Quantifying the health impacts of ambient air pollution: methodology and input data

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Front page picture:
A large fraction of the European urban population is exposed to air pollution concentrations above the EU and WHO reference concentrations. Urban green spaces may have positive health effects.
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1 Introduction

Since the 1970's European ambient air quality has largely improved as a result of the ongoing reduction in emissions from stationary and mobile sources. However, currently the exposure to ambient air pollution still leads to adverse effects on human health and ecosystems. Epidemiological studies have shown that especially the exposure to pollutants such as particulate matter, ozone and nitrogen dioxide is associated with cardiovascular and respiratory diseases leading to increased sickness, hospital admissions and premature death (Pope et al., 2006, Jerrett et al., 2009; Hoek et al., 2013; Beelen et al., 2014). Exposure to particulate matter favours the development of lung cancer (Loomis et al., 2013; Raaschou-Nielsen et al., 2016).

In integrated studies assessing the costs and benefits of air pollution abatement but also in papers related to informing stakeholders and the general public, an evaluation of the health consequences of the exposure to air pollution is essential. Quantification of the health impacts of ambient air pollution is frequently presented in reports of EEA and its ETC/ACM. In this Technical Paper, we describe the methodology used for estimation of the impacts as presented in EEA's *Air Quality in Europe – 2016 report* (EEA, 2016) and in related reports. The methodology aims at obtaining comparable data for the whole of Europe. It is recognised that at the national level more and more detailed information will be available. At the urban scale, for example, air quality data are generally available from direct measurements; health and population data is also generally available, see for example the APHEKOM project (Pascal et al., 2013) and the ESCAPE project (see http://www.escapeproject.eu/index.php or references cited in Brunekreef et al., 2015). However, in view of the comparability, international data sets (that is, available at the European or global level) are preferred as discussed below.

Assessment of the impact of air pollution on human health requires information on air pollution concentrations, demographic data and the relationship between the concentration of an ambient pollutant and a health outcome at population level. Lack of harmonised input data hampers the comparability of the assessments between various countries or regions. Such comparability is of importance when preparing a health assessment at the European level.

Data collected by international bodies will likely guarantee the best possible harmonized data sets. Note that use of international, harmonised data may come at a cost of increased uncertainties for those regions where more accurate/detailed data is available.

The health impacts of air pollution can be quantified and expressed as premature mortality and morbidity. Mortality reflects reduction in life expectancy owing to premature death as a result of air pollution exposure, whereas morbidity relates to illness occurrence and years lived with a disease or disability, ranging from minor effects, such as coughing, to chronic conditions that may require hospitalisation (EEA, 2016).

In this paper the approach to estimate the health impacts presented in the EEA's *Air Quality in Europe – report series* is described in chapter 2, together with a detailed description of the

concentration-response functions to estimate mortality endpoints (section 2.3.1) and morbidity endpoints (section 2.3.2). Chapter 3 provides a general overview of input data needed for the calculations.

Note that chapter 2 includes a description of health endpoints (in particular morbidity related endpoints) for which assessments have not been performed yet in the context of the EEA annual Air Quality Reports[1]. Nonetheless, for completeness of the methodology, the concentration response functions and the input data needed for these calculations are presented here.

The methodology has been described as if it would have been applied for the year 2013. By describing the preparation of input data for an actual year (2013), the approaches needed for gap-filling missing data can be shown in detail. It will be evident that, when selecting another reference year, various data sets might need to be updated. Web links to the most recent versions as used in the 2013 assessment, have been included in the reference list and may serve as a starting point in searching updated information.

[1] The EEA's Air Quality in Europe –report series up to year 2016 has only estimated the health impacts related to premature mortality. The assessment of the impact of air pollution on morbidity and postneonatal infant mortality may be included in future reports.

2 Methodology

2.1 Calculation of health outcomes

The burden of disease associated with ambient air pollution is estimated using health impact functions that relate concentration to health outcomes at population level (see section 2.2). It is assumed that concentration data is available in a spatially distributed form, e.g. as concentrations maps (see, for example, Horálek et al., 2016).

For quantifying the effect of air pollution, the relative risk (*RR*) in a population whose exposure is estimated by an average concentration *C* is given by the concentration response function:

$$RR = \exp\left[B\left(C - C_0\right)\right] \qquad [1]$$

where C_0 is a reference concentration (the background concentration that would exist without any man-made pollution determined by natural sources or a concentration below which no health effects are to be expected). *B* is the concentration-response factor, that is, the estimated effect of the pollutant on the health outcome (e.g. mortality from cardiopulmonary diseases). The relative risks are based on epidemiological studies and are given as an increase in incidence or prevalence per unit increase in concentration from which the factor *B* is obtained. This coefficient is generally assumed to be similar for the whole population.
Once the relative risks have been determined, the attributable fraction (*AF*) of health effects from air pollution for the exposed population is:

$$AF = \sum P_i(RR_i - 1) / \sum P_i RR_i \qquad [2]$$

where P_i is the proportion of the population at concentration category *i*;
RR_i is the relative risk in concentration category *i.*
However, as in general the total population is considered with only one concentration level, this relation can be written as:

$$AF = \left(RR - 1\right) / RR \qquad [3]$$

The expected burden of disease attributable to air pollution is given by:

$$E = AF \cdot \sum_i MR_i \cdot Pop_i \qquad [4]$$

where E is the expected burden of disease (e.g. number of premature deaths or number of hospital admissions due to outdoor air pollution);

MR_i is the baseline incidence of the given health effect for each age group *i* (e.g. the total or cause specific number of deaths per 100,000 people per year);

Pop_i is the size of population in each of the age classes *i*.

Equation [4] is evaluated for each 1*1 km grid cell of the air quality maps (see section 3.2 for further details on the air quality maps).

The expected burden of disease attributable to air pollution in a specific country (E_{CC}) is obtained by summation over all grid cells within that country:

$$E_{CC} = \sum_x E_x \qquad [5]$$

Note that *AF* and *Pop* are grid cell dependent whereas the baseline incidence *MR* is assumed to be constant over the country.

2.2 Health endpoints

In the EEA´s *Air Quality in Europe – 2016 report* (EEA, 2016) the mortality (non-accidental, all-cause) attributable to the long-term exposure of PM, NO_2 and ozone exposure are considered. The health endpoints are expressed in terms of *premature deaths* and *years of life lost* (YLL) (see box below). In future studies, the assessment of the impact of air pollution on morbidity may be included .

When evaluating morbidity, impacts are expressed in terms of hospital admissions, cases of diseases, working days lost etc. The selected endpoints are discussed in more detail in section 2.3.

The number of years of life lost due to premature mortality is calculated according to:

$$YLL = \sum(E_i \cdot L_i) \qquad [6]$$

where E_i is the number of deaths in age class *i* attributable to air pollution and L_i is the life expectancy at age of death (in years). The summation is done for each 5-year age class.

Premature deaths are deaths that occur before a person reaches an expected age. This expected age is typically the age of standard life expectancy for a country and gender. Premature deaths are considered to be preventable if their cause is eliminated.

Years of life lost may be defined as the years of potential life lost due to premature deaths. It is an estimate of the average years a person would have lived if he or she had not died prematurely. Years of life lost take into account the age at which deaths occur, giving greater weight to deaths at a

younger age and lower weight to deaths at older age. It gives therefore more information than the number of premature deaths.

2.3 Concentration response functions, baseline incidence data

The concentration-response functions (CRF) as recommended in the HRAPIE project (WHO, 2013a; Héroux et al., 2015) and as implemented in cost-benefit analysis for the European Commission (Holland, 2014) are described in this paper. The HRAPIE recommendations include a grouping of the effects according to the uncertainty related to CRF and the availability of the baseline health data. Effects for which enough data are available for quantification of the effects are labelled with an A; those effects for which there is more uncertainty are labelled with a B. Further, HRAPIE marked the pollutant-outcome pairs which contributes to the total effect (that is, it is assumed that their effects are additive) with an asterisk (*). The HRAPIE projects focuses on quantification of the health effects of the main pollutants PM, NO_2 and O_3. In ambient air, concentrations of these pollutants are usually correlated to some extent. The REVIHAAP project (WHO, 2013b) therefore proposed quantification of health effects associated with these pollutants only after adjustment for at least one of the others. In EEA's *Air Quality in Europe* reports (EEA, 2015, 2016) this adjustment is not implemented; premature deaths attributable to $PM_{2.5}$, NO_2 and O_3 individually are calculated. In particular the estimates of the effects of NO_2 may represent an overestimate in the likely range of 0-33% (WHO, 2013a).

Below a summary of the applied CRF, the definition of the exposed population fraction and the incidence data is given. For PM and ozone all pollutant-outcome pairs labelled in the HRAPIE report as A* or B* have been included except the effects of short-term PM_{10} exposure on incidence of asthma symptoms in asthmatic children (aged 5-19 years). However, an estimate of the effect asthma symptoms in asthmatic children is included in the estimated restricted activity days (WHO, 2013a). For NO_2 currently only the effects of long-term NO_2 exposure on all-cause mortality (group B*) has been included.

The input data sets from which information on baseline incidences is extracted, are described in section 3.1.

2.3.1 Concentration response functions for mortality

$PM_{2.5}$

To describe the health effect of long-term exposure to $PM_{2.5}$, on total **all-cause (natural) mortality** in ages above 30 the recommended risk ratio (RR) of 1.062 for a 10 µg/m^3 increase of $PM_{2.5}$ (95% confidence interval is 1.040-1.083; Group A*) is used. Non-violent (natural) mortality refers to the

ICD-10[2] codes A00-R99, or, when not available, refers to the MTL1[3] codes MTL1 1001-1094. Data has been extracted from European Mortality database (see 3.1).

The HRAPIE report recommends the quantification of health impacts at all concentrations. In the impact assessment made for the Clean Air Package (EC, 2013), impacts are estimated for the (modelled) anthropogenic contribution to $PM_{2.5}$ which implies that the background (natural) contribution is not considered. In the estimation of the impacts of $PM_{2.5}$, this might lead to higher impact estimates compared to other studies like the Global Burden of Disease study (Lim et al., 2012). In the GBD study, impacts are estimated only above a counterfactual concentration (that is, the concentration below which the risk could not be quantified due to a lack of data) of 5.8 to 8.8 µg/m^3 (Burnett et al., 2014).

Our approach is based on observed air pollution concentrations (see 3.2), which include a (small) contribution from natural sources. In addition to a calculation using annual $PM_{2.5}$ data without assuming a threshold, a sensitivity calculation using a threshold of 2.5 µg/m^3, that is the lowest annual mean concentration observed at any of the European stations, is recommended.

PM_{10}

Postneonatal infant mortality (defined as death between the ages of 1 and 12 months; Group B*) has been associated with PM_{10} exposure. Following HRAPIE, a relative risk of 1.04 (95% CI 1.02 – 1.07) per 10 µg/m^3 change in PM_{10} is chosen. Country specific data on the number of life births and postneonatal mortality rates are available from the European Health for All database (HFA-DB, see 3.1).

O_3

For ozone the health assessment is based on the average of the daily maximum 8-h mean over 35 ppb (SOMO35) for **all (natural) cause mortality** (all ages, Group A*) as health outcome. The recommended RR is 1.0029 (95% CI 1.0014-1.0043) per 10 µg/m^3 change in SOMO35. Non-violent

[2] The International Classification of Diseases (ICD) is the standard diagnostic tool for epidemiology, health management and clinical purposes. This includes the analysis of the general health situation of population groups. It is used to monitor the incidence and prevalence of diseases and other health problems, providing a picture of the general health situations in countries and populations. See also http://www.who.int/classifications/icd/en/

[3] Mortality tabulation list 1 of the ICD-10

(natural) mortality refers to the ICD-10 codes A00-R99, or, when not available, refers to the MTL1 codes MTL1 1001-1094. Data has been extracted from European Mortality database (see 3.1).

By using the SOMO35 values, a threshold value of 35 ppb (70 µg/m^3) is implicitly assumed.

NO_2

HRAPIE recommends a relative risk of 1.055 (95% CL 1.031 to 1.08) per 10 µg/m^3 based on the **all-cause (natural) mortality** rate (> 30 year, Group B*). Non-violent (natural) mortality refers to the ICD-10 codes A00-R99, or, when not available, refers to the MTL1 codes MTL1 1001-1094. Data has been extracted from European Mortality database (see 3.1).

Following further HRAPIE recommendation, the NO_2 impact has been calculated for levels above 20 µg/m^3. The material as available in the HRAPIE review did not suggest that there is no effect below 20 µg/m^3; the studies rather showed that the size of the effect is less certain below 20 µg/m^3. However, this recommendation might be too conservative, as indicated by Héroux et al. (2015).

A Danish study (Raaschou-Nielsen et al., 2012) showed a significant correlation between NO_2 concentrations and health outcomes throughout the full range of observed concentrations (10.5 to 59.6 µg/m^3 with a median of 15.1 µg/m^3).

The impacts estimated for each pollutant may not be added to determine the total impact attributable to exposure to air pollution, because concentrations are (sometimes strongly) correlated. This may lead to a double counting of up to 30 % of the effects of $PM_{2.5}$ and NO_2 (WHO, 2013a).

2.3.2 Concentration response functions for morbidity

PM

To quantify the effects of long-term PM_{10} exposure (annual mean) on new incidences of **chronic bronchitis in adults** (> 27 year, Group B*) a relative risk of 1.117 (95% CI 1.040 – 1.189 per 10 µg/m^3 change in PM_{10}) is recommended. In the absence of country-specific baseline rates for chronic bronchitis, HRAPIE recommended to use a baseline incidence of 3.9 cases per 1000 adults at risk.

Recommendation for effects of long-term PM_{10} exposure (annual mean) on prevalence of **bronchitis in young children** (6-12 year, Group B*) is a relative risk of 1.08 (95% CI 0.98 to 1.19) per 10 µg/m^3. The baseline rate of bronchitis from various countries ranged from 6.2% to 41.5%, the HRAPIE experts recommended the mean prevalence rate of 18.6%. Acute bronchitis lasts about 5-21 days; an averaged value of 10 days can be assumed here. This outcome contributes to the calculation of

restricted activity days (RAD), so when estimating the total burden of disease, RADs should be reduced accordingly.

A **restricted activity day** (RAD) is defined as a day when individuals reduce their normal activities, including days of missed work, absences from school and other more minor reductions in daily activity. For the effects of short term $PM_{2.5}$ exposure on the incidence of RAD (all ages, Group B*) a relative risk of 1.047 (95% CI 1.042 – 1.053) per 10 $\mu g/m^3$ change is recommended.

In the absence of country specific data on RAD, HRAPIE recommends a baseline rate of RAD of 19 days per person per year.

Note that RADs includes work loss days, days spent in hospital from respiratory and cardiovascular diseases and children's sick days related acute bronchitis. When estimating the total burden of disease, the number of RADs has to be corrected for these double countings.

The short-term effects of $PM_{2.5}$ on **respiratory hospital admissions** (all ages, Group A*) is described by a relative risks of 1.019 (95% CI 0.9982 – 1.0402) per 10 $\mu g/m^3$. All age categories are considered. Baseline data is available from the Hospital Morbidity Database (see 3.1). Respiratory diseases are defined by ICD10 codes J00-J99, or ISHMT code 1000 (International Shortlist of Hospital Morbidity Tabulation[4]).

Preferably, country –specific incidence data according to the ICD-10 classification is used; missing data can be – as far as possible- completed with data classified according to the ISHMT codes.

As yet, several countries were not included in the database. For the missing countries gap filling is proposed, by selecting data from neighbouring countries e.g. for Montenegro, Greece, Bosnia-Herzegovina, Albania and Kosovo[5] the averaged value of surrounding countries (Bulgaria, Croatia, Hungary, FYR of Macedonia, Romania and Serbia) can be selected. Missing data in Portugal and Belgium can be gap-filled by selecting the data from Spain and the Netherlands, respectively. Missing data for the smaller countries can be obtained as described in section 3.1

[4] http://www.who.int/classifications/icd/implementation/morbidity/ishmt/en/

[5] In this paper references to Kosovo shall be understood to be in the context of UN Security Council Resolution 1244/99.

The averaged hospital stay ranges from 0.03 day in Serbia to 9.4 day in Finland. The average length of stay for Serbia is likely in error. In such cases we suggest to use the median value (6.9 day) as calculated for the other countries.

The hospitalizations contribute to the calculation of restricted activity days (RAD), so when estimating the number of RAD, the time spent in hospital is to be subtracted.

For **cardiac hospital admissions** (all ages, ICD10 codes I00-I99, ISHMT 900, Group A*) the recommended RR is 1.0091 (95% CI 1.0017 – 1.0166) per 10 μg/m^3 change in $PM_{2.5}$. Baseline data is available from the Hospital Morbidity Database (see 3.1).

For both classification schemes, data for several countries were not included. For the missing Balkan countries (Albania, Bosnia-Herzegovina, Greece, Kosovo and Montenegro) we suggest selecting the averaged value of surrounding countries (Bulgaria, Croatia, Hungary, FYR of Macedonia, Romania and Serbia). Missing data for Estonia can be replaced by the averaged values of neighbouring Baltic countries (Finland, Latvia, Lithuania, and Poland). Missing data in Portugal and Belgium can be gap-filled by selecting the data from Spain and the Netherland, respectively. Data for the smaller countries can be obtained as described above (section 3.1).

The hospitalizations contribute to the calculation of restricted activity days (RAD), so when estimating the number of RAD, the time spent in hospital is to be subtracted.

The effect of short-term $PM_{2.5}$ exposure on the number of **work days lost** (absenteeism) is described by a relative risk of 1.046 (95% CI 1.039 – 1.053, Group B*). For baseline rates, country-specific data on absenteeism from work due to illness is provided by the European Health for All database (HFA-DB, see 3.1). Although baseline rates are available for most countries, the definitions and criteria used for registering sick leave differ significantly between countries, increasing the uncertainty of burden estimates. By using averaged data over a recent five-year period (2009-2013) the number of available countries can be optimized. The multi-annual averages show a large range, e.g. from 1 sick day in the UK to 47 days in Slovakia. Both extremes are not confirmed in other studies (Livanos and Zangelidis, 2010, Eurofound, 2010) which show a more uniform absence rate with 7-8 sick days in the UK and 2-8 days in Slovakia. However, the data presented in these studies (Livanos and Zangelidis, 2010, Eurofound, 2010) refer to the period 2004-2008 and should therefore not be applied here.

In preparing country-specific baselines, we propose to replace the UK number by a value of 4.6 days number (ONS, 2014). For the missing data for Italy we suggest a value of 5.7 days according to Scuppa and Vuri (2014); this value is slightly higher than the 2.0-3.4 days quoted by Livanos and Zangelidis (2010) and Eurofound (2010). For Slovakia and all other missing countries, a value of 9.6 days (being the averaged value of the 19 countries for which data are available) is proposed.

Baseline data on the exposed population (that is, the employment rate) is obtained from Eurostat (2016).

O_3

The short-term effects of ozone on **respiratory hospital admissions** (aged 65+ years, Group A*) is described by a relative risk of 1.0044 (95% CI 1.0007 – 1.0083) per 10 µg/m^3 change in O_3.

It's recommended using incidence data (available in the Hospital Mortality Database) according to the ICD-10 classification (ICD-10 classes ICD10 J00-J99), completed – as far as possible- with ISHMT (classes ISHMT 1000) data. However, for several countries information on the age-distribution of hospital admissions has not been reported or were not included at all in the database. For countries providing both information on the number of hospital admission for all age groups as well as on the number of admissions for 65 years and older, a high correlation (R^2 = 0.941) between the two parameters was observed. The found ratio (0.45) is in our approach suggested for gap-filling the missing countries.

The short-term effects of ozone on **cardiovascular hospital admissions** (aged 65+ years, Group A*) is described by a RR = 1.0089 (95% CI 1.0050 – 1.0127) per 10 µg/m^3 change in O_3. Hospital admissions related to stroke are not included. Baseline data is available from the Hospital Morbidity Database by selecting the ICD-10 code I00-I52 or, in case of missing data, by selecting the ISHMT codes 901-907, 911. These selections are not fully equivalent; a correction factor of 0.92 is obtained from countries providing data for both selections. When no age distribution data was available, we suggest a ratio of 0.56 between the admissions for 65+ age and all ages.

Whereas a restricted activity day (RAD) is defined as a day when individuals reduce their normal activities, for health-related reasons, the **minor restricted activity days** (MRAD) do not involve work loss or bed disability, but do include some noticeable limitation on "normal" activity. The short term effect of ozone on MRAD, all ages, (Group B*) is described by a RR = 1.0154 (95% CI 1.0060 – 1.0249) per 10 µg/m^3 change in O_3. By lack of information at a national level, a uniform baseline of 7.8 MRAD per year for all ages is proposed (WHO, 2013a).

3 Input data

In this chapter we have chosen to describe the methodology as it would have been applied for the year 2013. The input data sets show some missing data. By describing the preparation of input data for an actual year (2013), the approaches needed for gap-filling missing data can be shown in detail. It will be evident that, when selecting another reference year, various data sets might need to be updated. Web links to the most recent versions as used in the 2013 assessment, have been included in the reference list and may serve as a starting point in searching updated information.

3.1 Population data and demographics

Total population data is available from various sources, e.g Eurostat, and they are generally included in health statistical databases. However, for the population data, preference is given to demographic data bases. Total national populations for the year 2013 (as per 1 January) were obtained from Eurostat (2015). As the area for which concentration data is available does not cover in all cases the total area of some countries, a correction on national population totals was made: for France, the overseas territories, for Portugal, the Azores and Madeira, and for Spain, the Canary Island and the cities in North Africa (Ceuta and Melilla) were excluded.

Age distributions were taken from UN (2015). Note that for some countries (e.g. Andorra, Kosovo, Liechtenstein, Monaco, San Marino) the age distribution is not available in this UN database; gap-filling has been done on basis of the data from the neighbouring countries (France, Serbia, Austria, France, and Italy, respectively). Within a country, the same age distribution is assumed in all grid cells.

Country-specific life expectances – as used in the estimation of years of life lost, Eq [6] – is available from the World Population Prospect 2015 (UN, 2015) for each age group with a five-year interval; estimates representative for the period 2010-2015 have been selected.

The population density map as used in the air quality mapping procedure (in this case based on: Horálek et al., 2015) has a resolution of 1x1 km. It is primarily based on Geostat 2011 grid dataset (Eurostat, 2014). For regions, which are not included in the Geostat 2011, alternative sources were used. As a first option, JRC (Joint Research Centre) population data in resolution 100x100 m were used (JRC, 2009; Gallego, 2010). The JRC 100x100 m population density data has been spatially aggregated into the reference 1x1 km EEA grid. For regions, which are not included neither in the Geostat 2011 nor in the JRC database, we used population density data from ORNL LandScan Global

Population Dataset (ORNL, 2008). This dataset is in 30x30 arcsec resolution; its values are based on the annual mid-year national population estimates for 2008 from the Geographic Studies Branch, US Bureau of Census (http://www.census.gov). The ORNL data is projected and converted from its original 30x30 arcsec grids into the reference 1x1 km EEA grid. See Horálek et al. (2015) for a more detailed discussion on the population density map.

The demographic data as extracted from sources mentioned above is not always consistent with each other. The required input data is therefore converted to relative quantities (for example, as percentage or as cases per capita). In the final step of the calculation, the relative numbers have been scaled with the national totals given by Eurostat (2015) to absolute numbers.

Baseline incidence

Background health data is mainly extracted from three different databases:

1. the European detailed mortality database (WHO, 2016). This database contains mortality data by cause of death, age and sex, submitted to the WHO by the European Member States. Data are extracted from the latest available update (July 2016).

   For some countries (Andorra, Kosovo, Liechtenstein, Monaco, San Marino) no health information is available in this database; gap-filling has been done on basis of the data from the neighbouring countries (France, Serbia, Austria, France, Italy, respectively).

2. the Hospital Morbidity Database (HMDB; WHO,2015b), containing hospital discharge data by detailed diagnosis, age and sex, which were submitted by European countries to the WHO Regional Office for Europe. The latest available update is from January 2015.
3. the European health for all database (HFA-DB; WHO, 2015a) provides easy and rapid access to a wide range of basic health statistics for the 53 Member States of the WHO European Region. It was developed by the WHO Regional Office for Europe (WHO/Europe) in the mid-1980s to support the monitoring of health trends in the Region. The database is a helpful tool for international comparison and for assessing the health situation and trends in any European country in an international context. The December 2015 version is used here.

As much as possible data for 2013 has been extracted, or, when 2013 data was missing, the latest available year is used. Where needed, country-level data was gap-filled on the basis of the data from neighbouring countries; details have been given in describing the concentration-response functions. Within a country, the same baseline incidence is assumed in all grid cells.

3.2 Air Quality data

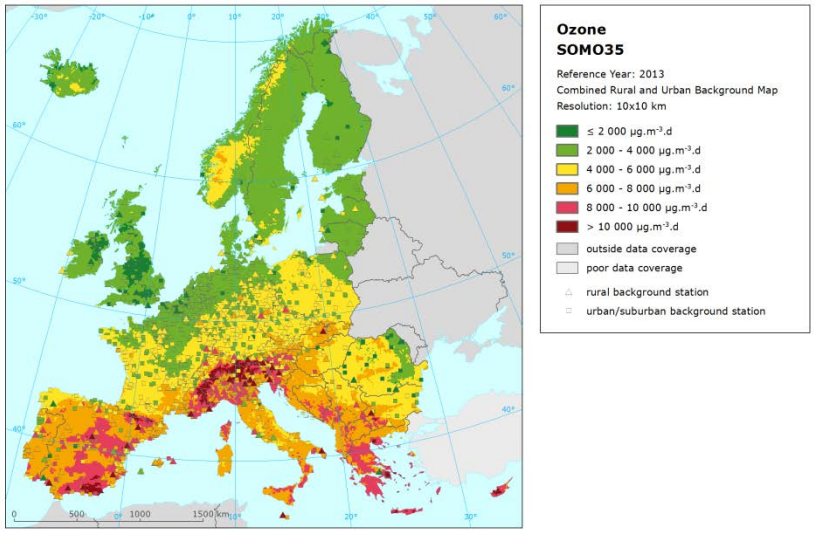

Figure 1. Example map showing a concentration field (in this case, ozone SOMO35 values)

used as input data in the health impact assessment

The air quality maps (annual mean concentration for $PM_{2.5}$, PM_{10} and NO_2; SOMO35 for ozone) prepared by the European Topic Centre on Air and Climate Change (ETC/ACM) have been used as input to the health impact assessment.

The maps can be briefly described as:

- reference year 2013;
- interpolation methodology: co-kriging of observed concentrations using additional spatial information (EMEP model results, meteorological data, altitude, population density map). To increase the spatial coverage of measurements, pseudo $PM_{2.5}$ stations data were used in addition to measured $PM_{2.5}$ data. Pseudo $PM_{2.5}$ stations data are estimated using PM_{10} measured data, surface solar radiation, latitude and longitude;
- a computational spatial resolution 1*1 km is used; note that the uncertainties inherent to the followed spatial interpolation procedure do not allow a presentation of the results at a 1x1 km scale.

Details of the mapping procedures and the data used are described by Horálek et al (2016). In the health impact assessment the computations are made on the 1x1 km grid; in this way the known correlation between air pollution levels and population density is (partly) taken into account. It also allows for the introduction of a threshold value, e.g. an assumed threshold value below which no

health impacts are to be expected or a “correction” for natural contributions. However, as population and health input data are available only at a national scale, results are presented at the national level only.

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