6. Particulate Matter Formation

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The impact assessment method for assessing damage to human health due to primary PM2.5 and PM2.5 precursor emissions is described based on Van Zelm et al. (2016).

6.1. Areas of protection and environmental mechanisms covered

The cause and effect pathway (Figure 6.1) of particulate matter formation starts with an emission of NO_x , NH₃, SO₂, or primary PM_{2.5} to the atmosphere, followed by atmospheric fate and chemistry in the air; NO_x, NH₃, and SO₂ are transformed in air to secondary aerosols. Subsequently, PM_{2.5} can be inhaled by the human population, leading to an increased number of mortality cases and final damage to human health.

Figure 6.1: Cause and effect pathway from primary PM2.5 and PM2.5 precursor emissions to damage to human health

The intake of a pollutant by the population is described by intake fractions (iF, in kg intake per kg emission) that quantify the relationship between an emission and intake (Van Zelm et al. 2008). Here, a global chemical transport model was applied to determine human intake fractions for 56 emission and receptor regions. Second, region-specific mortality rates, background concentrations and years of life lost were used to determine human health effect factors. Here, we included cardiopulmonary and lung cancer mortality due to particulate matter with a diameter of less than 2.5 μ m (PM_{2.5}) for two reasons: first, these contribute by far the most to overall disability adjusted life years (DALYs) for these two pollutants (as e.g. shown in previous research (Van Zelm et al. 2008)), and second, for these the most up-to-date and least uncertain data related to relative risks and years of life lost are available (see e.g. Anenberg et al. 2010, Friedrich et al. 2011, Murray et al. 2012, WHO 2013).

6.2. Calculation of the characterization factors at endpoint level

The endpoint characterization factors (CFs) for human health damage due to particulate matter formation caused by emitted substance x in world region i (CFx,i in DALY∙kg-1) are defined as the yearly change in Disability Adjusted Life Years (DALY) of all inhabitants (dDALY in yr∙yr-1) due to a change in emission of substance x in source region i (dMx,i in kg∙yr-1). This CF for human health damage is composed of a dimensionless intake fraction (iFx, $i\rightarrow j$), providing the population intake of PM2.5 in receptor region j (in kg/yr) following an emission change of substance x in source region i (in kg/yr), an effect factor (EFe), describing the cases of health effect *e* per kg of inhaled PM2.5, and a damage factor (DFe), which describes the years of life lost per case of health effect *e*. In equation this reads:

$$
CF_{x,i} = \sum_{j} \left(\left(iF_{x,i \to j} \right) \sum_{e} \left(EF_{e,j} \cdot DF_{e,j} \right) \right)
$$

Equation 6.1.

6.2.1. From emission to human intake

The intake fraction is determined as the change in exposure to PM_{2.5} in region j (dEXP_i), due to a change in emission of substance x $(dM_{x,i})$. dEXP was retrieved by multiplying the change in concentration of PM_{2.5} in each receptor region (dC_i) with the population (N_i) in the receptor region j and the average breathing rate per person (BR) of 4745 m³·yr⁻¹ (13 m³·d⁻¹ as recommended by USEPA (1997):

$$
iF_{x,i\to j} = \frac{dEXP_j}{dM_{x,i}} = \frac{dC_j \cdot N_j \cdot BR}{dM_{x,i}}
$$
 Equation 6.2.

Population numbers (year 2005) were taken from the United Nations (2011). Since all data for the effect factor are based on the population ≥ 30 years of age, the population number was adjusted for the population share ≥ 30 years of age in 2005 (United Nations 2011) assuming no effects for younger people.

The emission–concentration sensitivities matrices for emitted precursors and relevant end pollutants (or pollutant metrics) from the global source-receptor model TM5-FASST (FAst Scenario Screening Tool for Global Air Quality and Instantaneous Radiative Forcing), based on perturbation runs with TM5 (Van Dingenen et al. 2009; Krol et al. 2005) were used to derive the change in ambient concentration of a pollutant after the emission of a precursor. TM5 is a global chemical transport model hosted by the European Commission Joint Research Center (JRC). TM5-FASST takes into account spatial features at the emission site as well as dispersion characteristics for the whole world. In this model, the world is divided into 56 emission source regions. The regions correspond to countries or a group of countries (see Table 6.1). The TM5 model output consists of the change in concentration for each region, derived from gridded 1°×1° concentration results, following a change in emission. This change is determined by lowering the year 2000 emissions (Lamarque et al. 2010) by 20% for each of the 56 source regions sequentially. The emission-normalized differences in pollutant concentration between the unperturbed and perturbed case, aggregated over each receptor region, are stored as the emission – concentration matrix elements. This procedure was performed for each (precursor) substance. i.e. $NH₃$, NO_x, SO₂, and primary PM_{2.5}.

6.2.2 From human intake to human health damage

The human effect factor (dINC/dEXP) for health effect e caused by $PM_{2.5}$ in receptor region j, representing the change in disease incidence due to a change in exposure concentration in ambient air, was determined by dividing the concentration-response function (CRF in m³·yr⁻¹·kg⁻¹) by the breathing rate BR ($m^3 \cdot yr^{-1}$) (Gronlund et al. 2015) (equation 6.3).

$$
EF_{e,j} = \frac{dINC_j}{dEXP_j} = \frac{CRF_{e,j}}{BR}
$$
\nEquation 6.3

Region-specific CRFs were calculated as follows (equation 6.4):

$$
CRF_{e,j} = \frac{(RR_e - 1) \cdot MR_{e,j}}{(RR_e - 1) \cdot C_j + 1}
$$
 Equation 6.4

where RR_e is the relative risk to obtain health effect e due to exposure to PM_{2.5} (per µg⋅m⁻³), MR_{e,j} is the mortality rate for health effect e in region j (deaths/person/yr), and C_i is the yearly average background concentration of PM_{2.5} in a region (μ g·m⁻³).

We followed recommendations for RRs by Anenberg et al. (2010) and Friedrich et al. (2011), who focus on the world and Europe respectively, based on North American cohort studies. RRs for cardiopulmonary (1.013 per μ g⋅m^{−3}), and lung cancer (1.014 per μ g⋅m^{−3}) mortality from Krewski et al. (2009) were used. This study is the latest reanalysis of the American Cancer Society (ACS) PM_{2.5} studies (see e.g. Pope et al. 2002) and has by far the largest population of the available PM2.5 cohort studies, and this latest update involves better exposure data, longer follow-up (i.e. more deaths) and more comprehensive statistical analyses.

Mortality rates per health effect (year 2005) were taken from the World Health Organization (WHO 2015a), and simulated background concentrations per region for the year 2000 were taken from the TM5-CTM reference run with the Lamarque et al. (2010) year 2000reference emission scenario.

The Damage factor De,j is defined as the Disability Adjusted Life Years (DALY) associated to the health effect e per incidence case, which were estimated per receiving region j from the world health organization (WHO) world health estimates, year 2012 (WHO 2015b):

$$
DF_{e,j} = \frac{dDALY_{e,j}}{dINC_{e,j}}
$$
 Equation 6.5

For the DALY no discounting was included and uniform age weights were applied.

6.3. Uncertainties

The CFs were derived from emission-concentration sensitivities (dC/dM) obtained from a 20% emission perturbation. For a limited number of representative source regions the dC/dM coefficients were calculated for large perturbations of inorganic pollutants (-80%, +100%) and compared to the extrapolated 20% perturbation (Van Zelm et al. 2016). Relatively small maximum absolute deviations were seen, up to 5%.

TM5 includes the various emission stack heights. However, it does not differentiate between them to derive the CFs. Stack-height specific intake fractions can differ 2 orders of magnitude, as shown by Humbert et al. (2011).

The native TM5 resolution of 1x1 degree at the receptor level does not reflect possible sub-grid gradients in PM and ozone that are expected when large population gradients occur within the grid (like isolated urban areas), leading to a possible underestimation of exposure. Van Zelm et al. (2016) compared area-weighted and population-weighted concentration and found that, aggregated at the level of the receptor regions used in this study, the largest deviations in exposure concentrations were found for Australia, Philippines, and Japan with population-weighted concentrations 12-19% higher compared to area-weighted concentrations. For all other regions, the deviation (over- or underestimation) between area and population-weighted PM2.5 was less than 10%.

In this research, only effects of lung cancer and cardiopulmonary mortality were included, neglecting morbidity due to, e.g. COPD and chronic bronchitis. The choice was made to include mortality with the largest share to human health damage caused by PM2.5, and of which the most certain epidemiological data are available. Due to this, total human health damage is slightly underestimated. Van Zelm et al. (2008) showed, for example, that 99% of DALYs due to PM10 is caused by chronic mortality.

6.4. Value choices

6.4.1. Time horizon

For human health damage due to fine dust, time horizon is not of importance as only short-living substances are involved.

6.4.2. Level of robustness

As outlined by De Schryver et al. (2011), evidence for effects from primary PM is available (Pope et al. 2009) and therefore considered robust. There is evidence concerning human health risks at ambient concentrations of secondary PM from SO₂, NO_x and NH₃ is available. However, the level of effect is still under debate (De Schryver et al. 2011). Reiss et al. (2007) do show that there are more studies indicating health effects from secondary PM from $SO₂$ than from NO_x or $NH₃$.

6.5. Resulting characterization factors

Figure 6.2 shows the region-specific characterization factors for human health for $PM_{2.5}$ precursor emissions. Lowest factors were obtained for emissions of NO_x on the Southern Hemisphere, while largest factors were obtained for primary $PM_{2.5}$ emissions in Central Asia. The emission weighted average for the world for PM_{2.5} is 6.29⋅10⁻⁴ DALY⋅kg⁻¹ (with a minimum of 9.40⋅10⁻⁶ and a maximum of 4.02∙10⁻³ DALY∙kg⁻¹). The emission weighted average for the world for NH₃ is 1.61∙10⁻⁴ DALY∙kg⁻¹ (3.30∙10⁻⁶ to 1.34∙10⁻³ DALY∙kg⁻¹), for NO_x 7.62∙10⁻⁵ DALY∙kg⁻¹ (4.43∙10⁻⁷ to 3.65∙10⁻⁴ DALY∙kg⁻¹), and for SO₂ 1.83⋅10⁻⁴ DALY⋅kg⁻¹ (1.40⋅10⁻⁵ to 9.45⋅10⁻⁴ DALY⋅kg⁻¹). For each country the region-specific factor was allocated to it. Table 6.1 provides the characterization factors for each country. Table 6.2 provides the continent-specific emission weighted average characterization factors.

Primary PM2.5 NH₃

 NO_x SO₂

Figure 6.2.: Characterization factors for human health damage caused by fine dust formation (10-6 DALY∙kg -1) (Taken from Van Zelm et al. 2016).

South Africa	RSA	3.15E-04	5.53E-05	2.11E-06	4.64E-05
South Korea	COR	6.96E-04	5.17E-04	2.71E-05	1.45E-04
Spain	ESP	6.10E-04	1.06E-04	6.34E-05	1.52E-04
Sri Lanka	NDE	3.36E-03	1.73E-04	3.16E-04	8.32E-04
Sudan	EAF	1.41E-04	7.92E-06	2.67E-06	1.08E-04
Suriname	RSAM	7.11E-05	1.98E-05	3.52E-06	6.52E-05
Swaziland	RSA	3.15E-04	5.53E-05	2.11E-06	4.64E-05
Sweden	SWE	3.10E-04	1.11E-04	9.09E-05	7.05E-05
Switzerland	CHE	1.48E-03	1.34E-03	2.26E-04	2.07E-04
Syria	MEME	7.55E-04	1.96E-04	3.21E-05	1.78E-04
Sao Tomo and Principe	WAF	2.44E-04	1.48E-05	3.25E-06	9.31E-05
Taiwan	TWN	3.51E-04	2.25E-04	9.01E-06	1.31E-04
Tajikistan	RIS	1.08E-03	3.55E-04	4.20E-05	1.48E-04
Tanzania, United Republic of	EAF	1.41E-04	7.92E-06	2.67E-06	1.08E-04
Thailand	THA	2.31E-04	1.11E-05	9.91E-06	8.83E-05
Togo	WAF	2.44E-04	1.48E-05	3.25E-06	9.31E-05
Tonga	PAC	1.14E-05	6.92E-06	2.52E-06	1.02E-04
Trinidad and Tobago	RCAM	1.58E-04	2.46E-05	6.85E-06	4.72E-05
Tunisia	NOA	6.63E-04	6.39E-05	3.39E-05	1.58E-04
Turkey	TUR	8.14E-04	2.28E-04	1.45E-04	1.99E-04
Turkmenistan	RIS	1.08E-03	3.55E-04	4.20E-05	1.48E-04
Uganda	EAF	1.41E-04	7.92E-06	2.67E-06	1.08E-04
Ukraine	UKR	1.34E-03	3.91E-04	1.80E-04	1.71E-04
United Arab Emirates	GOLF	5.63E-04	1.44E-04	4.71E-05	2.09E-04
United Kingdom	GBR	1.27E-03	3.99E-04	6.31E-05	1.07E-04
United States	USA	4.55E-04	1.53E-04	1.41E-05	5.29E-05
Uruguay	ARG	2.13E-04	6.44E-06	4.43E-07	6.34E-05
Uzbekistan	RIS	1.08E-03	3.55E-04	4.20E-05	1.48E-04
Vanuatu	PAC	1.14E-05	6.92E-06	2.52E-06	1.02E-04
Venezuela	RSAM	7.11E-05	1.98E-05	3.52E-06	6.52E-05
Vietnam	VNM	9.61E-04	7.21E-05	1.43E-05	2.11E-04
Western Sahara	NOA	6.63E-04	6.39E-05	3.39E-05	1.58E-04
Yemen	GOLF	5.63E-04	1.44E-04	4.71E-05	2.09E-04
Zambia	SAF	6.26E-05	4.08E-06	8.46E-07	4.59E-05
Zimbabwe	SAF	6.26E-05	4.08E-06	8.46E-07	4.59E-05

Table 6.2: Continent-specific endpoint characterization factors for human health damage due to particulate matter formation (DALY∙kg-1) (Van Zelm et al. 2016).

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