LCIA OF IMPACTS ON HUMAN HEALTH AND ECOSYSTEMS

Health effects of fine particulate matter in life cycle impact assessment: findings from the Basel Guidance Workshop

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Received: 11 February 2014 /Accepted: 5 November 2014 / Published online: 21 November 2014 \oslash Springer-Verlag Berlin Heidelberg 2014

Abstract

Purpose Fine particulate matter (PM_{2.5}) is considered to be one of the most important environmental factors contributing to the global human disease burden. However, due to the lack of broad consensus and harmonization in the life cycle assessment (LCA) community, there is no clear guidance on how to consistently include health effects from $PM_{2.5}$ exposure in LCA practice. As a consequence, different models are

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F. Hurley : B. G. Miller Institute of Occupational Medicine, Edinburgh EH14 4AP, UK currently used to assess life cycle impacts for $PM_{2.5}$, sometimes leading to inconsistent results. In a global effort initiated by the United Nations Environment Programme (UNEP)/ Society for Environmental Toxicology and Chemistry (SETAC) Life Cycle Initiative, respiratory inorganics' impacts expressed as health effects from $PM_{2.5}$ exposure were selected as one of the initial impact categories to undergo review with the goal of providing global guidance for implementation in

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life cycle impact assessment (LCIA). The goal of this paper is to summarize the current knowledge and practice for assessing health effects from $PM_{2.5}$ exposure and to provide recommendations for their consistent integration into LCIA.

Methods A task force on human health impacts was convened to build the framework for consistently quantifying health effects from $PM_{2.5}$ exposure and for recommending $PM_{2.5}$ characterization factors. In an initial Guidance Workshop, existing literature was reviewed and input from a broad range of internationally recognized experts was obtained and discussed. Workshop objectives were to identify the main scientific questions and challenges for quantifying health effects from $PM_{2.5}$ exposure and to provide initial guidance to the impact quantification process.

Results and discussion A set of 10 recommendations was developed addressing (a) the general framework for assessing PM_{2.5}-related health effects, (b) approaches and data to estimate human exposure to $PM_{2.5}$ using intake fractions, and (c) approaches and data to characterize exposure-response functions (ERFs) for $PM_{2.5}$ and to quantify severity of the diseases attributed to $PM_{2.5}$ exposure. Despite these advances, a number of complex issues, such as those related to nonlinearity of the ERF and the possible need to provide different ERFs for use in different geographical regions, require further analysis. Conclusions and outlook Questions of how to refine and improve the overall framework were analyzed. Data and models were proposed for harmonizing various elements of the health impact pathways for $PM_{2.5}$. Within the next two years, our goal is to build a global guidance framework and to determine characterization factors that are more reliable for incorporating the health effects from exposure to $PM_{2.5}$ into LCIA. Ideally, this will allow quantification of the impacts of both indoor and outdoor exposures to $PM_{2.5}$.

Keywords Air pollution . Exposure-response function . Fine particulate matter \cdot Global guidance \cdot Human health effects \cdot Intake fraction . Life cycle impact assessment (LCIA)

1 Health effects from fine particulate matter: towards global guidance in life cycle assessment

Life cycle assessment (LCA) is a structured, comprehensive, and internationally standardized method to assess potential

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environmental impacts and resources used throughout the life cycle of a good or service in a comparable way (ISO [2006\)](#page-10-0). LCA thereby aims for best estimates in the modeling of all relevant impacts on the natural environment, human health, and resources in the life cycle impact assessment (LCIA) phase (EC [2010a](#page-10-0); Finnveden et al. [2009](#page-10-0)). To help identify the best LCA practice, Phase III (2012–2016) of the United Nations Environment Programme (UNEP)/Society for Environmental Toxicology and Chemistry (SETAC) Life Cycle Initiative¹ has launched a flagship project aiming to provide global guidance and consensus on a limited number of LCIA indicators. The Glasgow Scoping Workshop in May 2013 (Jolliet et al. [2014](#page-10-0)) focused on establishing a tentative short list of impact category indicators that would be addressed during two consensus building periods. These indicators included the impacts of respiratory inorganics expressed as health effects from exposure to primary and secondary particulate matter (PM), which is considered to be one of the most important environmental stressors contributing to the global human disease burden (Hänninen et al. [2014;](#page-10-0) Lim et al. [2012](#page-10-0)). Primary PM refers to directly emitted particles. Secondary PM refers to organic and inorganic (e.g., ammonium nitrate, ammonium sulfate) particles formed through reactions of precursor substances including nitrogen oxides (NO_x) , sulfur oxides (SO_x) , ammonia (NH_3) , and semivolatile and volatile organic compounds (VOC), of which the latter are the most important for secondary organic aerosol formation. PM is further distinguished according to aerodynamic diameter, i.e., respirable particles (PM_{10}) with <10 μ m, fine particles (PM_{2.5}) with <2.5 μ m, and ultrafine particles (UFP) with \leq 100 nm aerodynamic diameter (WHO [2006](#page-11-0)). PM_{2.5} was chosen to provide international recommendations regarding the consistent integration of its health effects into LCIA because it might best describe the component of particulate matter responsible for adverse health effects (Harrison and Yin [2000](#page-10-0); Lim et al. [2012;](#page-10-0) Lippmann and Chen [2009](#page-10-0)).

2 Assessing fine particulate matter in the context of life cycle impact assessment

In epidemiological studies, exposure to $PM_{2.5}$ is associated with various adverse health effects and reduction in life expectancy including chronic and acute respiratory and cardiovascular morbidity, chronic and acute mortality, lung cancer, diabetes, and adverse birth outcomes (Beelen et al. [2014;](#page-9-0) Brook et al. [2010](#page-9-0); Chen et al. [2008;](#page-9-0) COMEAP [2010;](#page-10-0) Dadvand et al. [2013](#page-10-0); Hoek et al. [2013](#page-10-0); Künzli et al. [2000;](#page-10-0) Lippmann and Chen [2009;](#page-10-0) Loomis et al. [2013;](#page-10-0) Mehta et al. [2013;](#page-11-0) Pelucchi et al. [2009](#page-11-0); Pope III et al. [2009;](#page-11-0) Pope III et al. [2011](#page-11-0); Straif et al. [2013\)](#page-11-0). Furthermore, toxicological studies

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¹ <http://www.lifecycleinitiative.org/activities/phase-iii>

support the observation that exposure to $PM_{2.5}$ can exert effects on key biological systems, with some evidence that not all particles are likely to cause the same health effects (Harrison and Yin [2000](#page-10-0); Kelly and Fussell [2012;](#page-10-0) Rohr and Wyzga [2012;](#page-11-0) Stanek et al. [2011](#page-11-0)). Several existing LCIA methods already characterize health effects associated with ambient PM or $PM_{2.5}$ concentrations (EC [2010c](#page-10-0)), mostly based on ambient $PM_{2.5}$ intake estimated from simple exposure or intake fraction models and using health effect data from the Harvard Six Cities and American Cancer Society studies (Krewski et al. [2000](#page-10-0); Laden et al. [2006;](#page-10-0) Pope III et al. [2002\)](#page-11-0). A few studies include spatial allocation of emissions and modeling of air dispersion and chemical reactions to predict downwind $PM_{2.5}$ concentrations (Hill et al. [2009](#page-10-0); Tessum et al. [2012\)](#page-11-0). Whenever emission locations are known, these spatially explicit approaches can be applied in LCIA. It is anticipated in the future to fully assess $PM_{2.5}$ impacts using such spatially explicit approaches. In the current absence of this capacity, a consistent and globally harmonized approach for LCIA should be based on the most recent science to simultaneously address environmental fate, human exposure, and health effects of $PM_{2.5}$ concentrations resulting from emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors (Hauschild et al. [2013](#page-10-0)).

One of the challenges in LCA is that impacts are linked to emissions via intake, whereas in epidemiology, impacts are related to concentrations. Generally, when assessing the health response of a population, the most accurate and efficient approach is to relate observed concentrations to population response. This also constitutes the basis for the LCA framework. However, this approach needs to be adapted for the emission-based LCA context for which the impact of an additional kilogram emitted by multiple sources in different, often unknown locations needs to be evaluated (Finnveden et al. [2009](#page-10-0); Hauschild [2005](#page-10-0)). For such emission-based assessments, the human intake fraction (iF) as the fraction of an emitted mass ultimately taken in by the total exposed population is well adapted, accounting directly for a temporally and spatially integrated concentration multiplied by nominal human intake rates. Intake fraction is a time- and spaceintegrated metric, easy to understand, to communicate, and to combine with chemical emissions. Emission source types can be associated with a specific iF, which is easier to interface and combine at the level of exposure than a field of concentrations over a certain distance around the source.

With respect to assessing the particular health effects from PM_{2.5} exposure, the effort of an earlier UNEP/SETAC working group has designed a framework and proposed a set of default iF associated with $PM_{2.5}$ emissions for use in LCIA (Humbert et al. [2011](#page-10-0)). This effort is limited to the steps of the impact pathway from emissions to concentration and human intake but does not cover the steps from human intake to health effects. In addition, due to the lack of broad

consensus and harmonization in the LCA community, there is no clear guidance on how to include health effects from PM_{2.5} exposure in LCA practice. As a consequence, different models are currently used leading at times to inconsistent life cycle impact results reported for this category. This reveals the importance of pursuing consensus building, based on the initial work of Humbert et al. [\(2011\)](#page-10-0) and combining it with latest exposure-response and severity data to yield revised guidance on the development and use of human health characterization factors for both primary and secondary $PM_{2.5}$ including precursor substances. Ultrafine particles are currently not separately considered in LCA.

To meet our needs for global guidance and harmonization regarding health effects from $PM_{2.5}$ exposure in LCIA, the UNEP/SETAC Life Cycle Initiative established a task force on human health impacts. The aim of the task force is to build within the next 2 years a framework and determine factors recommended for incorporating human health effects from PM_{2.5} exposure into LCIA and addressing both outdoor and indoor releases. In order to provide a starting point for the task force effort, the workshop participants summarize in this paper the current knowledge on and practice in assessing the health effects from $PM_{2.5}$ exposure including related recommendations.

3 The Basel Guidance Workshop: identifying and addressing the key questions

Within the task force on human health impacts, an initial Guidance Workshop was organized back-to-back with the ISEE/ISES/ISIAQ Environment and Health Conference in Basel, Switzerland, in August 2013. Based on a literature review and expert input, the workshop organizers reached out to a broad range of internationally recognized experts in PM exposure and health effects. Sixteen of these experts agreed both to participate in the process and attend the Basel workshop (in person or by phone). This included experts from Canada, Denmark, Finland, Germany, Poland, Spain, Switzerland, the UK, and the USA. Many others have agreed to contribute in some form to the task force activities.

The specific objectives of the workshop were to first identify and discuss the main scientific questions and challenges for quantifying human health effects from $PM_{2.5}$ exposure and then to provide initial guidance to the impact quantification process. Three main topics were addressed at the workshop: (a) the general assessment framework as proposed by Humbert et al. [\(2011\)](#page-10-0), (b) approaches and data to determine human exposure to $PM_{2.5}$ expressed as intake fractions, and (c) approaches and data to determine exposure-response functions (ERFs) for $PM_{2.5}$ along with disease severity. For these topics, the workshop participants discussed a set of key questions that had been established with selected experts in three pre-workshop phone conferences. Table [1](#page-4-0) summarizes these key questions, which are discussed in detail in the following.

4 General assessment framework recommendations

An overall picture of the approach currently proposed for health effects attributed to $PM_{2.5}$ exposure in LCIA including the findings of the Basel Guidance Workshop is presented in Fig. [1.](#page-5-0)

4.1 Overall assessment approach

There was agreement among the workshop participants to build upon the general framework proposed by Humbert et al. ([2011\)](#page-10-0). In this framework, human intake fractions for primary and secondary $PM_{2.5}$ are provided, emissions from low and high stacks are differentiated, and dominant influences for generic landscape characteristics are parameterized. Humbert et al. ([2011](#page-10-0)) thereby start from emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors into the environment, m (mass emitted), and multiply these emissions with *intake fractions*, iF (mass of $PM_{2.5}$ inhaled by the affected population per mass of primary $PM_{2.5}$ or secondary PM_2 , precursor emitted, respectively), an *exposure-re*sponse factor derived from epidemiological studies linking health effects in the affected population to ambient $PM_{2.5}$ concentrations,² ERF (disease rate per unit mass concentration), and a severity factor, SF (disability-adjusted life years (DALY) per disease case), to arrive at a human health-related impact score, IS (DALY):

$$
IS = m \times \underbrace{\text{i}F \times \text{ERF} \times \text{SF}}_{CF} \tag{1}
$$

Intake fraction, exposure-response factor, and severity factor can be represented by the characterization factor, CF (DALY per mass emitted). A key assumption implicit in this framework is the linear, no-threshold ERF. While not uncontroversial, this assumption reflects current practice and recent recommendations in LCIA (EC [2010b;](#page-10-0) Potting et al. [2007](#page-11-0)) and is also applied in other studies as discussed, e.g., in COMEAP [\(2009\)](#page-9-0).

4.2 Exposure metrics

Two exposure metrics, (i) ambient $PM_{2.5}$ concentration and (ii) population intake of $PM_{2.5}$, were considered as possible starting points for assessing health impacts from $PM_{2.5}$ exposure. It should be noted that, when all populations are assigned the same population breathing rate, the exposure expressed as either ambient concentration or intake fraction is exactly proportional. In other LCIA areas, health impacts are typically assessed using population intake as exposure metric (Udo de Haes et al. [2002\)](#page-11-0). This approach can be justified for many endpoints, e.g., cancer risk assessment for genetic carcinogens, where risk is proportional to cumulative intake (often expressed as applied dose), i.e., where there are no population thresholds and no appreciable nonlinearities in the relationship between intake and response. However, in cases where there are thresholds, i.e., concentrations or intakes below which health effects are not induced even in the most sensitive individuals or significant nonlinearities in describing response as a function of concentration or cumulative intake, this simple approach may not provide a satisfactory representation of the effect of changes in exposure on population health risk. To make the approach more appropriate in such cases, the population intake fraction can be used as a measure of the population's ambient $PM_{2.5}$ exposure. For population exposure to PM_{2.5}, it is reasonable to assume no threshold, but there are possibilities for nonlinear response for highly exposed populations (Burnett et al. [2014](#page-9-0)).

Epidemiological studies of the health impacts of exposure to $PM_{2.5}$ typically report the relative risk of morbidity or mortality (i.e., the ratio of the risk among the exposed to that among the unexposed) as a function of the concentration of PM_{2.5} measured at fixed site monitors (see, for example, COMEAP [2010\)](#page-10-0). They are not based on concentrations found through personal exposure monitoring (Hurley et al. [2005](#page-10-0)). In LCIA, the impact of an additional kilogram often emitted by multiple sources at different, often unknown locations over the life cycle is evaluated, making it effectively impossible to report the related concentrations.

Recognizing the need for a population-scale exposure metric often without access to site-specific emissions data, workshop participants recommended the use of population intake fraction, which is equivalent to population exposure concentration, as the default measure for computing $PM_{2.5}$ health risks in LCIA. Population intake estimates computed using iF reflect the change in population-weighted intake of the ambient outdoor concentration. Thus, intake estimates are directly related to concentrations underlying epidemiological estimates of mortality and morbidity risks from $PM_{2.5}$ exposure, although this requires knowledge about background concentrations when using nonlinear exposure-response functions.

4.3 Health metrics

Various health metrics were discussed, including total and premature mortality, years of life lost (YLL), and DALY.

 $2 PM_{2.5}$ concentrations can be converted to intake using the breathing rate of the exposed population. How to average the breathing rate for different activities, age, etc. remains to be further discussed.

Most workshop participants felt that when death is the outcome of interest, YLL is a better measure of mortality impacts than the number of deaths. The view was that information on the number of deaths is more challenging to interpret because reduced $PM_{2.5}$ intake cannot affect the fact of death, but only its cause and timing (Leksell and Rabl [2001](#page-10-0); Rabl [2005](#page-11-0)). When it is necessary to combine mortality and morbidity impacts into a single summary measure, two approaches can be used. The first approach is to use DALY combining YLL and years lived with a disability (YLD) weighted by the quality of life during the period of disability (Murray and Lopez [1996a,](#page-11-0) [b](#page-11-0)). The second approach, which is frequently preferred by economists, is to use weights reflecting societal willingness to pay to avoid small incremental risks of mortality and morbidity.

The workshop participants see no reason to reconsider this matter. In summary, YLL and DALY seem to be appropriate health metrics for use in LCIA, since they focus attention on actions with the greatest potential to lead to improvement in the number of healthy life years lived by the exposed populations (Wang et al. [2012\)](#page-11-0). In addition, selecting a preferred approach is an issue that affects all analyses of health impacts in LCIA. Typically, LCIA has relied on the DALY metric (EC [2010b\)](#page-10-0) without age weighting and/or discounting.

Fig. 1 Proposed framework for assessing human health effects from fine particulate matter exposure in life cycle impact assessment, adapted from Humbert et al. [\(2011\)](#page-10-0)

4.4 Other framework discussion points

Two additional aspects were briefly discussed at the workshop: (i) whether and, if so, how to address the dynamics when expressing of health impacts attributable to $PM_{2.5}$ exposure and (ii) how to account for differences between average and marginal impacts on health of primary and secondary PM_{2.5} precursor emissions, which may occur when either emissions-exposure or exposure-response functions exhibit thresholds or significant nonlinearities. The workshop participants agreed that in the long term, both issues require further attention.

5 From emissions to concentration and human intake: determining intake fractions

5.1 Archetype structure

In LCIA, it is common practice to make use of archetypal exposure scenarios, e.g., urban vs. rural scenarios (Riley et al. [2002\)](#page-11-0), rather than site-specific exposure assessments, especially when emission locations are unknown. The workshop discussion focused on identifying the key factors influencing iF and determining how to address these in the context of quantifying $PM₂$ -related health effects in LCIA. Table 3 in Humbert et al. ([2011\)](#page-10-0) proposed one such archetypal structure in which population density (urban, rural, remote) and emission height (high-stack, low-stack, ground-level) serve as the main determinants of iF. Humbert et al. also provided a default set of iF values corresponding to these archetypes.

The workshop participants agreed to adopt this structure as starting point but pointed out that additional refinements in terms of archetypes need to be explored. Refinements can thereby build on applying a sensitivity analysis to a range of aspects that influence the variability of iF. This includes, for example, distinct urban areas based on work by Apte et al. [\(2012\)](#page-9-0) and different emission sources, such as traffic-related sources (Greco et al. [2007](#page-10-0); Lobscheid et al. [2012;](#page-10-0) Marshall et al. [2005\)](#page-11-0), stationary emissions from coal/gas-fired power plants (Heath et al. [2006](#page-10-0); Levy et al. [2002](#page-10-0), [2003](#page-10-0)), or indoor emissions from wood burning (Ries et al. [2009](#page-11-0)). The participants also agreed to include additional archetypes reflecting exposure from indoor emissions of $PM_{2.5}$ based on work by Hellweg et al. [\(2009\)](#page-10-0).

5.2 Geographical differentiation

Despite the availability of studies that examine the influence of geographical location and spatial resolution on $PM_{2.5}$ concentrations and exposures (Kheirbek et al. [2013](#page-10-0); Zhou et al. [2006\)](#page-12-0), questions remain about the level of geographical differentiation appropriate for LCIA and about how to properly characterize in LCIA the effects of differences in population age structure and disease incidence rates. Both issues appear to require further discussion. Based on that, the workshop participants agreed that it would be useful to develop regional and/or continental sets of archetype-based iF to account for differences in environmental conditions (e.g., climate, precipitation, background concentration of secondary $PM_{2.5}$ precursors), exposure conditions (e.g., population density, stack height), and receptor attributes (e.g., population age structure, disease incidence rates).

To account for differences in spatial scales, the workshop participants suggested developing LCIA methods that differentiate between near-field (e.g., occupational settings; within 10 m), neighborhood (scale of a block; order of 100 m), urban

(cities; order of 10–100 km), regional (order of 100– 1000 km), and continental scales (up to 10,000 km), thereby refining the archetypes used in Humbert et al. [\(2011](#page-10-0)).

The workshop participants also discussed the complex interactions between emissions of NH_3 , NO_x , and SO_x with respect to the formation and intake of secondary nitrates and sulfates. At the regional-continental scale, in areas with little agriculture and significant industrial activity (for example, along the east coast of the USA), emissions of $NH₃$ are a limiting factor for secondary $PM_{2.5}$ formation, whereas in rural areas dominated by agriculture, NO_x and SO_x are more commonly the factors limiting the formation of secondary PM2.5 (Paulot and Jacob [2014;](#page-11-0) Squizzato et al. [2013](#page-11-0); Xu and Penner [2012\)](#page-12-0). It was noted that geographically resolved data for primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursor emissions and iF for different emission heights are available for some regions (Apte et al. [2012;](#page-9-0) Levy et al. [2002;](#page-10-0) Pregger and Friedrich [2009\)](#page-11-0) but are not consistently available at the global level. It was agreed that in any attempt to differentiate geographical regions, particulate matter type (primary vs. secondary) is an important aspect to consider—secondary $PM_{2.5}$ iF are less sensitive than primary $PM_{2.5}$ iF to near source environmental, exposure, and receptor characteristics (Humbert et al. [2011;](#page-10-0) Levy et al. [2003\)](#page-10-0).

5.3 Aggregation of intake fractions

When combining iF from multiple sources, the appropriate approach is to multiply each emission's iF by the magnitude of that emission, sum this product for all emissions being combined, and then divide by the total emissions to obtain the emission-weighted iF for all the individual emissions that are linked by their association with a given functional unit in an LCA. In cases where emissions are not well characterized, it can be assumed that emissions (e.g., from vehicles or energy production) are proportional to population (Humbert et al. [2011](#page-10-0)). Population-weighted iF have been used in some studies as a proxy for emission-weighted iF (Apte et al. [2012](#page-9-0); Humbert et al. [2009](#page-10-0)), but other source- or sector-specific emission weights exist to account for spatial correlations between source locations and population patterns (Levy et al. [2002](#page-10-0); Lobscheid et al. [2012\)](#page-10-0).

For all cases where the region, emission sources and locations, and/or population exposure conditions are unknown, it was agreed to use an emission-weighted average iF (i.e., sitegeneric) in the context of LCIA, as population intake is the result of multiplying iF by the corresponding emissions. To arrive at such emission weights, the workshop participants suggested that the iF of each region/area (e.g., Indochina, Scandinavia) should be weighted according to the proportion of the contribution of this region to the total emission in the considered geographical domain (typically continental or global scale). This approach would be entirely consistent with

previous efforts to develop iF values intended to be used to quantify the impact of $PM_{2.5}$ or $PM_{2.5}$ precursor emissions on ambient PM_{2.5} concentrations (Humbert et al. [2011](#page-10-0); Levy et al. [2003](#page-10-0); Marshall et al. [2003;](#page-11-0) Tainio et al. [2009](#page-11-0)).

6 From concentration and human intake to health effects: defining appropriate exposure-response functions

6.1 Effect assessment starting point

In LCIA, ERF link estimates of population exposure with estimates of health effects. Whereas some guidance is available on deriving $PM_{2.5}$ intake fractions for use in LCIA (Humbert et al. [2011\)](#page-10-0), guidance has not yet been established on the development of $PM_{2.5}$ exposure-response to support LCIA.

Workshop participants agreed that models developed in support of the Global Burden of Disease Study (GBD) 2010 (Lim et al. [2012\)](#page-10-0) may provide a reasonable framework for calculating health effects of $PM_{2.5}$ exposure. GBD 2010 provides estimates of the health effects (expressed in DALY) caused by 67 risk factors for both 1990 and 2010. GBD estimates are provided for each of 21 world regions (based on epidemiological homogeneity and geographical contiguity) and are disaggregated by age (20 groups) and sex. $PM_{2.5}$ as one of the considered risk factors was associated with five adverse health effects—ischemic heart disease, cerebrovascular disease, cancers of the trachea/bronchus or lung, chronic obstructive pulmonary disease among adults $(\geq 25$ years old), and lower respiratory infections among young children (\leq) years old). For these effects, risk estimates were developed using an integrated exposure-response (IER) function which provided cause-specific estimates of the relative risk as a function of the ambient $PM_{2.5}$ concentration over a broad range of exposures from the counterfactual or threshold level to concentrations on the order of 100 μ g/m³ (Burnett et al. [2014\)](#page-9-0). This model was labeled "integrated" because it combined evidence from studies of the health effects of ambient $PM_{2.5}$ with studies of the effects of active and passive smoking. Other health effects were not considered because epidemiological evidence was either inconclusive or absent.

GBD 2010 not only computes the relative risks of various health effects as a function of ambient $PM_{2.5}$ concentrations but also assigns DALY to each of the five health outcomes studied. In their 2010 analysis, GBD uses DALY that (a) are neither age-weighted nor discounted, (b) are derived using a counterfactual life expectancy at birth of 86 years for both males and females derived from the lowest age-specific death rates observed in any country (Murray et al. [2012\)](#page-11-0), and (c) using disability weights derived from population-based household surveys involving 13,902 participants from

Bangladesh, Indonesia, Peru, South Africa, Tanzania, and the USA and an Internet-based survey of 16,328 participants from 167 countries, 44 % of whom were from the USA (Salomon et al. [2012](#page-11-0)). The approach applied in GBD 2010 to derive DALY that are not age-weighted or discounted is consistent with current LCIA practice (EC [2010c](#page-10-0)).

In summary, the workshop participants consider the GBD 2010 models for the relative risks of the five health effects as a function of ambient $PM_{2.5}$ concentrations as suitable starting points for developing ERF for use in LCIA. Because PM_{2.5} exposures associated with LCA applications and populations differ from those addressed in the GBD study, the question of whether the GBD 2010 disability weights for $PM_{2.5}$ are well suited to be directly applied in LCIA requires further discussion. Currently, the workshop participants consider the GBD 2010 disability weights a useful starting point.

6.2 Health effects

Health effects associated with $PM_{2.5}$ exposure include a wide range of diseases. To date, PM exposure-response functions used in LCIA have focused on chronic and acute mortality and acute respiratory and cardiovascular morbidity associated with exposure to PM_{10} (van Zelm et al. [2008](#page-11-0)) or on cardiopulmonary mortality and lung cancer attributable to chronic exposure to $PM_{2.5}$ (Gronlund et al. [2014\)](#page-10-0). ERF have been derived using several approaches discussed in EC ([2010c\)](#page-10-0), primarily based on results from the Harvard Six Cities and American Cancer Society studies (Krewski et al. [2000;](#page-10-0) Laden et al. [2006;](#page-10-0) Pope III et al. [2002\)](#page-11-0). Although the impact of $PM_{2.5}$ exposure on asthma has been reported in several epidemiological studies (Brauer et al. [2002](#page-9-0); Kheirbek et al. [2013](#page-10-0); Künzli et al. [2000\)](#page-10-0), asthma is usually not considered in LCIA. At the workshop, it was noted that evidence linking PM2.5 exposure with new asthma incidences is inconclusive, whereas it does support a link between $PM_{2.5}$ exposure and the exacerbation of existing asthma (Donaldson et al. [2000](#page-10-0); Gavett and Koren [2001;](#page-10-0) Pope III et al. [1995](#page-11-0)). However, since it is unclear how to differentiate between induction of new cases and exacerbation of existing disease, there was no agreement on whether, and if so how, to include asthma as a health effect in LCIA.

It was emphasized that, in addition to the GBD 2010 effort, there is a large European movement to decide which health effects associated with $PM_{2.5}$ exposure to quantify. This involves two projects³—the Health Risks of Air Pollution in Europe, HRAPIE (WHO [2013a](#page-12-0)), and Review of Evidence on Health Aspects of Air Pollution, REVIHAAP (WHO [2013b\)](#page-12-0). These projects aim to provide advice in support of the

comprehensive review of the European Union's air quality policies scheduled for 2013. A consensus document reflecting this effort was published at the end of 2013 (WHO [2013a\)](#page-12-0). Whereas the GBD 2010 effort focuses on cause-specific mortality, the HRAPIE/REVIHAAP projects recommend allcause analysis as primary choice and cause-specific analysis as alternative method based on similarity of the frequency of the causes of death linked with exposure between considered cohorts and countries. It can be argued that a cause-specific assessment is particularly important in global assessments because of the large geographical variability in the relative importance of various causes of death. This view is supported by several studies (Lipsett et al. [2011](#page-10-0); Miller et al. [2007;](#page-11-0) Puett et al. [2009](#page-11-0), [2011\)](#page-11-0).

Considering these different approaches, the workshop participants agreed to recommend that LCIA should assess causespecific mortality, when feasible, whereas all-cause mortality along with an appropriate assessment of uncertainty might still be useful in case of inconclusive allocation to causes. Furthermore, health effects considered in GBD 2010 and in the HRAPIE consensus document should serve as a starting point.

6.3 Shape of exposure-response functions

In current LCIA practice, the shape of population ERF is usually assumed to be linear with no threshold. This approach is supported by several studies which find no evidence of a departure from linearity (Chen et al. [2013](#page-9-0); Schwartz et al. [2008;](#page-11-0) Stafoggia et al. [2013](#page-11-0); WHO [2006\)](#page-11-0) and no evidence suggesting a threshold at the population level (COMEAP [2009,](#page-9-0) [2010](#page-10-0)). Despite this, when these linear functions are applied to the very high $PM_{2.5}$ levels often found in developing countries, the estimates of risk are so high as to be implausible (Abrahamowicz et al. [2003](#page-9-0); EC [2010b](#page-10-0)). Recently, several research groups have suggested nonlinear ERF that could be applied across a large range of $PM_{2.5}$ concentrations, from very low to very high $PM_{2.5}$ concentrations. These are typically steep at low concentration levels and relatively flat at high levels (Abrahamowicz et al. [2003;](#page-9-0) Burnett et al. [2014;](#page-9-0) Ostro [2004](#page-11-0); Pope III et al. [2009](#page-11-0)). Whether and, if so, how this approach can be adapted for use in LCIA needs to be further discussed, acknowledging that LCA aims to support decisions in regions with low concentration levels and also in regions with high concentration levels. From a sustainability point of view, intervention in highly polluted areas may be a priority despite the lower response per unit exposure. Significant departures from linearity would imply that iF would need to be reconstructed in a manner that is stratified by $PM_{2.5}$ concentration or other relevant factors. In making such a change, it is also important to realize that the shape of the ERF might be effect-specific for example, nearly linear for lung cancer but substantially

³ [http://www.euro.who.int/en/what-we-do/health-topics/environment](http://www.euro.who.int/en/what-we-do/health-topics/environment-and-health/air-quality/activities/health-aspects-of-air-pollution-and-review-of-eu-policies-the-revihaap-and-hrapie-projects)[and-health/air-quality/activities/health-aspects-of-air-pollution-and](http://www.euro.who.int/en/what-we-do/health-topics/environment-and-health/air-quality/activities/health-aspects-of-air-pollution-and-review-of-eu-policies-the-revihaap-and-hrapie-projects)[review-of-eu-policies-the-revihaap-and-hrapie-projects](http://www.euro.who.int/en/what-we-do/health-topics/environment-and-health/air-quality/activities/health-aspects-of-air-pollution-and-review-of-eu-policies-the-revihaap-and-hrapie-projects)

nonlinear for cardiovascular mortality (Pope III et al. [2011](#page-11-0)). In GBD 2010, effect-specific, integrated ERF are proposed for $PM_{2.5}$ (Lim et al. [2012\)](#page-10-0). These ERF express relative risk as an exponential function (or a power function) of $PM_{2.5}$ concentration (Burnett et al. [2014](#page-9-0)). In order to apply such nonlinear ERF in LCIA, nonlinear models can either be directly applied as, e.g., in van Zelm et al. [\(2008\)](#page-11-0) for ozone formation or be decomposed into piecewise linear functions. The workshop participants explained that methods for applying this approach are currently being developed.

In summary, it was agreed to further discuss how the ERF from GBD 2010 together with recommendations from the HRAPIE project can be adapted to serve as starting points. Thereby, the workshop participants acknowledge that the slope of any linear ERF will vary as a function of different PM2.5 concentration ranges. LCIA methods will therefore need to be developed which can account for the variation in background levels of ambient $PM_{2.5}$ around the world. This is challenging because in an LCA framework, the exact geographical locations of individual emission sources are typically unknown (Finnveden et al. [2009;](#page-10-0) Hauschild [2005](#page-10-0); Humbert et al. [2011](#page-10-0)). Even if the source locations were known, the LCA analyst would need to integrate concentrations (and risks) over large areas, including individuals quite close to the source as well as those far from the source, to capture the entire exposed population. In principle, this can be addressed by treating the location of the emission source as uncertain and computing the distribution of possible impacts and recognizing this as a source of uncertainty in estimates of health impact.

6.4 Particle characteristics and differential toxicity

PM_{2.5} mass is commonly used as an indicator of the risk associated with exposure to a mixture of particle-related pollutants (of different sizes below 2.5 μm diameter) from diverse (primary or secondary) sources and in different environments (COMEAP [2009;](#page-9-0) Lim et al. [2012;](#page-10-0) Pope III et al. [2009,](#page-11-0) [2011\)](#page-11-0). This approach, which implicitly assumes equal toxicity of PM2.5 constituents per mass unit, is commonly used in LCIA (Potting et al. [2007\)](#page-11-0). There is currently no scientific consensus on the relative toxicity of various constituents of PM_{2.5}. This, however, does not suggest that all particle constituents are in fact equally toxic, but instead that the toxicological and epidemiological evidence of differential toxicity is inconclusive (Hurley et al. [2005](#page-10-0)). One study found differential toxicity of multiple particle constituents for short-term exposure effects on hospital admissions (Levy et al. [2012](#page-10-0)), but further research is required to address other health outcomes, long-term exposure, and other geographical settings (Rohr and Wyzga [2012](#page-11-0)).

In view of this, it was agreed to use $PM_{2.5}$ mass as an indicator of exposure without differentiating between and

among primary and secondary $PM_{2.5}$ and without differentiating between different $PM_{2.5}$ constituents in terms of toxicity for cause-specific chronic mortality effects. However, the workshop participants understood that given the current state of scientific uncertainty about this matter, it would be important to develop an approach for characterizing the uncertainty of the toxicity of various constituents of $PM_{2.5}$ which reflects the lack of knowledge about which constituents of $PM_{2.5}$ are in fact responsible for the toxicity of the mixture.

Another aspect in the discussion of particle characteristics is particle size. Experimental studies suggest that health effects from exposure to the ultrafine particle (UFP) fraction differ from those of larger particles due to distinct deposition patterns in the lung and clearance mechanisms (Oberdörster et al. [2005](#page-11-0)). There is epidemiological and toxicological evidence for specific adverse respiratory and cardiovascular effects from exposure to UFP (Delfino et al. [2005](#page-10-0); Weichenthal et al. [2007\)](#page-11-0). However, the limited evidence currently available is inconsistent for short-term exposure and does not yet address the impacts of long-term exposure (Rückerl et al. [2011\)](#page-11-0). Thus, it is not yet possible to determine how health effects associated with exposure to UFP differ from those associated with exposure to larger particles (HEI [2013](#page-10-0)). Moreover, there is only limited literature that would allow for calculating iF for UFP, which is generally characterized by particle number rather than particle mass.

As a result, the workshop participants decided not to separately incorporate UFP into LCIA at present but suggested that in the future, a correction factor might be introduced to account for the distribution of particle sizes.

7 Conclusions and next steps

7.1 Conclusions

The workshop participants discussed the questions shown in Table [1](#page-4-0) in an effort to find ways to refine and improve the overall framework and to suggest data and models that could harmonize the analysis of health impacts from exposure to ambient particulate matter. This discussion constituted a first step towards developing recommendations for addressing the health effects from exposure associated with emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors in LCIA. A set of 10 recommendations reflecting the consensus of the workshop participants are summarized as follows:

- & The intake fraction framework proposed by Humbert et al. [\(2011](#page-10-0)) provides a useful starting point for assessing health effects of ambient PM in LCIA with a focus on $PM_{2.5}$.
- & Human intake fractions can be used to estimate emissionrelated population exposure. In conjunction with

population-averaged breathing rates, intake fractions can be used to estimate intake from air concentrations.

- Disability-adjusted life years without age weighting or discounting, which aggregate mortality and morbidity, can be used as a summary health metric.
- For most cases, where emission locations are unknown, exposure scenario archetypes provide a useful approach to account for factors, such as population density, emission height, and exposure to PM_{2.5} from indoor sources, which influence human intake fractions. The decision whether additional archetypes are necessary should be based on a sensitivity analysis that considers the importance of these additional factors in reducing uncertainty in exposure estimates. When the exact emission location is known, spatially explicit fate and transport models should be used.
- Geographical archetypes of intake fractions should be established for indoor, near-field, neighborhood, urban, regional, and continental scales. Geographical differentiation should be further discussed and analyzed with respect to scale and nonlinear chemical processes in the formation of secondary $PM_{2.5}$.
- Emission-weighted average intake fractions should be used in cases where the nature of the emission sources and/or exposure conditions is unclear.
- The Global Burden of Disease Study 2010 is considered to provide a useful starting point for developing exposureresponse functions for assessing $PM_{2.5}$ -related health effects in LCIA.
- Cause-specific mortality can provide a more informative basis for developing LCIA characterization factors than all-cause mortality. Assumptions for age- and causespecific disability weights should be further discussed and analyzed.
- & Nonlinear exposure-response functions are recommended in the Global Burden of Disease Study 2010, whereas linear functions are used in the consensus document of the Health Risks of Air Pollution in Europe projects. There remains a need for discussion about whether and, if so, how to integrate nonlinear (or piecewise linear) exposureresponse functions into LCIA.
- $PM_{2.5}$ mass can be used as the indicator of the health risk associated with PM inhalation exposure in LCIA. There is no justification at this time to differentiate between different primary/secondary PM_{2.5} sources or between different PM2.5 particle sizes regarding toxicity. However, analyses should report the uncertainties inherent in any assumptions made about the relative toxicity of various types of particles.

7.2 Next steps

Within the next 2 years, the goals of the task force on human health impacts are to build a global guidance framework and

to determine characterization factors for incorporating the health effects from exposure to $PM_{2.5}$ in LCIA and for including both indoor and outdoor releases. As next steps towards these goals, the first set of recommendations from the Basel Guidance Workshop will be taken. Open questions and unsolved problems that were pointed out by the workshop participants will be further studied, and the proposed framework will be refined based on the best available data and methods. The harmonized framework and related results will finally be presented at a Pellston Technical Workshop⁴ in 2015.

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⁴ Pellston Workshops are preeminent workshops held by the SETAC, each of which brings together leading scientists from academia, business, and governments around the world and focuses on a relevant environmental topic with proceedings published as a peer-reviewed report, book, or journal article compilation.

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