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ICARUS

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D5.3 – Methodology Report on the Relationship between Policies and Measures

WP5 Integrated assessment for short to medium term policies and measures

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1 INTRODUCTION

This deliverable describes the methods employed in estimating the relationship between policies and measures. It includes a detailed description of assumptions and mathematical operators for estimating effect, costs and utility losses for use in the ICARUS project.

Analysis of policies and measures requires a careful investigation of the options at hand. This report is structured as follows. First, an overview of the policies and measures to be considered in ICARUS is presented, drawing on the outputs of Deliverable 5.2. Then, different conceptual models are presented which could be used in ICARUS to link environmental change arising from pollution mitigation measures to impacts – be they in terms of environment or health. Then methods for measuring and valuation of the key impacts are presented. The concept of resilience planning is then discussed, before methods for dealing with uncertainty are identified.

This Deliverable will assist in the development of cost-benefit analysis for the different policies and in examining urban futures developed as part of the ICARUS project. By its nature, it will need to be a living document – taking in new dose-response functions and valuation studies as they arise from emerging studies in the literature.

2 Overview of Policies and Measures

Many cities in Europe are affected by air pollution and are at the same time major contributors to the emissions of air pollutants and greenhouse gases. Effective city, national, EU level policies and measures to reduce emissions will have to consider the interest of citizens for clean air and will simultaneously have to rely on the feasibility of interventions of achieving these very goals.

This section provides an overview of urban measures and policies chosen for further evaluation in ICARUS. It is therefore based on Deliverable 5.2 – two databases of policies and measures at the city level, which presents the inventarisation of air pollution reduction policies and measures for each participating city in ICARUS. After the finalization of Deliverable 5.2 further important improvements regarding the classification of the city-specific measures have been made, which is why this section presents the status of policies and measures as they are currently stored in the databases and ready for detailed assessment of impacts in WP5. The database set up is oriented on the Guidelines for reporting on policies and measures by Member States under Regulation (EZ) No525/2013 (EEA 2018), the MURE database of energy efficiency policy measures (ISINNOVA 2018) and the inventory of measures in German air quality plans (Diegmann et al. 2015). The structure of the databases with all information stored in the data sheets can be taken from ICARUS Deliverable D5.2.

During the first phase of measure collection in WP5 over 720 potential policies and measures have been identified in relation to the incentives of the EU, national and municipal authorities as well as transport and energy providers. Phase 1 was followed by a selection of approximately 10 policies and measures per city (97 altogether) which was made based on predefined selection criteria. The criteria consisted of (1) compliance of both AQ limit values and WHO health-based guidelines, (2) reduction in long-lived GHG and short-lived climate pollutant (SLCP) emissions and (3) changes in sequestration (i.e. CO2). The policies and measures chosen for further detailed consideration cover a wide range of possible abatement options. Therefore, the first categorization of the measures and policies aims to identify commonly addressed sectors and overarching strategies (policy thematic categories). The first level of classification refers to seven ***macro-sectors*** and ***sub-sectors*** of the activities involved. The list presented below is not exhaustive but shows the sectors relevant for the ICARUS city measures:

1. Buildings and households
    a. Non-residential buildings
    b. Residential buildings
2. Tertiary
    a. Non-residential buildings
3. Transport
    a. Cycling/walking
    b. Public transportation (metro/bus)
    c. Road transport
    d. All transport modes affected
    e. Stationary energy consumption
4. Energy supply
    a. Energy distribution
    b. Power plants
5. Industry

6. Waste management
7. Other

The city-specific policies and measures can be traced back to universal objectives and strategies that are common among all ICARUS cities. They can be supported by different policy interventions and ecopolitical instruments. These common ***policy thematic categories*** indicate the intention why a specific policy intervention will be implemented but are not equal to the response from population subgroups or emission source operators, since the actual response can differ from the expected or targeted objective.

The policy thematic categories in the database are as follows:

Table 2-1 Policy thematic categories in the database

Sector	Policy thematic category
Buildings and households	Enhanced energy conscious behavior
	Increase of building renovation and efficient design
	More environmental friendly heating technologies
	Other energy related investments
	Efficiency improvement of appliances
Tertiary	Increase of building renovation
	Enhanced energy conscious behavior
Transport	Car-independent lifestyles
	Alternative fuels and driving technologies (e-mobility, hybrids, CNG, LPG)
	Increase of vehicles with high emission standard
	Retrofitting of old cars
	Efficient logistics
	Sustainable mobility plans
	Demand management strategies
	Traffic management and optimization
	Efficiency improvement of appliances
Energy supply	Promotion of district heating

Sector	Policy thematic category
	Switch to less carbon-intensive fuels
Industry	Use of fuel alternatives
Waste management	Eco-friendly waste management with citizens participation
	MSW incineration and energy recovery
Other	Regeneration of neighborhoods
	Climate change adaption
	Promotion of green spaces
	Reduction of other emission sources

The second categorization of the measures and policies chosen by the cities aims to find a coherent description of policy interventions to allow the modelling of effect and costs.

During the collection of city-specific measures and policies, it was found that the term *measure* is predominantly used to describe a policy intervention that can be put into practice by municipal and national authorities or by the European Union. In this context the term *measure* is mainly influenced by abatement options specified in air quality plans, climate actions plans and energy concepts. Therefore, it does not directly imply the reactions of population subgroups or emission source operators when confronted with the measure. However, different responses to the same measure can occur among the city inhabitants and vary also across different regions (Miranda et al. 2015). In integrated assessment modeling no uniform definition of the term *measure* is given, but it is commonly referred to as changes in activities and/or emission factors that are based on the actions of individuals or emission source operators (Catarina Sternhufvud, Stefan Åström 2006; Sternhufvud et al. 2006; UCL 2004). Both definitions of a *measure* therefore address different levels (city level – individual/operator level). Thus, in the following the term *policy intervention* will be used for the options and measures an authority can take and the term *response* for the expected reactions that can be expressed as activity and/or emissions factor changes. The underlying assumption is that to estimate the effect of a specific measure, both the general framework (options an authority can take) and the expected response of individuals and emission source operators that lead to the emission reduction need to be described (cf. Figure 2-1).

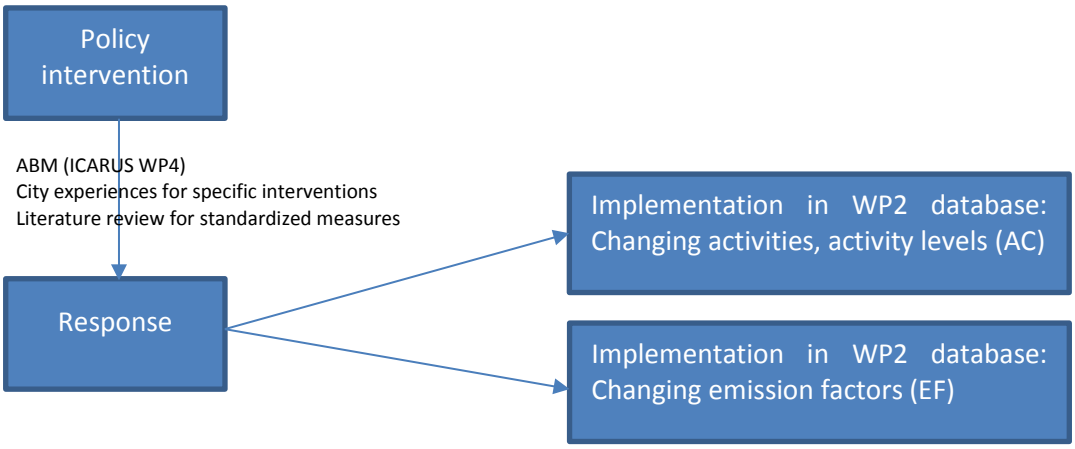

Figure 2-1 Term measure: policy intervention and responses

The next level of categorization in the database describes the ***policy intervention***. Policy interventions are defined as the use of eco-political instruments to enforce environmental protection aims (here referred to as policy thematic categories) by different authorities. The term *policy intervention* refers to urban policies as well as regional and/or EU wide policies, as long as the latter have a considerable effect on air pollution in cities.

The *policy interventions* presented in the databases are accompanied by a description of the **ecopolitical instrument** used (command and control, regulations, certificates, permits, financial incentives, taxes and charges, subsidies, liability law, information/education/training, voluntary agreements, corporate social responsibility) and the **authority** responsible for the implementation (companies or administrative institutions like the city, federal state, government, and the EU).

The collected city-specific *policy interventions* can be clustered into ***standardized measures*** (e.g., low emission zones, highway speed limits, fuel shifts) to allow inter-comparability (Diegmann et al. 2015; examples given in D'Elia et al. 2009; Gulia et al. 2015). The database therefore shows the specific policy intervention in each city named in accordance with the input of each city partner and the assigned standardized measure. This approach allows the definition of uniform responses for standardized measures which will be used to model the emission mitigation potential. Assumptions for responses that are based on experiences of one particular city can be assigned to the city-specific policy interventions.

Several studies show that emission source sectors which are most relevant for air quality at the urban scale are road transport, off-road transport, machinery, residential heating and with a minor contribution also energy production and industry (Viaene et al. 2016; Fuller et al. 2013). In accordance with these findings the city emission inventories of ICARUS WP2 indicate the particularly high contribution of *road transport* to the overall city emissions. The second most important emission source sector, especially for PM_{10}, is *small and medium combustion plants* that are included in the buildings and households sector. Households also account for a considerable amount of cities' CO_2 emissions. Furthermore, policy interventions related to energy consumption and supply as well as transportation are the two areas that have the most direct impact on both urban air quality and climate change and thus highlight the importance of an integrated assessment. Source apportionment methods with respect to pollution sources can be used to quantify the contribution of different source

groups (like road traffic, industrial sources, domestic combustion, other traffic) to direct urban air pollution levels (Diegmann et al. 2015). The results of the analysis in ICARUS WP3 will be compared to the first assessment of important sectors at the city level which might lead to a revision of selected measures.

Table 2-2 shows the policy thematic categories and current standardized measures. An analysis of the distribution of chosen policy interventions among the different source sectors shows that about 40 % of the selected city measures are referring to the source group *buildings and households* and *tertiary* and further 40 % to the source group *transport*. The remaining measures aimed with decreasing importance at the source sectors *energy supply*, *other*, *waste management* and *industry*.

The sectors *buildings and households* and *tertiary* mainly comprise policy interventions focusing on the thematic categories insulation and renovation of private, municipal and tertiary buildings, a switch to more environmental friendly combustion technologies, energy conscious behavior among citizens, the use of energy efficient appliances and other energy related investments like the installation of photovoltaic systems on buildings. Policy interventions related to the implementation of green roofs on are also included in the sector buildings and households. About two thirds of the policy interventions in buildings and households target at energy efficient building renovation and more environmental friendly heating technologies.

Policy interventions related to transportation mainly focus on road transport and address a variety of different objectives such as car-independent lifestyles, alternative fuels and driving technologies, high emission standards among the urban vehicle fleet and retrofitting of old cars, and management of demand, traffic and logistics. More than one third of the transport interventions focus on the promotion of car independent lifestyles and one quarter on demand management strategies. The development of car-independent lifestyles is supported by measures like the introduction of new underground railway/metro lines, the expansion of bus lanes network, improving cycle networks, pedestrian friendly networks, price reductions in public transport and increased use of car sharing.

The *energy supply* sector comprises policy interventions regarding the expansion of district heating including co-generation plants and natural gas use. Not included in the selected policy interventions but also belonging to this category are possible interventions for a penetration with renewable energies and efficiency improvements of power plants.

The *industry* sector covers policy interventions aiming at the use of fuel alternatives, while chosen policy options in the *waste management* sector target at energy recovery and waste prevention.

The sector *other* comprises various policy interventions regarding regeneration of neighborhoods, climate change adaption, urban green spaces other emission sources not included in the primary emission source sectors.

Table 2-2 Standardized measures in the database

Sector	Policy thematic category	Standardized measure (policy intervention)
Buildings and households	Enhanced energy conscious behavior	Energy conscious use of appliances
		Energy conscious use of domestic heating
	Increase of building renovation and efficient design	Energetic renovation of municipal buildings/properties
		Energetic renovation of residential buildings
		Energy efficient design of new buildings
		Implementation of green roofs
	More environmental friendly heating technologies	Switch to gas boilers
		Switch to solar heating
		Switch to heat pumps
		Switch to district heating
		Switch to biomass burning
	Other energy related investments	Penetration of PVs
	Efficiency improvement of appliances	Use of CLF and LED lamps
		Use of air conditioners with new inverter technology Use of energy efficient appliances
Tertiary	Increase of building renovation	Energetic renovation of tertiary buildings
	Enhanced energy conscious behavior	Energy conscious use of appliances
Transport	Car-independent lifestyles	Introduction of new underground railway/metro lines
		Expansion of bus lanes network
		Improving cycle networks

Sector	Policy thematic category	Standardized measure (policy intervention)
		Pedestrian friendly networks
		Price reductions in public transport
		Increased use of car sharing
	Alternative fuels and driving technologies (e-mobility, hybrids, CNG, LPG)	Penetration of electric vehicles
		Penetration of hybrid vehicles
		Penetration of bio-fuel
		Penetration of CNG
		Penetration of LPG
	Increase of vehicles with high emission standard	Withdrawal of old cars
	Retrofitting of old cars	Hardware update of diesel EURO 5
		Software update of diesel EURO 5
	Efficient logistics	Urban logistic concepts
	Sustainable mobility plans	Low emission zone
		City toll/congestion charge
	Demand management strategies	Intelligent traffic management systems
		Optimization of traffic flow
		Parking regulations
	Traffic management and optimization	Use of LED lamps in municipal lighting systems
	Efficiency improvement of appliances	-
Energy supply	Promotion of district heating	Expansion of networks
		Combined heat and power systems

Sector	Policy thematic category	Standardized measure (policy intervention)
	Switch to less carbon-intensive fuels	Switch from coal to gas power plants
Industry	Use of fuel alternatives	Use of refuse derived fuel
Waste management	Eco-friendly waste management with citizens participation	Eco-friendly waste management with citizens participation
	Biochemical treatment of MSW	Promotion / incentives for enhanced use of anaerobic digestion of the MSW organic fraction and of the biorefinery concept for production of high-added value chemicals and pharmaceuticals
	MSW incineration and energy recovery	MSW incineration and energy recovery
Other	Regeneration of neighborhoods	-
	Climate change adaption	-
	Promotion of green spaces	Bioclimatic renovation of public areas
	Reduction of other emission sources	Fireworks reduction

For the integrated impact assessment, the specific policy interventions for each ICARUS city and standardized measure have to be translated into emission reductions and impacts on air quality using different modeling tools. To allow the modeling of changes in activity levels and emission factors, the underlying assumptions of possible responses to policy interventions need to be identified and described. This is all the more important since many end-of pipe measures are adopted at an European or national decision level so that regional and local authorities often have to rely on non-technical interventions resulting in a lot of different responses (Viaene et al. 2016). ***Responses*** in the ICARUS database are therefore defined as the reactions of population subgroups or emission source operators that are induced by a specific policy intervention; for example, investment in new equipment, share of inhabitants switching to public transport or deciding to renovate their building.

Policy interventions can lead to multiple types of responses with a certain probability distribution (Miranda et al. 2015). For example, the introduction of low emission zones motivates some inhabitants to buy a new car (expressed as changes in the vehicle stock) and others to change their mode of

transportation (switch from private car to public transport). The responses to standardized measures will be based as far as possible on elasticities and agent-based modeling results of ICARUS WP4. The responses to city-specific policy interventions can be derived from local experiences, accompanying studies, and impact assessments of air quality plans and individual measures. Changes in levels of activities and emission factors will be determined for each response and summarized to an evaluation of the emission reduction potential of each policy intervention. To calculate the cost/benefit of the selected emission reduction measures, information on costs of the measures will also be included.

The acceptability and feasibility of the measures will be thoroughly discussed after the first assessment of costs and benefits (CBA), which could serve as an additional filter in the selection of measures which will undergo the final CBA and feasibility analysis in tasks T5.3 and T5.4. This approach aims avoiding the exclusion of measures that might have a high pollution reduction potential but are not yet considered by the authorities or because of their unpopularity. Further steps will provide the answers whether the measures and policies identified actually have the potential of being implemented in a particular city along with the prediction of improvement (air quality, climate, health) and at what total societal costs. The comparative evaluation of alternatives will allow identification of the most appropriate strategies/options for a particular city which will then be a subject of integrated modelling that would lead towards the applicability of win-win solutions in the 9 ICARUS cities followed by the 892 cities in EU with over 50.000 inhabitants, and finally for the entire EU.

3 Conceptual models: Methodologies for linking policies and measures to effects

Different conceptual models exist that have been used to attempt to link policies, measures and effects. These models help in the identification of potential impact pathways and in identifying potential actions (policies or measures) which can reduce potential stressors on environment and health. At the base there are two conceptual models that have been widely used in the context of environment and health:

- The Driver-Pressure-State- Impact-Response model (or DPSIR); and
- The Driver- Pressure-State-Exposure-Effect-Action model (or DPSEEA).

DPSIR has been widely used in the development of models for air pollution – for example it formed the basis of the methodological framework for the ExternE series of projects. It has advantages in that it is flexible and can be applied for a range of environmental endpoints (not just health). DPSIR was developed. However, for health related issues, the DPSEEA model, developed by the WHO, has perhaps been more applied.

3.1 Driver-Pressure-State- Impact-Response model

The Driver-Pressure-State-Impact-Response model was developed for the European Environment Agency and links, in a fairly simple way, the key factors that influence environmental pressures, changes in the state of the environment and impacts (see figure below).

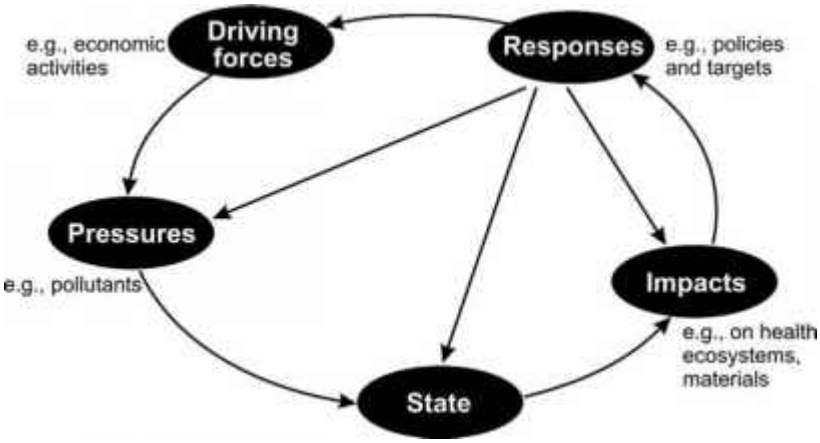

Figure 3-1: DPSIR framework

The impact pathway approach, which stemmed from the DPSIR model, is shown in Figure 3-2 below.

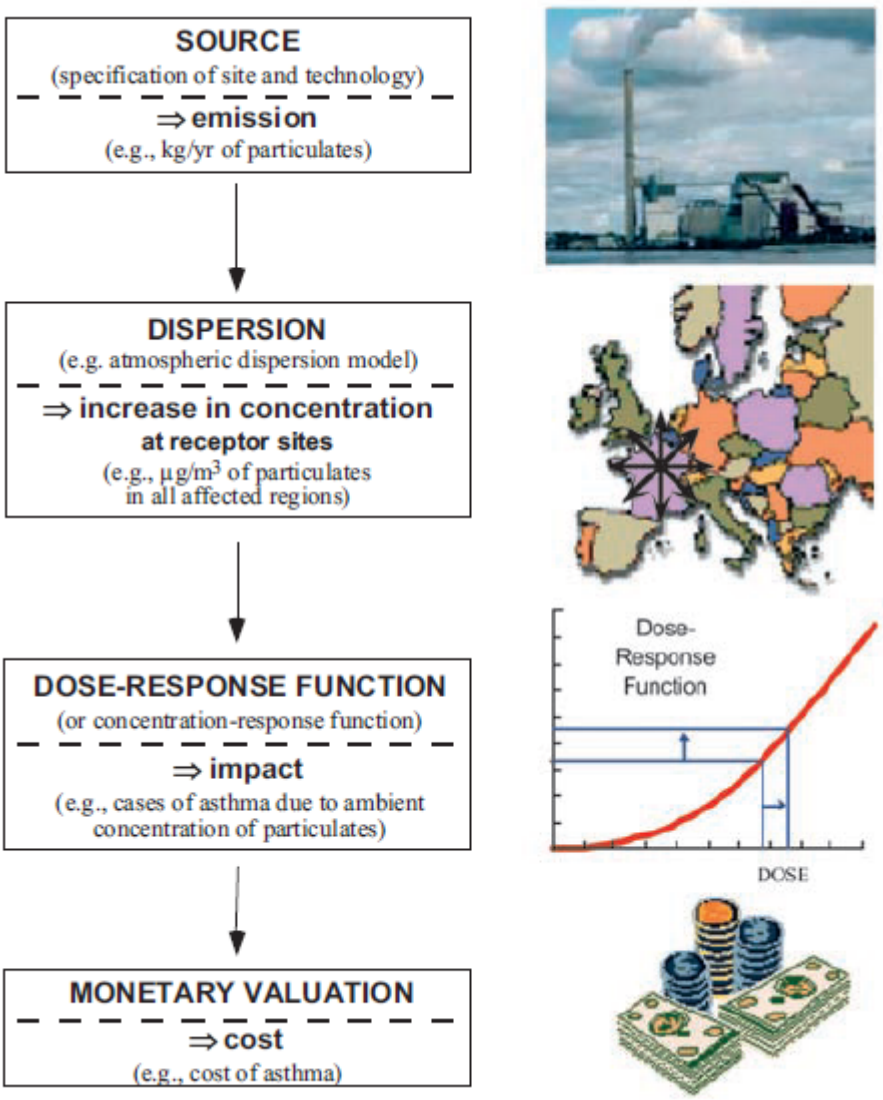

Figure 3-2: Impact Pathway Approach Source: ExternE (2005)

3.2 Driver-Pressure-State-Exposure-Effect-Action model

Building on the DPSEEA model, Morris et al (2006) proposed a modified DPSEEA approach. Under this modified DPSEEA, the importance of context was particularly highlighted (see Figure 3-2). This helps to further unpick the interrelationships between environment and health, as there are a range of contextual factors that impact on the degree to which changes in state lead to changes in exposures, and in the impact of exposures on health endpoints. These include factors such as:

- Socioeconomic status;
- Cultural norms;

- Weather conditions; and
- Demographic aspects.

All of these factors need to be considered when developing an appropriate policy to address a change in environmental pressure in a given country.

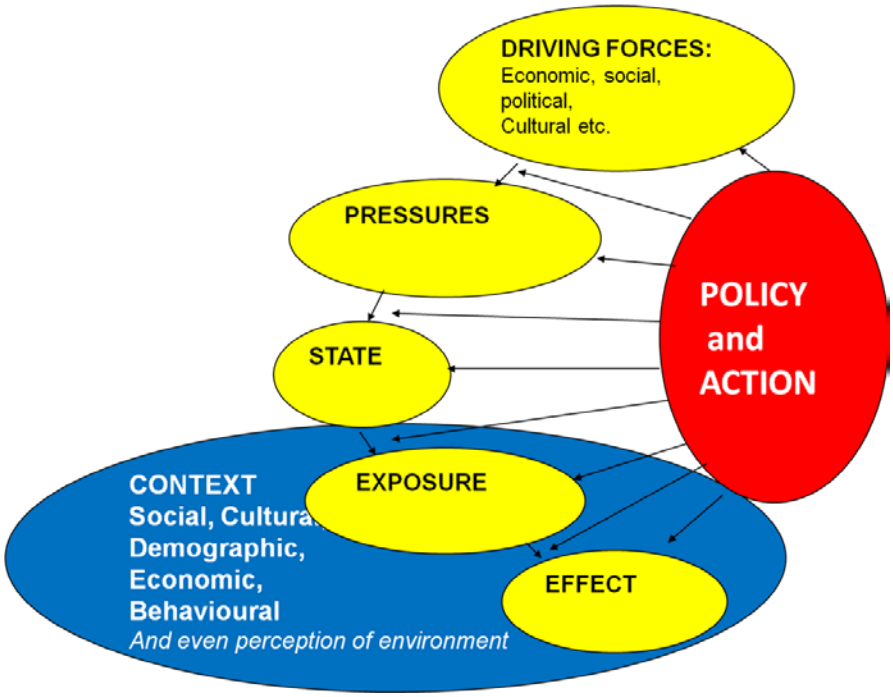

Figure 3-3: The modified DPSEEA model Source: Morris et al, 2006

The ecosystems-enriched DPSEEA looks further into the wider impacts of changes in the environment and assesses the consequences for health not only of direct impacts on health of such changes, but also impacts that are mediated by ecosystems and the services they provide (Figure 3-3).

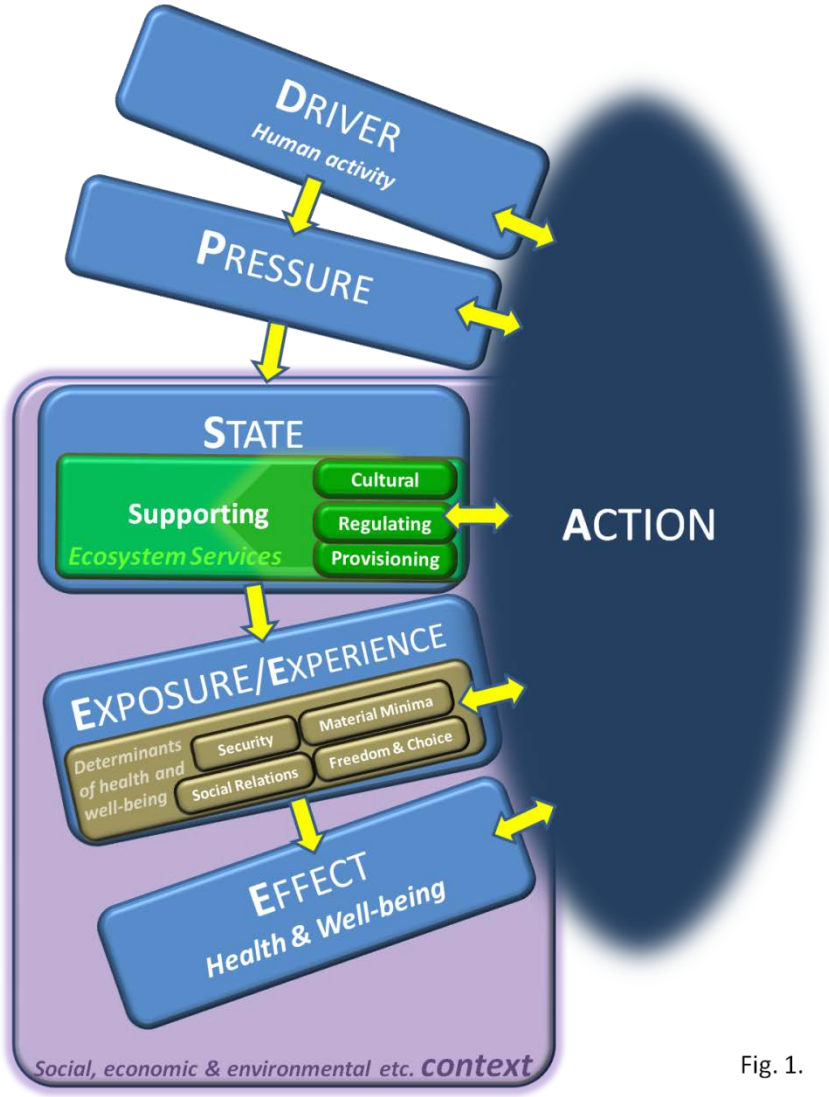

Fig. 1.

Figure 3-4 eDPSEEA model Source: Reis et al (2015)

3.3 Summary

The table below gives a summary of the key conceptual models that could be applied in the ICARUS project.

Table 3-1: Summary of key conceptual models

Model	Key contribution	Critical references
DPSIR	Links environmental change and impacts across a range of impact categories	ExternE (2005)

DPSEEA	Presents simple linear environmental health pathway.	Kjellstrom and Corvalan, 1995 Corvalan et al (1999)
Modified DPSEEA (mDPSEEA)	Explicitly considers the role of context in modifying exposure risk.	Morris et al (2006)
Ecosystems-enriched DPSEEA (eDPSEEA)	Considers wider threats to health through changes in ecosystems and considers wider spatial and temporal impacts	Reis et al (2015)

4 Methods for measuring key impacts

4.1 Health impacts

4.1.1 Exposure

The health status of persons, especially regarding chronic diseases caused by environmental pressures, is influenced by the exposure to toxic substances. More precisely, these chronic diseases are not caused by short-term exposures to stressors, but by the long-term exposure over the whole life time. However existing concentration response relationships are mainly derived from epidemiological studies that measure the health status and the concentration of pollutants for a short period of the life of the probands only. Moreover, the application of CRFs ignores the fact that ambient concentration does not represent the true exposure of individuals since people spend most of the time indoors.

To support the health impact assessment, we developed and applied a probabilistic model to estimate the temporal course of the external exposure for population subgroups that are characterized by certain features (e.g. age, gender, employment status, income). This methodology starts with individual exposure assessment which integrates model for assessing ambient concentration, time-activity patterns and models for simulating concentration in different microenvironments. The exposure modelling is applied to representative population from EU-SILC data. This methodology is implemented for air pollutants including fine particulate matter (PM2.5), nitrogen dioxide (NO_2) and ozone (O_3).

4.1.2 Model overview

The model starts with individual exposure simulation with the indirect approach, or the microenvironment approach, which can be interpreted as:

$$E = \sum_{i=1}^{n} c_i t_i$$

where c_i is the pollutant concentration in microenvironment i, t_i is the time that individual spends in microenvironment i, and n is the number of different microenvironments.

The microenvironments are classified as:

- home indoor
- work indoor
- school indoor
- outdoor
- travel/commute
- other indoor

“Other indoor” refers to indoor locations if it does not fit into one of the other categories. In this model, the time spent in “other indoor” is assigned to “home indoor”.

To simulate the indoor pollutant concentration, we assume the concentration indoors is under steady state and apply the mass balance model (Dockery & Spengler 1981):

$$c_{in} = \frac{c_{out} pa + \frac{\sum_{i=1}^{n} E_i}{v}}{a + k}$$

Where C_{in} indoor concentration [µg m^{-3}]
C_{out} ambient pollutant concentration [µg m^{-3}]
p penetration factor
a air exchange rate [h^{-1}]
k decay rate [h^{-1}]
E_i emission rate for source i [µg h^{-1}]
v room volume [m³]

For indoor microenvironments equipped with HVAC system, the mass balance model is modified by Thornburg *et al.* (2001) as:

$$c_{in} = \frac{c_{out} pa + \frac{\sum_{i=1}^{n} E_i}{v}}{a + k + \eta NT}$$

Where η filter efficiency
N recirculated air exchange rate [h^{-1}]
T duty cycle of HVAC system

The equations can be further presented as (Hänninen *et al.* 2004):

$$c_{in} = F_{INF} c_{out} + c_{ig}$$

Where C_{in} indoor concentration [µg m^{-3}]
C_{out} ambient pollutant concentration [µg m^{-3}]
F_{INF} infiltration factor
C_{ig} concentration generated from indoor sources [µg m^{-3}]

To simulate the pollutant concentration in transport, method from ICF Consulting & TRJ Environmental (2000) and Gens (2012) is applied in this thesis, which can be interpreted as:

$$c_{trans} = ME \cdot c_{out}$$

Where C_{trans} concentration in transport [µg m^{-3}]
C_{out} ambient pollutant concentration [µg m^{-3}]
ME *traffic ME factor*

The individual exposure method is applied to population samples taken from EU-SILC (see chapter 3.1). Each sample of the EU-SILC data is matched with individual diaries of activity pattern data from MTUS (see chapter 3.3) based on variables including gender, age and household income level. The matched diaries are used as input data for exposure assessment. For each diary, a set of realizations are made based on the distribution of the model parameters. The model parameters are represented by a certain distribution instead of single values (information see chapter 3.4). In this model, Monto Carlo sampling is used to draw a large number of realizations from each of the parameter distribution. The outcome of individual exposure assessment is aggregated by socioeconomic variables based on the weights given in EU-SILC data and the statistical analysis methods are applied.

4.1.3 Model input

4.1.3.1 EU-SILC

The European Union Statistics on Income and Living Conditions (EU-SILC) is a reference source for cross-sectional and longitudinal multidimensional microdata concerning income, poverty, social exclusion and living conditions (EUROSTAT 2013). Information covering social exclusion and housing condition (including housing, material deprivation and household income) is collected at household level. Moreover, education, health and labor information is collected at personal level. The samples selected are considered as representative of the whole population.

EU-SILC data are demanded to stay anonymous and the address of respondents cannot be accessed by public. This hinders acquisition data of ambient pollutant concentration and other model parameters. The way to compensate data scarcity is to determine the distribution of the input data based on variable DB040, Region and DB100, Degree of urbanization. In EU-SILC, regions are determined by Nomenclature of Territorial Units for Statistics (NUTS) and the lowest territorial unit available is NUTS2. Besides, degree of urbanization is classified into three categories by EUROSTAT as highly populated area, intermediate area and thinly populated area. Thus each territorial region is further classified into three sub-regions according to the degree of urbanization. The Figure 4-1 shows the NUTS2 regions and degree of urbanization in Spain as an example.

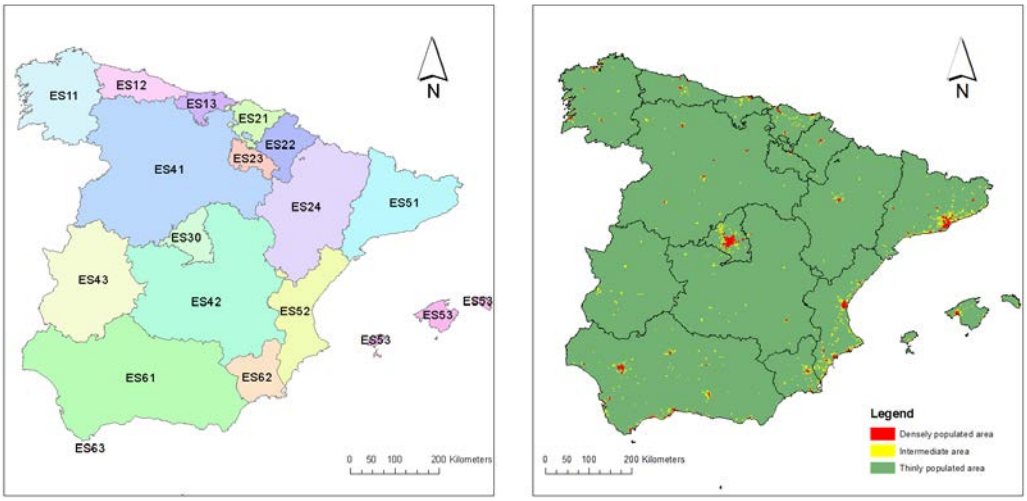

Figure 4-1: NUTS2 regions and degree of urbanization of Spain.

4.1.3.2. Concentration

The ambient concentration fields are basic input data for exposure modelling. The concentration data are generated back to 1930s since oldest respondents in EU-SILC are aged around 80 in 2012. The results from EMEP chemical transport model for Europe are widely applied and publically available for 1980 onwards. However, the modelling performance of EMEP has been evaluated and especially for PM2.5, an overall trend of underestimation has been reported. To modify the EMEP modelling results, either an interpolation method (Horálek et al. 2008) or a multiplicative bias adjustment (McKeen et al. 2005) is implemented. We also used the emission data from EDGAR HYDE as input data for ECOSENSE

to generate concentration fields from 1930 to 1970, which are further adjusted by multiplicative bias adjustment factor. All the concentration fields are aggregated by NUTS regions and degree of urbanization as defined in EU-SILC.

4.1.3.3. Time activity pattern

The simulation of individual exposure requires information of time that person stays in each microenvironment. Data for time activity pattern are derived from Multinational Time Use Study, which is a concept initially proposed by Professor Jonathan Gershuny in 1970s to create a multi-nationally harmonized set of time use surveys (Fisher et al. 2012). The MTUS includes three data formats: Harmonized simple file (HSF), Harmonized aggregate files (HAF) and Harmonized episode file (HEF).

The data availability of MTUS for European countries is displayed in Table 4-1. Since no information is available for children under three years old in Europe, data from US survey is utilized as supplementary.

Even though HSF data covers most countries within three files, its unavailability of location information hinders the microenvironment based modelling. In this model, we used HEF and HAF and joined the two datasets based on identifier variables (including household identifier, person/diarist identifier and diary identifier). The fused data profit from the structure of the HEF with detailed microenvironment information. Simultaneously, accurate socioeconomic information is inherited by the merged file from HAF.

Table 4-1: Data availability of Multinational Time Use Study (MTUS) during several periods. [- = no data available, ● = HAF and HEF].

	1975-1979	1980-1984	1985-1989	1990-1994	1995 - 1999	2000 - 2004	2005+
Austria	-	-	-	●	-	-	-
France	-	-	-	-	●	-	-
Germany	-	-	-	●	-	-	-
Italy	-	-	●	-	-	-	-
Netherlands	●	●	●	●	●	●	●
Spain	-	-	-	●	●	●	●
United Kingdom	●	-	●	-	●	●	●
United States	-	-	-	●	-	-	-

4.1.4 Model parameters

4.1.4.1. Infiltration parameters

It has been proved by studies that pollutant infiltrated from outdoors play an important, even dominant role in the composition of indoor concentration for both PM2.5 and NO2. The parameters

for simulating the infiltration factor and their definitions are listed in Table 4-2. For simplicity, we exclude the influence of infiltration and indoor sources for ozone.

Studies have revealed the influence of advanced ventilation system on improving indoor air quality (Hänninen et al. 2005, Hodas et al. 2016, Thornburg 2004). Buildings are classified into three types, namely "old dwellings", "insulated dwellings" and "new dwellings". The "new dwellings" are renovated and equipped with heating, ventilating and air conditioning system systems (HVAC) while the "insulated dwellings" are well insulated, but without additional ventilation or air conditioning.

Table 4-2: List of parameters for infiltration factor.

Parameter	Definition	Source
Air exchange rate	Measurement of how much indoor air is placed by outdoor fresh air in a given time period	Bouhamra, Elkilani & Abdul-Raheem 1998
Penetration factor	Fraction of pollutant in the infiltration air that passes through the building shell	Chen & Zhao 2011
Decay rate	Loss rate of air pollutants due to all processes	Gens 2012
Filter efficiency	Removal efficiency of the HVAC filter	Thornburg 2004
Recirculated air exchange rate	Measurement of how much air is removed from a space and reused in a given time period	ASHRAE 2007
Duty cycle of HVAC system	Fraction of time that the HVAC fan is operating	Thornburg 2004

4.1.4.2. Air exchange rate

The data for air exchange rate for dwellings with natural ventilation system are taken from EXPOLIS (Hänninen et al. 2004). We follow the method of Gens (2012) to assign and extrapolate these values to four geographical regions (Southern - Athens, Central – Prague, Northwestern – Basel, Northern-Helsinki). For offices and schools, data are generated from project OFFICAIR and SINPHONIE respectively and assumed to follow a triangular distribution. For insulated and new buildings, an average value of 0.5 is applied considering the studies available (Földváry et al. 2017, Villi et al. 2013).

Table 4-3: Values for air exchange rate (h^{-1}).

Microenvironment	Old buildings	Insulated buildings	New buildings

home	0.83 (± 0.46); lognormal; for Basel 1.29 (± 1.09); lognormal; for Athens 0.75 (± 0.43); lognormal; for Prague 0.81 (± 0.85); lognormal; for Helsinki	0.5 (± 0.1); lognormal	0.5 (± 0.1); lognormal
Office	(0.1, 0.4, 1.8); triangular	0.5 (± 0.1); lognormal	0.5 (± 0.1); lognormal
Schools	(0.3, 0.4, 0.7); triangular	0.5 (± 0.1); lognormal	0.5 (± 0.1); lognormal

4.1.4.3. Penetration factor

In buildings where mechanical ventilation systems have been rare, outdoor particles penetrate indoors very efficiently. Studies (Özkaynak et al. 1997; Wallace 1996, Lewis 1995, Thatcher & Layton 1995) have reported penetration factor close to unity. Thus we assume the penetration factor for PM2.5 is lognormally distributed with mean of 1 and standard deviation of 0.1. For NO2, we assumed that P=1 according to the data from Fabian, Adamkiewicz & Levy (2012) and Emmerich & Persily (1996).

4.1.4.4. Decay rate

The distribution for this variable is assumed to apply to all indoor buildings. Data for decay rate for PM2.5 are assumed based on studies from Gens (2012), Thornburg et al. (2001) and Burke, Zufall & Özkaynak (2001). For NO2 the data are taken from Emmerich & Persily (1996) and applied to all building types (see Table 4-4).

Table 4-4: Values for decay rate (h^{-1}).

Pollutant	Old dwellings	Insulated dwellings	New dwellings
PM2.5	0.39(± 0.09); lognormal	0.25(± 0.15); lognormal	0.25(± 0.15); lognormal
NO2		0.17-2.07; uniform	

4.1.4.5. Other parameters for infiltration factor

The range of data for HVAC related parameters varies considerably. For example, the HEPA filter for PM2.5 has rated an efficiency over 99%, however, some studies indicate that filters have a maximum efficiency less than 10% (Fisk et al. 2002, Moyer et al. 2002). For NO2, data are generated from studies from Choi et al. (2012) and Lin & Chen (2014). Both values are assumed to follow a uniform distribution.

For recirculated air exchange rate, typical values used in models and experiments in the literature range from 0.67 to 24 h-1 (Stephens, Siegel & Novoselac 2011, Zuraimi et al. 2007, Waring & Siegel 2008). Duty cycle, i.e. fractional operation time data for residential and other buildings are generated from Stephens et al. (2011), Thornburg et al. (2001), Kassas (2015) and California Energy Commission (2008). All the data are shown in Table 4-5.

Table 4-5: Values for other parameters.

Pollutant	Filter efficiency	Recirculated air exchange rate (h^{-1})	Duty cycle of HVAC system
PM2.5	Residential: 0.1-0.6; uniform Other: 0.1-0.5; uniform	5 (± 2); lognormal	Residential: 0-1.0; uniform Other: constant = 0.42
NO_2	0-0.7; uniform		

4.1.4.6 Building volume

For residential buildings, data for floor space are generated from EU-SILC data for 2012. The data are aggregated by country and income level and assumed to follow a lognormal distribution. The room height is assumed to follow a uniform distribution from 2.4 to 3 m (European Commission 2011).

For offices, data for room volume are derived from the OFFICAIR Questionnaire Survey (Ploskas 2013). Offices are categorized as either cellular or landscape and data are given in Table 6. For both categories, the office sizes are assumed to follow a triangular distribution.

For schools, data are derived from SINPHONIE (Csobod et al. 2000) and summarized by country. For countries with no data available in SINPHONIE, their distributions were estimated using the average data across the countries in corresponding geographical region (see chapter 3.4.1.1).

Table 4-6: Values for office sizes (m^3).

Pollutant	Min	max	mode
Landscape	44.4	1680.0	466.8
Cellular	23.4	176.0	77.3

4.1.5 Indoor sources

Except for outdoor air, great contribution of indoor sources has been observed by PM2.5 and NO2 (Spengler et al. 1983, Levy 1998, Kornartit et al. 2010). In this model, the following sources are included:

4.1.5.1. Cigarettes

Smoking has been revealed by many studies as one of the most important indoor sources (Spengler et al. 1985, Lai et al. 2006, Wigzell, Kendal & Nieuwenhuijsen 2000, Koistinen et al. 2001). The emission rate for cigarettes is calculated with the following equation:

$$E_{cig} = \frac{S_{cig} N_{cig}}{T}$$

Where Ecig emission rate of cigarettes [µg h-1]
Scig source strength of cigarettes [µg min-1]
Ncig number of cigarettes
T total time in microenvironment [h]

The data for average daily cigarettes consumption for 2010 are taken from the Eurobarometer 385 (European Commission 2012). The information for cigarette consumption for earlier years is very limited. According to the studies from Hill (1992 & 2005), the individual cigarette consumption kept increasing from 1930s and reached a peak around 1985. It is assumed that the daily cigarette consumed per person changes 0.2 annually. Exposure to ETS is only considered at home and at work environments. Data for proportion of population exposed to ETS at home and at work are given by Flash Eurobarometer (European Commission 2009).

The source strength of PM2.5 per cigarette is taken from the PTEAM study (Özkaynak et al. 1997) and Daisey, Mahanama & Hodgson (1994). Results are normally distributed with mean of 10950 µg cig-1 and standard deviation of 2000 µg cig-1. For NO2 the source strength is assumed to follow a uniform distribution with a range from 80 to 3510 µg cig-1 (Searl 2004, Jenkins & Gill 1980, Graham, Grant & Folinsbee 1997 and Rickert, Robinson & Collishaw 1987).

4.1.5.2. Cooking

Studies have also reported cooking as another significant source (Özkaynak et al. 1997, Gerharz, Krüger & Klemm 2009, Wallace 1996). The emission rate for cooking is calculated with the following equation:

$$E_{cooking} = \frac{S_{cooking} t_{cooking} (1 - CE)}{T}$$

Where Ecooking emission rate of cooking [µg h-1]
Scooking source strength of cooking [µg min-1]
tcooking time of cooking activities [min]
CE capture efficiency
T total time in microenvironment [h]

Table 4-7 shows the data for cooking related parameters. Large differences have been found by source strength of cooking, which is dependent on oven type, sort of fat used and temperature reached during the cooking process (Buonanno, Morawska & Stabile 2009, He et al. 2004, Gens 2012). The data applied are generated based on studies from He et al. (2004), Fabian et al. (2012), Burke et al. (2001) and Fortmann, Kariher & Clayton (2001). We also assume that 10% to 60% of the whole food preparing

time is spent on cooking and frying. The capture efficiencies are generated from Lunden, Delp & Singer (2015) and Delp & Singer (2012).

Table 4-7: Values for cooking related parameters.

Pollutant	Source strength (µg min^{-1})	Capture efficiency
PM2.5	1560 (±400); lognormal	11%-70%; uniform
NO_2	55-3360; uniform	15%-98%; uniform

4.1.5.3. Wood burning

Wood burning is also revealed by many studies as one important indoor source for both PM2.5 and NO2. In this model, we follow the method in project NEEDS (Torfs et al. 2007) to calculate the emission rate of wood burning:

$$E_{wood} = \frac{S_{wood} t_{wood} H_{demand} VR_{removal}}{T}$$

Where Ewood	emission rate of wood [µg h-1]
Swood	source strength of wood [µg kJ-1]
twood	time of burning wood [h]
Hdemand	heat demand [kJ m-3 h-1]
V	room volume [m3]
Rremoval	removal ratio

The values for parameters are derived from Torfs et al. (2007) and Struschka et al. (2008). More detailed data are given in Table 4-8.

Table 4-8: Values for simulation of emission rate for wood.

Pollutant	S_{wood} (µg kJ^{-1})	t_{wood} (h)	H_{demand} (kJ m^{-3} h^{-1})	$R_{removal}$
PM2.5	13-146; uniform	0.3-2.8; uniform	85	0.995-0.95; uniform
NO_2	58-185; uniform			

4.1.5.4. Candles

Emission from candle is also included in this model and calculated with the following equation:

$$E_{candle} = \frac{S_{candle} t_{candle}}{T}$$

Where Ecandle	emission rate of candle [µg h-1]
Scandle	source strength of candle [µg min-1]
tcandle	time of burning candles [min]
T	total time in microenvironment [h]

Data from Sørensen et al. (2005), Hu, Singer & Logue (2012) and Stabile, Fuoco & Buonanno (2012) are used. We assumed that 10% of the population is exposed to PM2.5 emissions from candle burning. The candle burning time ranges from 5 to 120 minutes. The source strength is assumed to be uniformly distributed with a range between 5.5 to 910 µg min^{-1}.

4.1.6 Suspension

Human activity tends to generate short duration, high concentration particle events, which can result in indoor PM concentrations (Ferro, Kopperud & Hildemann 2004). In this model, emission rate of resuspension is calculated with the following equation:

$$E_{resuspension} = \frac{\sum_{i=1}^{n} S_{res,i} t_{res,i}}{T}$$

Where Eres emission rate of resuspension [µg h-1]
Sres, i source strength of resuspension for activity i [µg min-1]
tres, i time spending on activity i [min]
T total time in microenvironment [h]

Sources for resuspension include some indoor activities e.g. setting table, cleaning, other domestic work and walking. Data for PM2.5 resuspension are taken from He et al. (2004) and Ferro et al. (2004).The source strength of resuspension for activity is assumed to follow uniform distribution and data are given in Table 4-9.

Table 4-9: Values for simulation of emission rate for resuspension

Activity	Min (µg min^{-1})	Max (µg min^{-1})
Set table, wash/put away dishes	20	180
Cleaning	90	440
Laundry, ironing, clothing repair	20	180

4.1.7 Traffic ME factor

In this model, we use the microenvironment (ME) factors to simulate the pollutant concentration in transport. Data applied in the model are based on studies from Zuurbier et al. (2010), van Roosbroeck et al. (2006), ICF Consulting & TRJ Environmental (2000), Samoli et al. (2016), Riediker et al. (2003), Adams et al. (2001) and Zagury, Le Moullec & Momas (2000). We assume for PM2.5 and NO2 the ME factor follows a normal distribution of 2 (±0.5) and 1.8 (±0.5) respectively.

4.2 Quantification of health impacts – Dose-response relationships for pollutants of interest

4.2.1 Particulate matter

Rationale

Particulate Matter is generally categorised on the basis of the size of the particles that reflects their aerodynamic diameter (e.g PM2.5 refers to particles with an aerodynamic diameter of less than 2.5µm). PM is made up of a wide range of components and are formed from a variety of sources and processes. Ambient air levels of PM comprise primary particles emitted directly into the atmosphere from combustion sources and secondary particles formed by chemical reactions in the air. Ambient air PM are released from both anthropogenic and natural sources (such as sea spray, Saharan dust or volcanos). The most common anthropogenic sources are stationary fuel combustion and transport. Road transport gives rise to primary particles from engine emissions, as well as various non-exhaust emissions such as tire and brake wear. Secondary PM is formed from emissions of ammonia, sulphur dioxide and oxides of nitrogen as well as from emissions of organic compounds from both combustion sources and vegetation. Both short-term and long-term exposure to ambient levels of PM are consistently associated with respiratory and cardiovascular illness and mortality as well as other adverse health effects. It is not currently possible to discern a threshold concentration below which there are no effects on public health. Fine particles are deposited in the lowest part of the human respiratory tract, where they can cause inflammation and a worsening of the condition of people with heart and lung diseases. In addition, they may carry surface-absorbed carcinogenic compounds into the lungs. Based on the above, the CRFs for both short-term and long-term health effects are given below.

Long-term health effects for PM

Table 4-10. Concentration response Functions (CRFs) for PM regarding long term health effects

Health endpoint	CRF	Reference	Background rate	Age group
Mortality (all causes)	6.2% (95% CI: 4%, 8.3%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	8417 annual deaths/881288 population (EUROSTAT, 2011; WHO, 2008), Life table data (WHO, 2014a)	Adults aged 30 years and older
Infant Mortality	4% (95% CI: 2%, 7%) change per 10 µg/m^3 PM_{10}	(Hurley et al., 2005; IOM, 2011)	145 post-neonatal deaths per 100,000 live births (9141 annual births) (EUROSTAT, 2011; WHO, 2008)	1 month to 1 year

Chronic bronchitis (adults)	11.7% (95% CI: 4%, 18.9%) change per 10 µg/m^3 PM_{10}	(WHO, 2013)	390 new cases annually per 100,000 adults at risk (adjusted for remission - remission rate of 56.2%) (Schindler et al., 2009)	Adults aged 18 years and older
Chronic bronchitis (children)	1.08% (95% CI: -2%, 19%) change per 10 µg/m^3 PM_{10}	(Hoek et al., 2012; WHO, 2013)	18.6% (Hoek et al., 2012)	Children aged 6–12 years
Incidence of chronic bronchitis			Annual incidence 3.9 per 1000 adults based on the Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA)	Adults aged 18 years and older
Cardiac hospital admissions	0.6% (95% CI: 0.3%, 0.9%) change per 10 µg/m^3 PM_{10}	(Hurley et al., 2005; IOM, 2011)	723 emergency cardiac admissions per 100,000 population, all ages, per year (Hurley et al., 2005):	All Ages
Respiratory hospital admissions	0.9% (95% CI: 0.7%, 1.0%) change per 10 µg/m^3 PM_{10}	(Hurley et al., 2005; IOM, 2011)	617 emergency respiratory hospital admissions per 100,000 population, all ages, per year (Hurley et al., 2005):	All Ages

Short-term health effects for PM

Table 4-11. Concentration response Functions (CRFs) for PM regarding short-term health effects

Health endpoint	CRF	Reference	Background rate	Age group
Mortality (all causes)	1.23% (95% CI: 0.45%, 2.01%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	City or country specific data	All ages
Cardiac hospital admissions	0.91% (95% CI: 0.17%, 1.66%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	723 emergency cardiac admissions per 100,000 population, all ages, per year(Hurley et al., 2005):	All Ages
Respiratory hospital admissions	1.9% (95% CI: 0.99%, 4.02%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	617 emergency respiratory hospital admissions per 100,000 population, all ages, per year(Hurley et al., 2005):	All Ages
Restricted activity days (RADs)	4.7 % (95% CI: 4.2%, 5.3%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	19 RADs per person per year: baseline rate from the Ostro and Rothschild (1989) study	All Ages
Work days lost	4.6% (95% CI: 3.9%, 5.3%) change per 10 µg/m^3 $PM_{2.5}$	(WHO, 2013)	European Health for All database	Working- age population (age 20–65 years)
Incidence of asthma symptoms in asthmatic children	2.8% (95% CI: 0.6%, 5.1%) change per 10 µg/m^3 PM_{10}	(WHO, 2013)	(Lai et al., 2009) – western Europe: 4.9%; northern and eastern Europe: 3.5%. Daily incidence of symptoms in this	Children aged 5–19 years

			group: 17% (interpolation from several panel studies)	

4.2.2 Black carbon

Rationale

Black Carbon (BC) is an term aiming at describing carbon as measured by light absorption methods. BC originates from multiple sources related to combustion engines, mostly related to diesel, as well as other less elaborate combustion processes such as biomass and coal burning, which in turn is associated with many sectors of urban activities relevant to the ICARUS project. As a result, BC is in practice a more generic indicator of the multitude composition of combustion sources of particulate matter. At the moment there is limited toxicological in vivo and in vitro information on BC, however it is hypothesized that BC does not exert direct properties, but is acting as a substrate for strong adsorption of toxic components such as PAHs, quinones, nickel, vanadium and arsenic, related to emissions from various sectors involving combustion. As such, BC levels are associated with both PM mass concentrations, as well as their toxic burden. The latter is highlighted on the proposed CRF for BC associated to all-causes mortality, where an increase of 10 μg/m^3 of BC, has been associated with an increase of 60% of all-causes mortality.

Long-term health effects of black carbon

Table 4-12. Black Carbon (BC) related all-cause mortality

Health endpoint	CRF	Reference	Background rate	Age group
Mortality, All causes	60% (95% CI: 10%, 110%) change per 10 μg/m^3 Black Carbon	(Janssen et al., 2011)	City or country specific data for all-cause mortality	30+ years

4.2.3 Ozone

Rationale

O_3 is the tri-atomic form of molecular oxygen. It is a strong oxidising agent, and hence highly reactive. Background levels of O_3 in Europe are usually less than 15 ppb but can be as 100 ppb during summer time photochemical smog episodes. In the UK ozone occurs in higher concentrations during summer than winter, in the south rather than the north and in rural rather than urban areas. According to the Ambient Air Quality Directive (EU, 2008), a maximum daily 8-hour mean threshold of 120 μg/m^3 has been established. Based on the health effects of ozone, several CRFs have been widely accepted for both long-term and short-term health effects.

Long-term health effects of ozone

Health endpoint	CRF	Reference	Background rate	Age group
Mortality, respiratory diseases	1.4% (95% CI: 0.5%, 2.4%) change per 10 µg/m^3 O_3	(WHO, 2013)	MDB (WHO, 2013c), ICD-10 codes J00–J99	30+ years

Short-term health effects of ozone

Health endpoint	CRF	Reference	Background rate	Age group
Mortality, all (natural) causes, (>35 ppb (>70 µg/m³))	0.29% (95% CI: 0.14%, 0.43%) change per 10 µg/m^3 O_3	(WHO, 2013)	MDB (WHO, 2013c), ICD-10 chapters I–XVIII, codes A–R	All ages
Mortality, all (natural) causes, (>10 ppb(>20 µg/m³))	0.29% (95% CI: 0.14%, 0.43%) change per 10 µg/m^3 O_3	(WHO, 2013)	MDB (WHO, 2013c), ICD-10 chapters I–XVIII, codes A–R	All Ages
Mortality, CVDs and respiratory diseases (>35 ppb (>70 µg/m³))	CDV: 0.49% (95% CI: 0.13%, 0.85%) change per 10 µg/m^3 O_3 respiratory: 0.29% (95% CI: 0.099%, 0.7%) change per 10 µg/m^3 O_3	(WHO, 2013)	MDB (WHO, 2013c), ICD-10 codes CVD: I00–I99; respiratory: J00–J99	All Ages
Mortality, CVDs and respiratory diseases (>10 ppb (>20 µg/m³))	CDV: 0.49% (95% CI: 0.13%, 0.85%) change per 10 µg/m^3 O_3 respiratory: 0.29% (95% CI: 0.099%, 0.7%) change per 10 µg/m^3 O_3	(WHO, 2013)	MDB (WHO, 2013c), ICD-10 codes CVD: I00–I99; respiratory: J00–J99	All Ages

Hospital admissions, CVDs (excluding stroke) and respiratory disease(>35 ppb (>70 µg/m³))	CDV: 0.89% (95% CI: 0.5%, 1.27%) change per 10 µg/m^3 O_3 respiratory: 0.44% (95% CI: 0.07%, 0.83%) change per 10 µg/m^3 O_3	(WHO, 2013)	European hospital morbidity database (WHO, 2013f), ICD-9 codes CVD: 390–429; respiratory: 460–519 (ICD-10 codes I00–I52; J00–J99)	Age 65+ years
Hospital admissions, CVDs (excluding stroke) and respiratory disease(>10 ppb (>20 µg/m³))	CDV: 0.89% (95% CI: 0.5%, 1.27%) change per 10 µg/m^3 O_3 respiratory: 0.44% (95% CI: 0.07%, 0.83%) change per 10 µg/m^3 O_3	(WHO, 2013)	European hospital morbidity database (WHO, 2013f), ICD-9 codes CVD: 390–429; respiratory: 460–519 (ICD-10 codes I00–I52; J00–J99)	Age 65+ years
Minor restricted activity days (MRADs) (>35 ppb (>70 µg/m³))	1.54% (95% CI: 0.60%, 2.49%) change per 10 µg/m^3 O_3	(WHO, 2013)	7.8 days per year, based on Ostro and Rothschild (1989)	All Ages
MRADs(>10 ppb (>20 µg/m³))	1.54% (95% CI: 0.60%, 2.49%) change per 10 µg/m^3 O_3	(WHO, 2013)	7.8 days per year, based on Ostro and Rothschild (1989)	All Ages

4.2.4 NO_2

Rationale

The main health effects associated to exposure to NO_2 are: shortness of breath or coughing and enhanced risk of respiratory disease. NO_2 is associated with several respiratory adverse effects on human health. At high levels NO_2 causes inflammation of the airways. Long-term exposure may affect lung function and respiratory symptoms. NO_2 also enhances the response to allergens in sensitive individuals. Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infections such as influenza. Continued or frequent exposure to concentrations that are typically much higher than those normally found in the ambient air may cause increased incidence of acute respiratory illness in children. The CRFs for long term and short health effects of NO_2 are given below.

Long-term health effects of NO_2

Health endpoint	CRF	Reference	Background rate	Age group
Mortality, all (natural) (causes>20 µg/m³)	5.50% (95% CI: 3.10%, 8.0%) change per 10 µg/m^3 NO_2	(WHO, 2013)	MDB (WHO, 2013c), rates for deaths from all natural causes (ICD-10 chapters I–XVIII, codes A–R) in each of the 53 WHO Regional Office for Europe countries, latest available data	Age 30+ years
Prevalence of bronchitic symptoms in asthmatic	2.1% (95% CI: 0.099%, 6.00%) per 1 µg/m³ change in annual mean NO2	(WHO, 2013)	Background rate of asthmatic children, "asthma ever", in Lai et al. (2009) – western Europe: 15.8%, standard deviation (SD) 7.8%; northern and eastern Europe: 5.1%, SD 2.7%, with a recommended alternative of "severe wheeze" in Lai et al. (2009) – western Europe: 4.9%; northern and eastern Europe: 3.5% Prevalence of bronchitic symptoms among asthmatic children 21.1% to 38.7% (Migliore et al., 2009; McConnell et al., 2003)	Children aged 5–14 years
Diabetes	14.8 %(95% CI: 2.4%, 28.8%) change per 10 µg/m^3 NO_2	(Eze et al., 2014)		Age 30+ years

Short-term health effects of NO_2

Health endpoint	CRF	Reference	Background rate	Age group
Mortality (all natural causes) (daily maximum 1-hour mean)	0.27% (95% CI: 0.16%, 0.38%) change per 10 µg/m^3 NO_2	(WHO, 2013)	MDB (WHO, 2013c), rates for deaths from all natural causes (ICD-10 chapters I–XVIII, codes A–R) in each of the 53 countries of the WHO European Region, latest available data	All ages
Hospital admissions, respiratory diseases (daily maximum 1-hour mean)	0.15% (95% CI: 0.099%, 0.38%) change per 10 µg/m^3 NO_2	(WHO, 2013)	European hospital morbidity database	All Ages
Hospital admissions, respiratory diseases (24-hour mean)	1.8% (95% CI: 1.15%, 2.45%) change per 10 µg/m^3 NO_2	(WHO, 2013)	European hospital morbidity database	All Ages

4.2.5 Relative risk, attributable burden and health impact calculation

Relative risk is calculated for the average concentration X, using the following formula:

$$RR = CRF^{\left(\frac{X}{10}\right)}$$

The exponential is used because the original analyses used a proportional hazard, i.e. log-linear, regression model. From this relative risk was derived the attributable fraction ***AF*** was derived as follows:

$$AF = \left(\frac{RR-1}{RR}\right)$$

and this was multiplied by the background rate of disease ***BR*** to derive the estimated health impact ***HI*** as the number of cases expected to present the respective adverse health outcome in the population of interest ***P***. Thus, the health impact ***HI*** is given as:

$$HI = AF \cdot BR \cdot F$$

The actual burden of mortality, (i.e. the attributable death due to $PM_{2.5}$ exposure) was expressed in years of life lost (***YLL***). To estimate *YLL*, life tables (WHO, 2014b) were employed, where national population and the corresponding mortality data per age band are used. These data are used to derive the population-weighed annual average concentration for a pollutant *C*. The attributable fraction *AF* is multiplied with the background mortality rate *D* for the given age band in order to compute the number of attributable deaths (***AD***) per age band.

$$AD = AF \cdot D$$

The number of attributable deaths *AD* per age band was then multiplied with the expected years of life (Y_{LE}) for the given age band

$$YLL = AD \cdot Y_{LE}$$

Demographic data regarding the population and mortality/morbidity rates distribution for the several age groups were obtained from the EUROSTAT databases (EUROSTAT, 2011). Additional data for background rate of disease and mortality were obtained by WHO (2008).

4.3 Other air pollutants

4.3.1 Benzene (in the view of BTEX mixture)

Rationale

Benzene is a colourless, clear liquid compound. It is fairly stable but highly volatile, i.e. it readily evaporates. Ambient concentrations of benzene are typically between 1 - 50 ppb. Levels close to major emission sources can be as high as several hundred ppb. The urban background mean concentration of benzene is 1 to 2 ppb (3 to 6 µg/m^3); rural areas average 0.5 to 1 ppb (1.5 to 3 µg/m^3). Mean annual concentration can be 5 ppb (15 µg/m^3) on urban roadsides. The limit value for benzene is set as an annual mean, given that C_6H_6 is a carcinogen with long-term effects. The target value for benzene is set at 5 µg/m^3. Nevertheless, C6H6 is measured at a relatively small number of stations in EU. The concentrations above the limit value is limited to a few local areas with higher concentrations (2 µg/m^3) which are often close to traffic or industrial sources. No exceedances of the limit value were observed (EEA, 2016).

Benzene leukemia risk

For estimating the health effects of benzene, it is important to take into account its highly dynamic toxicokinetics and to evaluate tissue levels of benzene, benzene oxide (BO), phenol (PH), and hydroquinone (HQ), as well as the total amounts of muconic acid (MA), phenylmercapturic acid (PMA), phenol conjugates, hydroquinone conjugates, and total catechol produced in the target tissues. Details on the toxicokinetics of benzene and more specifically on the interaction of the benzene, toluene, ethylbenzene and xylenes quartenary mixture, have been provided in the Sarigiannis and Gotti Sarigiannis and Gotti (2008) study. The high potential toxicity of benzene metabolites associated with leukaemia risk in humans, suggested taking into account in more detail the metabolic chain from benzene to its key metabolites through a more refined PBPK model for that chemical. The whole

metabolic chain of benzene was modelled starting from previously developed PBPK models for benzene metabolism in mice and its extrapolation to humans. The model evaluates tissue levels of benzene, benzene oxide (BO), phenol (PH), and hydroquinone (HQ), as well as the total amounts of muconic acid (MA), phenylmercapturic acid (PMA), phenol conjugates, hydroquinone conjugates, and total catechol produced. For benzene oxide, phenol, and hydroquinone, the body is divided into five compartments: kidney; liver; fat; rapidly perfused tissues (RTP), and slowly perfused tissues (PPT).

The final building block for addressing the source to health outcome continuum is the definition of mathematical models of human pathology. Chronic exposure to low levels of benzene may produce reversible decreases in blood cell numbers but, at higher levels, an irreversible bone marrow depression, with pancytopenia, may establish. This pathological condition is called aplastic anemia. Pancytopenia can occur also in the so-called myelodisplastic syndrome (MDS). Benzene MDS usually proceeds to leukemia, mostly acute myeloid leukemia (AML). The approaches taken to assess the cancer risk from benzene exposure have been varied and have resulted in risk estimates that range considerably in magnitude. The U.S. EPA (2000) used the Goodyear Pliofilm study (Rinsky et al., 1987; Rinsky et al., 1981) for their quantitative risk estimation. They estimated a range of $2.2 \cdot 10^{-6}$ to $7.8 \cdot 10^{-6}$ as the increase in the lifetime risk of an individual who is exposed for a lifetime to 1 ug/m^3 benzene in air. This is based on a linear model and extrapolates to air concentrations of 1.3 to 4.5 ug/m^3 for a risk level of 1 in 100,000. The approach used by Crump directly linked external exposure to cancer risk using the Area Under the Curve (AUC) as the dose metric. Finally, an empirical statistical D-R model based on Maximum Likelihood Estimation (MLE) is derived, based on experimental data about cancer incidence as function of the exposure. The dose-response model developed by Crump (1994) takes the following form:

$$P(x) = 1 - e^{\left[-\left(0.00145x + 0.00013x^2\right)\right]}$$

where ***P(x)*** represents the cancer probability attributable to ***x*** mg/kg/day of administered benzene to male mice. This equation implies that, at very low administered doses, the risk varies linearly with dose. To extend it to humans the authors of this study assumed that the same administered quantities of benzene "produce equal cancer risk in humans and animals, independent of the route of exposure". In this work we applied a method, originally developed by Cox Jr. (1996), based on the decomposition of the dose-response relationship into a set of causal micro-relations, each one describing a separate biologic process. Instead of evaluating the relationship between administered dose and cancer risk 'directly' through an empirical-statistical model, this relationship is thus decomposed into two different parts: the first one links the administered dose to the total amount of metabolites produced (internal dose), while the second connects the internal dose to the probability of cancer. The first relation is provided by the results of the PBPK/PD model, which has already been validated against human biomonitoring data (Sarigiannis and Gotti, 2008). The statistical relation between internal dose and cancer probability was calculated using a parameterized function (1) from Crump and Allen (1984). In particular, the administered dose was calculated assuming an average person of 70 kg (adult) who inhales 10 m^3 of air in 8 hours for an occupation period of 40 years over a life of 70 years. The next step was to derive an empirical statistical relation linking the internal dose to cancer probability. This was found to be the following:

$$P(y) = 1 - e^{\left[-0.04296940\,y + 0.02633730\,y^2 - 0.00764081\,y^3\right]}$$

The standard error for the model parameters varied from 23% (for the linear term) to 9% (for the cubic term). This relationship incorporates the results of the PBPK/PD models that allow us to estimate the actual biologically effective dose (BED) of benzene metabolites in the bone marrow (the main target tissue for leukemia).

4.3.2 Benzo[a]pyrene and PAHs

Rationale

Several epidemiological studies have shown the adverse health effects of airborne particulate matter deposited in the human respiratory tract (HRT) (Kennedy, 2007; Pope Iii and Dockery, 2006). HRT deposition of a particular particle depends on its aerodynamic diameter (d_p). Particulate matter can be divided to coarse particles (d_p>2.5 μm), which are mainly deposited in the upper respiratory system, fine particles (0.1<d_p<2.5 μm), which are deposited in the tracheobronchial region of the human respiratory tract, and ultrafine particles (d_p<0.1 μm) which are deposited in the pulmonary/alveolar region (Lin et al., 2008). As a result, xenobiotics contained in ultrafine particles can be easily translocated in the human body via systemic circulation. Genotoxic effects of inhaled particulate matter are mainly attributed to adsorbed polycyclic aromatic hydrocarbons (PAHs). PAHs include a variety of semi-volatile organic compounds of low vapor pressure that can be transferred in long distances as they are mostly adsorbed in fine and ultrafine particles (Dvorská et al., 2012; Venkataraman et al., 1994). Such compounds may be retained for long in human tissue due to their high lipophilicity. About 90% of PAHs are emitted by vehicles (Nielsen, 1996). Other sources include industry, biomass combustion, coke and tar production, as well as tobacco smoke (Freeman and Cattell, 1990; Masclet et al., 1987). Including benzo[a]pyrene (B[a]P), the only PAH classified as known carcinogen to humans by IARC, the most hazardous PAHs are mainly distributed in the particulate phase (IARC, 2010). After human exposure to particulate-bound PAHs, the compounds are distributed in alveolar (80%) and tracheobronchial region (20%) of the HRT. However, the ultimate dose of more toxic substances and their carcinogenic metabolites is much greater in the latter region due to the lower rate of diffusion through the bronchial epithelium.

PAHs cancer risk

HRT particle deposition modeling is applied for the determination of PM deposition fraction (DF) to the three parts of the pulmonary system in order to estimate the internal dose of PAHs. Major mechanisms of PM deposition across HRT include diffusion, sedimentation and impaction. Secondary mechanisms involve interception and electrostatic deposition. Different HRT regions involve different deposition mechanisms, with regard to different PM size as follows:

- Naso-pharyngeal region (or upper respiratory tract – URT): impaction, sedimentation, electrostatic (particles > 1 μm)
- Tracheo-bronchial (TB) region: impaction, sedimentation, diffusion (particles < 1 μm)
- Pulmonary (P) region: sedimentation, diffusion (particles < 0.1 μm)

Several parameters affect HRT deposition, including PM properties (concentration and size distribution), air flow parameters (lung capacity and breathing frequency) and HRT physiology (structure and morphology). All of these parameters have been taken into account in the approach proposed herein.

HRT deposition was carried out using the Multiple Path Particle Deposition (MPPD) v. 2.1 model (de Winter-Sorkina and Cassee, 2002). Age-specific lung geometries representing 10 distinct ages from 3 months old to 21 years old are also provided. An idealized symmetric single-path model as well as a 5-lobe symmetric multiple-path model are available for use with each age setting (Mortensen, 1983a; Mortensen, 1988; Mortensen et al., 1983b). Software inputs include morphological parameters of pulmonary system – functional residual volume (FRC), tidal volume (TV), upper respiratory tract (URT) volume, as well as breathing frequency (BF) for each age group

In its Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons (EPA/600/R-93/089, July 1993) and regional guidance, EPA recommends that a toxicity equivalency factor (TEF) be used to convert concentrations of carcinogenic polycyclic aromatic hydrocarbons (cPAHs) to an equivalent concentration of benzo(a)pyrene when assessing the risks posed by these substances. Calculation of the overall toxicity of the mixture of the 19 PAHs is done using Toxic Equivalent Factors (TEFs), based on the assumption that the *TEF* for B[a]P is equal to 1 (Nisbet and LaGoy, 1992).

TEQ values are calculated according to Eq. 3 using the median value of the measured concentrations, since the concentrations of individual compounds follow an asymmetric distribution:

$$TEQ = \sum_{i=1}^{19} (C_i \times TEF_i) \qquad [3]$$

Genotoxic effects of PAHs are estimated through inhalation cancer risk (*ICR*) assessment. *ICR* is expressed as a linear function of ambient *TEQ* concentration and $IUR_{B[a]P}$ (Eq. 4), as the exposure-cancer risk relationship is considered linear in the low dose region (EPA, 2005). California Environmental Protection Agency recommends an $IUR_{B[a]P}$ value of 1.1×10^{-3} $m^3/\mu g$ (CEPA, 2004).

$$ICR = TEQ \times IUR_{B[a]P} \qquad [4]$$

Equation 4 is adapted to include the exposure and dose parameters discussed earlier for each age group.

In order to calculate the risk of cancer that can be attributed to PAHs , we need to estimate the amount of *TEQ* deposited across the middle (tracheobronchial) and lower (alveoli) HRT regions (Bostrom et al., 2002). This is calculated as the sum of the products of the different size fractioned PM mass deposited across the different HRT regions, multiplied to the *TEQ* estimated for the specific size fraction.

$$TEQ_{uptake} = \sum_{1}^{n} PM_{Tr\_bronch-i} \cdot TEQ_{Tr\_bronch-i} + \sum_{1}^{n} PM_{Alveoli-i} \cdot TEQ_{Alveoli-i} \qquad [5]$$

The cancer risk function implemented is given in Equation 6, where BW_i is the average body weight of each age group and *SF* is the B[a]P slope factor, derived from the assumption that $IUR_{B[a]P}$ refers to a human of 70 kg inhaling 20 m^3 of ambient air per day. *SF* is equal to 3.85×10^{-6} (kg day)/ng B[a]P.

$$ICR = TEQ_{uptake} \cdot \frac{IR_i}{BW_i} \cdot SF \qquad [6]$$

4.3.3 Dioxins and furans

Rationale

Dioxins and furans (PCDDs/PCDFs) are characterized by high carcinogenic potency (Cole et al., 2003). Because PCDDs/PCDFs appears to be acting like a potent and persistent hormone agonist, it appears reasonable to incorporate mechanistic information on receptor-mediated events in risk assessments for TCDD. This information may be obtained from steroid receptor action and from molecular data on the Ah receptor (Lucier et al., 1993). This receptor-based toxicity, results in sex-dependent sensitivities, as a result of a set of sex-specific PCDD/PCDF-responsive genes. However, the estimation of the additional probability of cancer due to the additional exposure burden is quite difficult (Dong et al., 2016). A major obstacle is that an elevated short term external exposure associated to the accidental event, has to be translated into long term risk estimates. Considering the significant persistence and bioaccumulation of PCDDs/PCDFs in the human body, assessing the actual internal dosimetry of this complex mixture is of particular importance. The biokinetics of TCDD are relatively well understood in adult humans (Kerger et al., 2006; Michalek and Tripathi, 1999; Milbrath et al., 2009). However, the impact of pregnancy and lactation on the elimination of TCDD and other dioxins is not clear (Emond et al., 2016). Additional insights regarding the biological perturbations induced by PCDDs/PCDFs exposure are provided by transcriptomics and metabolomics analysis, where altered levels of endogenous steroid metabolites and modified urinary bile acids profiles were identified as a result of acute exposure to PCDDs/PCDFs (Jeanneret et al., 2014). Taken together, these findings are compatible with an increased expression of cytochrome P450s, persistent hepatotoxicity, bile acid homeostasis dysregulation and oxidative stress.

PCDDs/PCDFs cancer risk

The biokinetics of TCDD are relatively well understood in adult humans (Kerger et al., 2006; Michalek and Tripathi, 1999; Milbrath et al., 2009) and several key parameters (tissue partition coeficients and clearance rates) were used from literature. The key aspects characterizing TCDD biokinetics are very high lipophilicity (adipose:tissue blood patrition coefficient is equal to 220) and very slow elimination rate, resulting in a half-life elimination rate of 7.5 years, explaining its long persistance and bioaccumulation potential. Another important issue that needed to be addressed was the transfer of PCDDs/PCDFs through the placenta during pregnancy and maternal milk during lactation. The model describes mother fetus interactions by modelling the intra-placental properties that govern the transfer of xenobiotics and their metabolites from the mother to the fetus as it grows. The anthropometric parameters of the models are time dependent, so as to provide a lifetime internal dose assessment, as well as to describe the continuously changing physiology of the mother and the developing fetus. The model include diffusive flow from the uterus to the placenta and back during pregnancy (Beaudouin et al., 2010). Excretion via lactation is described as an output from the mammary tissue compartment through a partitioning process between mammary tissue and milk, and

milk withdrawal by suckling, as described for PCBs in rats (Lee et al., 2007) and further adopted for humans (Verner et al., 2008). Mixtures of PCDDs/PCDFs are complex environmental mixtures of 210 interrelated chemicals composed of different dioxins and furans. For PCDD/PCDF mixtures, the reference chemical is 2,3,7,8 – tetrachlorodibenzo-p-dioxin (2,3,7,8–TCDD) because it is the most toxic and best-studied of the 210 PCDDs/PCDFs. The toxicity equivalence factor (TEF) methodology was developed by the U.S. Environmental Protection Agency to evaluate the toxicity and assess the risks of a mixture of structurally related chemicals with a common mechanism of action. A TEF is an estimate of the relative toxicity of a chemical compared to a reference chemical. Toxic Equivalents, or TEQs, are used to report the toxicity-weighted masses of mixtures of PCDDs/PCDFs. The TEQ method of PCDDs/PCDFs reporting is more meaningful than simply reporting the total number of grams of a mixture of variously toxic compounds because the TEQ method offers toxicity information about the mixture. Within the TEQ method, each PCDDs/PCDFs compound is assigned a Toxic Equivalency Factor, or TEF. This factor denotes the toxicity of a given dioxin compound relative to the toxicity of 2,3,7,8-TCDD, which is assigned the value of one. Other dioxin compounds are given equal or lower numbers, with each number roughly proportional to its toxicity relative to that of 2,3,7,8-TCDD. Developed by the World Health Organization, TEFs are used extensively by scientists and governments around the world (Van den Berg et al., 1998), finally expressing the so-called TEQ WHO (toxicity equivalent concentration in accordance with the methodology of the World Health Organization), that uses units of grams-TEQ. The EPA uses TEQ WHO to report emissions of PCDDs/PCDFs from known sources to the open environment in its Inventory of Sources of Dioxin in the United States and similar practices have been adopted worldwide, including all the data presented in this study. To obtain the number of grams-TEQ of a dioxin mixture, one simply multiplies the mass of each compound in the mixture by its TEF and sums them up. EPA has classified 2,3,7,8-TCDD as a Group B2, meaning a probable human carcinogen (USEPA, 1985). With regard to 2,3,7,8-TCDD, EPA has calculated an inhalation cancer slope factor of $1.5 \cdot 10^{5}$ $(mg/kg/d)^{-1}$ and an inhalation unit risk estimate of 3.3×10^{-5} $(pg/m^{3})^{-1}$ for 2,3,7,8-TCDD.

4.3.4 Cadmium

Rationale

The assessment of cancer risks in occupational cohorts exposed to cadmium is constrained by the small number of long-term, highly exposed workers, the lack of historical data on exposure to cadmium and the inability to define and examine a gradient of cumulative exposure across studies. Few studies could control the confounding effect of co-exposure to other substances, particularly arsenic and nickel; however, the analyses of workers with low levels of exposure to arsenic still showed an increased lung cancer risk associated with cadmium exposure. Additional support for a cadmium-linked lung cancer risk comes from a prospective population-based study in environmentally polluted areas in Belgium (Nawrot et al., 2006). The results of the studies on cadmium exposure and the risk of prostate cancer are suggestive of an association, but the results are inconsistent. In studies of occupational cohorts exposed to cadmium, studies of people residing in cadmium-contaminated areas and case–control studies of individuals with prostate cancer, some studies reported an increased risk for prostate cancer, while other studies did not indicate the same (Armstrong and Kazantzis, 1985; Sahmoun et al., 2005; Sorahan and Esmen, 2004). The results from cohort studies are supported by a hospital-based case–control study that included highly exposed subjects (Vinceti et al., 2007). Case–control studies suggest that other cancer sites, such as the kidney, and perhaps also the bladder, the breast, and the endometrium may show increased risks associated with dietary or respiratory cadmium exposure

(Åkesson et al., 2008; Antila et al., 1996; Hu et al., 2002; Jarup et al., 1998; Kriegel et al., 2006; Pesch et al., 2000; Sorahan and Esmen, 2004). The International Agency for Research on Cancer reevaluated the evidence for carcinogenicity of cadmium in 2009 and reaffirmed its earlier conclusion that there was sufficient evidence of cadmium's carcinogenicity in humans. The evidence was classified as sufficient for lung cancer and limited for prostate and kidney cancer (Straif et al., 2009).

Cadmium cancer risk

For the parameterization of the generic PBTK model, information on previous well validated models have been used. Cadmium has specific toxicokinetic properties, such as that cadmium metal and cadmium salts are not well absorbed; approximately 25, 1–10, or <1% of the dose is absorbed following inhalation, oral or dermal exposure. Inhalation and oral absorption can be influenced by several factors. Absorbed cadmium is excreted very slowly, with urinary and fecal excretion being approximately equal, 0.007 and 0.009% of the body burden, respectively, per day (Kjellström et al., 1978). The largest amount of cadmium ingested or inhaled and transported to the gut via mucociliary clearance is not absorbed and is excreted through feces. The half-time of cadmium in the whole body in humans is estimated to be higher than 26 years (Shaikh and Smith, 1980), while half-times in mice, rats, rabbits, and monkeys have been calculated from several months to several years (Kjellström and Nordberg, 1985). The highest concentrations of cadmium are found in human liver and kidney and other tissues (particularly muscle, skin, and bone). According to Kjellström and Nordberg (1985), half-times were estimated in the range of 6–38 years for the human kidney and 4–19 years for the human liver. The Nordberg-Kjellström (1978) model is a linear multi-compartment model that describes the disposition of cadmium via oral and inhalation routes of exposure. For inhalation exposure, the model accounts for different deposition patterns in nasopharyngeal, tracheobronchial and alveolar regions of the respiratory tract depending on the size of particles. For oral exposure, cadmium may enter the gastrointestinal tract via food or water or as cadmium particles embedded in mucus from the respiratory tract via the mucociliary/tracheobronchial escalator. For either route of exposure, the model assumes that cadmium enters any of the three blood compartments (B); a) B1 represents the plasma compartment where cadmium may bind to plasma components (i.e., albumin and other organic constituents), b) B2 is the red-blood cell compartment, which represents the accumulation of cadmium in erythrocytes and c) B3 represents the binding of cadmium to metallothionein. The model does not take into account the metallothionein induction after cadmium exposure. Cadmium is assumed to be distributed to the major accumulation sites of either liver, kidney, or "other tissues" via blood while its elimination occurs via feces or urine. The transport of cadmium between compartments is assumed to follow first-order exponential functions and is driven by concentration-dependent gradients.

The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day (U.S. EPA, 1987). Since, there are no positive studies of orally ingested cadmium suitable for quantification, the quantitative estimate of carcinogenic risk from inhalation exposure was only considered. A mortality study of 292 cadmium production workers employed for a minimum of 2 years was used for estimating risks regarding respiratory and prostate cancer (Thun et al., 1985). To examine further the mortality experience of these workers, investigators from the National Institute for Occupational Safety and Health extended the study to include 602 white males with at least 6 months of production work in the same plant between 1940 and 1969. Vital status was determined through 1978, which included the addition of 5 years to the original follow-up. The unit risk should not be used if the air concentration exceeds 6 ug/m^3, since above this concentration the

unit risk may not be appropriate. The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects. It was considered that the use of available human data was reliable because of species variations in response and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide). For cadmium, the slope factor of 6.3 $(mg/kg/day)^{-1}$ was used for estimating the respective UR_{SF}. As a result, the respective CDI_{AR} was equal to 0.00016 μg/kg_bw/d, that was translated in an URSF of 0.18 μg*h/L. To estimate the cancer risks associated with cadmium exposure, the lifetime internal exposure levels will be used.

4.3.5 Lead

Rationale

Accumulating evidence since 1991 suggests that children's intellectual ability is adversely affected at blood lead concentrations < 10 μg/dL (Jusko et al., 2008). To examine some of this evidence in detail, a working group (Weitzman et al. 2004) was convened by the CDC, and the fifth revision of the CDC's Preventing Lead Poisoning in Young Children was issued in 2005 (CDC 2005). The working group concluded that the "*overall weight of evidence supports an inverse association between blood lead levels < 10 μg/dL and the cognitive function of children*," with the caveat that the available data were limited by the small number of "*directly relevant cohort studies*"—studies that include multiple measures of lead exposure throughout early life and key covariate information to reduce the potential for residual confounding (CDC 2005).

Lead neurodevelopmental disorders risk

Approximately 95% of deposited inorganic lead that is inhaled is absorbed. The extent and rate of gastrointestinal absorption of inorganic lead are influenced by the physiological state of the exposed individual and the species of the lead compound. Gastrointestinal absorption of lead is higher in children (40–50%) than in adults (3–10%). The presence of food in the gastrointestinal tract decreases absorption. In adults, about 94% of the total amount of lead in the body is contained in the bones and teeth versus about 73% in children. The elimination half-lives for inorganic lead in blood and bone are approximately 30 days and 27 years, respectively. However, independently of the route of exposure, absorbed lead isexcreted primarily in urine and feces. With regard to the associated neurodevelopmental effects, it has been found (Jusko et al., 2008) that at environmentally relevant lifetime average blood lead concentration 7.2 μg/dL, was inversely associated with Full-Scale IQ ($p = 0.006$) and Performance IQ scores ($p = 0.002$). Compared with children who had lifetime average blood lead concentrations < 5 microg/dL, children with lifetime average concentrations between 5 and 9.9 microg/dL scored 4.9 points lower on Full-Scale IQ (91.3 vs. 86.4, $p = 0.03$). Thus, the assessment of scenarios where lead levels are going to be affected will be based on assessment of the lead blood levels using the respective toxicokinetic model.

4.3.6 Associating internal dose with health effects

A way to associate internal dose with cancer health effects is to use an established slope factor and to translate the intake-based slope factor into a lifetime Area Under the Curve (AUC) related cancer potency factor, defined as AUC_{SF}. Hence, the respective cancer risk (R_C), will be estimated by

multiplying the actual AUC_E for a given period of time as defined by the exposure scenario, with the related unit risk UR_{SF}, that results in risk associated to 10^{-6}.

$$R_C = \frac{AUC_E}{UR_{SF}} \cdot 10^{-6}$$

The area under the curve / slope factor (UR_{SF}) is defined as the AUC that results in risk associated to 10^{-6}. This in turn is derived as follow:

- Starting from the slope factor of the respective chemical, the chronic daily intake (CDI) that results in cancer risk equal to 10^{-6} is estimated. It has to be noted that this level of environmental risc is characterized as acceptable, hence, this intake levels is now defined as CDI_{AR}.
- The CDI_{AR} is used as an input to the respective PBPK model, and the AUC for a period of 70 years is estimated; the product of this computation, describes the AUC that corresponds to a risk of 10^{-6} and is defined as UR_{SF}, since it is originally based in the initial slope factor.

This method has clear advantages, since it allows us to incorporate all key parameters that induce inter-individual variability related to physiology (e.g. bodyweight, genetic polymorphism of enzymes associated with metabolism), as well as related to the exposure scenario, such as route dependent bioavailability differences. Moreover, considering that AUC is by definition the integral of internal exposure over time, the effect of highly dynamic exposure scenarios (including short term accidental events) to internal dose fluctuations are effectively captured and incorporated in the risk calculation. This is of particular importance for compounds that are not rapidly metabolized or eliminated, where short term exposure events result in long term internal exposure changes. Up to now, the method has been effectively applied in the case of dioxins release in an accidental fire of a plastic recycling plant (Sarigiannis, 2017).

It has to be noted that cancer risk estimates derived by this method are more conservative to the ones derived by original BBDR models. BBDR models translate human epidemiological data into micro relationships, associating them with internal dose; in contrast, the AUC/slope factor association method, starts from an animal-based slope factor, which is already conservative in its nature.

4.4 Noise

4.4.1 Rationale

Noise according to WHO (Berglund, 1999) is a harmful environmental pollutant with adverse psychosocial and physiologic effects on public health. Common psychosocial effects are annoyance and sleep disturbance (Babisch, 2005; Passchier-Vermeer and Passchier, 2000). WHO suggests that daytime and nighttime limits at 55 dB(A) and 40 dB(A) respectively, should not be exceeded in order to prevent possible such psychosocial effects (Murphy and King, 2010). Focusing on the road generated noise, it is estimated that more than 30% of the EU citizens are exposed to noise levels above those regarded as acceptable by the World Health Organization (WHO) and about 10% report severe sleep disturbance because of transportation noise at night (EEA, 2003). Furthermore, recent studies suggest

that 24 million people in the European Union are highly annoyed by road traffic noise (EEA, 2000). The effects of the primary sleep disturbance include amongst other, the difficulty in falling asleep (increased sleep latency time), awakenings and alterations in the sleep stages or depth. Other primary physiological effects can also be induced by noise during sleep, including increased blood pressure, increased heart rate, increased finger pulse amplitude, vasoconstriction, change in respiration, cardiac arrhythmia and an increase in body movements. According to WHO (Berglund, 1999), exposure to night-time noise also induces secondary effects, including reduced perceived sleep quality, increased fatigue, depressed mood or well-being and decreased performance. Noise annoyance can be defined as "*a feeling of displeasure associated with any agent or condition, known or believed by an individual or group to adversely affect them*" (Lindvall & Radford 1973; Koelega 1987). However, apart from "annoyance", according to WHO (Berglund, 1999) people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion . The effects of night-time annoyance are expressed in the following day, where studies have shown that people living in areas exposed to night-time noise have an increased use of sedatives or sleeping pills. Other frequently reported behavioural effects of night-time noise include closed bedroom windows and use of personal hearing protection. It is noted that sensitive groups of the above health effects include the elderly, shift workers, persons especially vulnerable to physical or mental disorders and other individuals with sleeping difficulties.

4.4.2 Noise response functions

Sleep disturbance, is classified as low, medium and high, according to the noise level the population is exposed to; it is computed via the polynomial equations 1-3 (Miedema and Vos, 2007),

$$LSD = -8.4 + 0.16*L_{night} + 0.01081*(L_{night})^2 \quad (1)$$

$$SD = 13.8 - 0.85*L_{night} + 0.01670*(L_{night})^2 \quad (2)$$

$$HSD = 20.8 - 1.05*L_{night} + 0.01486*(L_{night})^2 \quad (3)$$

where the LSD is the low sleep disturbance exposed to L_{night} in the range of 0 – 45 dB(A), SD is the sleep disturbance exposed to L_{night} in the range of 45 – 65 dB(A) and HSD is the high sleep disturbance exposed to L_{night} above 65 dB(A).

The sleep annoyance due to road transport is computed from equation 4 (Miedema and Oudshoorn, 2001),

$$HA[\%] = 0.5118*(L_{den} - 42) - 1.436*10^{-2}*(L_{den} - 42)^2 + 9.868*10^{-4}*(L_{den} - 42)^3 \quad (4)$$

where HA are the highly annoyed persons

Myocardial infraction for an L_{day} in range of 57 to 80 dB(A) is computed from the Odd Ratio presented by equation 5 and used for the calculation of the relative risk presented by equation 6 (Babisch, 2008).

$$OR = 1.63 - 6.13*10^{-4}*(L_{day},16h)^2 + 7.36*10^{-6}*(L_{day},16h)^3 \quad (5)$$

$$RR = (e^{((OR-1)/10)*(Lday,16h)} - 1) / e^{((OR-1)/10)*(Lday,16h)} * BHE * EP \quad (6)$$

Where the *BHE* is the background health effect per year i.e. 1.4 hospital admissions per 1000 population (WHO, 2011b) and *EP* is the fraction of the exposed population.

4.5 Disability adjusted life years lost (DALY)

The Disability Adjusted life Years or DALY for a disease or a health condition, are utilized defined as the sum of the Years of Life Lost (YLL) due to premature mortality in the population and the Years lost due disability (YLD) for people living with the health condition or its consequence, i.e.

DALY = YLL + YLD

Where YLL corresponds to the number of deaths multiplied by the standard life expectancy at the age at which death occurs and YLD to the number of incident cases in that period, multiplied by the average duration of the disease and a weight factor that reflects the severity of the disease on a scale from 0 (perfect health) to 1 (dead), as defined in the following equations.

YLL = N * L

Where N is the number of deaths and L the standard life expectancy at age of death in years

YLD = I * DW * L

Where I is the number of incident cases, DW is the disability weight and L is the average duration of the case until remission or death in years.

It is noted that, for the mortality induced cases, DALY are computed solely from the YLL, where as for morbidity incidences the YLD are utilized with appropriate use of a disability weight and duration of the case.

4.5.1 Valuation

The valuation of health endpoints will build on previous studies – for example Hunt et al (2011) presented values for a range of health endpoints – see below.

Table 4-13: Health unit values in HEIMSTA and INTARESE studies

Health End-Point	Low	Central	High	Unit (2010) per case	Reference
Sleep disturbance	480	1,240	1,570	Euro/year	Godet-Cayré et al. (2006); Ozminkowski et al. (2007)
Hypertension	880	950	1,110	Euro/year	Ramsey et al. (1997); Berto et al. (2002)
Acute myocardial infarction	4,675	86,200	436,200	Euro	Moïse et Jacobzone, 2003; Yasunga et al. (2006)
Increased mortality risk (infants)	1,120,000	2,475,000	11,200,000	Euro	Holland et. al. (2004)
Chronic bronchitis	43,000	60,000	100,000	Euro	Krupnick and Cropper (1992)
Severe COPD	70,000	120,000	260,000	Euro	Maca et al. (2011)
Increased mortality risk - Value Of Life Years acute	60,820	89,715	220,000	Euro	Alberini et. al. (2006)
Increased mortality risk - Value of Prevented Fatality acute	1,120,000	1,650,000	5,600,000	Euro	Alberini et. al. (2006)
Life expectancy reduction - Value of Life Years chronic	37,500	60,000	215,000	Euro	Alberini et. al. (2006); Desaigues et. al. (2011)
Respiratory hospital admissions	2,990	2,990	8,074	Euro	Navrud (2001); Holland et. al. (2004)
Cardiac hospital admissions	2,990	2,990	8,074	Euro	Navrud (2001); Holland et. al. (2004)
Work loss days (WLD)	441	441	441	Euro	Navrud (2001); Holland et. al. (2004)
Restricted activity days (RADs)	194	194	194	Euro	Navrud (2001); Holland et. al. (2004)
Minor restricted activity days (MRAD)	57	57	57	Euro	Navrud (2001); Holland et. al. (2004)
Lower respiratory symptoms	57	57	57	Euro	Navrud (2001); Holland et. al. (2004)
LRS excluding cough	57	57	57	Euro	Navrud (2001); Holland et. al. (2004)
Cough days	57	57	57	Euro	Navrud (2001); Holland et. al. (2004)
Medication use / bronchodilator use	74	80	96	Euro	Maca et al. (2011)
Lung cancer	70,000	720,000	4,200,000	Euro	Weissflog et al. (2001); Serup-Hansen et al. (2003); Scasny (2008); Jeanrenaud and Priez (1999); Aimola (1998)
Leukaemia	2,050,000	4,000,000	7,000,000	Euro	Aimola (1998)
Neuro-development disorders	4,500	15,000	33,000	Euro/year	Ščasný et. al. (2008)
Skin cancer	11,000	14,000	27,000	Euro	Aimola (1998)
Osteoporosis	3,000	5,700	8,100	Euro	Kudma and Krška (2005); Werner and Vered (2002)
Renal dysfunction	23,000	30,400	41,000	Euro	Bartaskova et al (2005); Sun-Mi et al. (2006)
Anaemia	750	750	750	Euro	Ossa et. al. (2007)

Source: Hunt et al, 2011

4.6 Greenhouse gases

Monetary valuation of greenhouse gas emissions has taken a number of different routes:

a. The social cost of carbon;
b. Use of market prices for carbon; and
c. The standard price approach based around marginal abatement costs.

4.6.1. Social cost of carbon

The estimation of the social cost of carbon has taken several forms:

- Model based estimation of damages arising from climate change and linking this back to atmospheric concentrations and hence emissions (e.g. the FUND or DICE model); and
- Direct estimation of the willingness to pay to avoid carbon emissions.

Model based estimates of the social cost of carbon have been the subject of much debate. A recent study by Nordhaus using a revised version of the DICE model (DICE-2016R) suggests a social cost of carbon at present of $31 per ton of CO_2. There is significant uncertainty in the estimates of the social cost of carbon, with factors such as the rate of time preference leading to values for the social cost of carbon to range from $13 per ton to $201 per ton (Tol, 2016). Given the uncertainties and limited number of studies that have attempted to quantify the global cost of climate change, other methods of valuation may be preferred.

Attempts to value the willingness to pay to avoid carbon emissions include studies using computer-based panels. Alberini et al (2018) find a willingness to pay per ton of CO_2 emissions avoided of €133 for Italy and €94 for the Czech Republic.

4.6.2. Market prices for carbon

Carbon is traded in the EU ETS. The UK government uses a hybrid approach based on Business-As-Usual emissions scenarios and marginal abatement cost curves from the POLES model and the market prices of EU Allowance futures contracts to estimate short term and long term carbon values for policy analysis. Current short-term values are presented in the table below.

Table 4-14: BEIS updated short-term traded sector carbon values for policy appraisal in real 2016 terms, £/tCO2e

Year	Low	Central	High
2016	0.00	4.18	4.18
2017	0.00	4.22	4.22
2018	0.00	4.25	4.61
2019	0.00	4.41	7.22
2020	0.00	4.58	9.14
2021	3.87	11.86	19.83
2022	7.74	19.14	30.52
2023	11.61	26.42	41.21
2024	15.47	33.70	51.90
2025	19.34	40.98	62.60
2026	23.21	48.25	73.29
2027	27.08	55.53	83.98
2028	30.95	62.81	94.67
2029	34.82	70.09	105.36
2030	38.68	77.37	116.05

Source: BEIS (2017)

4.6.3. Standard price approach

The standard-price approach involves taking the agreed aims for GHGs reduction and assuming decisions were taken after consideration of (a) current knowledge about impacts and mitigation and adaptation measures, and (b) the possibilities of additional unknown risks (precautionary principle). Marginal abatement costs (MAC) to reach these aims will be estimated; in particular the European Commission goal to reduce greenhouse gas emissions by 40% from 1990 to 2030 and to limit the temperature increase to 2°C within an international strategy. Numerous existing studies to estimate the marginal costs per t of CO_{2eq} can be used.

As a new element in this assessment we can also take into account the global warming effect of short-lived greenhouse gases like ozone, black carbon and aerosols.

4.6.4. Global warming potentials of greenhouse gases

The Global Warming Potential is based on the cumulative radiative forcing over a particular time horizon. Table 4-15 shows global warming potentials as given in the IPCC Fifth Assessment Report (IPCC AR 5). Global warming potentials for further pollutants can be taken from IPCC 2013: Myhre et al. 2013, Appendix 8.A: Lifetimes, Radiative Efficiencies and Metric Values.

Table 4-15 Global warming potentials (source: IPCC AR5: Myhre et al. 2013)

	20 years	100 years	Reference
CO_2	1	1	IPCC AR 5: Myhre et al. 2013
CH_4	84	28	IPCC AR 5: Myhre et al. 2013
N_2O	264	265	IPCC AR 5: Myhre et al. 2013
BC	3200 (270 to 6200)	900 (100 to 1700)	IPCC AR 5: Myhre et al. 2013, Bond et al. (2013)
OC	–160 (–60 to –320)	–46 (–18 to –19)	IPCC AR 5: Myhre et al. 2013, Bond et al. (2011)
VOC global	14	4.5	IPCC AR 5: Myhre et al. 2013, Fuglestvedt et al. (2010), Collins et al. (2002)
CO	6 to 9.3	2 to 3.3	IPCC AR 5: Myhre et al. 2013, Fuglestvedt et al. (2010)
NO_x global	19	-11	IPCC AR 5: Myhre et al. 2013, Fuglestvedt et al. (2010)
SO_2	-140	-40	Fuglestvedt et al. (2010) based on Schultz et al (2006)

Atmospheric drivers like black carbon and ozone have relatively short atmospheric lifetimes but significant warming impacts on the climate. As it can be seen in Figure 4-2 the short-lived gases and aerosols account for an important part of the total anthropogenic radiative forcing. Emissions of black carbon have a positive radiative forcing through aerosol–radiation interactions and black carbon on snow. Tropospheric O_3 forcing is driven by and broadly attributable to emissions of precursors such as NO_x, CO, CH_4 and NMVOCs. However, the climate forcing effect of ozone has often been overlooked in air quality and vice versa. Despite some attempts have been made, to take the effect of short lived climate forcers like ozone into account, up to now there is no internationally approved and standardized method. The IPCC AR5 (2013) adopted a methodology that demonstrated the direct link between the precursors of ozone and the resulting radiative forcing. Thus, the radiative forcing of ozone will be included in the assessment through the emissions of precursor substances (cf. Table 4-15). Furthermore, the global warming potential of black carbon will be included in the assessment (cf. Table 4-15).

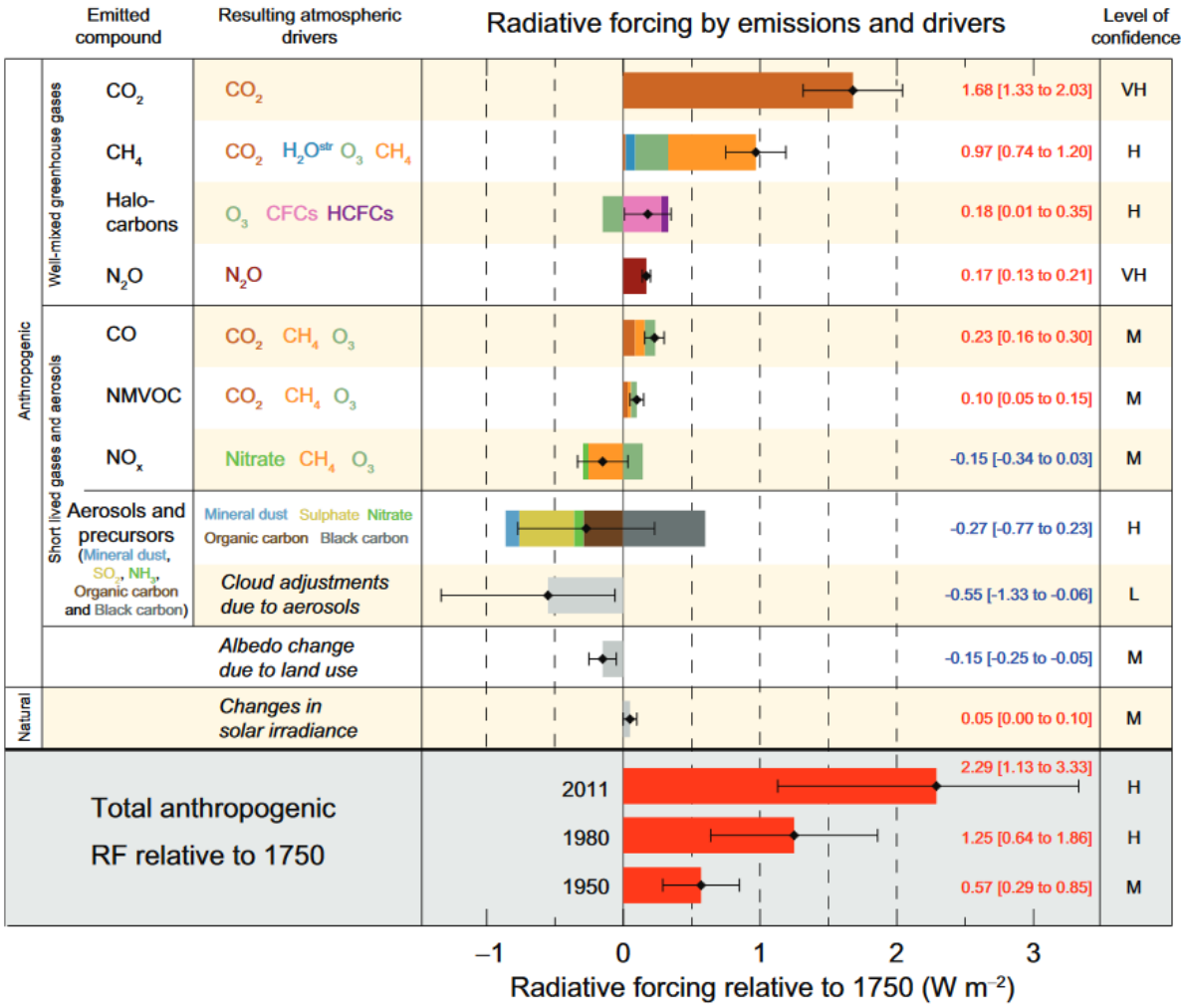

Figure 4-2 Radiative forcing estimates in 2011 relative to 1750 and aggregated uncertainties for the main drivers of climate change (source: IPCC 2013)

4.7 Time losses

For the valuation of time losses, we can build on existing meta-analyses of the values attributable to time losses for different transport modes and travel types (e.g. commuting, business, leisure travel). There are also EIB values for a number of these factors – and studies have compared the values from meta-analyses to these values (Wardman et al, 2012). The table below gives indicative values for a selection of countries.

Table 4-16:Comparison between Value of Time from EIB, Official sources and Meta-analysis model

	Commute			Other			Business		
	Official	Meta	EIB	Official	Meta	EIB	Official	Meta	EIB
Norway	Original prices and incomes 2009. Figures supplied by Farideh Ramjerdi								
Car Short (<100)	11.39	22 = 13.69	14.58	9.75	22=11.78	9.79	48.10	22=41.01	50.56
Car Long (>100)	25.31	125=17.46	14.58	18.48	218=16.25	9.79	48.10	218=56.54	50.56
PT (Bus) Short (<100)	7.59	19=7.22	4.28	5.82	19=6.12	9.34	48.10	19=21.31	46.59
Rail Long (>100)	19.74	125=13.16	16.73	11.65	303=13.38	10.84	48.10	303=46.57	46.36
Bus Long (>100)	13.04	125=10.29	4.28	9.24	232=9.8	9.34	48.10	232=34.12	46.59
Air				22.79	1386=31.85	27.67	56.33	1386=110.8	72.28
Netherlands	Originally based on HCG (1998). http://www.rijkswaterstaat.nl/kenniscentrum/economische_evaluatie/kengetallen/								
Car	10.51	20=8.58	9.99	7.27	13=6.59	7.03	36.43	17=23.30	33.56
Train	10.58	45=7.06	10.84	6.52	45=6.07	7.55	22.40	45=19.16	31.85
Bus/Tram	9.85	17=4.59	8.58	6.22	13=3.70	6.50	17.16	15=12.28	30.55
Germany	Bundesministerium für Verkehr, Bau und Stadtentwickelung (2005) Die gesamtwirtschaftliche Bewertungsmethodik des Bundesverkehrswegeplan 2003, Bonn, Druckerei des BMVBW.								
Car	5.95	20=7.98	9.42	5.95	14=6.53	6.68	33.44	19=21.58	31.39
Train	5.95	40=6.46	10.14	5.95	39=5.53	7.12	23.88	80=20.05	30.01
Bus	5.95	10=3.89	8.31	5.95	15=3.56	6.14	25.72	41=13.62	28.50

Source: Wardman et al (2012)

4.8 Health co-benefits of measures

4.8.1. Valuing Physical Activity

HEAT is the WHO-Europe Health Economic Assessment Tool, developed for analysis of the health benefits of walking and cycling via estimation of effects on mortality[1]. The HEAT tool uses estimates of the Value of a Statistical Life and studies on the mortality risk benefits of exercise to come up with values for the health benefits of walking and cycling.

The model is designed for application in situations such as:

- When planning new cycling or walking infrastructure. HEAT attaches a value to the estimated level of cycling or walking when the new infrastructure is in place. This can be compared to the costs of implementing different interventions to produce a benefit–cost ratio (and help to make the case for investment).
- To value the reduced mortality from past and/or current levels of cycling or walking, such as to a specific workplace, across a city or in a country. It can also be used to illustrate economic consequences from a potential future change in levels of cycling or walking.
- To provide input into more comprehensive economic appraisal exercises, or prospective health impact assessments. For example, to estimate the mortality benefits from achieving targets to increase cycling or walking, or from the results of an intervention project.

[1] http://www.heatwalkingcycling.org/

The following table summarises results from HEAT policy studies in a number of European countries and the USA. The HEAT model has been widely used and officially adopted by the Government of Sweden.

Table 4-17. Examples of use of the HEAT model[2]

Country	Scenario	Benefits
Austria	Assessment of benefits of existing modal share for cycling of 5%, with average journey distance of 2km	412 lives saved/year Benefits of $US560 million/year
Czech Republic	Pilsen: 2% of inhabitants of city of take up cycling, 2 trips per day	Discounted benefits of $US1.2 million
Estonia	Pamu: Estimation of cyclists using a new route proposed as part of infrastructure development	0.17 deaths / year, current value of benefit equal to $US155,000/year
Finland	Kuopio: Assessment of benefits of existing cycling levels	0.29 to 5.66 deaths prevented / year, with average annual benefit of $US0.56 – 11 million
Italy	Florence: Existing levels of walking and cycling to work Modena: Existing and future levels of cycling	Lives saved annually: 16.8 (walking), 5.3 (cycling) with total benefits of $US37 million/year 5% decrease in mortality risk (0.35 deaths/year). Annual benefits of $US0.56 million
Sweden	Swedish road administration has adopted HEAT for their economic assessment of cycling infrastructure.	
UK	Brighton: 30% increase in cycling Glasgow: Existing levels of cycling Sustrans: Existing levels of walking and cycling across the UK's National Cycle Network	Mean annual benefit of $US370,000 Annual benefit of $US 5 million in 2009, increasing to $US 7 million by 2012 Lives saved in 2011: 144 (walking), 264 (cycling) with total benefits of $US 480 million/year

[2] summarised from http://www.euro.who.int/en/health-topics/environment-and-health/Transport-and-health/activities/guidance-and-tools/health-economic-assessment-tool-heat-for-cycling-and-walking/examples-of-applications-of-heat with all values converted to US dollars for consistency though this paper.

USA	Boston: Reduction in walking through fares increase on public transport	9 – 14 additional deaths per year from reduced walking, valued between $75 – 117 million
Central Europe (Austria, Czech Republic, Hungary, Italy, Slovakia and Slovenia)	Increased cycling or walking in 12 Central European cities	Reaching target of 15% modal share for cycling by 2015 in 6 cities prevents between 2 and 33 deaths per year in each city with annual benefits of $US1.8 – 33 million. Cost per life saved = $US 0.56 – 2.2 million, with benefit cost ratios of 5 -16.

4.8.2. Valuing wellbeing

Improving well-being can have a number of impacts on individuals and society. Figure 4-3 gives an overview of some of the key linkages and the values that can be attached to these. There has been increasing attention paid to the valuation of the direct benefits of changes in wellbeing and these are discussed later – but in our view it is the co-benefits that are probably of most interest to local policy makers. The assessment of the co-benefits [3] to health, crime, productivity, education and environments of wellbeing improvements are likely to yield potentially significant gains over and above the direct benefits to individuals.

As part of the URGENCHE project, Hiscock et al (2014) reviewed the wellbeing impacts of different options to mitigate greenhouse gases. There are a number of weaknesses in this literature including:

- The conceptualization of causality in existing work in this area – including issues with the direction of causality;
- Concerns about the generalizability of study findings with a plethora of measures being used in both reporting and in the measurement of wellbeing outcomes; and
- Though a number of studies were found to exist that had assessed the wellbeing implications of urban environmental factors, there was insufficient evidence to allow for wellbeing assessment of particular interventions.

Monetary valuation of wellbeing is still in its relative infancy (Cox et al, 2012). One method that has been used is to equate a loss of wellbeing with a level 3 mental health problem (i.e. severe problem) and use Quality Adjusted Life Year (QALY) weights assessed by health economists (Centre for Mental Health, 2010). QALYs are one way economists use to estimate the varying types of health outcomes in a common metric – with a value of 1 indicating a year in full health and 0 indicating death. Taking the loss of QALYs from a severe mental health condition (0.352) and multiplying by the NICE Cost

[3] The term "co-benefits" is used in this case as the outcomes are not the main objective of the intervention – the main or primary outcome targeted is taken to be that of wellbeing. Benefits to education may occur, and be considered by schools, but these are secondary or co-benefits.

Effectiveness threshold[4] of £30,000 gives a value of £10,560 per year for overall wellbeing. There is some evidence in the literature that children's health is value more highly than that of adults. OECD (2006) suggest that a factor of 2 should be applied – so this might imply a value of overall children's wellbeing of £21,120 per annum.

Another method for valuing changes in subjective wellbeing using the "life satisfaction approach" has been developed in recent years. This approach uses survey-based methods to estimate changes in life satisfaction using, for example, the ONS's Integrated Household Survey. Using analysis of the relationship between life satisfaction and income, this method can be used as a way of valuing changes in provision of a range of non-marketed goods (HMT, 2011). Fujiwara et al (2014) use these methods in the context of the valuation of the wellbeing benefits of culture and sport – finding that engagement with the arts led to higher wellbeing, with a value of £90 per person per month, and engagement with sports led to wellbeing benefits worth £94 per person per month.

The What Works Centre for Wellbeing is currently exploring the use of life satisfaction measures compared to the costs – in a way this is similar to the methods used by NICE for decisions on treatment provision in the health setting, where a cost per Quality Adjusted Life Year is used to estimate whether a particular treatment is "cost-effective" or not.

For the valuation of the co-benefits of wellbeing, a recent review (Maccagnan et al, 2017) gives a good overview of the evidence and of methods that can be used to value these impacts. Table 4-18 gives a summary of the evidence on co-benefits of improved wellbeing.

Table 4-18: Summary of empirical evidence on the co-benefits of improved wellbeing

Co-benefit dimension	Empirical evidence of key linkages between subjective wellbeing and:
Health	Mortality (4 to 10 additional life years) Prevention of new diseases (particularly stroke, ischemic heart disease, cold, pregnancy outcomes) Survival from illnesses (mixed results, depending on seriousness of illness) Doing physical exercise (in turn having a protecting effect against development of illnesses)
Social	Criminal and antisocial activities: higher delinquency is associated with lower subjective wellbeing levels Prosocial behaviours: blood donation, voluntary work and money to charities Social activities: more time spent with other, more quality time, higher enjoyment of social interactions
Employment	Lower levels of absenteeism and work turnover

[4] The NICE cost-effectiveness threshold represents the value for money that a drug or intervention can have to be funded. The normal maximum threshold is £30,000 per QALY – i.e. the NHS will fund interventions with cost-effectiveness up to this level, but interventions that cost more than this per QALY gained will not be funded under normal circumstances. There are exceptions – but in general this is the case.

	Better worker performance in terms of manager assessments, income and productivity
Education	Better educational achievements: better results in Key Stage tests and GCSEs exams
Environment	Environmental-friendly household behaviours More money to charities for environmental purposes

Source: Maccagnan et al (2017)

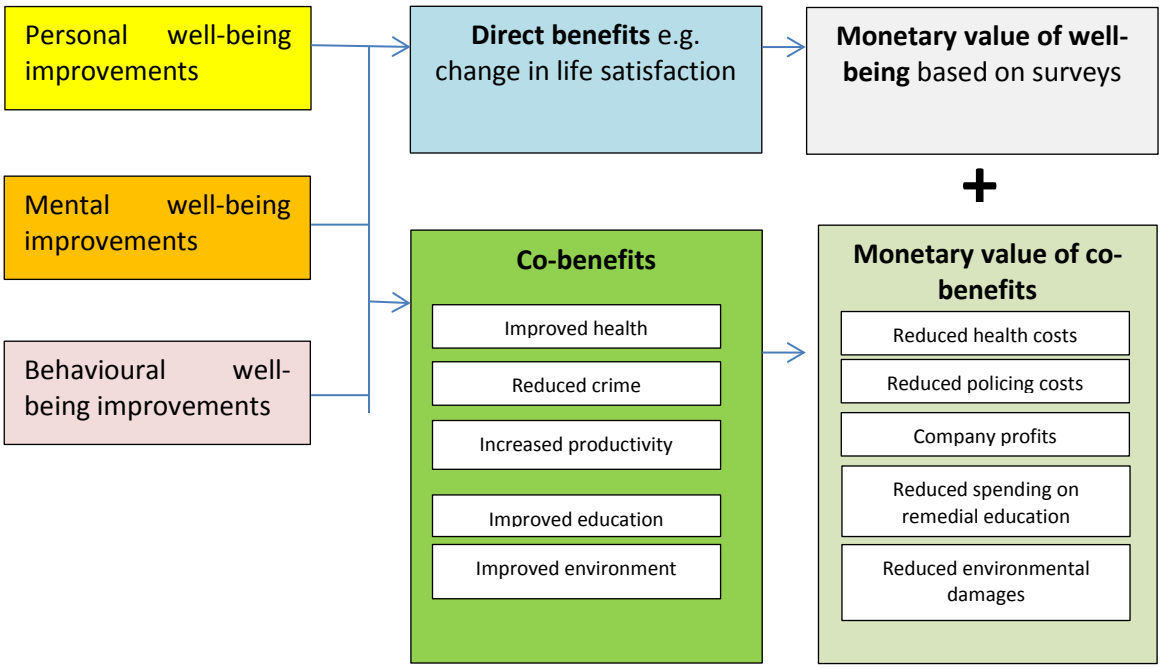

Figure 4-3: The impacts of wellbeing and associated values

5 Resilience planning

A resilience approach to sustainability focuses on how to build capacity to deal with unexpected change[5]. It is important, first, to identify and set the principles that are crucial for building resilience of a complex system (such as a city or community that is made up of many elements) and then discuss how these principles could be best practically applied. It is worth mentioning that systems are dynamic and not static and that disruptions are the essential part of the adaptation cycle and change is fundamental to resilience building. We should want to make ourselves adaptive, flexible and capable of absorbing disruptions and converting them into change that contributes to the systems' overall functioning and purpose[6].

The City of Athens, through a competitive process, was selected in 2014 to join the 100 Resilient Cities (100RC) network. The city engaged in an intense and participatory process in order to draft and implement a holistic, robust and realistic strategy that supports and enhances the resilience of the city for the upcoming decades.

Each city's resilience journey begins with a diagnostic assessment that delineates its own significant challenges. For several months, in the beginning of the program citizens were interviewed, from experts, artists and community leaders to industrialists, homeless and women migrants, on what they thought made the city resilient and how they envisioned a resilient Athens in 2030. In the spring of 2015, Athens introduced the concepts and methods of the 100 Resilient Cities program by convening an Agenda Setting Workshop (with 130 Athenians). February 2016, the city started its resilience-building journey by establishing the Office of Resilience and Sustainability (ORS) and drafting a robust Stakeholder Engagement Plan. The ORS became a part of the new organizational structure of the municipality gaining official status as part of the Mayor's Office in December 2016. The following activities have been achieved in Phase I, II & III of Athens' resilience journey.

Phase I - Kick-starting Resilient Athens Office, Team and Investigations

- February 2016 –June 2016: Focus Groups with municipal and local stakeholders
  Several focus groups were organized with city districts' council members, advisors to the Mayor, architects, start-uppers, tour guides, young entrepreneurs, CSOs and NGOs as well as migrant women, homeless street paper vendors and elders. The participants described city's assets, vulnerabilities and their personal wishes for the future Athens.

- July 2016: Athens's Preliminary Resilience Assessment (PRA)
  The PRA set the resilience baseline for Athens, introducing 5 discovery areas that the city had to explore more in order to discover opportunities that would help it built its resilience. Numerous people including experts, representatives from the local, regional and central government, city district leaders and other civil society and community ambassadors, platform partners and the strategy partner actively participated in the process. Through desktop

[5] Principles for Building resilience: Sustaining Ecosystem Services in Social-Ecological Systems, Reinette Biggs, Maja Schlüter, Michael L. Schoon, Read more at http://www.cambridge.org/9781107082656
[6] The Resilience Dividend: Being strong in a world where things go wrong, Judith Rodin, New York, Public Affairs, 2014

research, interviews, working group meetings, workshops and conferences, the specific opportunities and challenges that started emerging were examined.

- International Workshops: During its first year of operation, the Resilient Athens team initiated and hosted two international meetings with fellow 100RC cities, partners and experts. In July 2016, Athens hosted the European leg of the first Trans-Atlantic Policy Lab aiming to identify and propose policies that mitigate social inequity in the United States and Europe. In September 2016, Athens, in partnership with the 100 Resilient Cities and Chief Resilience Officers (CRO) from eight of its member cities launched the Athens Network Exchange: Cities and the Global Migration Crisis.

Phase II – Co-creating and launching Athens Resilience Strategy

- September 2016-February 2017: Identification of resilient opportunities
  Resilient Athens Team explored deeper each discovery area, engaging in numerous meetings, discussions and research as well as organizing workshops, events and conferences. This process resulted in 138 resilient opportunities.

- February 2017: Field Of Opportunities Workshop (FoO)
  138 resilient opportunities with two groups of local and international partners were prioritized during the Workshop. The FoO ensured the Athens Resilient Strategy is fact-based and data-driven but also, thought its making, a result of transparent and extensive dialogue and consultation with many relevant stakeholders. After the FoO, a set of 65 resilient actions were reshaped and organized under 4 pillars and 13 Resilience goals.

- March 2017–June 2017: Drafting and presenting the Athens Resilience Strategy
  The document was drafted and presented in the Mayor of Athens, the city Council, the municipal Executive Committee and the RSC as well as to the municipal political parties before its official public launch in June 2017.

The Athens Resilience Strategy[7], developed with the support of 100 Resilient Cities – Pioneered by the Rockefeller Foundation –, presents a series of distinct yet connected actions with a clear vision of how the city can best cope with the increasing interdependency of shocks and stresses. It links new resilience-building actions with existing efforts, and relies heavily on our urban communities, who proved effective and resilient during times of crisis. This work does not stand alone; it is supported by several documents that have been instigated by or produced in alliance with the city's resilience journey. The first one came out of the 100RC Athens Network Exchange in September 2016 under the title "Global Migration: Resilient Cities at the Forefront," and the second is a set of policy proposals "Advancing Equity for Athens' Resilience" created for the city by Transatlantic Policy Lab program as a 100RC offering and funded by the Bertelsmann Foundation. Finally, the Athens Climate Change Adaptation and Mitigation Action plan was produced through a unique collaboration between C40 and 100RC. These two documents together with the existing Athens strategic and operational plans, frame this resilience strategy. Athens' vision is that *"By 2030 Athens strives to be a responsive, embracing and inspirational city that is proud, green and citizen led. We nurture creativity and innovation, creating*

[7] http://www.100resilientcities.org/strategies/athens/

prototypes of belonging, bridging history and progress. Athens is a city that listens and speaks with the world."

The Athens Resilience Strategy also draws upon other local, regional, national and international strategic documents as well as the Urban Agenda for the EU, the New Urban Agenda – Habitat III and the UN Sustainable Development Goals. From early on, the ORC collaborated closely with the C40 Advisor for Athens, leading to the first Athens Climate Change Adaptation and Mitigation Plan (April 2017). The connections with other strategic documents elevates the Athens Resilient Strategy's integration and robustness, bridging the gaps between different institutions as well as gaps that exist between the city's everyday management and its long-term policy commitments and goals.

Phase III – Developing the One Year Action Plan

- February 2018: The one-year Action Plan, developed by the ORS, acts as a detailed project plan setting out the tasks that will be undertaken during the next period of the Resilience Strategy Implementation for Athens, including timescales, roles and responsibilities. This document follows from the research and stakeholder consultations undertaken for the Athens Resilience Strategy Development during Phase I & II of the Strategy Development process.

This Action Plan will explain how the CRO and the Resilience Team of Athens will approach the following for each Action:

- The integrated approach that will be taken to the activities in Phase III, given the multiple Actions;
- How other local resilience issues, processes, timelines and imperatives will be integrated into the research and analysis;
- How the team will conduct further stakeholder engagement and participation, including how it will adapt structures of new or existing committees and working groups;
- How Phase III activities and processes will be governed to ensure concurrence, support and sign-off from key decision-makers;
- How the city will approach decision-making about the city's goals and initiatives, while incorporating insights and information from Phase II;
- Details on the roles, responsibilities and commitments of local partners, strategy partners and platform partner resources, including MoU where relevant.

6 Uncertainty

6.1 Introduction

Evaluation of uncertainty in complex impact assessment of air pollution and climate change policies is usually disregarded regardless of its importance. The reasons behind this arise due to the inherent complexity in the processes involved, the lack of knowledge on the parameter distribution and most importantly due to the lack of a solid framework to assess the uncertainty.

The approach we will employ in ICARUS will be based on a tiered approach, that is, the analysis can begin with a simple qualitative uncertainty characterisation and subsequently progress to semi-quantitative and finally a complex quantitative assessment. The latter could follow when a lower tier analysis indicates a high degree of uncertainty for certain identified sources, the sources are highly influential to final result(s), and sufficient information and resources are available to conduct quantitative uncertainty assessment. This is not to suggest that quantitative uncertainty analyses should always be performed in all the impacts assessments. The decision regarding the type of uncertainty characterisation performed depends on the scope and purpose of the assessment, on whether the selected analysis will provide additional information to the overall impact assessment, whether sufficient data are available to conduct a complex quantitative analysis, and if time and resources are available for higher tier characterisations.

More specifically the proposed uncertainty framework consists of a combination of qualitative and quantitative assessment tools clustered into three tiers. Tier 1 corresponds to the qualitative part, where all sources of uncertainty are tabulated in a matrix, by annotating direction, level and appraisal of the knowledge-base. Tiers 2 and 3 involve the quantitative evaluations of those sources with the highest degree of uncertainty and the highest influence to the final result(s), provided that sufficient information and resources are available. Methods employed at the tier 2 and 3 include screening methods for global sensitivity assessment (tier 2), the Bayesian statistics and Monte Carlo analysis (tier 3).

6.2 Qualitative uncertainty assessment – Tier 1

Qualitative uncertainty assessment is organised in three distinct steps:

1. Identification of all uncertainty sources;
2. Qualitative characterisation of uncertainty comprising three dimensions:
   a) Assessing the direction and magnitude of the influence of the uncertainty source on the result(s)
   b) Assessing the knowledge base of the uncertainty source;
   c) Assessing the subjectivity of the choice of uncertainty sources
3. Qualitative uncertainty reporting.

The following chapters summarise the three basic sequential steps that need to be performed to characterise the uncertainty of each component in the full chain of environmental health impact

assessment, as well as the uncertainty associated with the final result(s) of the full chain. The same three steps are repeated in sequence at each tier of the uncertainty assessment composing the harmonised methodology developed in ICARUS; this methodology integrates in a tiered system qualitative, semi-quantitative and quantitative uncertainty assessment.

Systematic identification of uncertainty sources, according to the stages composing the full chain process, can form the basis for all the tiers of uncertainty assessment.

Organisational coherence is a crucial issue. The steps described in this report must be performed by the same analysts and facilitator(s) who performed the full chain of environmental health impact assessment under the overview of the full chain coordinator. Coherence prevents inconsistencies in the assessment scores when different modules and several teams of analysts are involved. Inconsistency can arise from different identification of uncertainty sources among the different modules composing the full chain, or even from a different focus in the uncertainty assessment.

6.2.1 Identifying uncertainty sources

Uncertainty refers to lack of knowledge which could be reduced by further investigation. Three main sources of uncertainty are recognised in exposure and hazard assessments:

i. **Scenario uncertainty** refers to the description of the context (scenario setting) as a prerequisite for either modelling or measuring experimental data. It includes descriptive errors, aggregation errors, errors in selection of the assessment tier and errors due to incomplete analysis. It often includes the purpose of the environmental health impact assessment and consistency between the scenario definition and the scope and purpose of the assessment.

ii. **Model uncertainty** reflects the limited ability of mathematical models to represent the real world accurately and may also reflect lack of sufficient knowledge. It is principally associated to model boundaries, extrapolation limits, modelling errors and correlation (dependency) errors. It also includes errors due to the implementation of tools and software.

iii. **Parameter uncertainty** refers to data values that are not known with precision due to measurement error or limited observations (sampling error). Sometimes it consists of variability as an inherent property of the heterogeneity or diversity in the parameter, such as parameters expressed as a function of the entire population. Usually, variability cannot be reducible through further investigation. It is also possible for the uncertainty and variability of parameters to be combined.

Classification using the three categories defined above is not as strict as it may seem, and uncertainties may arise in overlapping areas. Thus it is sometimes difficult to reach a clear decision as to whether the uncertainty is related to the scenario, model or parameters because there may be overlaps and even expert opinions may differ. Identification of the sources of uncertainty is a matter of interpretation and depends on the clarity with which the scope and purpose of the assessment are given.

A comprehensive list of relevant sources of uncertainty composing the environmental health impact assessment should be assembled. At this stage, it is not necessary to be concerned about the quantification of individual components; the aim is to be completely clear about what should be considered. In practical terms, all potential sources of uncertainty should be identified at a first instance; judging whether a particular source is important enough to be included in the assessment can be done after evaluating the associated uncertainty at a later stage.

Table 6-1. Overall qualitative assessment matrix on level of uncertainty – here only the sources of uncertainty are filled in

Sources of uncertainty	Dimensions of uncertainty		
Policy scenario			
Conceptual model			
Mathematical model			
Parameters			

In forming the required list of uncertainty sources, it is usually convenient to start with the basic expression(s) used to calculate the result(s) of the impact assessment chain or of each part of the chain as appropriate from intermediate values of the chain or part thereof (Figure 6-1). In addition there may be other parameters that do not appear explicitly in the expression(s) used to calculate the result(s), but which nevertheless affect the results. All these different sources should be included.
The cause and effect diagram as a hierarchical structure culminating in a single outcome can be a very convenient way of listing the uncertainty sources, showing how they relate to each other and indicating their influence on the result(s). It also helps to avoid double counting of sources (Figure 6-2). The 'branches' leading to the outcome are the contributory effects that are potential sources of uncertainty. Each branch may in turn have further contributory effects that will be additional potential sources.

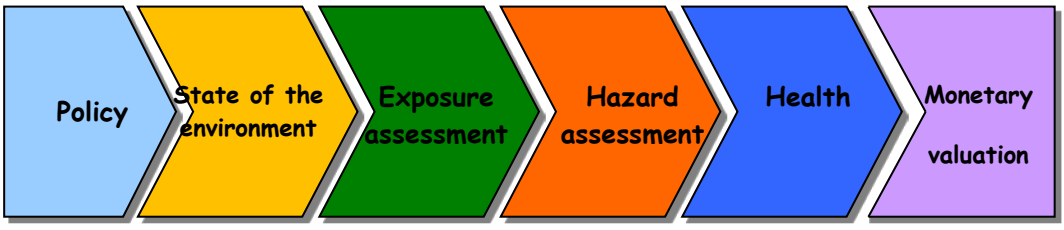

Figure 6-1 Identification of the 'macro' sources of uncertainty

In Figure 6-2, only three modules have been decomposed into three main sources of uncertainty. The initial list is refined to simplify presentation and ensure that effects are not unnecessarily duplicated. The individual sources that have been identified and selected can then be regrouped into the three categories defined above.

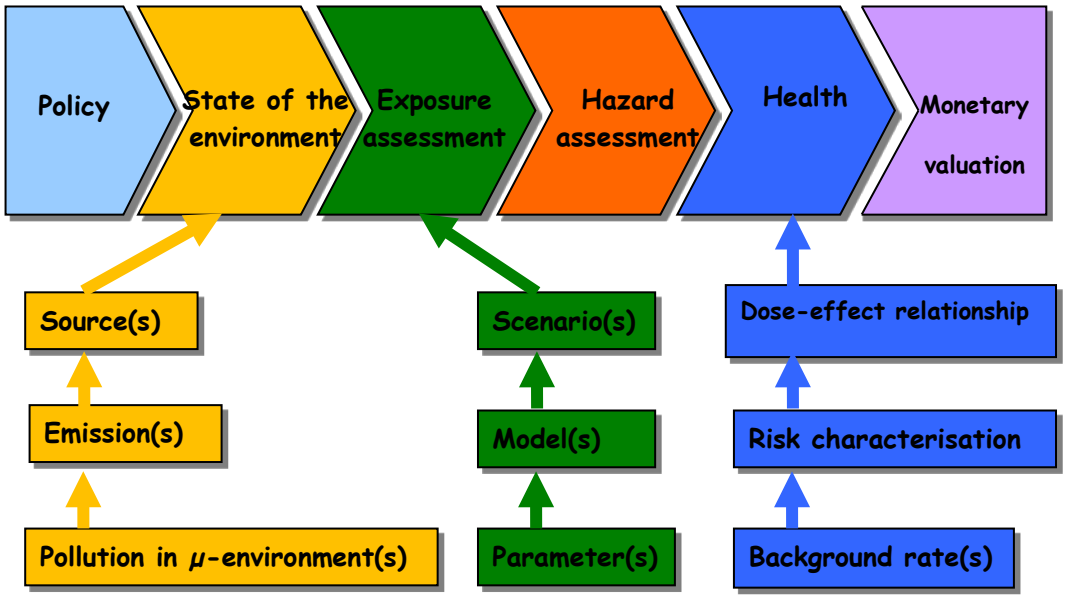

Figure 6-2. Identification of the 'micro' sources of uncertainty

The following text outlines how the qualitative assessment methodology should be interpreted and implemented in the AQ and/or climate change policies. As outlined above, qualitative assessments of uncertainty should be done for all parts of each full chain separately, as well as for the whole full chain.

6.2.2 Assessment of direction and magnitude of uncertainty

First off, the direction of uncertainty, i.e. the tendency of the specific uncertainty source to over- or under-estimate the final result of the specific module (part of the full chain) has to be evaluated. The score that can be assigned can be over (if the specific uncertainty source tends to over-estimates the result) or under (if it tends to under-estimate the result) – see table 6-2. In cases where it is difficult to judge whether the uncertainty would lead to over- or under-estimation of the result, the classification is not known and should be left blank in the qualitative assessment matrix.

Table 6-2. Direction of uncertainty related to the influence of the source of uncertainty on the result(s)

Score	criteria
	direction
Over	the source is judged to over-estimate the final result
Under	the source is judged to under-estimate the final result

The metric of influence evaluates the overall impact of the uncertainty on the final outcome of the assessment. The magnitude of uncertainty is rated low when it is judged that large changes within the source of uncertainty would have only a small effect on the assessment results and when the values

of the data sets needed for the assessment are known. A designation of medium implies that a change within the source of uncertainty is likely to have a moderate effect on the results and the values of the data sets needed for the assessment are unknown (completely or partially). A characterisation of high implies that a small change in the source would have a large effect on results and the values of the data sets needed for the assessment are unknown. The potential assignments and the respective criteria are summarized in Table 6-3.

Table 6-3. Scale of uncertainty related to the magnitude of influence of the source on the result(s)

Score	Criteria	
	magnitude	data sets
Low	known	known
Medium	known	unknown
High	unknown	unknown

6.2.3 Assessment of scientific consistency of the knowledge base

This dimension of qualitative uncertainty characterisation scales the knowledge base uncertainty associated with each identified source using a three level scale: low indicates significant confidence in the data, models and assumptions used and their applicability to the assessment; medium implies that there are some limitations regarding consistency and completeness of the data, models and assumptions used or scientific evidence presented; and high indicates that the knowledge base is extremely limited. In the case of the AQ and climate change policies assessment, only one criterion is to be used for this assessment, namely the scientific consistency of the assessment made in the specific module (part) of the full chain. Table 6-4 outlines the attributes that have to be used to qualify the scientific consistency of the assessment across the board.

Table 6-4 Scale of uncertainty related to the knowledge base

score	criteria
	scientific consistency
Low	extended scientific backing exists
Medium	independent scientific backing exists

High	no scientific backing

The following look-up table can be used to help score the scientific consistency of the knowledge base supporting the assessment consistently.

Table 6-5: Look-up table for assessing uncertainty related to the knowledge base

State of the knowledge base	Uncertainty Category
consistent extensive scientific evidence of many different types from many different sources	low
consistent extensive scientific evidence of a single type or from a single source	low
consistent scientific evidence but all suffering from the same limitation(s)	medium
scientific evidence is mixed; the bulk of it supports the conclusion	medium
consistent scientific evidence for related population/scenario but limited for this situation	medium
scientific evidence is limited or inconsistent	high
no external scientific evidence exists; internal analysis to support	high
no external scientific evidence exists; expert opinion is generally consistent	high
nothing is known	high

6.2.4 Overall qualitative uncertainty assessment matrix

The overall uncertainty of each module (part) of the full chain for each sector should be assessed qualitatively in a summary table that brings together the source and the respective dimensions of uncertainty. In this case, only three dimensions are to be considered, namely the uncertainty direction and level and the scientific consistency of the respective knowledge base; the evaluation can (and should be) accompanied by a few lines of text justifying the choice of the appropriate score among the options given in Table 6-6 below.

As already described in the description of the general methodology given above, the scoring of each source according to a specific dimension thus becomes a cell. The scoring value can be colored to

facilitate reading: red means *high*, orange means *medium*, yellow means *low* and green means *not applicable*.

Table 6-6. Overall qualitative assessment matrix on level of uncertainty

Sources of uncertainty	Dimensions of uncertainty			
	Direction of uncertainty	Level of uncertainty	Appraisal of knowledge base	Justification text
Policy scenario	O/U (if *not known*, then leave blank)	L/M/H	L/M/H	Textual description giving arguments to justify the selection
Conceptual model	O/U	L/M/H	L/M/H	
Mathematical model	O/U	L/M/H	L/M/H	
Parameters	O/U	L/M/H	L/M/H	

Nomenclature:

O/U: Over/Under (denoting the direction of the influence of the specific uncertainty source to the output of the estimation)

L/M/H: Low/Medium/High (denoting the level of the influence of the specific uncertainty source to the output of the estimation – in the case of the 2nd column)

L/M/H: Low/Medium/High (denoting the scientific consistency of the knowledge base underlying the assessment – in the case of the 3rd column).

The above table should be applied in each part (module) of the full chain impact assessment. This will be done by the scientists doing the actual impact assessment calculations. The summary tables constructed for each part of the full chain assessment should be compiled jointly in an overview table by the organisation leading the specific sectorial study. Once this is done, an overall summary table for the full chain assessment of each sectorial study will be developed in order to assess qualitatively the overall uncertainty of the final outcome of the sectorial study.

6.2.5 Conclusion

The levels of variability and uncertainty at each part of an impact assessment of policies and measures addressing AQ and climate change chain should be clearly characterised and reported to those involved in risk management and decision-making. Particular attention should be given to include the assumptions and subjective elements in the sources of uncertainty, clearly describing where contradictions in information occur, how the resultant uncertainty is resolved and justification of the uncertainty scoring. Transparency in these issues aids an informed assessment of uncertainty and enables better communication and understanding of the results by stakeholders.

This framework proposes a systematic and transparent methodology for characterising uncertainty in a qualitative manner and for reporting it. The methodology provides the basis for higher tiers of uncertainty assessment and should be applied consistently across the policies impact assessment.

6.3 Quantitative uncertainty

This qualitative framework illustrated in § 1.2 provides an analytic tool to prioritize uncertainties according to quantitative and qualitative insights in the limitations of available knowledge. Once this qualitative formulation is completed, depending on the data availability and complexity of processes involved, different tiers are considered. Sensitivity tools are used in Tier 2 to examine the contribution of each input source to model output, which helps to identify which uncertainty sources could be managed to reduce overall output uncertainty. Error propagation via Monte Carlo Simulation is implemented in Tier 3 in order to quantify the uncertainty levels through the calculation chain.

Uncertainty in atmospheric transport and dispersion is usually associated with the modelling input (e.g. the emissions and the meteorological input), the parameterization (e.g. the vertical turbulent mixing, the wet scavenging, the horizontal turbulent mixing, the dry deposition) and the practical solution of transport and diffusion equations (Dabberdt and Miller, 2000; Diez et al., 2014; Fox, 1984; Rao, 2005; Reen et al., 2014; Sax and Isakov, 2003; Yegnan et al., 2002). In all cases uncertainty is computed in accordance to the modelling components, since the chemical and physical processes modelled are not linear and some of the uncertainties present may compensate each other. Furthermore, in accordance to the 2008/50/EC Framework Directive any models used should be evaluated in combination with the available monitoring data. This directive does not provide guidelines on how to carry out model evaluations to achieve the specific quality requirements imposed; thus, the development of relevant guidelines was necessary for both modellers and authorities. Several attempts have been made towards the establishment of a set of guidelines based on a number of completed projects including AIR4EU (Denby et al., 2011) and FAIRMODE (Moussiopoulos et al., 2008). The latter is considered as the current reference point for model users and regulators, ensuring that a model meets the quality criteria as required by the EU legislation. The inherent error in prediction could be calculated using the Relative Directive Error (RDE) (Denby et al., 2011) or via the Relative Percentile Error (RPE) (Stern J., 2004).

In addition, the COST Action 732 (Schatzmann M., 2010) guide is widely used for assuring quality in flow and dispersion predictions in urban and industrial areas. According to the guidelines, a number of metrics could be used, including the Fractional Bias (FB), the Geometric Mean bias (MG), the Normalized Mean Square Error (NMSE), the Geometric Variance (VG), the correlation coefficient (R), the fraction of predictions within a factor of two observations (FAC2), the Figure of Merit in Space (FMS or threat score) and the Coefficient of Variation (CV). Another guideline in accordance with the AQ Directive is the use of the “90% of stations requirement”. Uncertainty in the model output is determined from the 90% of the available monitoring stations where outliers are excluded from the calculation. However, this guideline is not suitable for those cases where less than 10 monitoring stations are available; in this case all stations have to be considered. It is also noted that in order to communicate uncertainty, the model used should be adequately validated for the particular application, well documented and all the processes modelled should be suitable for the application, the scale and the type of pollutant investigated. In addition, the quality of the input data has to be sufficient, e.g. the relevant emission sources for the application need to be adequately represented and suitable meteorological data must be available.

Nevertheless characterizing, propagating and analyzing uncertainty in regional dispersion models is critical both to model development and to the effective application of model results in a decision-making setting. If not paid enough attention, it may lead to uncertainty in the model outputs since inputs to such models are inherently uncertain. Alternatively it may not be feasible to use error propagation techniques, as the many thousands of model evaluations required to sample the uncertainty space (e.g., via uniform or Latin hypercube sampling) present an intractable computational burden. A possible solution is the use of a surrogate simulation model in order to substitute regional dispersion models. Once uncertainty in the surrogate model is evaluated it could be updated and combined to other sources of uncertainty using Bayes' theory.

Similarly, errors and biases in exposure models (Fuentes, 2009) lead to poor exposure assessments. Exposure modeling is an important source of uncertainty to environmental health impact assessments (Martuzzi et al., 2003). Sources of error in the exposure models result from variability in human activity data, inaccurate model inputs, simplification in the physical, chemical and biological processes modelled and possible flaws in the conceptual model used. For those cases where input distributions representing variability and uncertainty are combined, the output represents a mixture of variability and uncertainty. In exposure assessments this can be read off as the probability of a randomly chosen individual being exposed to any given level. To this aim, it is recommended that a comprehensive list of relevant uncertainty sources is compiled.

In this context, it is advisable that probabilistic exposure models are used instead of deterministic, addressing thus most, if not all, the possible sources of uncertainty explicitly. In addition, in order to evaluate the effect of changes in ambient concentration to health outcome, a Concentration Response Function (CRF) is used. Here the confidence intervals to the mean CRF should be used to fit probability distributions in accordance to well-known statistical methods including the Kolmogorov–Smirnov, the Anderson–Darling and the Chi-Squared.

6.3.1 Tier 2

At tier 2 the interaction between input parameters and outputs is evaluated and where possible, the number of influencing parameters is reduced. Furthermore, the recommended methods depend on the type of interaction, including linear, non-linear, monotonic and non-monotonic relationships. The variation in model output caused by specific model input is quantified using sensitivity methods (Morris, 1991) or scatter plots.

Global Sensitivity

Local sensitivity analysis techniques suffer from two key shortcomings (WHO, 2008): (a) no simultaneous variation of multiple model inputs and (b) no simulation of the model non-linearities generating interactions between the inputs. In this regard, they are not the ideal choice for assessing the relative significance of different uncertainty sources and their interactions on the overall impact assessment uncertainty. The use of global sensitivity tools is a clear improvement, since it takes into account the entire input variation and apportions output uncertainty to the input factors. This is performed within a probabilistic framework (McRae et al., 1982); (Mara, 2009; Saltelli, 2002; Sobol, 1993b; Tarantola et al., 2006) using the Monte-Carlo methods described in the following section(Isukapalli et al., 1998).

6.3.2 Tier 3

6.3.2.1. Monte Carlo simulation (MC)

At tier 3 advanced methods are recommended including the error propagation and Monte Carlo methods, provided that screening and interactions between input parameters and outputs are evaluated in the previous tier.

A Monte Carlo simulation (MC) involves a large number of drawings (typically hundreds of thousands) from the distribution of the input parameters in the model that are combined to obtain values for the output variables. As many values are available for the output parameters a probability distribution can be evaluated. The outputs from each run of the model are saved and a probability distribution for the output variables is generated. The output can be in the form of a Probability Density Function (PDF) or more often as a cumulative probability distribution, which is the integrated PDF. This allows the estimation of the probability of the occurrence of any particular value or range of values for the output variables. Based on the distribution of the output, the desired levels of probability could be identified, including the high and low end (e.g., 95th and 5th percentile), the central tendency (e.g., mean and median), or any other level of probability. It should be noted that Monte Carlo analysis does not require that probability distribution functions are defined for all input parameters. Where there is no basis for assigning a probability distribution function to particular parameters in multiple parameter models, it is acceptable to keep a fixed value for those parameters while assigning probability density functions to parameters where sufficient information is available. Well known probability density functions are the normal, the triangular, the uniform and the lognormal. The classic form of Monte Carlo simulation usually involves the use of the simple uniform random sample techniques, nevertheless, it is possible to apply other sampling methods to improve the coverage and efficiency of the Monte Carlo methods using the Latin Hypercube Sampling (LHS) (McKay, 1979) or the computationally efficient Stochastic Response Surface methods (SRSMs) (Isukapalli et al., 1998). This usually the case for complex models with correlated parameters. Other alternatives to the Monte Carlo Simulation is the 2D Fuzzy Monte Carlo Simulation (Kentel and Aral, 2005), which can allows for the utilization of incomplete information together with expert judgment.

6.3.2.2. Bayesian Markov Chain Monte Carlo

The Bayesian inference is a method of inference in which Bayes' rule (see equation 1) is used to update the probability estimate for a hypothesis as additional evidence is acquired.

$$P(H|E) = \frac{P(E|H) \cdot P(H)}{P(E)} \quad (1)$$

Where, $P(H|E)$ is the posterior probability of the prior probability of *H* given the evidence E, $P(E|H)$ is likelihood, i.e. the probability of observing *E* given *H*, $P(H)$ is the prior probability of *H* and $P(E)$ is the marginal likelihood.

The Bayesian approach is implemented in Markov Chain Monte Carlo (MCMC) framework which essentially are numerical approximation algorithms. They originated in statistical physics and they were used in Bayesian inference to sample from probability distributions by constructing Markov chains. In Bayesian inference, the target distribution of each Markov chain is a marginal posterior distribution. Each Markov chain begins with an initial value and the algorithm attempting to maximize the logarithm of the un-normalized joint posterior distribution and eventually arriving at each target distribution by multiple iterations. Each iteration is considered a state. A Markov chain is a random process with a finite state-space where the next state depends only on the current state, not on the past one.

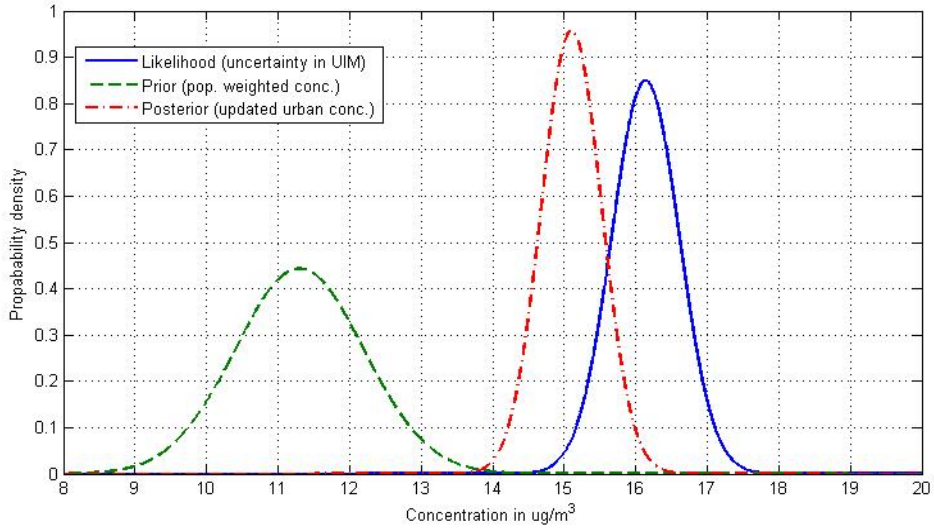

Figure 6-3: example of use of Bayesian inference to update uncertainty

The goal of MCMC is to design a Markov chain such that the stationary distribution of the chain is exactly the distribution that we are interesting in sampling from. The combination of the sampling technique settings leads to existing Metropolis Hasting techniques, including:

- Delayed Rejection Metropolis (DRM)
- Delayed Rejection Adaptive Metropolis (DARM)
- Adaptive Metropolis (AM)
- Componentwise Metropolis (CHM)
- Random-Walk Metropolis (RWM)

Differential Evolution (DE) is a genetic algorithm for numerical global optimization and it is a population Markov Chain Monte Carlo algorithm, in which parallel run for several chains is applied. The combination of DE and MCMC is called Differential Evolution Monte Carlo (DEMC). DEMC provides solutions to the choosing and the orientation of the jumping of the distribution that is an important practical problem in random walk Metropolis. In fact DEMC algorithm is based on a Metropolis Hasting and it is combined with a genetic algorithm called Differential Evolution (DE) with multiple chains and

each chain learn from another parallel chain. The crucial idea behind DE is an innovated generation of parameter vectors. DE adds a weighted difference vector between two population members in order to generate vectors. The vector yields an objective function value. Then the value is compared with the predetermined population and if the resulting value is lower than the existent, the new vector replaces the compared vector. Moreover, the evaluation of each generation can be done with the best parameter vector in order to retain track of progress during the minimization process.

6.3.2.1.1 MCMC algorithms

The goal of MCMC is to design a Markov chain such that the stationary distribution of the chain is exactly the distribution that we are interesting in sampling from. The combination of the sampling technique settings leads to existing Metropolis Hasting techniques. Table 6-7 presents the available MCMC algorithms based on Metropolis Hasting sampling that can been used.

Table 6-7. MCMC algorithms based on Metropolis Hasting

MCMC algorithms	Update mode:	Reference
Delayed Rejection Metropolis (DRM)	Multivariate	(Mira 2001)
Delayed Rejection Adaptive Metropolis (DARM)	Multivariate	(Haario, Laine et al. 2006)
Adaptive Metropolis(AM)	Multivariate	(Haario, Saksman et al. 2001)
Componentwise Metropolis (CHM)	Componentwise	(Haario, Saksman et al. 2005)
Random-Walk Metropolis (RWM)	Componentwise	(Gilks and Roberts 1996)

The Delayed Rejection Metropolis (DRM or DR) algorithm is a Random-Walk Metropolis (RWM) (Mira 2001). Whenever a proposal is rejected, the DRM selects one or more alternate proposals and corrects for the probability of this conditional acceptance. The delaying rejection enforces the decreased autocorrelation in the chains and the algorithm is encouraged to move. The additional calculations increase the computational cost of each iteration of the algorithm in which the first set of proposals is rejected, but the major benefit is the faster convergence to the optimal solution.

The Delayed Rejection Adaptive Metropolis (DRAM) algorithm is merely the combination of both Delayed Rejection Metropolis (DRM) and Adaptive Metropolis (AM) (Haario, Laine et al. 2006). DRAM has been demonstrated to be robust in extreme situations where DRM or AM fail separately. Haario et al. (2006) present an example involving ordinary differential equations in which least squares could not find a stable solution, and DRAM did well.

The Adaptive Metropolis (AM) algorithm of Haario et al. (2001) is an extension of Random-Walk Metropolis (RWM) that adapts based on the observed covariance matrix from the history of the chains. The algorithm is specified under adaptation and periodicity. Thus, the beginning of the iteration and the frequency in the periodicity in adaption have to be set. The adaption has to be controlled and immediate adaption has to be avoided since the algorithm is based on the observed covariance matrix of historical and accepted samples. Hence, a valid covariance matrix before adaptation has to be composed with a large number of samples. However, at the beginning of the algorithm, a small covariance matrix is commonly used to encourage a high acceptance rate.

The Componentwise Metropolis (CHM) is based on the Single Component Adaptive Metropolis (SCAM) that has been developed by Hario et al. (2005) and on the single component Metropolis – Hasting algorithm. In the SCAM the adaption is performed component by component. The chain is no more Markovian, but it remains ergodic. The SCAM can be used in many moderately high dimensional problems. Also, the algorithm does not need detailed prior knowledge of the target distribution and it can be used in numerous problems typically solved using pre-runs and hand tuning (Haario, Saksman et al. 2005). Also, the algorithm resembles basic single component Metropolis algorithm with Gaussian proposal distributions, the only exception being that the variances of the one-dimensional proposal distributions depend on time and the variance is been computing by a simple recursive formula. Moreover, in high dimension the updating of the proposal distribution performed demands only computations of component-wise variances. Hence, the additional computation brought in by the adaptiveness is negligible. Additionally, component-wise proposals usually indicate that a proposal is made for each parameter, without considering correlation. In case of that parameters are correlated, the problem of the distribution is faced with the rotation of the proposal distribution. Thus, the covariance matrix of the chain is computed and the principal vector direction is determined and it is used as sampling directions in the SCAM-algorithm. After the burn-in period of the algorithm, the proposal direction is fixed and the sampling is continued by only updating the size of the one-dimensional Gaussian proposal distribution. Hence, the SCAM is characterized as fully automatic algorithm. SCAM is widely applicable and general-purpose algorithm. It is appropriate to be performed to models with a small to medium number of parameters since the proposal covariance matrix grows with the number of parameters and the computation cost simultaneous increases.

The random walk algorithm of Metropolis is known to be an effective Markov chain Monte Carlo method for many diverse problems (Metropolis, Rosenbluth et al. 1953). The proposed Random-Walk Metropolis (RWM) is a multivariate extension of Metropolis-within-Gibbs (MWG) (Gilks and Roberts 1996). RWM is an algorithm the initials specification are not necessary though blockwise sampling. In fact RWM is a generic algorithm to draw a sample from a d-dimensional target distribution from a probability density function.The optimal scale of the proposal covariance is based on the asymptotic limit of infinite-dimensional Gaussian target distributions that are independent and identically-distributed (Gelman, Roberts et al. 1996). In case of multiple parameters the existence of correlations occurrences is very common. Hence, MCMC algorithms attempt to estimate multivariate proposals from a multivariate normal distribution taking into account correlations through the covariance matrix. The convergence of the algorithm is related with the proposal density. A small variance leads to slowly converge and conversely, if the variance is too large, the Metropolis algorithm will reject too high a proportion of its proposed moves (Roberts, Gelman et al. 1997).

6.3.2.1.1.1 Differential Evolution Monte Carlo

Differential Evolution (DE) is a genetic algorithm for numerical global optimization and it is a population Markov Chain Monte Carlo algorithm, in which parallel run for several chains is applied (Ter Braak 2006). The combination of DE and MCMC is called Differential Evolution Monte Carlo (DEMC) and the field has been explored among others by Liang and Wong (2001) Liang (2002) and Laskey and Myers (2003). DEMC provides solutions to the choosing and the orientation of the jumping of the distribution that is an important practical problem in random walk Metropolis. In fact DEMC algorithm is based on a Metropolis Hasting and it is combined with a genetic algorithm called Differential Evolution (DE) with multiple chains and each chain learn from another parallel chain. The crucial idea behind DE is an innovated generation of parameter vectors. DE adds a weighted difference vector between two

population members in order to generate vectors. The vector yields an objective function value. Then the value is compared with the predetermined population and if the resulting value is lower than the existent, the new vector replaces the compared vector. Moreover, the evaluation of each generation can be done with the best parameter vector in order to retain track of progress during the minimization process. The DE is described in detail by Storn and Price (1995) (1997) and the adaption of DE in MCMC is described and proofed by Ter Braak (2006).

6.3.2.1.2 DEMC algorithms

The applied DEMC algorithm is based on the Ter Braak (2006) algorithm.

Table 6-8 MCMC algorithms based on Differential Evolution method

MCMC algorithms	Update mode:	Reference
Differential Evolution Monte Carlo (DEMC)	Multivariate	(Ter Braak 2006)

DEMC is similar with Metropolis-within-Gibbs (MWG) but the main different consist in that DEMC updates by chain. The algorithm is specified under the number of chain that should be at least three and the thinning factor. The thinning factor provides the reduction of storage requirements and enhances the convergence of the chain to posterior distribution. In particular, the sampling is realized randomly and without replacement from a possibly thinned chain. Moreover, an adaptive step size can be used (ter Braak and Vrugt 2008) with the same contribution as it has been described to section 0. Also the snooker update fraction (Gilks, Roberts et al. 1994, Liang and Wong 2001, ter Braak and Vrugt 2008) can been specified providing to the sampler the ability to update along each coordinate axis in turn one axis at a time, with the specificity that this axis does not need to run parallel to the coordinate axes. Finally, it can be set the randomly uniform offset distribution that added to the creation of the DEMC proposal distribution.

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