to cause more than 3 million premature deaths worldwide each year[1].

As many other European agglomerations, Warsaw also suffers from high concentrations of air pollutants which are typical of the urban environment. These include particulate matter, sulfur dioxin (SO_2), nitrogen oxides (NOx), carbon monoxide (CO), benzo(a)pyrene (BaP), heavy metals (Pb, As, Cd, Ni), as well as polycyclic aromatic hydrocarbons (PAHs). In practice, the adverse impact of some particular pollutants on urban air quality depends on several individual factors, such as the city location, topography, the structure of the emission field, meteorology, etc. In Warsaw, the composition of the main polluting species, their spatial distribution and their maximum values also reflect the peculiar structure of the local emission field, which is determined by two dominating factors.

The first factor relates to coal, which is the main fossil fuel used in Poland for power generation and for the residential heating, Holnicki *et al.*[4]. Majority of Warsaw is covered by the district heating system, but in some peripheral districts and the neighboring area the coal fired small scale heating installations are used, which considerably contribute to worsening of air quality. This category of emission sources is responsible for particulate matter pollution (especially $PM_{2.5}$), SO_2, some heavy metals and BaP. The BaP pollution, which mainly

originates from the municipal sector, exceeds the limit value of the annual mean BaP concentration (EEA[3]) in the whole area of the Warsaw agglomeration.

The second factor relates to the key air pollution category, traffic. For example, in last decade number of cars registered in Warsaw increased 80% [4]. This trend is different from many other European cities, but representative for many global cities in low and middle income countries. Traffic originated emission is mainly responsible for NO_X, CO, benzene (C_6H_6) and partly Pb concentrations, but it also contributes to PM_{10} pollutions, mainly via the re-suspended particles (Dimitriou and Kassomenos[5], Kiesewetter et al.[6]). In particular, basing on the reports Holnicki and Nahorski [7], Holnicki *et al.* [4], related to the years 2005 and 2012, respectively – concentrations of NO_X and PM_{10} have been on the increase during the last decade and both exceed the annual average concentration limits.

The external inflow of some pollutants originating from distant sources also contributes significantly to the resulting air pollution in Warsaw, which mainly relates to the fine fractions of particulate matter, as shown in ETC/ACM[8], Wang *et al.*[9], Levy[10].

This study quantifies health burden caused by air pollution in Warsaw. We first estimate population average exposure for multiple air pollutants (particulate matter (PM), NO_X, SO_2, CO, C_6H_6, BaP and heavy metals (Pb, As, Cd, Ni)), and then predict changes in attributable deaths and disability-adjusted life-years (DALYs) due to this exposure. The modeling results of air pollution, which are utilized in an analysis of the negative health effects, are presented in more details in Holnicki *et al.*[4,11] and are not repeated in this study. Here we only recall the main assumptions and findings relevant for predicting health burden.

2. METHODS

2.1. The study area and spatial resolution

The base of this study is an air quality analysis for Warsaw agglomeration in the year 2012, presented in Holnicki *et al.*[4,11]. To simulate pollution dispersion processes, the

Gaussian puff model CALPUFF v.5, Scire *et al.*[12], was applied. It is a multilayer, non-stationary model designed for calculating concentrations of many substances, emitted by different types of sources. Meteorological fields are re-analyzed by the Weather Research and Forecasting Model (WRF) model, NCAR (National Center for Atmospheric Research)[13], and assimilated to the final resolution grid by the CALMET cooperating preprocessor. The aim of the simulation was to obtain the spatial maps of the year average concentrations of the main urban pollutants, to show districts/areas where the pollution limits are exceeded and to identify emission sources responsible for these violations. See Holnicki and Nahorski [7] and Holnicki at al.[4,11] for uncertainty estimates and assessment of the model performance.

2.2. The structure of the emission field

The Warsaw Metropolitan Area – about 520 km^2 within the administrative borders and total population 1715517 inhabitants[14] in the year 2012 – is shown below in Fig. 1. The study area is discretized for the numerical analysis with the homogeneous grid 0.5 × 0.5 km^2. To take into account specific technological characteristics of the different emission sources, the total emission field was split down into the following categories of sources: point (high and low emission height separately), area, and line (mobile). A separate class of the high point sources comprises the power/heating plants which operate within the district heating system, comprising the main part of the agglomeration. Thus, the aggregate emission field consists of the following categories, including the quantity of the individual sources in each category:

- High point sources (24) – energy generation;
- Low point sources (3880) – industrial plants;
- Area sources (6962) – residential combustion;
- Line sources (7285) – urban road traffic;
- Boundary conditions (the inflow of some pollutants due to the regional/national level emission based on the EMEP model results).

The total emission field encompasses the Warsaw area in the administrative borders and the surrounding belt of approximately 30 km wide (see Fig. 1b). The locations of the point sources are identified by the geographical coordinates. The area and line sources are represented as basic grid emission squares, 0.5 × 0.5 km^2, inside Warsaw administrative borders (Fig. 1a), and also in the aggregated grid, 1 × 1 km^2, in the surroundings (Fig. 1b). The local city areas in the suburban region are also represented by the nested fine resolution grid, as shown in Fig. 1b.

The computed annual mean concentrations of the polluting compounds listed in Table I have been recorded at 2248 receptors points, which coincide with the central points of the spatial resolution elements shown in Fig. 1a.

2.3. Concentrations and population weighted exposure of air pollutants

The annual mean concentrations of NO_X, PM_{10}, $PM_{2.5}$ and BaP exceed the EU limit values (CAFE[2] or ME[15]) in some districts[4]. The respective concentration maps are shown in Fig. 2. The other compounds listed in Table I, do not violate the air quality standards, but they also contribute to the final adverse health effects. Quantification of these effects is based on the population average concentration (exposure) of pollutants considered. Fig. 3 presents the population density map of Warsaw. The map for the year 2005 was previously used in Tainio *et al.*[16], and has now been modified for the year 2012 according to EEA[17], GUS[14] and Warsaw[18] data. The spatial resolution applied in the population density map is the same as that used in the forecasting model computations (0.5 × 0.5 km^2). Legend on the map represents a number of inhabitants in one elementary resolution square.

The population average exposure (E) for the individual emission sources and the specified pollutant, was calculated with the following formula:

$$E_{k,j} = \frac{1}{Pop} \sum_i C_{i,k,j} \bullet Pop_i \qquad (1)$$

where $E_{k,j}$ – exposure, $C_{i,k,j}$ – concentration, Pop_i – receptor population, i – receptor index, j – pollutant index, k – emission source index within emission category. The aggregated exposure index for each pollutant within the emission category is obtained by summing up in (1) with respect to the emission sources k

$$E_j = \frac{1}{Pop} \sum_k \sum_i C_{i,k,j} \bullet Pop_i \quad (2)$$

2.4. Estimation of health burden

The health risks due to air pollution were quantified following the methods described in Tainio[19]. For gaseous air pollutants and metals the health risks were estimated for an individual pollutant. For the particulate matter the health risks were calculated separately for two size fraction: $PM_{2.5}$ and $PM_{2.5\text{-}10}$. Thus, we assumed that the toxicity of the particles varies between primary ($PM_{2.5}$) and coarse ($PM_{2.5\text{-}10}$) fraction of the PM, but not between the source or chemical composition.

The calculation of the health risks followed the methods described in the supplementary material on Tainio[19] paper for $PM_{2.5}$, $PM_{2.5\text{-}10}$, SO_2, NOx, BaP, Cd, Ni and Pb. We updated the concentration-response function for non-accidental mortality for $PM_{2.5}$ following the recommendation of the World Health Organization (WHO), Héroux et al.[20]. The resulting relative risk (RR) used was 1.062 with the 95% confidence interval inform 1.040 to 1.083. For As, C_6H_6 and CO we used the following concentration-response functions. For CO we adopted CRF from Hosseinpoor *et al.* [21] study that estimated angina pectoris admissions in Tehran, Iran, for multiple pollutants. The RR was used in this study to estimate changes in ischemic heart disease related risks. For the As we adopted unit risk from Erraguntla *et al.*[22]. The unit risk for cancer mortality was estimated based on the three epidemiological studies, and the resulting unit risk factor was 1.5E-04 per $\mu g/m^3$. For C_6H_6

we adopted the unit risk value for leukemia from Hänninen and Knol[23] study that estimate the burden of disease due to environmental stressors in Europe.

Two measures of health were used, that are the number of attributable deaths and disability-adjusted life-years (DALY). The advantage of DALY measure is that it combines mortality and morbidity impact to one measure of health, allowing us to compare e.g., mild mental retardation, caused by Pb, with the increased mortality, caused by $PM_{2.5}$.

For the background DALY and mortality data, we used the year 2013 Global Burden of Disease country file for Poland[24] (Table II). The data were adjusted from national to Warsaw population by using age and gender specific differences in the population as a guide.

The health calculations were done with the Monte Carlo simulation program Analytica (Lumina Decision Systems, Inc.), version 4.6. Uncertainty was propagated through the model with 50,000 iterations.

3. RESULTS

3.1. Concentration and exposure

The spatial distribution of exposure values for selected pollutants, representing the line and area emission categories in the considered domain, is shown in Fig. 4. All the sources presented are split down into two groups: those located inside the square domain indicated in Fig. 1b (blue dots) and those located outside this square (red dots). The aim is to assess the share of emission sources located outside the close vicinity of Warsaw. For the line emissions (left panels) the dominating share of the intra-urban sources is seen, including high-traffic roads in the close vicinity of Warsaw. For the area emissions, the share of the intra-urban sources is low, mainly due to residential emission of the peripheral districts (right panels). On the other hand, a significant contribution of the sources located in a direct vicinity of Warsaw (blue color) can be observed. This is not only due to their emission intensity, but also results

from a coarse spatial resolution in this case, where each emission source is represented by an element of 1×1 km^2 instead of 0.5×0.5 km^2 for other sources.

The trans-boundary inflow contributes significantly to the final exposure. Aerosols $SO_4^=$ and NO_3^- are the secondary pollutants (Table I) where the share of the local sources is minor, mainly due to the time which is required to aerosol formation. The contribution of the aerosol's inflow from distant sources is greater, due to longer times when they are transported and transformed in the atmosphere. Also contribution of the inflow particulate matter, which contains aerosols as components, is considerable.

The population weighted concentration (exposure) for the studied air pollutants is presented in Table III for four local emission categories and the trans-boundary inflow from distant emission sources. For most pollutants, local emission sources caused larger exposure than external inflow outside the study area. The main exceptions were secondary sulfate and nitrate aerosols for which inflow contribution was dominating in the resulting exposure (76% for $SO_4^=$ and 81% for NO_3^-). Due to the spatially limited receptor area, the time interval when the local pollutants remain in the domain is too short for complete transformation. The time required for aerosol formation is a key factor in this case. Almost half of the exposure is also due to external inflow in the cases of $PM_{2.5}$ and CO.

3.2. Health burden

Air pollution was estimated to cause approximately 2800 (95% confidence interval: 2100 to 3500) attributable deaths per year in the study area (Table IV) and 51 000 (95% CI: 39 000 to 62 000) DALYs (Table V). Approximately 82% of the total attributable deaths are due to $PM_{2.5}$ air pollution and 16% due to NOx. About 1% of the deaths were due to all other pollutants. Air pollution influx outside the study area caused 45% of the deaths and local emissions 55% (Table IV). Of local emission sources the area sources (residential) were most

important one, followed by the line sources (traffic) (Table IV). Point sources (high and low combined) caused about 1% of the attributable deaths. The DALY results presented in Table V were similar to attributable deaths with most of the DALYs (84%) due to PM air pollution, followed by NOx (14%). From the morbidity outcomes, chronic bronchitis (COPD) caused highest health burden.

4. DISCUSSIONS

In this study, we quantified the health burden due to air pollution in the city of Warsaw, Poland, with high resolution of 0.5 km x 0.5 km. The emission field comprises the city territory, the area surrounding the city with the diameter of 90 km, as well as the pollution inflowing from outside of the study area. Several pollutants typical of the town atmosphere were considered, including particular matters, oxides, heavy metals, as well as benzene and benzo(a)pyrene. Due to differentiated land use characteristic of the town and different types of urban development, the pollution is very diverse in different town quarters, hence the high resolution estimation improves the quality of results. To quantify the health effect, the health risk was calculated for each individual pollutant.

4.1. Meaning of the study

The study provides important background information for developing mitigation strategies for air pollution in Warsaw. The magnitude of the health burden, 2800 deaths per year and 51 000 DALYs per year indicate that air pollution is a significant environmental health problem in Warsaw. These present 15% and 9% of all deaths and DALYs in the study area, respectively (Table II). Approximately 45% of the attributable deaths were due to air pollution inflow outside the Warsaw and 55% due to local emissions sources, indicating that local, national and international mitigation strategies are required to reduce the health burden. From local sources, the area sources, representing residential emissions, caused 47% of the

burden and the linear sources (traffic) 50%. This clearly indicates that local mitigation actions should target these two emission categories, while point sources had a nearly insignificant direct impact to health burden.

The study also provides information on the relative weight of different air pollutants for causing health risks, and the health outcomes they cause. For both attributable deaths and DALYs, $PM_{2.5}$ was causing almost the entire health burden, and for DALYs most of the health burden due to $PM_{2.5}$ was associated with non-accidental mortality. Thus, most important air pollution is $PM_{2.5}$ which causes non-accidental mortality. Other pollutants and health outcomes have minor impact to health. This result is similar to the European Environmental Agency (EEA)[25] air quality in Europe report that estimated attributable deaths due to $PM_{2.5}$, ozone (O_3) and NO_2. For Poland the number of deaths were 44 600, 1100 and 1600, respectively, for three different pollutants. Thus, also in their analysis most of the attributable deaths (94%) were due to $PM_{2.5}$. Similarly, European Environmental Burden of disease study, that included $PM_{2.5}$, benzene, lead, together with several other environmental stressors, concluded that most of the health burden was due to $PM_{2.5}$.

Although presented results relate to Warsaw agglomeration, the conclusions are likely applicable to other central-eastern European cities, and also to many cities in low- and middle-income countries around the world. Warsaw has seen rapid growth of private car ownership in the past decade[4], while at the same time houses are still warmed by coal[4]. In addition to local emissions, the influx of pollutants from outside the city plays also an important role in reducing the air quality.

4.2. Strengths and limitations of the study

The strength of this study is the use of well-established dispersion modeling system, based on CALPUFF and emission data, the combination of fine scape population data with

the resulting air pollution concentration, and estimating the health burden for multiple air pollutants and health outcomes. The main strength lies in a combination of these methods to one assessment with one purpose.

The main limitation of the study is lack of ozone concentration impact on the considered health burden. Together with $PM_{2.5}$ and NO_2 ozone is among most important air pollutant from a health effect point of view. CALPUFF system, due to its linear structure, is not an appropriate modeling tool to analyze the tropospheric ozone formation. On the other hand, the acquaintance of ozone concentrations is an important driving force in other urban atmospheric processes. Hence, in this study the ozone concentrations are based on the measurements[26] for the year 2012. The sequence of 1-h observed values at 8 stations located in the study area (Fig. 1) are entered and interpolated to the computational grid. The hourly variability range of the measured ozone concentrations is 3–90 $\mu g/m^3$. The annual mean values are 24–30 $\mu g/m^3$, depending on the measurement point, and the similar mean for the summer period with the highest occurrence of ozone, are within the range of 28–38 $\mu g/m^3$. The Polish reference value[26] of 1-h ozone concentration is set to 150 $\mu g/m^3$. As indicated in an earlier study[27], ozone is estimated to cause 1100 attributable deaths in Poland and likely tens or hundreds of cases in Warsaw. Even if we assume that ozone would cause hundreds of attributable deaths in Warsaw, the total health burden in Table IV would be in the same magnitude. Hence, the health burden would be much smaller than for the $PM_{2.5}$ and at maximum in same level with NOx. Moreover, lacking the modelling results, it is not known how much of the ozone concentration was due to distant sources, so the contribution of local sources to local level ozone can be even lower.

The attributable deaths and DALYs due to air pollution were calculated by combining the impact of individual pollutants together, although some of the pollutant categories used in this study overlap. For example, metals disperse through the air in particulate format and the

metal emissions are therefore included also in PM emissions. This could lead to overestimation of the impact. However, since the total burden caused by all the heavy metals and BaP combined is still less than 1% of the total burden, we assume that the potential impact of double counting is small. In some environments, where the concentrations of heavy metals are higher, the method used here could lead to higher overestimation of the burden.

4.3. Comparison to other studies, discussing important differences in results

Two previous studies have estimated the health burden due to transport related air pollution in the same study area with substantially different methods and results. Tainio[19] estimated that transport related air pollution cause 25 000 DALYs a year (in this study 15 000 DALYs, Table V) using methods and data similar to this study. The main reason for lower burden in this study is the update of the concentration-response function for all-cause mortality for $PM_{2.5}$, from Beelen *et al.*[28] to Héroux *et al.*[20]. Adamkiewicz *et al.*[29] used roadside measurements to estimate the contribution of local traffic to atmospheric PM10 and NOx concentration in the study area, and the Life Cycle Impact Assessment tool ReCiPe (http://www.lcia-recipe.net/) to estimate the health burden due to these two pollutants. Their estimate for health burden is 1700 DALYs, about one magnitude smaller than our estimate. However, if we compare Adamkiewicz *et al.*[29] results with the health burden caused by $PM_{2.5-10}$ and NOx, then the difference in results is much smaller (5400 DALYs in this study versus 1700 DALYs in Adamkiewicz *et al.*[29]. This might indicate that Adamkiewicz *et al.*[29] results are smaller because they didn't include fine particulate matter $PM_{2.5}$ in their analysis.

Few studies have estimated burden of disease due to air pollution in Poland. In the EEA[25] air quality report the total attributable deaths in Poland were assumed to be 47 300 deaths per year and another study estimating impact of Poland 39 800 attributable deaths for the year 2000[27]. When scaled for population of Poland (38.6 million) to population of

Warsaw (1.72 million), the attributable deaths would be 2100 and 1800 cases per year, respectively, by assuming that the burden is equally distributed around the country. In Global Burden of Disease (GBD) Study 2013[24] the impact of air pollution in Poland was 433 000 DALYs and similarly the contribution of Warsaw would be 19 300 DALYs (versus 51 000 DALYs estimated in this study). The result of the EEA is similar to this study (2100 versus 2800 deaths) when taking into account that urban areas are more polluted than country on average. The GBD 2013 estimate is much smaller. GBD Integrated Risk Function (IRF) method[30] used uncertain threshold value between 5.8 and 8.8 µg/m3 to set up counter-factual scenarios for five individual disease outcomes (ischemic heart disease (IHD), stroke, chronic obstructive pulmonary disease (COPD), lung cancer, and acute lower respiratory infection). The use of individual diseases might lead to smaller impact than the use of all-cause mortality, defined as natural mortality in this study, but without detailed analysis of GBD results that cannot be quantified.

The results between different pollutants and health outcomes were similar in Tainio[19] study that estimated health effects of local transport related air pollution in Warsaw. Also in that study most of the air pollution related health effects were due to non-accidental mortality due to $PM_{2.5}$. However, in the present study the relative contribution of $PM_{2.5}$ was smaller, due to updated concentration-response function for $PM_{2.5}$. This increased importance of NO_x, but had minor impact to other pollutants. Another study from Finland[31] estimated burden of air pollution for 14 different pollutants, including all the main pollutants from this study and ozone. They found out that from the total health burden (33 000 DALYs), 63% was due to $PM_{2.5}$, PM_{10}, ozone and NO_2 contributed 91% of the total burden. Thus, in their analysis, the total contribution of $PM_{2.5}$ alone was slightly smaller than in our study (71%) but within similar magnitude when taking into account that they included the effect of ozone.

4.4. Unanswered questions and future research

We also considered only exposure to outdoor air pollution in the assumed home addresses. Most of this exposure to outdoor air pollution occurs indoors and is impacted by the indoor sources of air pollutants. For example, Asikainen et al.[32] estimated that in Poland 66% of the burden of disease from residential indoor exposure is due to $PM_{2.5}$ from the outside air. Other significant sources were indoor generated $PM_{2.5}$, radon and home dampness.

5. CONCLUSIONS

The modelling results indicate that air pollutions cause 2800 deaths a year in Warsaw. From this 45% are due to inflow from outside the study area and the rest due to local emissions. From all the local emissions, area sources (residential) caused 46% of the burden and linear sources (transport) 52%. Impact of point sources was around 1%. Nearly all the deaths (91%) were due to PM2.5, highlighting importance of this pollutant for population health. When morbidity effects were included in the calculations, non-accidental mortality due to PM2.5 accounted of about71% of the total DALYs (36 000 DALYs out of total impact of 51 000 DALYs).

A large fraction of the $PM_{2.5}$ pollution in Warsaw comes from the sources located outside of the Warsaw borders. In this study, about half of the related health risks in Warsaw were due to the local emission sources and the other half due to inflow. Thus, the dominating risk factor relates to high exposure to the fine particular matter, coming both from local and external sources. Since Poland is one of a few EU countries responsible for the highest $PM_{2.5}$ (including BaP)[3] emissions, appropriate government decisions are essential for decreasing the health risk level. With reference to the housing sector, policies that could reduce emissions would include e.g.: (i) assisted replacement of the coal-fired installations by natural

gas ones (ii) subsidized installations of low-emission coal furnaces, (iii) considerable raise of norms of the coal quality for domestic use. Moreover, since the other emission categories also significantly contribute to the inflow of pollutants (85% of the energy in Poland is generated by coal combustion), the increase of the share of renewable sources in the national level could also improve air quality in Warsaw, and elsewhere in Poland.

ACKNOWLEDGEMENTS

MT: The work was also undertaken by the Centre for Diet and Activity Research (CEDAR), a UKCRC Public Health Research Centre of Excellence. Funding from the British Heart Foundation, Cancer Research UK, Economic and Social Research Council, Medical Research Council, the National Institute for Health Research, and the Welcome Trust, under the auspices of the UK Clinical Research Collaboration, is gratefully acknowledged. Agata Hościło from the Institute of Geodesy and Cartography in Warsaw is acknowledged for elaborating the population data for Warsaw.

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TABLES

Table I. Primary and secondary pollutants discussed (according to Holnicki *et al.*[4, 11])

Primary pollutants	**Secondary pollutants**
SO_2 – sulfur dioxide	$SO_4^{=}$ – sulfate aerosol
NO_X – nitrogen oxides	NO_3^{-} – nitrate aerosol
PPM_{10} – primary PM, $\Phi \leq 10$ μm	
PPM_{10_R} – re-suspended PPM10	
$PPM_{2.5}$ – primary PM, $\Phi \leq 2.5$ μm	
$PPM_{2.5\_R}$ – re-suspended PPM2.5	
CO – carbon monoxide	
C_6H_6 – benzene	
Pb – lead	
As – arsenic	
Cd – cadmium	
Ni – nickel	
BaP – benzo(a)pyrene	
Particulate matter	
$PM_{10} = PPM_{10} + PPM_{10\_R} + SO_4^{=} + NO_3^{-}$	
$PM_{2.5} = PPM_{2.5} + PPM_{2.5\_R} + SO_4^{=} + NO_3^{-}$	

Table II. Background burden in the study area.

Disease	Age group	Burden measure	#	Factor	Details
All causes	All	DALY	536,529	-	Total (All causes).
All causes	All	Deaths	18,254	-	Total (All causes).
Non-accidental mortality	30+	YLL	267,244	$PM_{2.5}$ NOx	Communicable, maternal, neonatal, and nutritional disorders; Non-communicable diseases
Non-accidental mortality	30+	Deaths	17,016	$PM_{2.5}$ NOx	Communicable, maternal, neonatal, and nutritional disorders; Non-communicable diseases
Lung cancer	All	DALY	23,373	SO_2	Trachea, bronchus, and lung cancer.
Lung cancer	All	Deaths	1,081	SO_2	Trachea, bronchus, and lung cancer.
Ischemic heart disease	All	DALY	52,377	CO	Ischemic heart disease.
Ischemic heart disease	All	Deaths	3,487	CO	Ischemic heart disease.
Ischemic heart disease	15-79	DALY	40,456	Pb	Ischemic heart disease.
Ischemic heart disease	15-79	Deaths	1,759	Pb	Ischemic heart disease.
Cerebrovascular disease	15-79	DALY	27,732	Pb	Cerebrovascular disease.
Cerebrovascular disease	15-79	Deaths	1,350	Pb	Cerebrovascular disease.
Hypertensive heart disease	15-79	DALY	4,427	Pb	Hypertensive heart disease.
Hypertensive heart disease	15-79	Deaths	188	Pb	Hypertensive heart disease.
Other cardiac diseases	15-79	DALY	13,097	Pb	*
Other cardiac diseases	15-79	Deaths	481	Pb	*

*) Cardiomyopathy and myocarditis; Atrial fibrillation and flutter; Aortic aneurysm, Peripheral vascular disease; Endocarditis, Other cardiovascular and circulatory diseases

Table III. Population weighted exposure for emission categories.

Pollution	Unit	Point sources		Line	Area	Local sources	External inflow	Total. exposure
		High	Low					
SO_2	[µg/m³]	0,71	0,27	1,32	3,68	5,99	1,46	7,45
SO_4		0,01	0,00	0,05	0,13	0,20	0,64	0,84
NOx		0,43	0,41	16,10	2,31	19,25	1,86	21,10
NO_3		0,01	0,01	0,53	0,12	0,67	2,87	3,54
PPM_{10}		0,06	0,23	1,93	10,30	12,52	10,15	22,67
PPM_{10}_r		-	-	9,14	-	9,14	-	9,14
$PPM_{2.5}$		0,02	0,10	1,30	8,02	9,44	7,35	16,84
$PPM_{2.5}\_r$		-	-	1,30	-	1,30	-	1,30
PM_{10}		0,08	0,24	11,66	10,55	22,52	13,66	36,18
PM_{25}		0,04	0,12	3,18	8,27	11,60	10,86	22,51
CO		0,14	0,48	145,40	7,57	153,60	132,88	281,67
C_6H_6		0,29	0,12	0,70	0,00	1,11	0,00	1,11
Pb		0,00	0,00	0,01	0,01	0,014	0,00	0,015
As	[ng/m³]	0,00	0,00	0,00	0,73	0,735	0,00	0,735
Cd		0,00	0,05	0,01	1,06	1,120	0,04	1,164
Ni		0,06	0,11	0,67	3,35	4,194	0,00	4,194
BaP		0,01	0,02	0,14	1,12	1,286	0,66	1,946

Table IV: Attributable deaths (number of deaths per year) in the study population by source, pollutant and cause of mortality: mean and (95% CI)

Pollutant	Point High	Point Low	Line	Area	Inflow	Total	%
PM2.5: Non-accidental mortality	4 (3 - 5)	12 (9- 14)	388 (291- 480)	691 (517- 856)	1,210 (906 - 1,498)	2,304 (1,725- 2,853)	82%
NOx: Non-accidental mortality	7 (2 - 12)	8 (2 - 14)	380 (87 - 665)	35 (8 - 61)	27 (6- 47)	457 (104- 800)	16%
SO2: Lung cancer	1 (-4 to 5)	0 (-1 - 2)	2 (-9 -12)	3 (-17 to 22)	1 (-7- 10)	8 (-38 - 50)	0%
BaP: Lung cancer	<1	<1	<1	2 (1- 2)	1 (0 - 1)	3 (1 -4)	0%
Cd: Cancer	<1	<1	<1	1 (0- 2)	<1	1 (0 - 2)	0%
Ni: Cancer	<1	<1	<1	<1	-	<1	0%
Pb: Cardiovascular diseases	<1	<1	7 (3-12)	4 (2 - 7)	0 (0 - 1)	11 (6 - 21)	0%
As: Lung Cancer	<1	<1	-	<1	-	<1	0%
CO: Ischemic heart disease	<1	<1	7 (3 - 11)	<1	4 (2 - 6)	11 (4 - 17)	0%
C6H6: Leukemia	<1	<1	<1	<1	-	<1	0%
Total	**12 (4- 19)**	**20 (13 - 27)**	**783 (476 - 1,085)**	**736 (559 - 903)**	**1,244 (938- 1,533)**	**2,794 (2,111 - 3,455)**	**100%**

Table V: DALY, due to air pollution in Warsaw, by source, pollutant and cause of morbidity or mortality: mean and (95% CI)

Pollutant	Point High	Point Low	Line	Area	Inflow	Total
PM2.5: Non-accidental mortality	59 (44 - 73)	182 (136-226)	6,094 (4,563 - 7,546)	10,852 (8,126 - 13,438)	19,000 14,228 - 23,528)	36,186 (27,098 - 44,811)
PM2.5: Chronic bronchitis (COPD)	5 (1- 9)	15 (3 - 28)	509 101 - 927)	906 (179 - 1,651)	1,586 (314 - 2,891)	3,021 (598 - 5,505)
PM2.5: Restricted activity days (RAD)	1 (1 - 1)	4 (3 - 4)	122 110 - 133)	217 (196 - 237)	379 (343 - 415)	722 (654 -791)
PM2.5: LRS symptoms days (School children)	<1	1 (1 - 1)	31 (19 - 43)	55 (33 - 76)	96 (58- 133)	182 111- 252)
PM2.5: LRS symptoms days (adult)	1 (0 - 1)	2 (1 - 4)	71 (21 - 134)	126 (37 - 239)	220 (65 - 418)	419 (124 - 797)
PM2.5-10: LRS symptoms days (School children)	<1	1 (1 - 1)	88 (54 - 122)	15 (9 - 21)	19 (11- 26)	123 (75 - 170)
PM2.5-10: LRS symptoms days (adult)	1 (0 - 1)	2 (1 - 4)	202 (60 - 384)	36 (11 - 68)	43 (13 -81)	283 (84 -538)
PM2.5-10: Chronic bronchitis (COPD)	4 (1 - 8)	16 (3 - 29)	1,455 (288 - 2,652)	256 (51 - 467)	307 (61- 559)	2,038 (403 - 3,715)

Table V:(continued)

Pollutant	Point High	Point Low	Line	Area	Inflow	Total
NOx: Non-accidental mortality	111 (25 - 194)	123 (28 - 215)	5,966 (1,364 - 10,448)	548 (125 - 959)	423 (97 - 741)	7,170 (1,639 - 12,557)
SO2: Lung cancer	16 (-80 - 105)	6 (-31 - 41)	39 (-197 - 260)	72 (-359 - 473)	31 (-156 - 206)	164 (-823 - 1,086)
BaP: Lung cancer	0 (0 -o 1)	1 (0 - 1)	7 (2 - 10)	34 (12 - 48)	22 (8 - 31)	63 (22 - 91)
Cd: Cancer	<1	1 (0 - 2)	<1	16 (1 - 40)	1 (0 - 2)	18 (1 - 44)
Ni: Cancer	<1	<1	<1	0 (0 - 1)	-	1 (0 -1)
Pb: Mild mental retardation (children)	<1	<1	8 (3 - 17)	5 (2 -10)	1 (0 - 1)	13 (5 - 28)
Pb: Cardiovascular diseases (adult)	0 (0 - 1)	2 (1 - 4)	166 (81 - 301)	100 (49 - 181)	11 (6 - 20)	279 (137 - 507)
As: Lung Cancer	<1	<1	-	<1	-	<1
CO: Ischemic heart disease	<1	<1	98 (38 - 158)	3 (1 - 5)	59 (23 - 95)	160 (62 - 259)
C6H6: Leukemia	1 (0 - 1)	0 (0 - 1)	3 (2 - 4)	3 (2 - 4)	-	7 (4 - 9)
Total	200 (68 - 326)	357 (241 - 469)	14,856 (9,708 - 19,902)	13,241 (10,288- 16,065)	22,196 (17,177 - 26,998)	50,849 (39,270- 62,083)
%	0%	1%	29%	26%	44%	100%

and circulatory diseases

FIGURES

Fig. 1. The study domain: (a) resolution of the receptor area, (b) resolution of the total emission area.

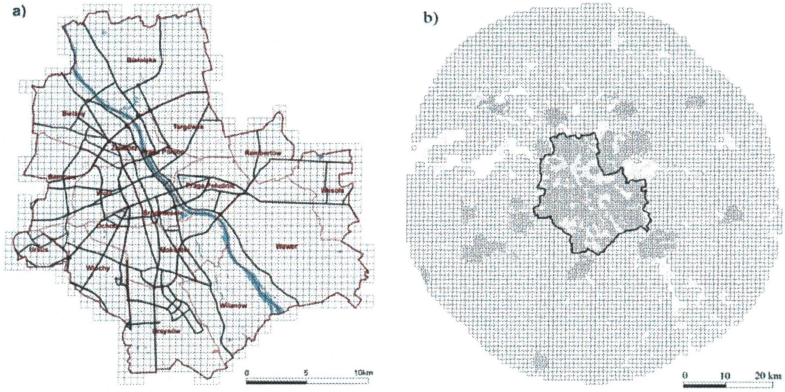

Fig. 2. The annual mean concentrations maps for PM_{10}, $PM_{2.5}$, BaP, where the limit values are exceeded (according to Holnicki *et al.*[4, 11])

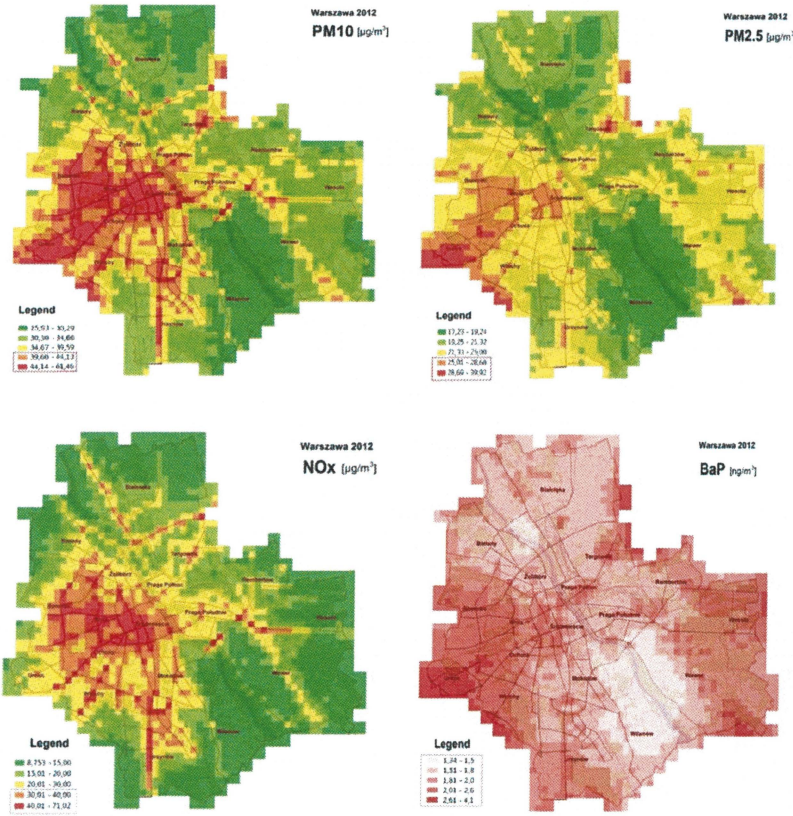

Fig. 3. Population density map for Warsaw

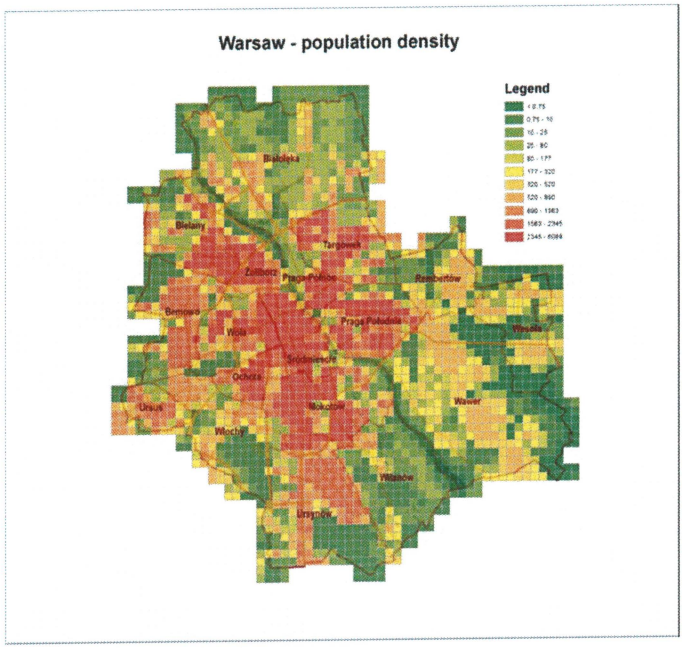

Fig. 4. Exposure of the selected pollutants attributed to the individual emission sources. X-axis represents the number of the source, Y-axis – the exposure in logarithmic scale. Left panels – 7285 line sources, Right panels – 6962 area sources. Positions of emission sources on the map and the related grid resolution are additionally explained below the X-axis description.