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Guidelines for assessing the health impacts of air pollution in European cities

Work Package 5

Deliverable D5

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Table of contents

Foreword.....	5
1. What are the health impacts of air pollution?	5
2. Why perform a health impact assessment of air pollution?.....	6
3. Which HIA for which purpose?	6
4. Guidelines for performing an HIA of the health impacts of urban air pollution.....	7
4.1. Definition of the study period.....	9
4.2. Definition of the study area	9
4.3. Exposure assessment.....	10
4.3.1. Selection of the stations.....	11
4.3.2. Computation of the exposure indicators	11
4.4. Choice of the health outcomes and population data.....	13
4.5. Choice of the concentration response functions	14
4.6. Definition of the scenarios.....	16
4.7. Calculating the number of cases.....	17
4.8. Calculations of the gain in life expectancy	18
5. Interpretation of HIA findings	20
6. Uncertainties.....	20
6.1. Uncertainty in exposure assessment	21
6.2. Uncertainty in health outcomes.....	22
6.3. Uncertainties in the CRFs	23
7. Communicating HIA findings	24
7.1.Choosing the metrics for reporting mortality results.....	24
Appendix 1 – Guidelines for using the Aphekom HIA short-term tool.....	26
1. Objectives	26
2. Methods	26
2.1. The health impact function	26
2.2. The short-term impacts of PM10.....	26
2.3. The short-term impacts of ozone	27
3. How to use the Excel tool	28
3.1. Format	28
3.2. Input data	28
3.3. Sheet “1-Environmental data”	28
3.4. Sheet “2-Health data”	28
3.5. Sheet “3-Population data”	29
3.6. Sheet “4-Hypothesis”	29
3.7. Sheet “PM10 results – Scenario 1”	29
3.8. Sheet “PM10 results – Scenario 2”	31
3.9. Sheet “Ozone results – scenario 1”	31

3.10. Sheet “Ozone results – scenario 2”	31
3.11. Sheet “Ozone results – scenario 3”	31
3.12. Graphs and summary tables	31
Appendix 2 – Guidelines for using the Aphekom HIA long-term tool	32
1. Objectives	32
2. Methods	32
3. How to use the Excel tools	34
3.1. Format	34
3.2. Input data	34
3.3. Sheet “1-Environmental data”	34
3.4. Sheet “2-Health data”	35
3.5. Sheet “3-Population data”	35
3.6. Sheet “4-Hypothesis”	35
3.7. Sheet “Descriptive data”	35
3.8. Sheet “PM2.5-Results-Total-Mortality-S1”	36
3.9. Sheet “PM2.5-Results-Total-Mortality-S2”	37
3.10. Sheet “PM2.5-Results-Cardiovascular-S1”	37
3.11. Sheet “PM2.5-Results-Cardiovascular-S2”	37
3.12. Graphs and summary tables	37
References	38

Foreword

This document provides guidelines for public-health professionals who wish to perform health impact assessments (HIAs) of urban air pollution. It follows the general framework recommended by the World Health Organisation for performing HIAs, and applies classical methods in the field of urban air pollution.

The guidelines start with a general overview of the health impacts of air pollution (Chapter 1). Chapter 2 details the uses and relevancies of HIAs in the field of air pollution, and Chapter 3 discusses the two approaches that can be used to perform an HIA. Chapter 4 describes all the practical steps that should be implemented to perform an HIA, while Chapter 5 discusses interpretation of the HIA results, Chapter 6 the uncertainties, and Chapter 7 the communication.

For each key step in the HIA process, theoretical considerations are described, and practical aspects are summarised in text boxes.

The guidelines develop the methods, tools and scenarios that the Aphekom project used to perform HIAs in 25 European cities. However, the methods can apply to other pollutants and other health outcomes provided that the relevant data and concentration response functions are available.

The guidelines are accompanied by two Excel spreadsheets that include all the equations needed to perform a complete HIA. One is for calculating short-term impacts, and one for calculating long-term impacts. Instructions on how to use the spreadsheets appear in Appendixes 1 and 2.

Finally, the guidelines focus on calculation of a number of health outcomes associated with air pollution. This number can then be valued economically. The method for doing so is described in Aphekom Deliverable D6: Guidelines on monetary-cost calculations related to air-pollution health impacts (www.aphekom.org).

We tried to be as clear as possible in these guidelines and the Excel spreadsheets so that these tools can be easily adapted to other situations.

1. What are the health impacts of air pollution?

Each day, adults inhale 10 000 to 20 000 litres of air, depending on their morphology and physical activities. Beside oxygen and nitrogen, which represent on average 99% of the composition of the air, pollutants from human and natural sources are also present in the air.

Because the mixture of pollutants in the air is complex, epidemiological studies focus on a few pollutants, such as particulate matter (PM) and ozone, which are used as markers/indicators of the air pollution mixture.

Since the 1990s, these indicators have been consistently associated with several health impacts. These impacts can occur within a short lag after exposure (short-term impacts), or as a cumulative exposure to air pollution over time (long-term impacts). Considering the short-term impacts of air pollution, strong evidence supports a link between exposure to ozone and particulate matter and daily mortality and morbidity. The Aphenia project compared the results obtained by multicity analyses in Europe, in the U.S. and in Canada. It found that estimates were relatively robust across studies (Katsouyanni, 2009) (Samoli, 2005). Considering the long-term impacts of air pollution, several cohort studies found evidence of an impact on mortality of exposure to particulate matter (Brunekreef *et al.* 2009; Pope and Dockery, 2006). The literature also provides evidence of observed benefits of reducing air-pollution levels (Clancy *et al.* 2002) (Bayer-Oglesby *et al.* 2005) (Heinrich *et al.* 2002).

2. Why perform a health impact assessment of air pollution?

Several scientific findings point to a causal relationship between exposure to air pollution and health. They also show that current levels of air pollutants observed in European cities are associated with health risks. The impact of the effects at the individual level may appear low compared to other risk factors. However, since the whole population is exposed to air pollution, this impact results in a non-negligible public-health burden. Any reduction in air pollution would therefore benefit a large number of people and would be associated with large health gains.

Drafting policies to reduce air pollution is not an easy task because many considerations are at play, such as economic and social constraints, political orientations and urban planning, as well as health. For the latter, health impact assessments (HIAs) provide an objective estimate of the impacts of improvements in air quality on a given population's health. HIAs use available epidemiological studies together with routine environmental and health data to help decision makers plan and implement measures to protect public health more effectively.

To achieve this goal, HIA methods must be transparent and HIA findings understandable. This is why any HIA should use a rigorous method, such as the ones described in these guidelines.

3. Which HIA for which purpose?

Several approaches can be used for assessing the health impact of air pollution (Künzli *et al.*, 2008):

- a *predictive* approach, where we want to know *what will be the health of the population in a given time framework in the future if we decrease pollutants levels*. This approach assesses the difference in health outcomes between what will be observed in the future if pollutant levels remain the same and what will be observed if pollutant levels decrease for example as the result of a given emission-control scenario. This is the richest approach, because it aims to assess the future health impact of a given policy, which is

the classical definition of a health impact assessment (HIA) (Mindell *et al.*, 2003). On the other hand this approach requires making assumptions about the future trends in population and health events, about the time required to achieve the decrease of pollutant levels, and about the lag between the decrease in pollutant levels and the occurrence of health benefits (Miller *et al.*, 2003). This was the approach used for example in the CAFE cost-benefit analysis (Hurley *et al.*, 2005) and in the first part of the recent COMEAP report on the mortality effects of long-term exposure to particulate air pollution in the United Kingdom (COMEAP, 2010). The approach also requires sophisticated tools like the IOMLIFET¹ tool developed by the Institute of Occupational Health (Miller *et al.*, 2008) or the older AIRQ² software developed for WHO.

- a *counterfactual* approach, which is a related but different approach (Mindell *et al.*, 2003). This approach assesses the difference in health outcomes between what is currently observed and what could have been observed if air-pollutant concentrations had been lower and the benefits for health effects had been achieved for the different age groups of the population. This approach gives an idea of the *current burden of air pollution on health*, with the assumption that policies targeting reductions in pollutant levels could lead to a reduction in the assessed health burden. The approach builds on the epidemiological concept of an attributable fraction defined at the population level as the proportion of disease cases that can be attributed to a given exposure level (Benichou, 2001). This was the approach used by the Apehis project (Medina *et al.*, 2009; Apehis, 2005; Boldo *et al.*, 2006; Ballester *et al.*, 2008) This was also the approach used in the second part of the COMEAP report (COMEAP, 2010) and in the WHO Global Burden of Disease project (Cohen *et al.*, 2005).

In Apekom WP5, we used the latter, *counterfactual* approach to update the Apehis results. Our aim was to assess the burden of particulate matter and ozone on mortality and hospitalisations by comparing current levels of these pollutants with WHO air quality guidelines (WHO, 2006). We also assessed the burden for a fixed decrement in pollutant levels.

4. Guidelines for performing an HIA of the health impacts of urban air pollution

The general principle of an HIA is to use a concentration-response function (CRF) linking the concentration of pollutants to which the population is exposed with the number of health events occurring in that population. This CRF is expressed in terms of a percent change in the number of health events per unit change in the pollutant concentration. This CRF is derived from epidemiological studies in a given population. Applied to a pollutant concentration and a baseline health outcome, the CRF allows computing the change in the health outcome associated with a change in the concentration of the pollutant. This process is illustrated in Figure 1.

1 IOMLIFET version 2008 is available at <http://www.iom-world.org/research/iomlifet.php>

2 AIRQ version 2.2.3 (2004) is available at <http://www.euro.who.int/en/what-we-do/health-topics/environmental-health/air-quality/activities/quantification-of-the-health-effects-of-exposure-to-air-pollution-the-air-quality-health-impact-assessment-software-airq-2.2/installation-instructions>

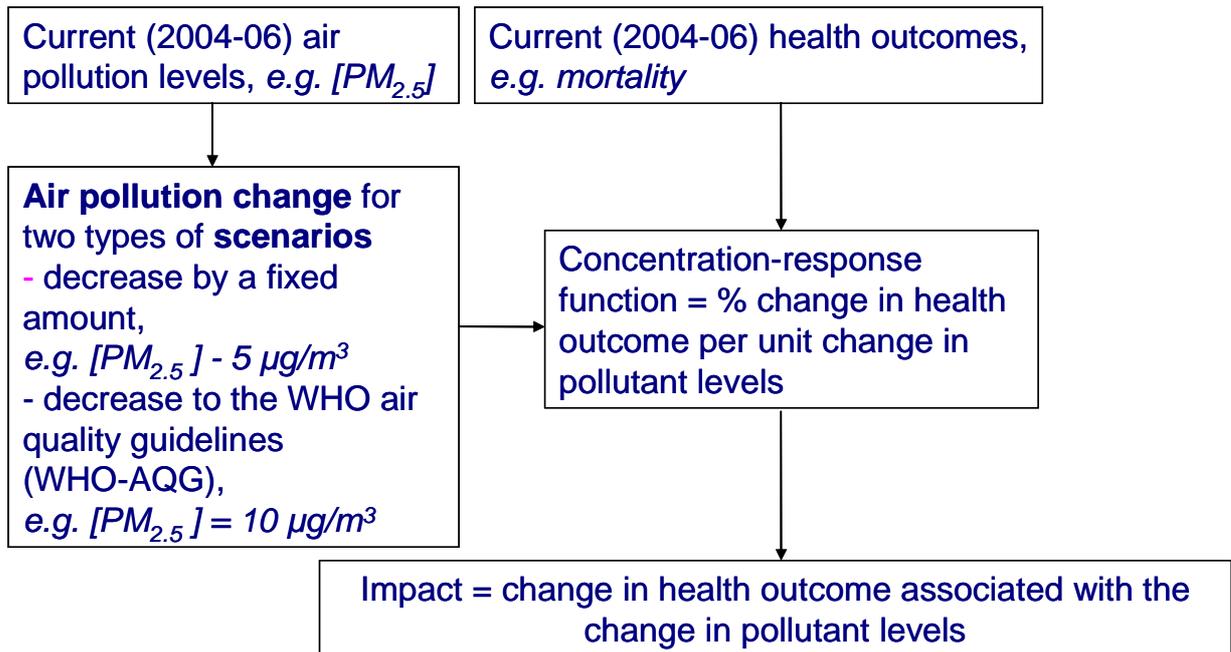


Figure 1: Basis of HIA method

Performing an HIA requires the following steps, which are detailed below;

- 1 - Definition of a study period
- 2 - Definition of a study area
- 3 - Selection of air pollution indicators, collection of environmental data and assessment of the exposure to air pollution
- 4 - Selection of health outcomes and collection of health and population data
- 5 - Selection of CRFs
- 6 - HIA calculations, using a standardised methodology

An HIA is an iterative process, and the steps should not be considered as independent, or to be performed in a chronological order. In fact, the main underlying rule of an HIA is to obtain the greatest similarity between the data used in the epidemiological study that provided the CRF and the data collected to perform the HIA. Therefore, the choices made in step 5 determine all the choices made in steps 1 through 4.

4.1. Definition of the study period

The study period is defined according to the availability of environmental and health data. Whenever possible, environmental and health data should be collected for the same period. If a common period does not exist, the lag between the collection period of environmental and health data should not exceed two years.

The objective is to obtain data representative of the usual situations observed in the cities. The minimum study period should cover a whole calendar year. However a period of two to three consecutive years is advisable in order to smooth year-to-year variations in pollutant concentrations associated with meteorological conditions.

Considering environmental data, it is advisable to favour periods when several monitoring stations are available for one pollutant. Doing so will allow calculating an average exposure across the city. Considering health data, it is advisable to select those years for which the coding methods for mortality or morbidity are identical.

The study period should exclude years that have been associated with unusual health events that may interfere with the health baseline (e.g., the management of the H1N1 pandemics may have interfered with hospital admissions data). Similarly, years associated with unexpected meteorological episodes (e.g., heat waves), or unusual air pollution (e.g., forest fires lasting several weeks) should be excluded.

In practice:

- Select a common study period for environmental and health data. If not possible, the lag between the two periods should not exceed two years.
- Favour two to three consecutive years with more than one air-pollution monitoring station available.
- Check that no unusual health, environmental or meteorological events were observed during the study period.
- Compare the meteorological characteristics of the study period with the usual climatic means.

4.2. Definition of the study area

The definition of the study area is a key step in the HIA process. The objective is to obtain a study area where the average of pollutant levels measured at fixed monitors will be a good estimate of the average population exposure. This means that:

- no part of the area is under the influence of a large stationary source, which would result in high heterogeneity of the pollutant levels
- there is no rupture of urbanisation within the study area, i.e., urban density is relatively homogeneous
- there are no main changes in topography that would modify the dispersion and concentration of pollutants
- a majority of the population lives and works within the study area.

In addition, health data must be available on a geographical scale consistent with the study area.

Finally, the choice of the study area should also take into account policy considerations (i.e. administrative boundaries, areas subjected to the same policies...)

In practice:

- Collect useful data that will help characterise the study area. This can include :
 - a precise and recent map of the urban area (scale 1/25,000) with topographic curves and indications on the urbanisation
 - Information on prevailing winds
 - List of the urban area's municipalities
 - Inventory of all urban background and suburban air monitoring stations and a map of their locations
 - Inventory of the main industrial plants emitting air pollution and a map of their locations
 - Population data for each municipality in the urban area
 - Data on daily commuting between home and place of work
 - Inventory of air monitoring stations and a map of their locations
 - Inventory of hospitals with cardiovascular and/or respiratory activity and a map of their locations.
- Define a first area, named zone 1, following the administrative boundaries of the city,
- Using data on commuting, you may include surrounding municipalities where more than 60% of inhabitants commute daily to zone 1. Doing so enables taking into account municipalities where significant portions of the population are exposed to central-city pollution on a daily basis.
- Check that relevant health data are available for the study area, especially if working with hospitalisation data.
- Take advice from experts at the air quality monitoring network of the area in order to check that calculating an average air-pollution-exposure level makes sense for the population's exposure of the whole selected area.

4.3. Exposure assessment

The exposure indicators must be representative of the population's average exposure to air pollution. Therefore, a discussion with the people in charge of the measuring network in the city can help select the relevant monitoring stations.

The hourly or daily data collected for each station must be averaged to give an estimate of global pollution. The indicator should be as close as possible to the one used in the epidemiological studies from which the CRFs are derived.

Rarely are series of data complete, i.e., without any missing values. To substitute missing values we propose a method that works using the available data. .

Finally, when performing an HIA simultaneously on different cities, the indicators should be calculated using the same protocol for all cities to be able to add the results for each city together and to compare results between cities.

4.3.1. Selection of the stations

Since the objective is to obtain an indicator of the mean exposure to air pollution, only monitoring stations representing background pollution should be included in the study. For PM10 and PM2.5, this means that urban background monitors should be selected; for ozone, urban and suburban background monitors should be selected. Traffic and industrial monitors should never be included in the study. The expertise of the people in charge of the monitoring network should be used to identify the correct stations. As some stations may be incorrectly labelled or may be faulty, we propose using simple criteria to help decide if a station should be included or not:

Whenever possible, at least two monitors should be available, which allows averaging the concentrations to obtain a better estimate of the exposure.

In practice:

- List all the available stations in the area:
 - Urban background stations for PM10 and PM2.5
 - Urban and suburban background stations for ozone
- Exclude stations with not enough data
 - Only stations with less than 25% missing data should be used
- Exclude stations that may not be representative of the mean exposure:
 - Exclude the stations if the interquartile range (P25-P75) of the data from one monitor does not overlap with the interquartile ranges of the other monitors.
 - Exclude the station if its correlation coefficients with the other stations are lower than 0.6
- Try to have more than one station per pollutant
- Validate the choice with an expert from the monitoring network

4.3.2. Computation of the exposure indicators

In this section we develop examples for calculating exposure indicators for the pollutants used in the Aphekom project, PM and ozone. However, the methodology can be applied to other pollutants.

The exposure indicators are calculated by averaging the data measured by the different monitoring stations. The indicators should be calculated to be as close as possible to the

indicators used in the epidemiological studies from which the CRFs are derived. The main aspect to consider is the comparability of the measurement techniques.

Measurement technique can be an issue for PM, as most epidemiological studies calculate the CRFs using concentrations measured with the gravimetric method. However, many cities routinely used the tapered element oscillating microbalance (TEOM) of the β -attenuation method to monitor PM. TEOM measurements underestimate semi-volatile components of the PM. To allow the application of CRFs from studies using gravimetric methods, it is then necessary to correct TEOM data, applying a correction factor to compensate losses of volatile particulate matter. In some cities, TEOM are real-time corrected using a gravimetric reference site or an FDMS device. If there is no real-time correction, a local correction factor can be estimated by the local monitoring networks based on local measurement campaigns. The correction factor may vary between seasons. Otherwise, a 1.3 European default correction factor must be applied as recommended by the EC Working Group on Particulate Matter (<http://europa.eu.int/comm/environment/air/pdf/finalwgreporten.pdf>).

In some places, PM_{2.5} is not measured routinely. It is possible to estimate the PM_{2.5} concentrations based on the PM₁₀ using a conversion factor. In Aphekom, we set this factor at 0.7, unless local data were available. However, doing so introduces large uncertainties in the HIA, so measured concentrations should be used when available,.

When working with PM, the exposure indicator is the daily or the annual mean calculated as the arithmetic mean of the daily concentrations of all the monitoring stations included in the study.

For ozone, we used the daily maximum 8-hour average, as in the WHO air quality guidelines (WHO, 2006). It is calculated as the arithmetic mean of the maximum 8-hour moving averages (daily maximum of the 24 8-hour running means) of the selected stations.

Monitoring stations can measure concentrations at different time intervals, e.g., every 15 minutes, 30 minutes, every hour, etc. Whatever the interval, the rule of more than 75% non-missing values should be applied. For example, from hourly data to daily averages, if data are available for less than 18 hours in a given day, the daily value of this day must be considered missing.

The daily exposure indicator for PM₁₀ and PM_{2.5} is calculated as the arithmetic mean of the daily concentrations of the selected stations.

In practice:

- Check that the measurement methods are consistent with the ones used in the epidemiological studies providing the CRFs.
- If needed, correct PM data obtained with TEOM data, applying a correction factor that can be obtained via the monitoring network
- For each station and each pollutant, aggregate the data to obtain daily data, applying the rule of more than 75% non-missing values

- For each pollutant, aggregate the daily data across all the stations using the arithmetic mean. When you have missing values, you can substitute them in accordance using the following procedure: The value x_{ijk} in a day i with missing data in a monitoring station j in the year k will be replaced by:

$$x_{ijk} = \frac{x_{ik} * x_{jk}}{x_k}$$

x_{ik} is the mean value on day i of year k among all monitors reporting
 x_{jk} is the mean value for monitor j in year k
 x_k and is the overall mean level in year k .

- For each pollutant, use the daily time series to calculate the relevant exposure indicator in agreement with the epidemiological studies providing the CRFs. It may be an annual mean of the number of days above a specific threshold.

4.4. Choice of the health outcomes and population data

The health outcomes should be chosen based on data availability in agreement with the data used in the epidemiological studies providing the CRFs. For the Aphekom project, the selected health outcomes are reported in Table 1.

When using mortality data, they should be selected on the main cause of death of the residents living in the study area regardless of the place of death.

When using hospitalisations data, only hospitals (public and private) located in the study area should be considered to avoid hospitalisations in another city during holidays for example. The place of hospitalisation must be close to the place of residence, because patients have to be exposed to air pollution levels in the study area in the few days preceding the hospital admission. However, according to the attractiveness of some hospitals located outside of the area of study in nearby municipalities, hospitalisations of residents in these hospitals may be selected. Therefore, the hospitals included in the analysis must be representative. Due to the differences in health-care systems and the roles of hospitals, in Aphekom we do not recommend comparing HIA findings on hospitalisations across cities.

To provide standardised rates, population data should be obtained for the same period, the same area and the same age groups.

In practice:

- Identify the relevant health outcomes in the epidemiological studies providing the CRFs
- Check data availability and quality
- Collect the data at the relevant geographical scale
- Collect corresponding population data (age-groups) that will be used to calculate standardised rates.

4.5. Choice of the concentration response functions

CRFs should be chosen favouring meta-analyses of multicentre studies in European cities when available. The HIA relies on the assumption that the chosen CRF is linear at the population level and lies in the range of concentrations observed in the original studies.

The choice of the CRFs directly affects HIA results. Since several studies provide estimates of CRFs in the literature, we recommend performing sensibility analyses to report how HIA findings vary when applying different CRFs.

Selected health outcomes and associated relative risks are presented in Table 1.

For long-term effects of PM_{2.5} on all-cause mortality, we suggest using a RR per 10 $\mu\text{g}/\text{m}^3=1.06$ (1.02 to 1.11) from the American Cancer Society cohort study (Pope *et al.*, 2002; Pope *et al.*, 2004) as done by Apheis (Medina *et al.*, 2009) and by other recent HIA studies in Europe (*e. g.* COMEAP, 2010). Due to its statistical power, the ACS CRF remains the best evidence we have on the long-term effects of chronic exposure to mortality. This estimate has been confirmed by a recent re-analysis of the ACS American cohort study data (Krewski *et al.*, 2009). It is interesting to note, though, that existing European cohort results support the use of ACS results for HIA. For example the results of the European NCLS-AIR study in the Netherlands (Beelen *et al.* 2008) which found a RR=1.06 (0.97 to 1.16).

For the long-term impact of PM_{2.5} on cardiovascular mortality, we suggest using the results from Pope *et al.* (2004). A recent update of the analysis of the ACS study provides a very close estimate (RR=1.15 instead of 1.12), but the RR estimated in the NLCS-study (Beelen *et al.*, 2008) is smaller (RR=1.04).

For the short-term effects of PM₁₀ on non-accidental mortality we suggest using a RR per 10 $\mu\text{g}/\text{m}^3=1.006$ [1.004 to 1.008], from the WHO metaanalysis (Anderson *et al.*, 2004). Other recent European studies have found similar results, the Aphenia project on 12 European cities (Katsouyanni *et al.*, 2009) reported slightly lower RR, ranging from 1.0027 to 1.0062 depending on the modelling strategy. In 10 Italian cities, the EpiAir project reported a RR of RR=1.0069 (1.0040 to 1.0098) (Stafoggia *et al.* 2009).

For the short-term effects of PM₁₀ on cardiac hospitalisations, we suggest using a RR per 10 $\mu\text{g}/\text{m}^3=1.006$ (1.003 to 1.009) from the analysis by Atkinson *et al.* (2005) on 8 APHEA cities. Similar RR were found in 8 French cities (RR=1.008 (1.002 to 1.014) (Larrieu *et al.* 2007), and in the EpiAir project (RR=1.0069 (1.0035 to 1.0103) (Colais *et al.* 2009). Larger estimates were found in the Emecas project on 14 Spanish cities (RR=1.0156 (1.0082 to 1.0231) (Ballester *et al.* 2006).

For short-term effects of PM₁₀ on respiratory hospitalisations, we suggest using a RR per 10 $\mu\text{g}/\text{m}^3=1.0114$ (1.0062 to 1.0167) from the analysis by Atkinson *et al.* (2005) on 8 APHEA cities. Similar RR were reported in the EpiAir cities (RR=1.0078 (1.0040 to 1.0116) (Colais *et al.* 2009).

For short-term effects of ozone on non-external mortality, we suggest using a RR=1.0031 (1.0017 to 1.0052) from the work by Gryparis et al. (2004) on 21 APHEA cities. This was the estimate reported in this study for the summer (April-September) period, but we decided to apply it to the whole year, as the winter estimate was very close after adjustment on PM10 or CO. This estimate is lower than the RR reported in EpiAir (1.007 (1.002 to 1.0121) (Stafoggia *et al.* 2009) but higher than what was observed in the Aphenia project, with RR ranging from 0.9998 to 1.0018, depending on the modelling strategy (Katsouyanni *et al.* 2009).

For short-term effects of ozone on respiratory hospitalisations, we suggest using a RR of 1.001 (0.991 to 1.012) for people aged 15-64 years, and 1.005 (0.998 to 1.012) for people older than 65 years (Anderson *et al.*, 2004). These values are similar to those found in the EpiAir project 1.0045 (0.9959 to 1.0131) (Stafoggia *et al.* 2009).

For long-term effects of ozone, the recent results from the ACS study (Jerrett *et al.*, 2009) on the link between average summer exposure to ozone and long-term respiratory mortality needs to be further explored for its use in HIAs as it is a result on only one study and the evidence is weaker than for PM.

These examples show that CRFs derived from multicity analyses in Europe are consistent for short-term and long-term effects of PM and ozone. The relative risks we chose may underestimate the risks compared to those found in other studies, so the results of our HIAs can be interpreted as “*at least*” estimates.

In practice:

- Perform a literature review to identify relevant publications
- Select the study most consistent with your needs
- Identify the CRFs and the associated health outcomes and exposure indicators.
- Select alternative CRFs to perform the sensitivity analysis

Table 1: Health outcomes and associated relative risks (RR) used in the Aphekom HIA for short and long term effects of air pollution

Short-term impacts of PM10				
Health outcome	ICD Codes	Ages	RR per 10 µg/m³	Reference
Non-external mortality	ICD9 001-799 ICD10 A00-R99	All	1.006 [1.004-1.008]	(Anderson <i>et al.</i> 2004)
Respiratory hospitalisations	ICD9 460-519 ICD10 J00-J199	All	1.0114 [1.0062-1.0167]	(Atkinson <i>et al.</i> 2005)
Cardiac hospitalisations	ICD9 390-429 ICD10 I00-I52	All	1.006 [1.003-1.009]	(Atkinson <i>et al.</i> 2005)
Short-term impacts of ozone				
Health outcome	ICD Codes	Ages	RR per 10 µg/m³	Reference
Non-external mortality	ICD9 001-799 ICD10 A00-R99	All	1.0031 [1.0017-1.0052]	(Gryparis <i>et al.</i> 2004)
Respiratory hospitalisations	ICD9 460-519 ICD10 J00-J199	15-64	1.001 [0.991-1.012]	(Anderson <i>et al.</i> 2004)
Respiratory hospitalisations	ICD9 460-519 ICD10 J00-J199	>=65	1.005 [0.998-1.012]	(Anderson <i>et al.</i> 2004)
Long-term impacts of PM2.5				
Health outcome	ICD Codes	Ages	RR per 10 µg/m³	Reference
Total mortality	ICD9 000-999 ICD10 A00-Y98	>30	1.06 [1.02-1.11]	(Pope, III <i>et al.</i> 2002)
Cardiovascular mortality	ICD9 390-459 ICD10 I00-I99	>30	1.12 [1.08-1.15]	(Pope, III <i>et al.</i> 2004)

4.6. Definition of the scenarios

The choice of a scenario of change in air pollution levels is a key step in the HIA process, and different scenarios can be considered to calculate this change. Two types of scenarios can be distinguished:

- scenarios where the concentrations are decreased by a fixed amount, for instance a decrease by 5 µg/m³
- scenarios where the concentrations are decreased to reach a specific value, for instance a decrease to reach the WHO air quality guidelines value.

In Aphekom, we applied scenarios using a fixed decrease in the concentrations, and decreasing the concentration to the WHO air quality guidelines. Scenarios are reported in Table 2.

Table 2: Scenarios used in the Aphekomb HIA

HIA	Scenario
Short-term impacts of PM10	Scenario 1: Decrease by 5 µg/m ³
	Scenario 2: Decrease to the WHO air quality guidelines (WHO-AQG) of 20 µg/m ³
Short-term impacts of Ozone	Scenario 1: All daily maximum values exceeding 160 µg/m ³ are decreased to the WHO-AQG of 160 µg/m ³
	Scenario 2: All daily maximum values exceeding 100 µg/m ³ are decreased to the WHO-AQG of 100 µg/m ³
	Scenario 3: Annual mean decreased by 5 µg/m ³
Long-term impacts of Ozone	Decrease by 5 µg/m ³ in the April-September average levels
Long-term impacts of PM2.5	Scenario 1: Decrease by 5 µg/m ³
	Scenario 2: Decrease to the WHO air quality guidelines (WHO-AQG) of 10 µg/m ³

In practice

- Define the scenarios consistently with the local needs and interests

4.7. Calculating the number of cases

For each health outcome and pollutant, the health impact is computed using the following health impact function;

$$\Delta y = y_0 (1 - e^{-\beta \Delta x})$$

Where;

Δy is the decrease in the health outcome associated with the decrease in pollutant concentrations

y_0 is the baseline health outcome

Δx is the decrease in the pollutant concentration in a given scenario

β is the coefficient of the concentration response function

A confidence interval (CI) of Δy can be calculated using the confidence interval of β , i.e.

$$\Delta y_{\text{sup}} = y_0 (1 - e^{-(\beta - 1.96s)\Delta x})$$

$$\Delta y_{\text{sup}} = y_0 (1 - e^{-(\beta+1.96s)\Delta x})$$

s is an estimate of the standard error of β . Note that the CI only takes into account the uncertainty in the estimate β in the original study but does not take into account other sources of uncertainty (exposure assessment, transfer from the context of the original study to the context of the city where the HIA is performed, etc.).

When assessing short-term impacts of air pollutants, baseline health-outcome data should be calculated using the same years as for air-pollution data.

When assessing long-term impacts of air pollutants, baseline health outcome data can be estimated using all available years, implicitly considering that the estimated chronic exposure from the available years is representative of the cumulative average population exposure.

Aphekom has developed two Excel sheets to calculate the short- and long-term impacts of air pollutants. These sheets focus on the short-term impacts of ozone and PM10 and the long-term impacts of PM2.5. Guidance on how to use these tools appears in Appendixes 1 and 2. An HIA online tool is also provided on the Aphekom Web site. The Excel can also be modified for use for other pollutants.

4.8. Calculations of the gain in life expectancy

When long-term impacts of exposure are considered, the HIA findings for a given scenario can be expressed in terms of annual number of deaths as a measure of the burden of air pollution on mortality. The impacts in terms of gains in life expectancy can also be estimated (Brunekreef et al., 2007; Miller et al, 2003).

There is a debate on which is the better way of expressing the long-term HIA findings (see section interpretation of findings). In Aphekom, we decided to use both, as they complement each other and provide two interesting types of information for stakeholders.

The impact on life expectancy of a decrease in the pollutant concentration can be calculated using a standard abridged life-table methodology.

Mortality data must be collected by age groups.

The life expectancy at 30 is calculated as detailed below, starting with a hypothetical cohort of 100,000 persons aged 30:

Y is the number of years used in the HIA.

x is the starting age in each group

n is the duration interval of each age group

n_{ax} is the average number of years lived by those who died during the interval $[x, x+n)$, which is estimated by $n/2$

${}_n N_x$ is the population in each age group.

${}_n D_x$ is the total number of deaths in each age group averaged over Y years.

${}_n M_x$ is the mortality rate in each age group, calculated as

$${}_n M_x = \frac{{}_n D_x}{{}_n N_x * Y}$$

${}_n q_x$ is the probability of dying in the age group estimated as

$${}_n q_x = \frac{n \times {}_n M_x}{1 + (n - n_{ax}) \times {}_n M_x}$$

For the last age group, which is open, ${}_n q_x = 1$, since everybody will die.

l_x is the number of people alive in the age group.

The number of people alive in the other age groups is calculated as:

$$l_{x+n} = l_x (1 - {}_n q_x)$$

${}_n d_x$ is the number of people who died in the age group calculated as:

$${}_n d_x = l_x * {}_n q_x$$

${}_n L_x$ is the number of person-years lived in each age group calculated for all age groups except the last one,

$${}_n L_x = n * l_{x+n} + n_{ax} * {}_n d_x$$

For the last age-group,

$${}_n L_x = \frac{l_x}{{}_n M_x}$$

${}_n T_x$ is the number of person years that the hypothetical cohort lives after reaching age x and is calculated recursively from the ${}_n L_x$;

$$T_x = T_{x+n} + {}_n L_x$$

e_{30} is the life expectancy at age 30 calculated as

$$e_{30} = \frac{T_{30}}{l_{30}}$$

The impacted life table, i.e. the life table modified by the change in air pollutant, is calculated using the same method, except that ${}_n D_x$ is estimated by:

$${}_n D_x^{impacted} = {}_n D_x * e^{-\Delta x * \beta}$$

The tool calculates the average gain in life expectancy at 30 (e_{30}), and the gain in annual total life years (e_{30} multiplied by an estimation of the population aged 30) by comparing the impacted and the base life tables. They are both summaries of the impact of the scenarios on the mortality experience of the study population.

As we said in section 2, our Excel sheets are adequate for doing a *counterfactual* HIA, as in Aphekom WP5. If you want to do a *predictive* HIA to assess health benefits in a given time framework in the future from a decrease today in pollutant levels, you will have to use tools like IOMLIFET or AIRQ (Miller, 2008).

5. Interpretation of HIA findings

HIA results from our counterfactual approach should be considered as “at least” estimates of the impacts of air pollution for two main reasons:

- The use of a single value to represent exposure for a whole city certainly smoothes the exposure indicators, and does not take into account within-city gradients of pollution. These gradients may be significant, with peaks of pollution especially near traffic. Taking into account within-city contrasts of exposure would require environmental data that are not routinely available in most cities. Aphekom WP4 has explored interesting new avenues in this direction (see Aphekom Web site for more information).
- Air pollution is likely to produce a wide range of health effects. As we are focusing on the most adverse ones (mortality and hospitalisations), we missed a large part of the population affected by less severe symptoms (e.g., restricted activities, reduced performance, medication intake, changes in the cardiovascular function, impaired respiratory function, etc.).

Interpretation of HIA findings should consider the sources of uncertainty in the HIA process including, among others, the question of causality between air pollution and health, uncertainties in exposure assessment and health outcomes, and the choice of the CRFs. Also, a qualitative assessment of these uncertainties is proposed in Aphekom WP7 (see Aphekom Web site for more information).

6. Uncertainties

Following are some elements that can be taken into consideration when discussing uncertainties in HIA.

The first issue is to distinguish between the uncertainties that can be reduced using careful design, and the uncertainties that are inherent to the methodology and cannot be avoided. It is essential to communicate both types of uncertainties and do whatever is possible to reduce manageable uncertainties.

Among the uncertainties that cannot be reduced is the key assumption that causality exists between exposure to air pollution and health outcomes. The number of cases attributable to air pollution can be estimated only if there are strong arguments for this causality as defined by Hill (Hill, 1965). In the case of air pollution, the most important arguments for this causality are: consistency of epidemiological findings reproduced over different geographic areas, periods and study designs; coherence of the observed effects; biological plausibility strengthened by clinical and toxicological studies; more recently the demonstration through intervention studies that changing the exposure causes a

change in the outcome and the analogy found with cigarette smoking (Pope, III *et al.* 2009).

6.1. Uncertainty in exposure assessment

Within-city contrasts

To limit the bias of transferring CRFs from the source studies, you can estimate the average exposure for the population in each city by averaging data from fixed monitors, as in the original studies. Doing so you probably underestimate the impact of chronic exposure to PM, since there can be significant within-city contrasts of exposure, notably in relation to road traffic, that are not taken into account.

Some recent epidemiological findings suggest that within-city contrasts can be even larger than between-cities contrasts and that using these within-city contrasts could give a higher estimate of the impact of PM_{2.5} on mortality (Jerrett *et al.*, 2005).

Taking into account within-city contrasts of exposure requires extensive work on local environmental data but Aphekom WP4 has explored interesting new avenues in this direction (visit www.aphekom.org for more information).

There is a clear need for robust CRFs applicable to within-city contrasts of exposure and health outcomes. Hopefully, the results of the European Escape³ project will be useful for this purpose, as this project is investigating within-cities contrasts of exposure, combining field PM and NO_x measurements and land-use regression modelling (Hoek *et al.*, 2008).

Correction factors

Various methods can be used to measure PM. The TEOM method underestimates the semi-volatile components of PM and various corrections may be applied: real-time correction with data from a station using a gravimetric method or an FDMS correction device; or fixed seasonal correction factors estimated from a local field study comparing TEOM and gravimetric methods. Sensitivity analysis of results to the correction method used can be performed if raw and corrected data is available.

Where PM_{2.5} data is not available, it is possible to estimate PM_{2.5} levels from PM₁₀ data using a 0.7 conversion factor, that was recommended by the Apehis exposure-assessment working group (Apehis, 2005). Earlier work by the Apehis project showed that this conversion can cause a small overestimation of the PM_{2.5} levels. For this reason and given the large share of PM_{2.5} in our HIA results, routine PM_{2.5} monitoring should be extended to more European cities.

3 See the Escape project webwite : <http://www.escapeproject.eu/>

PM chemical composition

Particulate matter is a complex mixture of primary and secondary components whose toxicity likely varies with its composition. As PM composition is also an indication of its source, better knowledge of PM composition and its impact on the health effects is needed, and some recent epidemiologic results are promising in this direction (Lippman *et al.*, 2009). In particular, results from a recent reanalysis of the ACS cohort study suggest it could be useful to use exposure to black carbon and sulfates in HIAs (Smith *et al.*, 2009). This research opens opportunities for new avenues for future work, provided that data on PM chemical composition becomes available for European cities.

Natural dust

In Athens and other southern cities a few days with very high levels of PM may be caused by Saharan dust episodes. In Aphekom, we did not exclude these days from the analysis, implicitly considering that Saharan dust has the same health effects as other PM components. Our decision is justified by recent work showing short-term effects of Saharan dust-related PM on mortality and hospitalisations (Perez *et al.*, 2008; Middleton *et al.*, 2009). Also considering long-term exposure, excluding the peak PM days would have had a minor influence on the average PM_{2.5} levels.

Nitrogen dioxide

Nitrogen dioxide (NO₂) is a toxic gas with effects on respiratory health that have been clearly demonstrated by toxicological and epidemiological studies (WHO, 2006).

However, it is generally agreed, that at the levels observed in an urban setting, which are lower than levels used in controlled human studies, the results of the (numerous) epidemiologic studies showing a link between NO₂ levels and various health effects, notably respiratory effects (see the recent review by USEPA, 2008), could be due in part to other traffic-related pollutants, *e.g.* ultrafine particles, for which NO₂ is a proxy (HEI panel on the health effects of traffic-related air pollution, 2010; Searl, 2004).

Also, performing HIAs on both NO₂ and PM can lead to do double counting, so for the time being we advise not to perform both HIAs, even if we probably underestimate the health impacts of urban air pollution, particularly in the short term.

Nevertheless, as NO₂ levels are currently increasing in Europe, due to an increasing trend in traffic-related primary NO₂ emissions (Carslaw *et al.*, 2007), the use of NO₂ in HIAs should be examined further in future work.

6.2. Uncertainty in health outcomes

Mortality

Mortality remains our first choice for outcomes, because it is robust, easy to obtain and, for all-cause mortality, not subject to misclassification. The impact on mortality accounts

for the larger share of the effects of air pollution when measured in economic terms (*see* www.aphekom.org).

Cause-specific mortality is more prone to misclassification, but chronic PM effects on cardiovascular health are now well documented, even if the CRFs vary between studies (Brook *et al.*, 2010).

Hospitalisations

To broaden the picture, we can also include in an HIA an estimation of the acute effects of PM and ozone on cardiac and respiratory hospitalisations. For comparison between cities hospitalisation data can be quite heterogeneous. This can be due to differences in the coding practices or due to differences in the use of hospitalisation in the health-care systems. However locally, hospitalisation data is a valuable indicator of the health impacts of air pollution beside mortality.

Other morbidity indicators

If available the impact of air pollution on chronic diseases would be a valuable information. Aphekom WP4 explored new avenues and investigated the impact of air pollution related to traffic on chronic diseases and exacerbations using the concepts framed by Künzli *et al.* (2008) (to learn more www.aphekom.org).

6.3. Uncertainties in the CRFs

The uncertainty in the CRFs should be taken into account when interpreting HIA findings. Considering the long-term impact of PM_{2.5} on mortality, the 95% confidence interval in the RR from Pope *et al.* (2002) is (1.02-1.11), which means 4% less or more than the central estimate (1.06). In terms of estimates of the total number of deaths postponed related to a decrease of PM_{2.5} levels to the WHO AQG, This translates into a range from 6,597 to 32,434 deaths (central estimate: 18,801). In terms of median gain in life expectancy at age 30 in the 25 Aphekom cities, this translates into a range from 2.0 to 10.5 months (central estimate: 5.8)⁴.

The original confidence interval, which reflects the uncertainty of the estimate for one particular study, does not capture uncertainties related to other aspects, such as representativeness of the population, shape of the CRF, omitted confounding variables or exposure misclassification.

Kinney *et al.* (2010) have shown how eliciting expert judgement on the uncertainty of the CRFs can inform HIA interpretation. As an indication of the benefits of using this elicitation approach, we used the results of work by COMEAP (2009), which aimed to elicit the view of seven of its experts on the uncertainty surrounding the CRF for long-term effects of PM_{2.5} on total mortality. The aggregated 95% plausibility interval ranged

⁴ A detailed sensitivity analysis using 95% confidence of RRs in the original studies for all pollutant and health outcomes used in Aphekom WP5 is presented in the Deliverable D7b.

from 1.00 to 1.15, *i. e.* approximately from 6% below to 8% above the central estimate (1.06). Translating that into the total number of deaths postponed would lead to a range from 0 to 42,229.65, and to a median gain in average life expectancy at age 30 of 0 to 14.1 months.

7. Communicating HIA findings

HIAs should serve the information and decision-making needs of multiple stakeholders including, among others, policy-makers, health professionals and the general population. As such, at Aphekom we feel careful attention should be paid to expressing HIA findings in terms that are as simple and clear as possible using language and concepts that all these audiences can easily understand, and expressing the findings with minimum use of scientific jargon.

We also suggest that your HIA findings be discussed with a view to their relevance for local policy making.

When communicating HIA findings, it is also important to detail the HIA process to ensure its transparency and the reproducibility of the exercise.

At Aphekom we provide detailed guidelines for performing HIAs of urban air pollution. We also produce single-city reports applying these guidelines that describe the study area, the sources of air pollution, characteristics of the population, data sources and quality controls, and main sources of uncertainty; and discuss local findings. These city reports are posted on the Aphekom Web site.

7.1. Choosing the metrics for reporting mortality results

As the main burden is on the long-term effects of PM chronic exposure on mortality, it is important to carefully choose the metrics for reporting mortality HIA results. This was the subject of a workshop Aphekom organized at the 2009 ISEE Conference (see proceedings on the Aphekom Web site).

There is a long tradition of using the number of “attributable” or “postponed” deaths to report the results of assessments of the burden on health of major risk factors (e.g., tobacco, alcohol and obesity) and also of air pollution (Künzli *et al.* 2000). It is true that, after the effects on mortality of a decrease in PM_{2.5} levels are achieved, as mortality rates decrease so does the annual number of deaths. But in the long run, decreasing mortality rates will make the population live longer, thus increasing the annual number of deaths (Miller *et al.*, 2003). This fact has led some authors to prefer focusing on the impact on life expectancy, because this impact will persist in the long run (Brunekreef *et al.*, 2007).

Period life expectancy at a given age, as a cross-sectional indicator, is both a good summary of the population mortality experience in a given year, and an estimate of average life expectancy for a subject at a given age. It has, for example, been used by Pope *et al.* (2009) to assess *ex post* the impact of the PM_{2.5} decrease in recent decades in

the U.S. In Aphekom, we suggest to focus on this metric, in terms of the potential average gain in period life expectancy in a given year, as an indicator of the burden of PM_{2.5} on mortality.

We use life expectancy at age 30 to make it clear that we only considered the impact after 30 years of age by applying the results of Pope *et al.* (2002, 2004) on the population 30 years of age and over. For example, we do not consider the effects of PM on infant mortality (Lacasaña *et al.*, 2005), both because the evidence is less robust in terms of CRFs and because data on the first year of life are statistically less reliable when working with local life tables.

Readers should be aware of the difference between life expectancy at birth and life expectancy at age 30. The latter is a conditional life expectancy, applicable for people still alive at 30. However, as the PM impact is on the group 30 years of age and older, the difference between gain in life expectancy at birth and gain in life expectancy at 30 is negligible (results not shown).

We also go beyond the average impact on life expectancy and quantify the total impact on the population in terms of the potential gain in total life years in the population (Brunekreef *et al.*, 2007). This is needed, in particular, for purposes of economic valuation.

To quantify this total impact, some authors have multiplied the number of “attributable” deaths in each age group by the conditional life expectancy at this age (COMEAP, 2010). This is hard to grasp, because it mixes the current situation with what the current situation will become when the benefits of the PM decrease are achieved.

In Aphekom we chose a different method building on the difference between two period life tables – the observed one and the counterfactual one – that could have been observed if the PM effects of the decrease in PM levels had been achieved. Our method essentially consists of multiplying the average gain in life expectancy at the starting age (here 30) by the size of the population at this starting age. The result can thus be interpreted as a summary indicator of the burden of PM on mortality in the given period.

Further work on comparing the different approaches to compute gain in total life years will be useful.

In these guidelines we also present the impact in terms of number of postponed deaths. Doing so enables your results to be compared with previous results using this metric. Indeed, the number of postponed deaths is also a useful indicator of the burden of air pollution on mortality.

Both metrics complement each other and provide two interesting types of information for stakeholders.

Appendix 1 – Guidelines for using the Aphekom HIA short-term tool

1. Objectives

The Aphekom HIA Excel sheets were developed as tools for performing standardised health impact assessments (HIAs) of air pollution using the latest scientific evidence. In order to use this tool, routine health and air quality monitoring data are required. Guidelines for creating appropriate databases are part of the WP5 deliverables.

The present document provides information on how to use the Excel sheet for calculating:

- the short-term impact of PM10 on total non-external mortality and on cardiac and respiratory hospitalisations
- the short-term impact of ozone on total non-external mortality and on cardiac and respiratory hospitalisations

While the tool has been designed to suit the objectives of the Aphekom project, particular attention was given to make it as flexible as possible, so that it can easily be adapted to other pollutants, scenarios, etc.

2. Methods

2.1. The health impact function

For the different outcomes, the health impact function will be:

$$\Delta y = y_0 (1 - e^{-\beta \Delta x})$$

Where Δy is the outcome of the HIA as a decrease in the number of health events

y_0 is the baseline health data

Δx is the decrease in the concentration defined by the scenario.

β is the concentration response function.

RR per 10 $\mu\text{g}/\text{m}^3 = \exp(10 * \beta)$

The results are then adjusted on the number of years N to give a yearly estimate

$$\Delta y_{outcome\ yearly}^{scenario} = \frac{\Delta y_{outcome}^{scenario}}{N}$$

The results are then adjusted on the total population to give a yearly estimate per 100 000.

$$\Delta y_{outcome\ yearlypop}^{scenario} = \frac{\Delta y_{outcome\ yearly}^{scenario}}{pop}$$

The method for calculating Δx differs for different pollutants.

2.2. The short-term impacts of PM10

Δx is calculated on yearly averages. Only the N years with less than 25% of missing values will be used for calculating the environmental data (Δx) health reference data (y_0).

Two scenarios are considered:

- Scenario 1, where the PM10 yearly mean is decreased by 5 $\mu\text{g}/\text{m}^3$. In that case, $\Delta x = 5 \mu\text{g}/\text{m}^3$
- Scenario 2, where the PM10 yearly mean is decreased to 20 $\mu\text{g}/\text{m}^3$. In that case,

$$\Delta x = ([PM10]_{\text{mean}} - 20 \mu\text{g}/\text{m}^3)$$

$$\Delta x = 0 \text{ if } [PM10]_{\text{mean}} \leq 20$$

2.3. The short-term impacts of ozone

Δx is calculated on the accumulated excess concentrations above a threshold, using the ozone 8-hour -daily values. We used the days for which a valid ozone concentration is available, within the years for which we have more than 75% of valid data. **Please check that missing data are not concentrated during the summer.**

Three scenarios are considered:

- Scenario 1, where all the maximum 8-hour concentrations >160 are decreased to $160 \mu\text{g}/\text{m}^3$. In that case, for each day i ,
if $[O_3]_i \geq 160 \mu\text{g}/\text{m}^3$, $O_i = ([O_3]_i - 160)$
if $[O_3]_i < 160 \mu\text{g}/\text{m}^3$, $O_i = 0$

$$\Delta x = \frac{\sum_{i=1}^N O_i}{N}$$

- Scenario 2, where all the maximum 8-hour concentrations >100 are decreased to $100 \mu\text{g}/\text{m}^3$. In that case
if $[O_3]_i \geq 100 \mu\text{g}/\text{m}^3$, $O_i = ([O_3]_i - 100)$
if $[O_3]_i < 100 \mu\text{g}/\text{m}^3$, $O_i = 0$

$$\Delta x = \frac{\sum_{i=1}^N O_i}{N}$$

- Scenario 3, where the ozone yearly mean is decreased by $5 \mu\text{g}/\text{m}^3$. In that case, $\Delta x = 5 \mu\text{g}/\text{m}^3$

Concentration-response functions (CRFs) are detailed in **Erreur ! Source du renvoi introuvable..**

Table 1- CRFs used in the Excel sheet.

HIA	Air pollution metrics	Health outcome	Ages	RR per $\mu\text{g}/\text{m}^3$	10 B	Ref
Short-term impacts of PM10	Annual average	All non-external mortality	All	1.006 [1.004-1.008]	0.000598207	WHO, 2004
	Annual average	All respiratory hospitalisations	All	1.0114 [1.0062-1.0167]	0.001133551	Atkinson et al, 2005
	Annual average	All cardiac hospitalisations	All	1.006 [1.003-1.009]	0.000598207	Atkinson et al, 2005
Short-term impacts of O₃	Daily max-8h concentrations	All non-external mortality	All	1.0031 [1.0017-1.0052]	0.00030952	Gryparis et al, 2004
	Daily max-8h concentrations	All respiratory hospitalisations	15-64	1.001 [0.991-1.012]	0.00009995	WHO, 2004
	Daily max-8h concentrations	All respiratory hospitalisations	≥ 65	1.005 [0.998-1.012]	0.000498754	WHO, 2004

3. How to use the Excel tool

The tool requires macros to work correctly. In Excel, you can set the level of security that is applied to a macro. We suggest that you set a medium level of security when you use the tool, and that you enable the macros.

To set the level of security, go to Tools -> options -> security -> macro security

3.1. Format

The Excel file comprises four sheets and uses a colour code:

Green = sheets that need your input: environmental data, health data and population data

Blue = sheets containing the hypothesis that will be used for the HIAs. By default, all parameters are set to the ones defined in the Aphekom project. However, you can modify these parameters. **Red** = results

A similar code is applied to the cells.

3.2. Input data

Prior to using the tool, you must define your study area and collect the environmental and health data as specified in the main section of this report.

In the examples below, we use data from Marseille for the years 2004-2006.

3.3. Sheet "1-Environmental data"

It is extremely important to respect the format of this sheet when inputting your data. Otherwise, the calculations may not work.

You must input your data column by column:

- dates column A, from cell A4
- O₃ concentrations column B, from cell B4
- PM10 concentrations column C, from cell C4

If you need to apply a correction factor to the PM data (correction from the TEOM to the gravimetric method), you have to input the factors in cells C1 (for winter) and G1 (for summer).

The sheets will clean your data, removing text values like NA for instance, and create two columns of valid concentrations for the HIA, labelled O3(year) and PM10(year). The tool will also calculate values of ozone for scenario 1, labelled O3 (year)-S1, and for scenario 2, labelled O3(year)-S2.

3.4. Sheet "2-Health data"

Again, it is extremely important to respect the format of this sheet when inputting your data. Otherwise, the calculations may not work.

You must input the annual number of cases for each of the health outcomes and age groups, and for years 2004 to 2006, as illustrated below:

Health Outcome	ICD-9 Codes	ICD-10 Codes	Age Group	2004	2005	2006
Total Non-external Causes Mortality	001-799	A00-R99	All Ages	8 182	8 371	8 126
Cardiac Hospital Admissions	390-429	I00-I52	All Ages	12 528	12 986	13 525
Respiratory Hospitalisations Admissions	460-519	J00-J99	All Ages	9 851	9 946	9 673
Respiratory Hospital Admissions	460-519	J00-J99	15-64	3 550	3 590	3 563
Respiratory Hospital Admissions	460-519	J00-J99	65 and over	3 456	3 668	3 273

3.5. Sheet “3-Population data”

Don't forget, it is extremely important to respect the format of this sheet when you input your data. Otherwise, the calculations may not work. Fill in the green cells as appropriate.

Total Population of the study Area	
All Ages	955 702
15-64	614 045
65 and over	170 302

3.6. Sheet “4-Hypothesis”

This sheet summarises the scenarios applied and the RR that will be used. If you want to modify the years used in the HIAs, the scenarios or the RR, please do it on this sheet only, and do not modify the others.

You can modify the years used in the HIA.

Years used in HIA
2004
2005
2006

If you want to modify the scenarios, you should change the parameter column.

Scenarios		
Ozone	Description of scenario	Parameter
Scenario 1	Values >160=160	160
Scenario 2	Values >100=100	100
Scenario 3	Annual mean decreased by 5	5
PM10	Description of scenario	Parameter
Scenario 1	Annual mean decreased by 5	5
Scenario 2	Annual mean decreased to 20	20

If you want to change the RR, you can use the RR columns. Be careful that RRs should be per 10µg/m³

RR	per	10	µg/m ³		
Pollutant	Indicators	Lower	Central	Upper	Ref
O3	Total non-external mortality	1.0017	1.0031	1.0052	Gryparis and al, 2004
	Respiratory hospitalisations (15-64)	0.991	1.001	1.012	WHO, 2004
	Respiratory hospitalisations (65 and over)	0.998	1.005	1.012	WHO, 2004
PM10	Total non external mortality	1.004	1.006	1.008	WHO, 2004
	Total respiratory hospitalisations	1.0062	1.0114	1.0167	Atkison and al, 2005
	Total cardiac hospitalisations	1.003	1.006	1.009	Atkison and al, 2005

3.7. Sheet “PM10 results – Scenario 1”

The tool automatically gives an overview of the exposure data for the whole year and by season (winter = October-March, summer = April-September), for all the years and year by year.

Distribution of exposure indicators

		2004	2005	2006
Exposure data	PM10 (all period)	PM10	PM10	PM10
Number of day	1096	366	365	365
Minimum	4	5	8	4
Percentile 5	14	13	14	14
Percentile 25	20	20	21	20
Median	27	27	28	27
Percentile 75	35	35	34	35
Percentile 95	48	48	47	48
Maximum	111	111	59	86
Daily mean	29	29	28	29
Standard deviation	11	12	10	12
% Valid data	100	100	100	100

Below you will find a summary of the number of days with valid data per year. The HIA only uses years with more than 75% of valid data. The annual means of PM10 per valid year are also reported.

Number of data (N) and the % of available data by year and by pollutant			
Number of days with valid data	% of days with valid data	Year used in the HIA	Annual mean of PM10 for years used in the HIA
366	100.00%	Yes	32
365	100.00%	Yes	32
365	100.00%	Yes	33

For the same N years, the tool reports the total health outcomes. You can check the values for all non-external mortality y_{0mort} , all respiratory hospitalisations y_{0resp} and all cardiac hospitalisations y_{0card} .

	2004	2005	2006	Annual mean
All Non-external Causes Mortality (All ages)	8182	8371	8126	8226
Cardiac Hospitalisations (All ages)	12528	12986	13525	13013
Respiratory Hospitalisations (All ages)	9851	9946	9673	9823

The sheet will calculate the annual mean and the associated Δx ,

	Scenario 1
[PM10]mean	32
ΔX	5

If you want to modify Δx , please modify the 4-hypothesis sheet and not this one.

Results are presented for each cause of death, with the lower and upper estimates corresponding to the IC95% of the RR.

	All external mortality lower	non - external mortality	All external mortality upper	non - external mortality
Total number of deaths avoided (Δy)	49.3	73.9	98.5	
Yearly number of deaths avoided (Δy yearly)	16.4	24.6	32.8	
Yearly number of deaths avoided per 100 000 (Δy yearly/pop)	1.7	2.6	3.4	

3.8. Sheet “PM10 results – Scenario 2”

The process is similar to the one described in scenario 1.

3.9. Sheet “Ozone results – scenario 1”

As for PM10, the tool automatically gives an overview of the exposure data for the whole year and by season (winter = October-March, summer = April-September), for all the years and year by year.

You can use this table to check that the number of missing values is not concentrated during summer, which might impair interpretation of the results.

The sheet calculates the accumulated concentration above $160 \mu\text{g}/\text{m}^3$ and the associated Δx .

Accumulated value >160	24
ΔX	0.02

If you want to modify Δx , please modify the 4-hypothesis sheet and not this one!

Results are reported as a total outcome, outcome adjusted on the number of years, and on the population /100 000 inhabitants, similar to PM10.

3.10. Sheet “Ozone results – scenario 2”

The process is similar to the one described in Ozone results - scenario 1.

3.11. Sheet “Ozone results – scenario 3”

The process is similar to the one described in PM10 results - scenario 1.

3.12. Graphs and summary tables

The tool produces graphs of the daily concentrations of PM10 (sheet Graph_ PM10) and ozone (sheet Graph_Ozone) and summary tables and graphs (sheet summary).

Appendix 2 – Guidelines for using the Aphekom HIA long-term tool

1. Objectives

The Aphekom HIA Excel sheets were developed as tools to help perform standardised health impact assessments (HIAs) of air pollution using the latest scientific evidence in the Aphekom project. The HIA requires routine health and air quality monitoring data. Guidelines for creating appropriate databases are part of these WP5 deliverables.

The present document provides information on how to use the Excel sheets for computing the long-term impacts of PM_{2.5} on total non-external mortality and cardiovascular mortality.

While the HIA long-term tool was designed to suit the objectives of the Aphekom project, particular attention was given to make it as flexible as possible, so that it can easily be adapted to other pollutants, scenarios, etc

2. Methods

The Excel tool includes a calculation of the loss of life expectancy.

The period life expectancy is calculated using standard actuarial methodology for 5-year age groups life tables as detailed below

Y is the number of years used, here 3

x is the starting age in each group

n is the duration interval of each age group

n_{ax} is the average number of years lived by those who died during the interval $[x, x+n)$, which is estimated by $n/2$

${}_nN_x$ is the population in each age group

${}_nD_x$ is the total number of deaths in each age group for years 2004, 2005 and 2006

${}_nM_x$ is the mortality rate in each age group, calculated as

$${}_nM_x = \frac{{}_nD_x}{{}_nN_x * Y}$$

${}_nq_x$ is the probability of dying in the age group estimated as

$${}_nq_x = \frac{n * {}_nM_x}{1 + (n - n_{ax}) * {}_nM_x}$$

For the last age group, which is open, ${}_nq_x = 1$, since everybody will die.

l_x is the number of people alive in the age group. We start from a hypothetical cohort of 100 000 people being alive at 30. The number of people alive in the other age groups is calculated as:

$$l_{x+n} = l_x (1 - {}_nq_x)$$

${}_nd_x$ is the number of people who died in the age group calculated as:

$${}_n d_x = l_x * q_x$$

${}_n L_x$ is the number of person-years lived in each age group calculated as:

$${}_n L_x = n * l_{x+n} + n_{ax} * d_x$$

For the last age-group:

$${}_n L_x = \frac{l_x}{M_x}$$

T_x is the number of person years that the hypothetical lives after reaching age x and is calculated recursively from the ${}_n L_x$:

$$T_x = T_{x+n} + {}_n L_x$$

e_x is the life expectancy at age x calculated as:

$$e_x = \frac{T_x}{l_x}$$

The impact life table is calculated using the same method, except that ${}_n D_x$ is the total number of deaths in each age group for years 2004, 2005 and 2006 when the concentrations of the specific pollutant are decreased.

$${}_n D_x^{impacted} = {}_n D_x * e^{-\Delta x * \beta}$$

Δx is the decrease in the concentration defined by the scenario

β is the concentration response functions.

RR per $10 \mu\text{g}/\text{m}^3 = \exp(10 * \beta)$

For PM2.5, two scenarios are considered

- Scenario 1, where the PM2.5 yearly mean is decreased to $5 \mu\text{g}/\text{m}^3$. In that case, $\Delta x = 5 \mu\text{g}/\text{m}^3$
- Scenario 2, where the PM2.5 yearly mean is decreased to $10 \mu\text{g}/\text{m}^3$. In that case, $\Delta x = ([\text{PM2.5}]_{\text{mean}} - 10 \mu\text{g}/\text{m}^3)$.
 $\Delta x = 0$ if $[\text{PM2.5}]_{\text{mean}} < 10$

Scenarios and concentration-response functions (CRFs) are detailed below and in Table 1.

Final results are expressed as reduction in the annual number of deaths per 100 000. For total mortality, results are also expressed as gains in life expectancy.

Table 1. Scenarios and CRFs used in the Excel sheet.

HIA	Air pollution metrics	Scenario 1	Scenario 2	Health outcome	Ages	RR per 10 $\mu\text{g}/\text{m}^3$	β	Ref
Long-term impacts of PM2.5	Annual average	Decrease by 5 $\mu\text{g}/\text{m}^3$	Decrease to 10 $\mu\text{g}/\text{m}^3$	All including non-external mortality	>30	1.06 [1.02-1.11]	0.0058269	Pope et al, 2002
	Annual average	Decrease by 5 $\mu\text{g}/\text{m}^3$	Decrease to 10 $\mu\text{g}/\text{m}^3$	All cardiovascular mortality	>30	1.12 [1.08-1.15]	0.0056664	Pope et al, 2004

3. How to use the Excel tools

The tools require macros to work correctly. In Excel, you can set the level of security that is applied to the macro. We suggest that you set a medium level of security when you use the tools, and that you enable the macros.

To set the level of security, go to [Tools -> options -> security -> macro security](#)

3.1. Format

The Excel file comprises four sheets and uses a colour code:

Green = sheets that need your input: environmental data, health data and population data

Blue = sheets containing data that you do not need to input, but that will be used in the calculations

Red = results

A similar code is applied to the cells.

3.2. Input data

Prior to using the tool, you must define your study area and collect the environmental and health data as specified in the main section of this report.

In the examples below, we use data from Marseille.

3.3. Sheet "1-Environmental data"

It is extremely important to respect the format of this sheet when you input your data. Otherwise, the calculations may not work.

You must input your data column by column:

- Dates: column A, from cell A4
- PM2.5 concentrations: column B, from cell B4
- PM10 concentrations, if you don't have PM2.5 concentrations; column C, from cell C4

You can add a correction factor for the PM measurement method.

If you want to use PM10 to estimate PM2.5 concentrations, you can modify the conversion factor in the tool.

Conversion factor PM10/PM2.5	0.7
PM correction factor (summer)	1.0
PM correction factor (winter)	1.0

The sheets will automatically summarise the period available, the total number of days and the number of days with valid data (i.e., not empty and not text).

3.4. Sheet “2-Health data”

Again, it is extremely important to respect the format of this sheet when you input your data. Otherwise, the calculations may not work.

You must input the annual number of cases for each group and age groups, and for years 2004 to 2006 as illustrated below. The total between 2004 and 2006 is automatically calculated.

3.5. Sheet “3-Population data”

Don't forget, it is extremely important to respect the format of this sheet when you input your data. Otherwise, the calculations may not work. Fill in the green cells as appropriate.

Total population of the study area	
30-34	63 741
35-39	66 215
40-44	66 161
45-49	62 875
50-54	60 041
55-59	57 183
60-64	46 607
65-69	40 160
70-74	40 826
75-79	37 366
80-84	29 838
85 and over	22 113

3.6. Sheet “4-Hypothesis”

This sheet is for information purposes only and summarises the scenarios applied and the RR that will be used.

You can modify the parameters to change the scenarios or the years used in the HIA.

Scenarios		
PM2.5	Description of scenario	Parameter
Scenario 1	Annual mean decreased by 5	5
Scenario 2	Annual mean decreased to 10	10

Years used in HIA
2004
2005
2006

3.7. Sheet “Descriptive data”

The tool automatically gives an overview of the exposure data for the whole year and by season (winter = October-March, summer = April-September), for all the years and year by year.

Exposure data	PM2.5
Number of days	748
Minimum	3
Percentile 5	7
Percentile 25	11
Median	16
Percentile 75	23
Percentile 95	35
Maximum	77
daily mean	18
Standard deviation	10

You can check the number of days available each year and the annual means of PM2.5 for these years.

Number of data (N) and the % of available data by year and by pollutant						
	N_PM2.5	% available_PM2.5 of	Year used in the HIA	Total number of days used in the HIA	Annual mean of PM2,5 for years used in the HIA	Number of years
2004	348	95.08%	Yes	348	17.45	
2005	313	85.75%	Yes	313	17.22	
2006	87	23.84%	No			
Total	748			661	17.34	3

The tool also gives you an overview of the data used in the HIA

	N_PM2.5	% available_PM2.5 of	Year used in the HIA	Total number of days used in the HIA	Annual mean PM2.5 for years used in the HIA	Number of years	Annual number of cases mortality CV
2004	348	95.08%	Yes	348	17.45		2 398
2005	313	85.75%	Yes	313	17.22		2 424
2006	87	23.84%	Yes	87	24.92		2 397
Total	748			748	19.86	3	7 219

3.8. Sheet "PM2.5-Results-Total-Mortality-S1"

You can check the scenario and the impact factor, calculated as $impactfactor = e^{\Delta x * \beta}$. If you want to modify the scenario, please use the hypothesis sheet.

[PM2.5]mean	20
ΔX	5

	All (including external) mortality		
	lower	central	upper
RR	1.02	1.06	1.11
□	0.001980263	0.0058269	0.010436
Impact factor	1.009950	0.971286	1.053565

The tool will automatically calculate the period life table from the population and health data.

NB: For convenience purposes, the period life table and the impacted life table are hidden in the results sheets. You can display them by doing the following;

- select the first twolines, which correspond to line 3 and line 65
- right click, and select "display"

Results are summarised as:

	lower	central	upper
Period Life Expectancy	52.11	52.11	52.11
Impacted Life Expectancy	51.98	52.47	51.46
Life Expectancy Gain	-0.12	0.37	-0.65
	lower	central	upper
Current Annual Number of Deaths	8226.33	8226.33	8226.33
Impacted Annual Number of Deaths	8308.19	7990.12	8666.98
Reduction in the Annual Number of Deaths (number)	-81.86	236.21	-440.65
Reduction in the Annual Number of Deaths (rate per 100,000)	-13.80	39.82	-74.29

3.9. Sheet "PM2.5-Results-Total-Mortality-S2"

This sheet presents the results for PM2.5, total mortality scenario 2.

3.10. Sheet "PM2.5-Results-Cardiovascular-S1"

This sheet presents the results for PM2.5, cardiovascular mortality scenario 1.

Life expectancy is not calculated for cardiovascular mortality.

3.11. Sheet "PM2.5-Results-Cardiovascular-S2"

This sheet presents the results for PM2.5, cardiovascular mortality scenario 2.

3.12. Graphs and summary tables

The tool produces graphs of the daily concentrations of PM2.5 (sheet Graph_PM2.5) and summary tables and graphs (sheet summary).

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