

Health impacts of air pollution

Assessing the risks to health from air pollution

The European Environment Agency (EEA) produces annual air pollution health risk assessments at the European level. These give an objective and comparable estimate of the impacts of air pollution on the population's health. This briefing provides an overview of the methodology followed in the assessments.



Key messages

- The EEA uses the best available air quality data, and information on population and health outcomes at European level to estimate health risk.
- World Health Organization (WHO) recommendations are used in the EEA's assessment. These include the relationships between the concentration of an air pollutant to which a population is exposed and a health outcome (for instance, mortality), and the counterfactual concentrations above which health impacts are considered.
- The estimates are a good indication of the magnitude of the health impacts of air pollution and a solid basis for measuring the impact of policies to improve air quality.
- Health impacts are estimated at population level, rather than for individuals so it is not possible to identify which individuals died because of air pollution.
- The estimated number of premature deaths are a measure of the general impact of air pollution across a given population.

Assessments of the health risk from air pollution estimate and communicate the impact of exposure to air pollution on the population's health. Such assessments may also include the impact of changes in air quality resulting from air quality improvement measures (WHO, 2016).

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The European Environment Agency's (EEA) annual health risk assessment is published in the *Air Quality in Europe* report and aims to:

- provide an objective and comparable estimate of the impacts of current air pollution (differentiated by pollutants but without focusing on any specific source) across Europe;
- identify any changes over time;
- provide input to the development and implementation of measures to improve air quality;
- raise awareness of the harmful effects of air pollution.

A health risk assessment involves three steps (WHO, 2016):

- Assess the exposure of the population to the pollutants in question;
- Estimate the health risk associated with that exposure;
- Estimate the uncertainty of the calculation.

Pollutants

The health risk assessment is made for long-term exposure to the three pollutants considered most harmful by the World Health Organization and for which the evidence of health effects is strongest (WHO, 2013a): particulate matter with a diameter below 2.5 µm (PM_{2.5}), nitrogen dioxide (NO₂) and ground-level ozone (O₃).

Air pollution is a complex mix of several pollutants, so the effects attributed to one pollutant may be partly caused by some others. Health experts consider the pollutants identified above to be proxies for the whole mixture. In particular, PM_{2.5} has been shown to be a robust indicator of the risk associated with exposure to particulate matter from diverse sources and in different environments.

Health effects

The effects of exposure to air pollution are diverse, ranging from subclinical effects such as inflammation to premature deaths (WHO, 2013a). For the EEA's health risk assessment, mortality is the health outcome analysed since it is the most serious effect of air pollution and the one for which the evidence is most robust. Mortality due to exposure to air pollution is estimated in terms of 'premature deaths' and 'years of life lost'.

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Premature deaths are deaths that occur before a person reaches an expected age. This expected age is typically the life expectancy for a country, stratified by sex. Premature deaths are considered preventable if their cause can be eliminated.

Years of life lost (YLL) are defined as the years of potential life lost due to premature death. YLL is an estimate of the number of years that people in a population would have lived had there been no premature deaths. The YLL measure takes into account the age at which deaths occur and therefore the contribution to the total is greater for a death occurring at a younger age and lower for a death occurring at an older age.

Determining population exposure to air pollution

To calculate the exposure of the European population to different levels of air pollution, information is needed on the spatial distribution of both air pollutant concentrations and the population.

Concentrations are estimated from measurements officially reported by EEA member and cooperating countries. Background stations in all areas (urban, suburban and rural), and traffic stations in the case of NO₂, are taken as a starting point (see step A in the example).

From the measurements at these stations, concentration maps covering all of Europe are made. Supplementary data such as altitude, meteorology and the results of air quality modelling delivered under the European Monitoring and Evaluation Programme are also used to produce the maps (ETC/ACM, 2016a, 2017). In the final map, concentrations are gridded at a scale resolution of 1x1 km² (see step B in the example).

Placing population density maps over the concentration maps at the same resolution produces a picture of population exposure (see step C in the example).

Using these maps, the percentage of the population exposed to the whole range of concentrations, in increments of 1 µg/m³, can be estimated for each country.

In addition to total population figures, information on the age and sex distribution of the population is also used in the calculation of the attributable mortality.

Estimating the risk to health

To calculate the health risks, the following information is also needed:

- Concentration-response functions;
- Baseline health statistics

Concentration-response functions have been established by epidemiological studies and represent the relationship between the concentration of an air pollutant to which a population is exposed and

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the risk of a health outcome. As such, concentration-response functions quantify the health impact per concentration unit of air pollutant (WHO, 2016).

The EEA uses the concentration-response functions defined by the World Health Organization (WHO, 2013b), which are based on the relative risks. Relative risks capture the increase in mortality that can be attributed to a given increase in the air pollutant concentration. Relative risks are defined at the population level (as statistical averages) and cannot be assigned to specific individuals. In the case of mortality it is therefore not possible to identify which individual cases are caused by air pollution (see point D in the example) (WHO, 2016; COMEAP, 2010).

Concentration-response functions are in general linear, but this may not be true for very low or very high concentrations. Also, the evidence on the impacts of human health for very low or very high concentrations may not be as robust, as for the 'intermediate' concentrations. This is why sometimes the impact cannot be analysed with the same confidence for the whole range of concentrations and an initial concentration or 'counterfactual concentration' is used. The counterfactual concentration is therefore the concentration above which the impacts are estimated. In the EEA's estimations, counterfactual concentrations as recommended for Europe by the WHO are used (WHO, 2013b):

- $0 \mu\text{g}/\text{m}^3$ for PM_{2.5}, which means the full range of concentrations is considered (see point E in the example);
- $20 \mu\text{g}/\text{m}^3$ for NO₂, since the evidence of the concentration-response function for lower concentrations is not sufficiently robust;
- For O₃, the SOMO35^[1] statistic is used, which implies a counterfactual concentration of $70 \mu\text{g}/\text{m}^3$ as SOMO35 only considers concentrations above that level.

Baseline health statistics include country-specific life expectancies, stratified by age and sex, and total mortality data for each country, also by age and sex. These statistics are characteristics of the population as a whole and cannot be applied at an individual level. As such, the estimated number of premature deaths derived using these statistics is a measure of the general impact of air pollution across a given population (WHO, 2016; COMEAP, 2010).

The relative risks allow the percentage of the baseline incidence that can be attributed to exposure to a pollutant to be determined. For mortality, the total number of deaths per year (see point F in the example) in a country is used as baseline incidence. The relative risks allow the expected burden of disease, in terms of premature deaths, to be estimated. These results are obtained at the grid level and then summed for all the grids in an area (as for instance a country or all of Europe) (see step G in the example) (ETC/ACM, 2016b).

To estimate years of life lost, the age at which the premature deaths occurred is also considered. For each death, the current age of death is subtracted from the life expectancy at that age to obtain the years of life lost due to that specific death. Summing up the years of life lost for all premature deaths results in the total years of life lost for the population (ETC/ACM, 2016b).

Sensitivity analysis of health risks

Because of evolving evidence, from the Air quality in Europe - 2017 report onwards, additional estimations are also carried out considering two other counterfactual concentrations:

- $2.5 \mu\text{g}/\text{m}^3$ for PM_{2.5}, which is the lowest annual mean concentration measured in Europe and could be considered as the minimum background concentration, that is, the natural concentration that would occur if all anthropogenic contributions were removed.
- $10 \mu\text{g}/\text{m}^3$ for NO₂, as the minimum concentration for which effects have been found in an epidemiological study (Raaschou-Nielsen et al., 2012) that appeared later than the WHO recommendations cited above.

Uncertainties of the estimation

The main uncertainty is associated with the concentration-response functions used in the health risk assessment (WHO, 2016; COMEAP, 2010). This derives from the assumptions made in the epidemiological studies on which the concentration-response function is based. Most epidemiological studies take into account other confounding factors that can also have an impact on mortality, such as smoking, diet or other life styles.

The uncertainty associated with the concentration-response function is calculated as a confidence interval around a mean or central estimate. The EEA uses a 95 % confidence interval. This implies that there is 95 % probability that the true value lies in the range defined by the interval. As an example, in the case of PM_{2.5} there is 95 % probability that the risk is between a 4 % and an 8.3 % increase in total mortality for each $10 \mu\text{g}/\text{m}^3$ increase in concentrations (with a mean or central estimate of 6.2 %).

The value that the EEA provides in its reports is the mean estimate, together with the limits of the confidence interval (see step H in the example).

Additional sources of known uncertainty, which should be born in mind in a qualitative way are (WHO, 2016; COMEAP, 2010):

The fact that air pollution is a complex mixture of several air pollutants, as mentioned before. The health impacts of some pollutants are correlated and that is why the premature deaths attributed to each pollutant cannot simply be added up. In particular, it has been estimated that adding premature deaths attributed to PM_{2.5} to those attributed to NO₂ could result in double counting of around 30 % (WHO, 2013b).

The uncertainties associated with air quality measurement and modelling data, and the methods used to combine them to produce air quality maps.

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The exposure data. It is assumed that anyone in the $1 \times 1 \text{ km}^2$ grid is exposed to the same concentration. It is also assumed that people are exposed to the ambient concentrations of the places where they live, without taking into account that many commute for work or leisure. This is another reason estimates are made for a defined population and not for individual risks. For individual exposures, a finer spatial resolution of exposure data is needed.

The baseline health data and the possible uncertainties in its compilation.

The counterfactual concentration when considering absolute numbers of premature deaths.

Various methods are used for population exposure estimation, concentration-response functions or counterfactual concentrations. These explain the differences between the EEA's estimates and those from other studies such as the Global Burden of Disease or national and local studies.

For example, in its 2016 publication *Ambient air pollution: A global assessment of exposure and burden of disease*, the WHO used ground air quality measurements from monitoring locations around the world, complemented with satellite remote sensing and chemical transport models. The counterfactual concentration was selected to be a uniform distribution with lower and upper limits of 5.9 and $8.7 \mu\text{g}/\text{m}^3$ respectively. It resulted in an estimate of 190 000 deaths in Europe attributable to PM_{2.5} air pollution in 2012.

In the 2015 *Global Burden of Disease* study, the Lancet Commission estimated population-weighted mean concentrations of PM_{2.5} at an approximate resolution of $11 \times 11 \text{ km}^2$, using satellite-based estimates, chemical transport models and ground-level measurements. The relative risk of mortality from ischaemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease, lung cancer and lower respiratory infections was estimated (instead of all cause mortality). The counterfactual concentration was assigned a uniform distribution of 2.4 - $5.9 \mu\text{g}/\text{m}^3$.

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A) Station

The area for which the health risk assessment will be calculated consists of four grids (1x1 km² each) and one monitoring station (S), which registered in year Y an annual mean PM_{2.5} concentration of 17 µg/m³

B) Concentration map

The concentration map from that monitoring station (S) and the supplementary data provides the result shown in B.

C) Population/exposure

The populations in the grids are shown in C. In grid 1, the 10 000 inhabitants are exposed to 15 µg/m³; in grid 2, the 5 000 inhabitants are exposed to 10 µg/m³; in grid 3, the 2 000 inhabitants are exposed to 10 µg/m³; and in grid 4, the 1 000 inhabitants are exposed to 5 µg/m³.

D) Relative risk

In the case of PM_{2.5}, the concentration-response function used for total (all cause) mortality in people above 30 years of age implies a relative risk of 1.062 per 10 µg/m³. This means that, assuming linearity, an increase of 10 µg/m³ of PM_{2.5} is associated with a 6.2 % increase in total mortality in the total population considered.

E) Counterfactual concentration

The counterfactual concentration for PM_{2.5} is 0 µg/m³, meaning that, for instance, for grid 1 the effect of the whole range of 15 µg/m³ will be estimated.

F) Mortality

The total mortality (incidence baseline) in the country for year Y and for the population over 30 years of age is 10 deaths per 1 000 inhabitants, so the number of deaths per grid are as shown in F.

G) Premature deaths

The number of deaths attributed to exposure to PM_{2.5} in each grid (assuming, according to the concentration-response function, an increment of 6.2 % in total mortality per 10 µg/m³) are as shown in G.

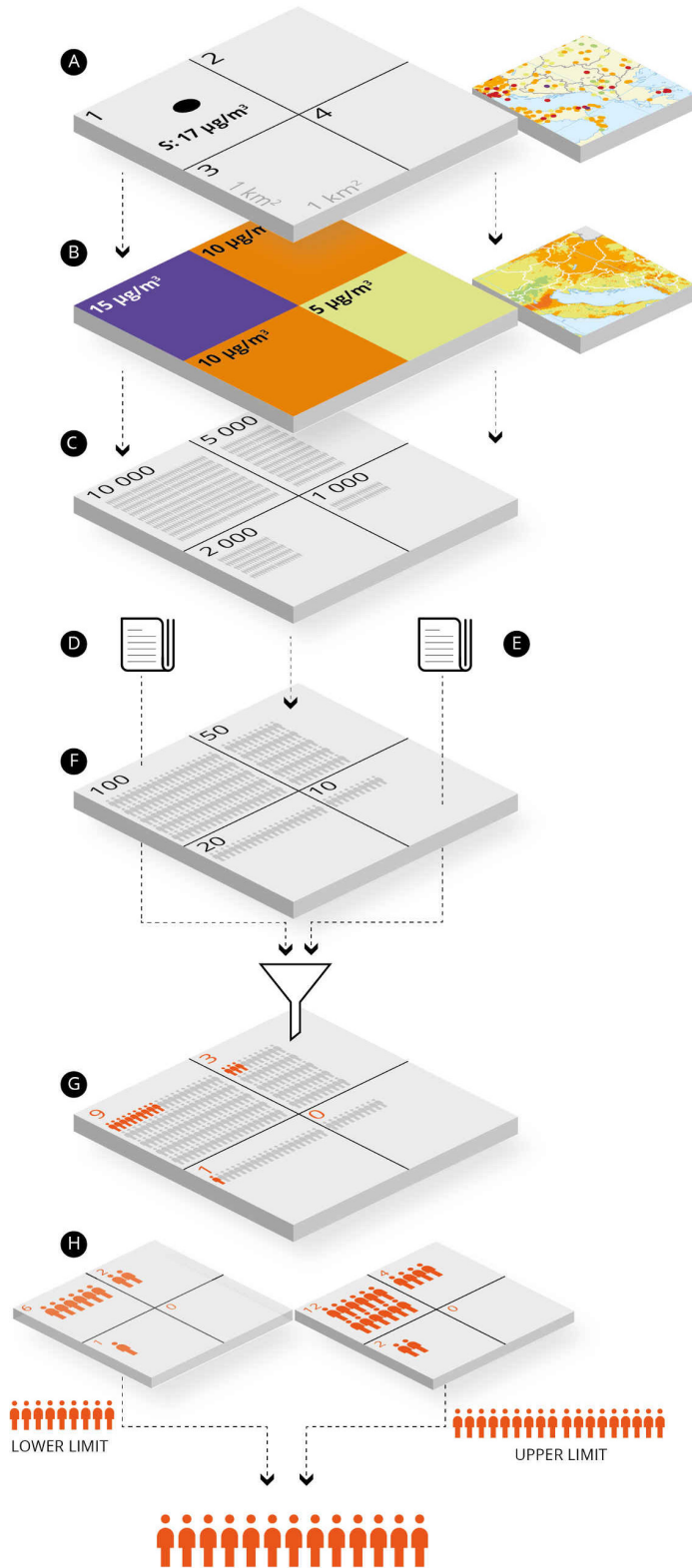
This is obtained from:
 Relative risk (RR) = exp (β * concentration) = exp (0.0062 * concentration). For grid 1: 1.097462
 The attributable fraction (AF) = (RR-1)/RR.
 For grid 1: 0.0888065
 Premature deaths (PD) = AF * mortality * pop.
 For grid 1: 8.88 ≈ 9.

And the total number of deaths attributed to PM_{2.5} in the whole area in year Y: 9+3+1+0=13.

H) Uncertainty range

The uncertainty range is calculated using the lower and upper limits of 1.040 and 1.083, instead of the relative risk of 1.062.

The total mortality is then expressed as 13 premature deaths with a 95 % confidence interval between the values of 9 and 18.



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Footnotes

[1] SOMO35 is the sum of means over 35 parts per billion (ppb) (daily maximum 8-hour). It is a way of aggregating ozone concentrations and is defined as the yearly sum of the daily maximum of 8-hour running average over 35 ppb ($70 \mu\text{g}/\text{m}^3$).

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