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Regionalized life cycle impact assessment of air pollution on the global scale: damage to human health and vegetation

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Abstract

We developed regionalized characterization factors (CFs) for human health damage from particulate matter (PM2.5) and ozone, and for damage to vegetation from ozone, at the global scale. These factors can be used in the impact assessment phase of an environmental life cycle assessment. CFs express the overall damage of a certain pollutant per unit of emission of a precursor, i.e. primary PM2.5, nitrogen oxides (NOx), ammonia (NH3), sulfur dioxide (SO2) and non-methane volatile organic compounds (NMVOCs). The global chemical transport model TM5 was used to calculate intake fractions of PM2.5 and ozone for 56 world regions covering the whole globe. Furthermore, region-specific effect and damage factors were derived, using mortality rates, background concentrations and years of life lost. The emission-weighted world average CF for primary PM2.5 emissions is 629 yr·kton⁻¹, varying up to 3 orders of magnitude over the regions. Larger CFs were obtained for emissions in central Asia and Europe, and smaller factors in Australia and South America. The world average CFs for PM2.5 from secondary aerosols, i.e. NOx, NH3, and SO2, is 67.2 to 183.4 yr·kton⁻¹. We found that the CFs for ozone human health damage are 2 to 4 orders of magnitude lower compared to the CFs for damage due to primary PM2.5 and PM2.5 precursor emissions. Human health damage due to the priority air pollutants considered in this study was 1.7·10⁻² yr·capita⁻¹ worldwide in year 2010, with primary PM2.5 emissions as the main contributor (62%). The emission-weighted world average CF for ecosystem damage due to ozone was 2.5 km²·yr·kton⁻¹ for NMVOCs and 8.7 m²·yr·kg⁻¹ for NOx emissions, varying 2-3 orders of magnitude over the regions. Ecosystem damage due to the priority air pollutants considered in this study was 1.6·10⁻⁴ km²·capita⁻¹ worldwide in 2010, with NOx as the main contributor (72%). The spatial range in CFs stresses the importance of including spatial variation in life cycle impact assessment of priority air pollutants.

Keywords: mortality; characterization factor; intake fraction, damage factor; photochemical ozone; PM2.5
Abstract graphic:

Highlights

- Years of life lost of fine dust and ozone in 56 world regions were determined
- Spatially explicit ozone damage to natural vegetation worldwide was also quantified
- Primary PM2.5 emissions contribute 62% to human health damage by fine dust and ozone
- NO, dominantly contributes (72%) to ozone damage in natural vegetation worldwide

1. Introduction

Air pollution causing primary and secondary aerosols and ozone in the atmosphere can have a substantial negative impact on human health, ranging from respiratory symptoms to hospital admissions and death (Bell et al. 2005; WHO 2006; Friedrich et al. 2011; Jerrett et al. 2009; Burnett et al. 2014; Lelieveld et al. 2015). Additionally, ozone can have a negative impact on vegetation, including reduction of growth and seed production, acceleration of leaf senescence and a reduced ability to withstand stressors (see e.g., Ashmore 2005; Gerosa et al. 2015).

To quantify the damage per unit of emission of a certain air pollutant, so called characterization factors (CFs) can be used. These factors can then be applied in the impact assessment phase of an environmental life cycle assessment (LCA) to quantify the damage caused by emissions due to activities connected to a product or service. This study will focus on human health damage due to fine particulate matter (PM2.5) and photochemical ozone formation (in years of life lost per kg of substance emitted), as well as ecosystem damage due to photochemical ozone formation (in area- and time-weighted potentially affected fraction of species per kg of substance emitted). For the calculation of CFs,
information on the intake of (human) or exposure to (vegetation) a pollutant is needed, as well as the
damage related to that intake or exposure.

The characteristics of the emission location (source region) determines where and in which
content a pollutant ends up (receptor regions), and thereby influences the exposure of receptors
in the corresponding regions. The characteristics of the receptors in each region, such as age
distribution, influence the total human health damage followed by intake or exposure. The differences in
vegetation types at the receptor region cause variation in the damage to vegetation from ozone
exposure.

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). Humbert
et al. (2011) provide iFs for three different emission location archetypes (urban, rural, remote), making a
distinction between three different stack heights (high-stack, low-stack and ground-level). Furthermore,
Apte et al. (2012) developed globally applicable iFs that were relevant for the urban environment and
pollutants emitted at ground level. However, using actual local characteristics for weather conditions
and population instead of archetypes will provide more precise results. Van Zelm et al. (2008) used a
source receptor model for this purpose. However, the developed intake fractions were for Europe only,
with no distinction between countries. Human effect factors are generally developed for one continent
only, e.g. Europe (Hofstetter 1998; Krewitt et al. 2001; Van Zelm et al. 2008) or North America (Gronlund
et al. 2015), while ecosystem effect factors for plant species were developed for Europe (Van Goethem
et al. 2013b). Finally, (Tang et al. 2016a, b) derived human health characterization factors for PM2.5 and
photochemical ozone, respectively, based on a global chemistry transport model. The world was divided
into 10 regions only and the effect factor was based on one world-generic concentration-response
function, while ammonia (NH₃) was not included as a precursor substance.

To provide more spatial detail on the global scale for both damage to human health and vegetation
of air pollution, the aim of this paper was to develop a set of globally applicable and spatially explicit
characterization factors for human health damage from particulate matter and ozone, and for damage to
vegetation from ozone. For this, we consistently applied one global chemical transport model and
determined human intake fractions and ecosystem fate factors for 56 emission and receptor regions.
Region-specific mortality rates, background concentrations and years of life lost were used to determine
human health effect factors. We included cardiopulmonary and lung cancer mortality due to PM2.5, and
respiratory mortality due to ozone.
2. Material and methods

2.1 Human health damage

Characterization factor

CFs for human health damage caused by substance x emitted in world region i (CF\(_{k,x,i}\) in yr\(^{-1}\)-kton\(^{-1}\)) are defined as the yearly change in years of life lost (YLL) of all inhabitants (dYLL in yr\(^{-1}\)) caused by pollutant k due to a change in emission of substance x in source region i (dM\(_{x,i}\) in kton\(\cdot yr\(^{-1}\)). This CF for human health damage is composed of a dimensionless intake fraction (iF\(_{k,x,i-j}\)), providing the population intake of pollutant k in receptor region j (in kg/year) following an emission change of precursor x in source region i (in kg/year), an effect factor (EF\(_{k,e}\)), describing the cases of health effect e per kg of inhaled pollutant k, and a damage factor (DF\(_e\)), which describes the years of life lost per case of health effect e. In equation this reads:

\[ CF_{HH,k,x,j} = \sum_j \left( iF_{k,x,i-j}\right) \cdot \sum_e \left( EF_{e,k,j} \cdot DF_{e,k,j}\right) \]  

(1)

Intake fraction

The intake fraction is the change in exposure of pollutant k in region j (dEXP\(_{k,j}\)), due to a change in emission of precursor x (dM\(_{x,i}\)). dEXP can be calculated by multiplying the change in concentration of pollutant k in each receptor region (dC\(_{k,j}\)) with the population (N\(_j\)) in the receptor region j and the average breathing rate per person (BR) of 4745 m\(^3\)-yr\(^{-1}\) (13 m\(^3\)-d\(^{-1}\) as recommended by U. S. EPA (1997):

\[ iF_{k,x,i-j} = \frac{dEXP_{k,j}}{dM_{x,i}} = \frac{dC_{k,j} \cdot N_j \cdot BR \cdot dM_{x,i}}{dM_{x,i}} \]  

(2)

For ozone, the “Maximal 6-month running average of 1-hr daily maximum ozone concentration” (M6M in \(\mu g\cdot m^{-3}\)) was used as metric of the concentration change. Corresponding to this, only 1/24\(^{th}\) of the breathing rate and only 6 months of the year were taken into account to be consistent with the M6M metric (98.85 m\(^3\)/yr). Population numbers (year 2005) were taken from United Nations (2011). Since all data for the effect factor are based on the population \(\geq 30\) years of age, the population number was adjusted for the population share \(\geq 30\) years of age in 2005 (United Nations 2011) assuming no effects for younger people.

To derive the change in ambient concentration of a pollutant after the emission of a precursor, we use 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). TM5 is a global chemical transport model hosted by the European Commission Joint Research Center (JRC) and applied before by Rao et al. (2012) and Chafe et al. (2014). 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 source and identical 56 receptor regions. The regions correspond to countries or a group of countries (see Table A1). The TM5 model output consists of the change in concentration for each receptor region, derived from gridded 1°×1° concentration results, following a change in anthropogenic emissions and 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, NOₓ, SO₂, and primary PM₂.₅ for PM₂.₅, and NOₓ and non-methane volatile organic compounds (NMVOCs) for ozone.

**Effect factor**

The human effect factor (dINC/dEXP) for health effect e caused by pollutant k in receptor region j, representing the change in disease incidence due to a change in exposure, was determined by dividing the concentration-response function (CRF in m³·yr⁻¹·kg⁻¹) by the breathing rate BR (m³·yr⁻¹) (Gronlund et al. 2015):

\[
EF_{e,k} = \frac{dINC_{e,k}}{dEXP_{k,i}} = \frac{CRF_{e,k,j}}{BR}
\]  

(3)

Region-specific CRFs were calculated as follows:

\[
CRF_{e,k,j} = \frac{(RR_{e,k-1})MR_{e,j}}{(RR_{e,k-1})C_{k,j+1}}
\]  

(4)

where RR_{e,k} is the relative risk to obtain health effect e due to exposure to pollutant k (per μg·m⁻³), MR_{e,j} is the mortality rate for health effect e in region j (deaths/person/yr), and C_{k,j} is the yearly average background concentration of pollutant k in a region (μg·m⁻³).

Here, we included cardiopulmonary and lung cancer mortality due to PM2.₅, and respiratory mortality due to ozone for two reasons: first, these contribute by far the most to overall human health damage 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).
Regarding the RR, we followed recommendations by Anenberg et al. (2010) and Friedrich et al. (2011), who focus on the world and Europe respectively, based on North American cohort studies. For PM2.5, RRs for cardiopulmonary (1.013 per μg·m⁻³) and lung cancer (1.014 per μg·m⁻³) mortality from Krewski et al. (2009) were used. This study is the latest reanalysis of the American Cancer Society (ACS) PM₂.₅ 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.

For ozone, an RR of 1.004 per μg·m⁻³ for death from respiratory disease based on data of daily 1-hr maximum ozone levels found by Jerrett et al. (2009) in an ACS cohort study of U.S. adults ≥ 30 years of age was used. Although many daily time-series epidemiology studies demonstrate short-term ozone mortality impacts (Anderson et al. 2004; Bell et al. 2005), Jerrett et al. (2009) provide the first clear evidence for long-term impacts.

Mortality rates per health effect (year 2005) were taken from the World Health Organization (WHO 2015a), and simulated background concentrations (M6M) per region for the year 2000 were taken from the TM5 chemical transport model reference run with the Lamarque et al. (2010) year 2000 reference emission scenario.

### Damage factor

The Damage factor \( D_{e,k,j} \) is defined as the years of life lost (YLL) associated to the health effect \( e \) per incidence case, which were estimated per region \( j \) from the world health organization (WHO) world health estimates, year 2012 (WHO 2015b):

\[
DF_{e,k,j} = \frac{dYLL_{e,k,j}}{dINC_{e,k,j}} \tag{5}
\]

For the YLL no discounting was included and uniform age weights were included. Table A1 in the appendix provides an overview of all region specific input data for the intake fraction and human health effect and damage factors.

### 2.2. Ecosystem damage

#### Characterization factor

CFs for ecosystem damage due to ozone formation are defined as the area-integrated change in Potentially Affected Fraction (PAF) of forest and natural grassland species due to a change in emission of substance \( x \) (in km²·yr·kton⁻¹). The CF consists of a Fate Factor (FF\(_{x,i\rightarrow e}\), unit: ppm·h·yr·kton⁻¹), quantifying
the relationship between the emission of precursor substances in region i and ozone exposure in 
receiving grid cell g, and an Effect Factor \( EF_{n,k,j} \) in \( \text{km}^2 \cdot \text{ppm}^{-1} \cdot \text{h}^{-1} \), quantifying the relationship between 
ozone exposure and the damage to natural vegetation n (forest and grassland). In equation this reads:

\[
CF_{ECO,k,x,j} = \sum_g \sum_n \left( EF_{k,x,j,g} \cdot EF_{n,k,g} \right)
\]

\( (6) \)

\[ \]

\( Fate \) factor

To determine the ecosystem fate factor, the AOT40, i.e. the sum of the differences between the 
hourly mean ozone concentration and 40 ppb during daylight hours over the relevant growing season in 
ppm·h, was used as metric of the cumulative concentration change, and derived with the TM5-FASST 
model. We chose to apply this indicator as it is a commonly used indicator to determine the risk for 
species groups or plant communities to ozone (see e.g. Musselman and Lefohn 2007; LRTAP 2004). The 
fate factor then represents the sum in the change in AOT40 in grid cell g due to a change of emission of 
precursor x in source region i (Van Goethem et al. 2013b):

\[
FF_{k,x,i,g} = \sum_g \frac{\Delta AOT40_g}{\Delta N_{x,i}}
\]

\( (7) \)

Monthly AOT40 concentrations per unit of emission of NOx and NMVOC were calculated on a 1°×1° 
resolution from hourly ozone concentrations resulting from the year 2000 reference run with TM5 
chemical transport model. For the Northern Hemisphere the same growing seasons for grassland and 
forest were taken as was done for Europe by Van Goethem et al. (2013b), namely May till July and 
April till September, respectively. For the Southern Hemisphere for grassland the months November till 
January and for forests the months October till March were taken.

\( Effect \) factor

Effect factors (EF) were derived from Van Goethem et al. (2013b), who determined EFs that describe 
the change in potentially affected fraction (PAF) of forest or grassland species due to the change in 
ground level ozone exposure:

\[
EF_{n,k,g} = \frac{\Delta PAF_{g,n}}{\Delta AOT40_g} \cdot A_{g,n}
\]

\( (8) \)

where \( A_{g,n} \) is the area (m²) occupied by vegetation type n in grid cell g. The effect factor was 
determined with data on AOT40 concentrations for which 50% reduction in productivity (EC50) was 
found for a number of forest or grassland species (taken from Van Goethem et al. (2013a, 2013b)). Here, 
we chose to use the linear ecosystem effect factor, assuming a linear change in PAF with changing AOT40
that represents the average effect between a PAF of 0.5 and 0 (Van Goethem et al. 2013b). The corresponding “AOT40 concentration per unit of yearly emission” values per region were multiplied by
the corresponding natural area of either grassland or forest per region based on the Global Land Cover
2000 (GLC2000) database (Bartholomé and Belward 2005). Allocation of GLC2000 types to grassland or
forest can be found in Appendix Table A2.

2.3 Normalization

Normalization of the characterization factor results gives insight in the relative magnitude of the
characterized impacts by relating them to a common reference situation and expressing them in a unit
common for multiple impact categories. In our case, we chose the load from society's total activities in
2010 as a reference situation. The normalization factors (NF) for worldwide PM2.5 and ozone impacts
were derived by multiplication of the region-specific characterization factors with the substance-specific
emissions in each region of 2010 (http://www.iiasa.ac.at/web/home/research/researchPrograms/ECLIPSEv5a.html), divided by the total
population in all regions:

\[
NF_{AoP} = \frac{\sum_x \sum_j \sum_i (M_{x,j} \cdot CF_{AoP,k,x,i})}{\sum_i (N_i)}
\]  

(9)

where \( NF_{AoP} \) is the normalization factor of the summation of all 56 regions per damage area (human
health or ecosystem health) and pollutant. \( M_{x,j} \) is the emission of precursor \( x \) in region \( j \) (in kton/yr),
\( CF_{AoP,k,x,i} \) is the characterization factor for the specific area of protection and pollutant \( k \), substance \( x \) in
region \( i \) and \( N_i \) is the number of inhabitants in region \( i \), which are summed to the world inhabitants
(United Nations 2011).

The normalization factor for human health damage equals the total years of life lost worldwide due
to emissions in 2010 per capita, while the normalization factor for ecosystem damage equals the area-
integrated potentially affected fraction of natural plant species worldwide due to emissions in 2010 per
capita (in PAF.m²/capita).
3. Results

3.1 Human health

Figure 1 shows the region-specific characterization factors for human health for fine dust precursor emissions. It can be seen that lowest factors were obtained for emissions of NOx on the Southern Hemisphere, while largest factors were obtained for primary PM2.5 emissions in Central Asia. Emissions in Australia and New Zealand lead to relatively low damages for all substances. For Chile, however, SO2 and NOx emissions lead to relatively low impacts, while PM2.5 and NH3 emissions lead to relatively large impacts. Also, Egypt does not show a specific trend. NOx emissions in Egypt lead to relatively low impacts, while PM2.5, NH3 and SO2 emissions lead to relatively large impacts. The emission weighted average for the world for primary PM2.5 is 629.2 yr·kton\(^{-1}\) (with a minimum of 9.4 and a maximum of 4018.6 yr·kton\(^{-1}\)). The emission weighted average for the world for NH3 is 160.6 yr·kton\(^{-1}\) (3.3 to 1.3\cdot10^3 yr·kton\(^{-1}\)), for NOx 76.2 yr·kton\(^{-1}\) (0.4 to 365.3 yr·kton\(^{-1}\)), and for SO2 183.4 yr·kton\(^{-1}\) (14.0 to 945.4 yr·kton\(^{-1}\)).

Emitting region specific intake fractions for PM2.5 range from 1.6\cdot10^{-9} to 9.6\cdot10^{-6}. This means that up to 10 mg PM2.5 is inhaled by the population in a region per kg of precursor substance emitted. Intake fractions for NOx were overall the lowest, about 1 order of magnitude lower than intake fractions from primary PM2.5. Largest intake fractions were found for PM2.5 emissions in South Asian regions and Japan. Lowest effect and damage factors for receiving regions were retrieved for PM2.5 in Thailand and South Korea due to relatively low mortality rates for all diseases. The largest effect and damage factors for PM2.5 were obtained for Kazakhstan and Russia, mainly due to a high mortality rate for cardiopulmonary disease. The full region-specific results are given in the Appendix (Tables A3-A5).
Figure 1: Characterization factors for human health damage caused by fine dust formation due to primary PM2.5, and NH₃, NOₓ, and SO₂ precursor emissions (yr⁻¹ kton⁻¹).

Figure 2 shows the region-specific characterization factors for human health for ozone precursor emissions. Overall, the CFs for ozone damage are 2 to 4 orders of magnitude lower compared to the CFs for damage due to PM2.5. It can be seen that lowest factors (apart from the negatives) were obtained for emissions of NMVOC in New Zealand, Australia, Indonesia, and South America, while largest factors were obtained for NOₓ emissions in South Asia, West-Africa, India and China. The emission weighted average for the world for NMVOC is 1.4·10⁻¹ yr⁻¹ kton⁻¹ (8.8·10⁻³ to 5.0·10⁻¹ yr⁻¹ kton⁻¹). The emission weighted average for the world for NOₓ is 9.1·10⁻¹ yr⁻¹ kton⁻¹ (-2.2·10⁻¹ to 5.7 yr⁻¹ kton⁻¹). Negative intake fractions and thus CFs were obtained for NOₓ emitted in Belgium, the Netherlands, Luxembourg, Great Britain, and Ireland. A negative value means that the emission of NOₓ leads to an overall reduction of ozone exposure. Region specific intake fractions range from -1.5·10⁻⁸ to 1.5·10⁻⁷. Largest region specific intake fractions were obtained for NOₓ emitted in Asia. Lowest effect and damage factors for receiving regions were retrieved for ozone in Thailand and South Korea, due to low mortality rates and a low number of life years lost for respiratory disease for South Korea. The largest effect and damage factors were obtained for South Africa and Western Africa due to high mortality rates for respiratory disease.
and the highest years of life lost for western Africa. All region-specific results are given in the Appendix (Tables A3-A5).

3.2 Natural vegetation

Characterization factors for damage to natural vegetation by tropospheric ozone are shown in Figure 3. The full region-specific results are given in the Appendix (Table A5).

The emission weighted average for the world for NMVOC is 2.5 km²·yr·kton⁻¹, while the emission weighted average for the world for NOx is 8.7 km²·yr·kton⁻¹. Lowest factors were obtained for emissions of NMVOC and NOx in New Zealand, and for NMVOC emissions in Mongolia, Australia, Indonesia, Argentina and Brazil. The largest CF was obtained for NOx emissions in Mexico (54.5 km²·yr·kton⁻¹).
Figure 3: Characterization factors for ecosystem damage caused by ozone formation (km²·yr·kton⁻¹).

3.3 Normalization

The normalization factor for human health damage due to global emissions of primary and secondary aerosols in 2010 is 1.7·10⁻² yr-capita⁻¹. Primary PM emissions contributed 62%, followed by SO₂ emissions (19%). NH₃ and NOₓ emissions contributed 10% and 9% to the total normalizations factors. Impacts on human health due to ozone damage are negligible compared to the damage caused by fine dust.

The normalization factor for ozone impact on natural vegetation due to global emissions of NOₓ and NMVOC in 2010 is 1.6·10⁻⁴ km²-capita⁻¹. NOₓ contributed 72% and NMVOCs 28%.

4. Discussion

4.1 Fate and intake factors

In this section model assumptions and uncertainties in the calculation of intake fractions and fate factors are discussed. Results of the global chemistry transport model TM5 were applied in this research to derive intake fractions and fate factors for PM2.5 and ozone exposure. TM5 was evaluated (Huijnen et al. 2010; Textor et al. 2006) and validated in a model intercomparison study (Van Loon et al. 2007). Textor et al. (2006) analyzed the atmospheric fate of sulfate, black carbon, and particulate organic matter, among others, highlighting the diversity of 16 global aerosol models. Huijnen et al. (2010) showed that TM5 simulated the seasonal and spatial variability of ozone and nitrogen dioxide in correspondence of measurements, to within 10 ppbv for ozone. Only for several tropical point measurements the model tends to underestimate ozone in the free troposphere (Huijnen et al. 2010). The model was found to predict concentrations well in agreement with measurements and realistic compared to other models (Van Loon et al. 2007). 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. We evaluated the possible error in exposed PM2.5 at the aggregated regional level used in this study, by making use of high-resolution (0.1x0.1 degree) year 2000 satellite-derived PM2.5 concentrations at the surface (Boys et al. 2014), in combination with population grids for the year 2000 at the same resolution (Janssens-Maenhout et al. 2012). These datasets allowed us to calculate the satellite-derived area-mean PM2.5 concentration for each 1x1 degree grid cell (i.e. the
equivalent of our modelled PM2.5 resolution), as well as the corresponding population-weighted mean PM2.5 at the 1x1 degree resolution. Comparing the area-weighted and population-weighted results we 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%. This implies that our characterization factors can give an underestimation of up to 19% for human health impacts caused by PM2.5 exposure due to possible sub-grid gradients within the 1x1 degree grids.

The negative intake fractions for ozone due to emissions of NOx are caused by the so-called titration-effect. As a result of the rapid reaction of ozone with NO to form NO2, concentrations of ozone tend to be lower close to sources of NO emissions, such as near dense urban traffic, major highways, and industrial sources (EEA 2005; Tong et al. 2006). Countries that show negative characterization factors for NOx therefore have relatively large characterization factors for NMVOC.

Because AOT40 is a threshold based concentration indicator, there is more uncertainty attached to it compared to the use of linear scaling concentrations (Van Dingenen et al. 2009). When a concentration is, for example, slightly above the threshold of 40 ppb and then reduced when looking at the 20% perturbation, this can have large impacts on the results.

Our CFs were derived from emission-concentration sensitivities (dC/dM) obtained from a 20% emission perturbation. Ozone chemistry is expected to behave non-linearly, especially to high emission changes, and this will be even more the case for threshold O3 metrics like AOT40. For a limited number of representative source regions the dC/dM coefficients were calculated for large perturbations of secondary pollutants (-80%, +100%) and compared to the extrapolated 20% perturbation. Results are summarized in Table A6. For PM2.5, relatively small maximum absolute deviations were seen, up to 5%. For M6M, precursor NOx a deviation up to 14% was seen. For AOT40, however, deviations can be large. The large deviation for AOT40 under an 80% reduction of NOx (36% average) is explained by the linear extrapolation of a threshold metric from a regime above threshold to a regime below threshold.

4.2. Effect and damage factors

Although Fantke et al. (2015) recommend to include health effects considered in GBD 2010 (Murray et al. 2012) and in the HRAPIE consensus document (WHO 2013) as a starting point, we chose to include the mortality with the largest share to human health damage caused by PM2.5 and ozone, and of which the most certain epidemiological data are available. Even though our numbers are based on mortality
only, the unit is comparable to other health damage included in life cycle impact assessment expressed in DALY. In a previous research, Van Zelm et al. (2008) showed that 99% of DALYs due to PM10 is caused by chronic mortality, while for ozone previous research has not shown significant morbidity effects.

Unlike previous global scale research (Tang et al. 2016a, b), we used region-specific data, except for the relative risk. In reality, relative risks are non-linear, and dependent on the background concentration of a region. GBD 2010 provides possibilities to compute relative risks of various health effects as a function of ambient PM2.5 concentrations (Burnett et al. 2014). Burnett et al. (2014) showed, for example, that there is a minimum of 5.8 μg·m⁻³ needed for health effects to occur, and in areas with high concentrations relative risks can be lower for some diseases. Table A1 shows that average concentrations in Finland, Norway, Iceland, Pacific Islands, and Papua New Guinea are below this minimum and no effects will occur.

There is no consensus yet on which type of ecosystem effect factor to use (Huijbregts et al. 2011). We chose the linear factor because (i) it is commonly used in ecotoxicology (Pennington et al. 2004), and (ii) we extrapolated the European based effect factor to the world, due to a lack of global data. It has to be noted however, that because the linear approach disregards the shape of the exposure-response curve, it is considered to be less reliable compared to the marginal (describing a marginal change in PAF due to the marginal change in ground level ozone exposure) or the average (describing the average distance between the current state and the preferred state) approach.

As outlined by Van Goethem et al. (2013a), a next step in effect modelling will be the use of flux-based ozone exposure experiments, which can take into account environmental conditions that are closer to observed conditions. For now, however, flux models are only available for a limited number of species. We refer to Van Goethem et al. (2013b) for a more comprehensive discussion on the derivation of the ecosystem effect factors, and allocation of forest or grassland to each region.

### 4.3 Characterisation factors

Emissions in Australia and New Zealand lead to relatively low damages for all substances as those countries are rather isolated and there is little transfer of pollution to neighboring regions. However, for other countries, not always a specific trend was shown. Primary PM2.5 has the lowest lifetime and is the most 'concentrated' in space, secondary PM is transported further than primary, while ozone has the largest lifetime and is transported at hemispheric scale. Source regions for which the concentration footprint of emitted precursors covers populated areas or ecosystems, as opposed to sea and deserts, will have a larger impact than relatively isolated source regions. A country like Egypt has its entire
population concentrated in a very limited part of the country and is more sensitive to the PM where the 'concentration footprint' coincides more or less with the populated areas. The concentration footprint of ozone from NO\textsubscript{x} on the other hand is spread out over a larger region of which a big portion causes no impact (e.g. in deserts). In a country like Chili, a complex partitioning system takes place where secondary PM formation is likely NH\textsubscript{3} limited due to high SO\textsubscript{2} emissions. NH\textsubscript{3} will preferentially associate with sulfate from SO\textsubscript{2} to form ammonium sulfate, leading to relatively larger impacts from NH\textsubscript{3} than from other precursor compounds.

Since no world average factors have been reported up to now, we derived European and US emission weighted averages to compare to European and US average factors available. Figure 4 shows the results of this comparison for PM2.5 damage to human health. It can be seen that for secondary particles, our factors are in the same order of magnitude as the ones derived by Van Zelm et al. (2008), ILCD recommended factors and Tang et al. (2016a) for Europe, and by Gronlund et al. (2015) and Tang et al. (2016a) for the US, with differences up to a factor of 3. These differences are caused by the application of different chemistry transport models, with different spatial variability, as well as the application of different effect factor input data. We are the first to determine region-specific effect and damage factors. For primary PM2.5, intake fractions of one order of magnitude larger are recommended by the ILCD. For future research it would be an additional value if a global chemistry transport model could be used that can differentiate CFs regarding stack heights and emission location as well to obtain archetype specific intake fractions on a global scale. Moreover, a finer source grid resolution would increase accuracy but would of course also increase data and computation demand.

The lighter parts of the columns in Figure 4a and Figure 5 of Tang et al. (2016a) reflect the damage caused by intake outside Europe. For NO\textsubscript{x} and SO\textsubscript{2} predictions of Tang et al. are slightly larger, i.e. 11\% and 33\% damage outside Europe versus 8\% and 24\% predicted in our study. For primary PM2.5, however, we predict 7\% damage outside Europe caused by emissions from our 23 European regions, whereas Tang et al. predict 29\% damage to occur outside their one European region.
For damage due to photochemical ozone formation, little research was done up to now determining endpoint characterization factors. Moreover, different ozone indicators can be used. This is the first time that human health damage from ozone formation was determined based on M6M. For NMVOC emissions, our European average factor of \(1.6 \times 10^{-3} \text{ yr-kton}^{-1}\) is close to the values obtained by Van Zelm et al. (2008) for 24-hr average concentration as well as maximum daily 8-hr average concentration, which was \(3.9 \times 10^{-2} \text{ yr-kton}^{-1}\) in both cases. When excluding impacts outside Europe, as done by Van Zelm et al. (2008) we obtain a value of \(8.1 \times 10^{-2} \text{ yr-kton}^{-1}\). Tang et al. (2016b) obtained a value of \(3.5 \text{ yr-kton}^{-1}\) (maximum daily 8-hr average concentration) by including illnesses as well, for which effect data are uncertain. For NO\(_x\), however, the outcome is much more influenced by the metric chosen, as negative intake fractions can be obtained due to the titration effect. Van Zelm et al. (2008) obtained a negative European characterization factor when using 24-hr average ozone concentrations \((-1.2 \times 10^{-2} \text{ yr-kton}^{-1})\), but a positive \((3.9 \times 10^{-2} \text{ yr-kton}^{-1})\) when using maximum daily 8-hr average concentration, just like Tang et al. (2016b) \((3.5 \text{ yr-kton}^{-1})\), while we obtained a European average factor of \(3.1 \times 10^{-1} \text{ yr-kton}^{-1}\) \((1.3 \times 10^{-1} \text{ yr-kton}^{-1}\) excluding impacts outside Europe).

The ecosystem damage CF for ozone formation due to NO\(_x\) for European regions is on average 3.4 times larger than the CF for NMVOC. These findings correspond with the results of Hauschild et al. (2006) and of Van Goethem et al. (2013b), who found larger CFs for NO\(_x\) by a factor of 3.6 and 3.5, respectively.
The European averages for NO$_x$ and NMVOC emissions of 13.2 and 4.1 km$^2$-yr-kton$^{-1}$ respectively correspond well with the obtained factors by Van Goethem et al. (2013b) of 16.4 and 4.8 km$^2$-yr-kton$^{-1}$.

4.4 Application in LCA

The presented characterization factors can be used in life cycle assessment studies to quantify the impacts of emissions of PM2.5, NO$_x$, NH$_3$, SO$_2$, and NMVOC on fine dust and ozone formation, along with the impacts of other stressors and/ or other impact types, like global warming damage due to carbon dioxide. Applying region specific characterization factors instead of the more commonly used generic factors can lead to significantly different outcomes of an LCA. Currently, European or USA averages are often used. Our research shows that applying a European or world average instead of a region specific value can lead to over- or underestimation of damage of up to 2 orders of magnitude. Damage due to PM2.5 caused by emissions in Australia is, for example, up to 150 times smaller than the European average.

The negative intake factors we derived for ozone formation due to NO$_x$ emissions do not imply that increased emissions have a positive influence on human health. NO$_x$ also attributes to secondary aerosol formation, for example. This secondary PM2.5 formation causes more health damage than that is ‘prevented’ due to a decrease in ozone formation. Additionally, NO$_x$ causes acidification and eutrophication that should be accounted for in life cycle impact assessment as well (see e.g. Azevedo et al. 2013; Roy et al. 2014).

Although the WHO (2013) provides recommendations for concentration-response functions for direct NO$_2$ effects, we did not include those here to prevent overlap with effects caused by ozone and fine dust formation due to NO$_2$ emissions.

4.5 Concluding remarks

Summarizing, we determined spatially explicit characterization factors for 56 world regions covering the whole globe, for human health damage of primary and secondary particulate matter driven by anthropogenic PM2.5, NH$_3$, NO$_x$, and SO$_2$ emissions, as well as for damage to human health and natural vegetation by tropospheric ozone driven by anthropogenic NO$_x$ and NMVOC emissions. For the first time a global chemistry transport model was applied, modeling fate of all these precursors simultaneously, and the first time factors were derived for photochemical ozone damage on natural vegetation worldwide. Our study provides a sound basis for life cycle impact assessment including effects of respiratory inorganics and photochemical oxidant formation.
Acknowledgements

The authors would like to thank Frank Dentener for providing the source-receptor runs of TM5-FASST, and Gea Stam, Joachim Roos, and Zoran Steinmann for their technical support, and Alexis Laurent and Francesca Verones for emission data compilation. This research was funded by the European Commission under the 7th framework program on environment; ENV.2009.3.3.2.1: LC-IMPACT – Improved Life Cycle Impact Assessment methods (LCIA) for better sustainability assessment of technologies, grant agreement number 243827.

Appendix A. Supplementary data

Supplementary data related to this article can be found at @@@

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