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1 **Health Effects of Fine Particulate Matter in Life Cycle Impact Assessment:**
2 **Findings from the Basel Guidance Workshop**

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36

37 **Abstract**

38 *Purpose* Fine particulate matter (PM_{2.5}) is considered to be one of the most important
39 environmental factors contributing to the global human disease burden. However, due to the
40 lack of broad consensus and harmonization in the life cycle assessment (LCA) community,
41 there is no clear guidance on how to consistently include health effects from PM_{2.5} exposure
42 in LCA practice. As a consequence, different models are currently used to assess life cycle
43 impacts for PM_{2.5}, sometimes leading to inconsistent results. In a global effort initiated by the
44 UNEP/SETAC Life Cycle Initiative, respiratory inorganics impacts expressed as health
45 effects from PM_{2.5} exposure were selected as one of the initial impact categories to undergo
46 review with the goal of providing global guidance for implementation in life cycle impact
47 assessment (LCIA). The goal of this paper is to summarize the current knowledge and
48 practice for assessing health effects from PM_{2.5} exposure and to provide recommendations for
49 their consistent integration into LCIA.

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

57 *Results and recommendations* A set of 10 recommendations was developed addressing:
58 (a) the general framework for assessing PM_{2.5}-related health effects, (b) approaches and data
59 to estimate human exposure to PM_{2.5} using intake fractions, and (c) approaches and data to
60 characterize exposure-response functions (ERF) for PM_{2.5} and to quantify severity of the
61 diseases attributed to PM_{2.5} exposure. Despite these advances, a number of complex issues,
62 such as those related to non-linearity of the ERF and the possible need to provide different
63 ERF's for use in different geographic regions, require further analysis.

64 *Conclusions and outlook* Questions of how to refine and improve the overall framework
65 were analyzed. Data and models were proposed for harmonizing various elements of the
66 health impact pathways for PM_{2.5}. Within the next two years, our goal is to build a global
67 guidance framework and to determine characterization factors that are more reliable for
68 incorporating the health effects from exposure to PM_{2.5} into LCIA. Ideally, this will allow
69 quantification of the impacts of both indoor and outdoor exposure to PM_{2.5}.

70

71 **Keywords**

72 fine particulate matter, air pollution, human health effects, intake fraction, exposure-response

73 function, global guidance, life cycle impact assessment (LCIA)

74

Accepted post-print

75 **1 Health effects from fine particulate matter: Towards global guidance in life cycle**
76 **assessment**

77 Life cycle assessment (LCA) is a structured, comprehensive, and internationally
78 standardized method to assess potential environmental impacts and resources used throughout
79 the life cycle of a good or service in a comparable way (ISO 2006). LCA thereby aims for
80 best estimates in the modelling of all relevant impacts on the natural environment, human
81 health, and resources in the life cycle impact assessment (LCIA) phase (EC 2010a, Finnveden
82 et al. 2009). To help identify best LCA practice, Phase III (2012-2016) of the UNEP/SETAC
83 Life Cycle Initiative¹ has launched a flagship project aiming to provide global guidance and
84 consensus on a limited number of LCIA indicators. The Glasgow Scoping Workshop in May
85 2013 (Jolliet et al. 2014) focused on establishing a tentative short list of impact category
86 indicators that would be addressed during two consensus building periods. These indicators
87 included the impacts of respiratory inorganics expressed as health effects from exposure to
88 primary and secondary particulate matter (PM), which is considered to be one of the most
89 important environmental stressors contributing to the global human disease burden (Hänninen
90 et al. 2014, Lim et al. 2012). Primary PM refers to directly emitted particles. Secondary PM
91 refers to organic and inorganic (e.g. ammonium nitrate, ammonium sulfate) particles formed
92 through reactions of precursor substances including nitrogen oxides (NO_x), sulfur oxides
93 (SO_x), ammonia (NH₃), semivolatile and volatile organic compounds (VOC), of which the
94 latter are most important for secondary organic aerosol formation. PM is further distinguished
95 according to aerodynamic diameter, i.e. respirable particles (PM₁₀) with <10 µm, fine
96 particles (PM_{2.5}) with <2.5 µm, and ultrafine particles (UFP) with <100 nm aerodynamic
97 diameter (WHO 2006). PM_{2.5} was chosen to provide international recommendations regarding
98 the consistent integration of its health effects into LCIA because it might best describe the
99 component of particulate matter responsible for adverse health effects (Harrison & Yin 2000,
100 Lim et al. 2012, Lippmann & Chen 2009).

101

102 **2 Assessing fine particulate matter in the context of life cycle impact assessment**

103 In epidemiological studies, exposure to PM_{2.5} is associated with various adverse health
104 effects and reduction in life expectancy including chronic and acute respiratory and
105 cardiovascular morbidity, chronic and acute mortality, lung cancer, diabetes, and adverse birth
106 outcomes (Beelen et al. 2014, Brook et al. 2010, Chen et al. 2008, COMEAP 2010, Dadvand
107 et al. 2013, Hoek et al. 2013, Künzli et al. 2000, Lippmann & Chen 2009, Loomis et al. 2013,

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

108 Mehta et al. 2013, Pelucchi et al. 2009, Pope III et al. 2009, Pope III et al. 2011, Straif et al.
109 2013). Furthermore, toxicological studies support the observation that exposure to PM_{2.5} can
110 exert effects on key biological systems, with some evidence that not all particles are likely to
111 cause the same health effects (Harrison & Yin 2000, Kelly & Fussell 2012, Rohr & Wyzga
112 2012, Stanek et al. 2011). Several existing LCIA methods already characterize health effects
113 associated with ambient PM or PM_{2.5} concentrations (EC 2010c), mostly based on ambient
114 PM_{2.5} intake estimated from simple exposure or intake fraction models and using health effect
115 data from the Harvard Six Cities and American Cancer Society studies (Krewski et al. 2000,
116 Laden et al. 2006, Pope III et al. 2002). A few studies include spatial allocation of emissions
117 and modeling of air dispersion and chemical reactions to predict downwind PM_{2.5}
118 concentrations (Hill et al. 2009, Tessum et al. 2012). Whenever emission locations are known,
119 these spatially-explicit approaches can be applied in LCIA. It is anticipated in the future to
120 fully assess PM_{2.5} impacts using such spatially explicit approaches. In the current absence of
121 this capacity, a consistent and globally harmonized approach for LCIA should be based on
122 the most recent science to simultaneously address environmental fate, human exposure, and
123 health effects of PM_{2.5} concentrations resulting from emissions of primary PM_{2.5} and
124 secondary PM_{2.5} precursors (Hauschild et al. 2013).

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

139 With respect to assessing the particular health effects from PM_{2.5} exposure, the effort of
140 an earlier UNEP/SETAC working group has designed a framework and proposed a set of
141 default iF associated with PM_{2.5} emissions for use in LCIA (Humbert et al. 2011). This effort

142 is limited to the steps of the impact pathway from emissions to concentration and human
143 intake, but does not cover the steps from human intake to health effects. In addition, due to
144 the lack of broad consensus and harmonization in the LCA community, there is no clear
145 guidance on how to include health effects from PM_{2.5} exposure in LCA practice. As a
146 consequence, different models are currently used leading at times to inconsistent life cycle
147 impact results reported for this category. This reveals the importance of pursuing consensus
148 building, based on the initial work of Humbert et al. (2011) and combining it with latest
149 exposure-response and severity data to yield revised guidance on the development and use of
150 human health characterization factors for both primary and secondary PM_{2.5} including
151 precursor substances. Ultrafine particles are currently not separately considered in LCA.

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

160

161 **3 The Basel Guidance Workshop: Identifying and addressing the key questions**

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

170 The specific objectives of the workshop were to first identify and discuss the main
171 scientific questions and challenges for quantifying human health effects from PM_{2.5} exposure,
172 and then to provide initial guidance to the impact quantification process. Three main topics
173 were addressed at the workshop: (a) the general assessment framework as proposed by
174 Humbert et al. (2011), (b) approaches and data to determine human exposure to PM_{2.5}
175 expressed as intake fractions, and (c) approaches and data to determine exposure-response

176 functions (ERF) for PM_{2.5} along with disease severity. For these topics, the workshop
 177 participants discussed a set of key questions that had been established with selected experts in
 178 three pre-workshop phone conferences. Table 1 summarizes these key questions, which are
 179 discussed in detail in the following.

180

181 **Table 1** Key questions discussed during the Basel Guidance Workshop

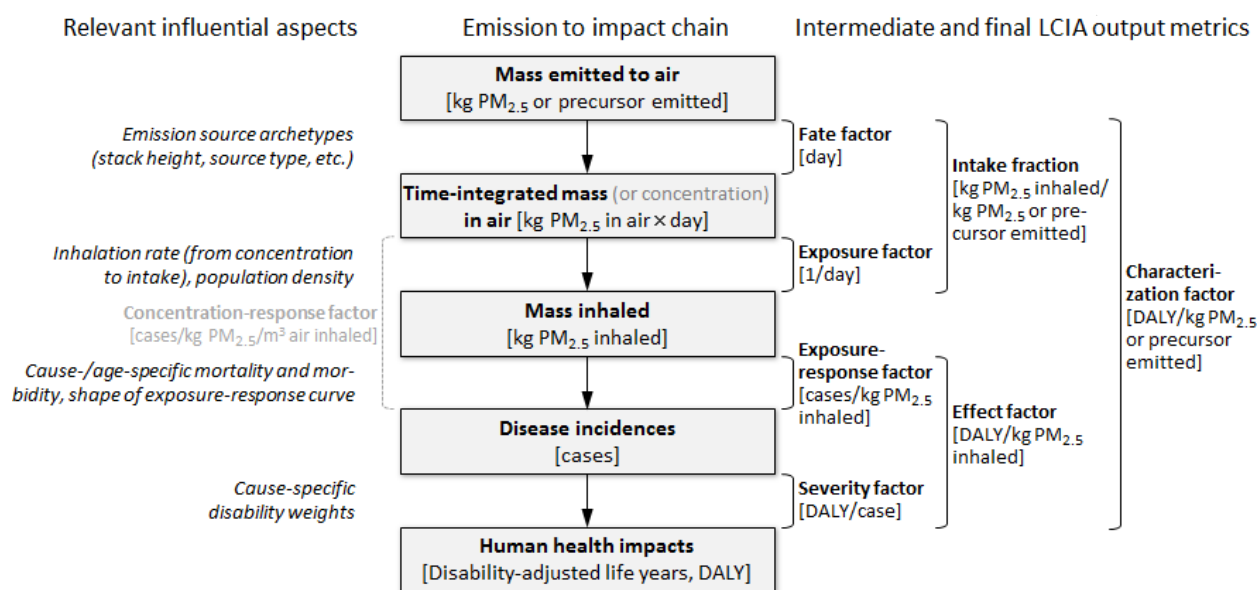
<p>1. General assessment framework</p> <ul style="list-style-type: none"> ▪ Can we use the framework that is proposed in Figure 1 based on work from Humbert et al. (2011) to include health effects from respiration of ambient particulate matter into life cycle impact assessment?
<p>2. Human intake fraction</p> <ul style="list-style-type: none"> ▪ What additional factors/aspects will we have to take into account, i.e. those that substantially influence intake fractions by at least a factor of two? ▪ Can we use archetypes to disaggregate aspects influencing intake fractions (emission stack height, primary/secondary particulates, particle size, and urban/rural/remote area) and what archetypal structure is meaningful? ▪ What is the added value of applying archetypes for emission sources (e.g. road transport) or specific regions (e.g. China)? ▪ How do we arrive at a consistent set of emission-weighted average intake fractions? ▪ Which existing studies, methods, and models are best or most usefully suited as starting points for arriving at a consistent set of intake fractions to improve factors stated in Humbert et al. (2011)? ▪ How can we properly address in life cycle impact assessment the combined environmental fate aspects of ammonia, nitrogen oxides, and sulfur oxides?
<p>3. Exposure-response functions and effect evaluation</p> <ul style="list-style-type: none"> ▪ What are the major studies that we need to take into account to determine exposure-response functions for relevant health effects? ▪ In addition to cardiovascular diseases and lung cancer, is it relevant to include other health effects, such as bronchitis or asthma in children? ▪ To what extent are exposure-response functions available for the fraction of particulates with an aerodynamic diameter below 2.5 µm and what alternative approach would be applicable? ▪ Are there any emerging studies that would challenge our default approach? ▪ Are there studies providing evidence and specific exposure-response functions for differentiated effects for primary and secondary particulates? ▪ What relevant exposure studies are available for exposure to particulates from indoor sources and for outdoor particulates emissions in different parts of the world? ▪ How far can studies focusing on the United States (or studies mentioned under the first question of point 3) be applied as a default for different parts of the world or for indoor exposure to particulates? ▪ How can we consistently account for the severity of different (mortality and morbidity) health effects based on disability weights? ▪ How can damage measures be suggested in order of priority in terms of health effects?
<p>4. Additional remarks</p> <ul style="list-style-type: none"> ▪ What additional comments or recommendations could improve the set of intake

fractions, exposure-response functions and severity factors?

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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 Figure 1.



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Figure 1 Proposed framework for assessing human health effects from fine particulate matter exposure in life cycle impact assessment; adapted from Humbert et al. (2011).

4.1 Overall assessment approach

There was agreement among the workshop participants to build upon the general framework proposed by Humbert et al. (2011). 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) 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_{2.5} precursor emitted, respectively), an *exposure-response 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*

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.

203 *factor*, SF (disability-adjusted life years, DALY per disease case), to arrive at a human health-
204 related impact score, IS (DALY):

$$205 \quad IS = m \times \underbrace{iF \times ERF \times SF}_{CF} \quad (1)$$

206 Intake fraction, exposure-response factor and severity factor can be represented by the
207 characterization factor, CF (DALY per mass emitted). A key assumption implicit in this
208 framework is the linear, no-threshold ERF. While not uncontroversial, this assumption
209 reflects current practice and recent recommendations in LCIA (EC 2010b, Potting et al.
210 2007), and is also applied in other studies as discussed e.g. in COMEAP (2009).

211

212 4.2 Exposure metrics

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

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

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

245

246 4.3 Health metrics

247 Various health metrics were discussed, including total and premature mortality, years of
248 life lost (YLL), and disability-adjusted life years (DALY). Most workshop participants felt
249 that when death is the outcome of interest, YLL is a better measure of mortality impacts than
250 numbers of deaths. The view was that information on the number of deaths is more
251 challenging to interpret because reduced PM_{2.5} intake cannot affect the fact of death, but only
252 its cause and timing (Leksell & Rabl 2001, Rabl 2005). When it is necessary to combine
253 mortality and morbidity impacts into a single summary measure, two approaches can be used.
254 The first approach is to use DALY combining YLL and years lived with a disability (YLD)
255 weighted by the quality of life during the period of disability (Murray & Lopez 1996b,
256 Murray & Lopez 1996a). The second approach, which is frequently preferred by economists,
257 is to use weights reflecting societal willingness to pay to avoid small incremental risks of
258 mortality and morbidity.

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

265

266 4.4 Other framework discussion points

267 Two additional aspects were briefly discussed at the workshop: (i) whether and, if so,
268 how to address the dynamics when expressing of health impacts attributable to PM_{2.5}
269 exposure, and (ii) how to account for differences between average and marginal impacts on
270 health of primary and secondary PM_{2.5} precursor emissions, which may occur when either

271 emissions-exposure or exposure-response functions exhibit thresholds or significant non-
272 linearities. The workshop participants agreed that in the long-term both issues require further
273 attention.

274

275 **5 From emissions to concentration and human intake: Determining intake fractions**

276 5.1 Archetypes structure

277 In LCIA, it is common practice to make use of archetypal exposure scenarios, e.g. urban
278 vs. rural scenarios (Riley et al. 2002), rather than site-specific exposure assessments,
279 especially when emission locations are unknown. The workshop discussion focused on
280 identifying the key factors influencing iF and determining how to address these in the context
281 of quantifying PM_{2.5}-related health effects in LCIA. Table 3 in Humbert et al. (2011)
282 proposed one such archetypal structure in which population density (urban, rural, remote) and
283 emission height (high-stack, low-stack, ground-level) serve as the main determinants of iF.
284 Humbert et al. also provided a default set of iF values corresponding to these archetypes.

285 Workshop participants agreed to adopt this structure as starting point, but pointed out that
286 additional refinements in terms of archetypes need to be explored. Refinements can thereby
287 build on applying a sensitivity analysis to a range of aspects that influence the variability of
288 iF. This includes, for example, distinct urban areas based on work by Apte et al. (2012) and
289 different emission sources, such as traffic-related sources (Greco et al. 2007, Lobscheid et al.
290 2012, Marshall et al. 2005), stationary emissions from coal/gas-fired power plants (Heath et
291 al. 2006, Levy et al. 2003, Levy et al. 2002), or indoor emissions from wood burning (Ries et
292 al. 2009). The participants also agreed to include additional archetypes reflecting exposure
293 from indoor emissions of PM_{2.5} based on work by Hellweg et al. (2009).

294

295 5.2 Geographical differentiation

296 Despite the availability of studies that examine the influence of geographical location and
297 spatial resolution on PM_{2.5} concentrations and exposures (Kheirbek et al. 2013, Zhou et al.
298 2006), questions remain about the level of geographical differentiation appropriate for LCIA
299 and about how to properly characterize in LCIA the effects of differences in population age
300 structure and disease incidence rates. Both issues appear to require further discussion. Based
301 on that, workshop participants agreed that it would be useful to develop regional and/or
302 continental sets of archetype-based iF to account for differences in environmental conditions
303 (e.g. climate, precipitation, background concentration of secondary PM_{2.5} precursors),

304 exposure conditions (e.g. population density, stack height), and receptor attributes (e.g.
305 population age structure, disease incidence rates).

306 To account for differences in spatial scales, workshop participants suggested developing
307 LCIA methods that differentiate between near-field (e.g. occupational settings; within 10 m),
308 neighborhood (scale of a block; order of 100 m), urban (cities; order of 10-100 km), regional
309 (order of 100-1,000 km), and continental scales (up to 10,000 km), thereby refining the
310 archetypes used in Humbert et al. (2011).

311 Workshop participants also discussed the complex interactions between emissions of
312 NH_3 , NO_x and SO_x with respect to the formation and intake of secondary nitrates and sulfates.
313 At the regional-continental scale, in areas with little agriculture and significant industrial
314 activity (for example, along the east coast of the US), emissions of NH_3 are a limiting factor
315 for secondary $\text{PM}_{2.5}$ formation, whereas in rural areas dominated by agriculture, NO_x and SO_x
316 are more commonly the factors limiting the formation of secondary $\text{PM}_{2.5}$ (Paulot & Jacob
317 2014, Squizzato et al. 2013, Xu & Penner 2012). It was noted that geographically-resolved
318 data for primary $\text{PM}_{2.5}$ and secondary $\text{PM}_{2.5}$ precursor emissions and iF for different emission
319 heights are available for some regions (Apte et al. 2012, Levy et al. 2002, Pregger & Friedrich
320 2009), but are not consistently available at the global level. It was agreed that in any attempt
321 to differentiate geographic regions, particulate matter type (primary vs. secondary) is an
322 important aspect to consider – secondary $\text{PM}_{2.5}$ iF are less sensitive than primary $\text{PM}_{2.5}$ iF to
323 near source environmental, exposure and receptor characteristics (Humbert et al. 2011, Levy
324 et al. 2003).

325

326 5.3 Aggregation of intake fractions

327 When combining iF from multiple sources, the appropriate approach is to multiply each
328 emission's iF by the magnitude of that emission, sum this product for all emissions being
329 combined and then divide by the total emissions to obtain the emissions-weighted iF for all
330 the individual emissions that are linked by their association with a given functional unit in an
331 LCA. In cases where emissions are not well characterized, it can be assumed that emissions
332 (e.g. from vehicles or energy production) are proportional to population (Humbert et al.
333 2011). Population-weighted iF have been used in some studies as a proxy for emission-
334 weighted iF (Apte et al. 2012, Humbert et al. 2009), but other source- or sector-specific
335 emission-weights exist to account for spatial correlations between source locations and
336 population patterns (Levy et al. 2002, Lobscheid et al. 2012).

337 For all cases where the region, emission sources and locations, and/or population
338 exposure conditions are unknown, it was agreed to use an emission-weighted average iF (i.e.,
339 site-generic) in the context of LCIA, as population intake is the result of multiplying iF by the
340 corresponding emissions. To arrive at such emission-weights, the workshop participants
341 suggested that the iF of each region/area (e.g. Indochina, Scandinavia) should be weighted
342 according to the proportion of the contribution of this region to the total emission in the
343 considered geographical domain (typically continental or global scale). This approach would
344 be entirely consistent with previous efforts to develop iF values intended to be used to
345 quantify the impact of PM_{2.5} or PM_{2.5} precursor emissions on ambient PM_{2.5} concentrations
346 (Humbert et al. 2011, Levy et al. 2003, Marshall et al. 2003, Tainio et al. 2009).

347

348 **6 From concentration and human intake to health effects: Defining appropriate** 349 **exposure-response functions**

350 6.1 Effect assessment starting point

351 In LCIA, ERF link estimates of population exposure with estimates of health effects.
352 Whereas some guidance is available on deriving PM_{2.5} intake fractions for use in LCIA
353 (Humbert et al. 2011), guidance has not yet been established on the development of PM_{2.5}
354 exposure-response to support LCIA.

355 Workshop participants agreed that models developed in support of the Global Burden of
356 Disease Study (GBD) 2010 (Lim et al. 2012) may provide a reasonable framework for
357 calculating health effects of PM_{2.5} exposure. GBD 2010 provides estimates of the health
358 effects (expressed in DALY) caused by 67 risk factors for both 1990 and 2010. GBD
359 estimates are provided for each of 21 world regions (based on epidemiological homogeneity
360 and geographical contiguity) and are disaggregated by age (20 groups) and sex. PM_{2.5} as one
361 of the considered risk factors was associated with five adverse health effects – ischemic heart
362 disease, cerebrovascular disease, cancers of the trachea/bronchus or lung, chronic obstructive
363 pulmonary disease among adults (≥25 years old), and lower respiratory infections among
364 young children (≤5 years old). For these effects, risk estimates were developed using an
365 integrated exposure-response (IER) function which provided cause-specific estimates of the
366 relative risk as a function of the ambient PM_{2.5} concentration over a broad range of exposures
367 from the counterfactual or threshold level to concentrations on the order of 100 µg/m³
368 (Burnett et al. 2014). This model was labelled “integrated” because it combined evidence
369 from studies of the health effects of ambient PM_{2.5} with studies of the effects of active and

370 passive smoking. Other health effects were not considered, because epidemiological evidence
371 was either inconclusive or absent.

372 GBD 2010 not only computes the relative risks of various health effects as a function of
373 ambient PM_{2.5} concentrations, but also assigns DALY to each of the five health outcomes
374 studied. In their 2010 analysis, GBD uses DALY that (a) are neither age-weighted nor
375 discounted, (b) were derived using a counterfactual life expectancy at birth of 86 years for
376 both males and females derived from the lowest age-specific death rates observed in any
377 country (Murray et al. 2012), and (c) using disability weights derived from population-based
378 household surveys involving 13,902 participants from Bangladesh, Indonesia, Peru, South
379 Africa, Tanzania and the United States and an internet-based survey of 16,328 participants
380 from 167 countries, 44% of whom were from the United States (Salomon et al. 2012). The
381 approach applied in GBD 2010 to derive DALY that are not age-weighted or discounted, is
382 consistent with current LCIA practice (EC 2010c).

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

390

391 6.2 Health effects

392 Health effects associated with PM_{2.5} exposure include a wide range of diseases. To date,
393 PM exposure-response functions used in LCIA have focused on chronic and acute mortality
394 and acute respiratory and cardiovascular morbidity associated with exposure to PM₁₀ (van
395 Zelm et al. 2008) or on cardiopulmonary mortality and lung cancer attributable to chronic
396 exposure to PM_{2.5} (Gronlund et al. 2014). ERF have been derived using several approaches
397 discussed in EC (2010c), primarily based on results from the Harvard Six Cities and
398 American Cancer Society studies (Krewski et al. 2000, Laden et al. 2006, Pope III et al.
399 2002). Although the impact of PM_{2.5} exposure on asthma has been reported in several
400 epidemiological studies (Brauer et al. 2002, Kheirbek et al. 2013, Künzli et al. 2000), asthma
401 is usually not considered in LCIA. At the workshop, it was noted that evidence linking PM_{2.5}
402 exposure with new asthma incidences is inconclusive, whereas it does support a link between
403 PM_{2.5} exposure and the exacerbation of existing asthma (Donaldson et al. 2000, Gavett &

404 Koren 2001, Pope III et al. 1995). However, since it is unclear how to differentiate between
405 induction of new cases and exacerbation of existing disease, there was no agreement on
406 whether, and if so how, to include asthma as a health effect in LCIA.

407 It was emphasized that, in addition to the GBD 2010 effort, there is a large European
408 movement to decide which health effects associated with PM_{2.5} exposure to quantify. This
409 involves two projects³ – the Health Risks of Air Pollution in Europe, HRAPIE (WHO 2013a),
410 and Review of Evidence on Health Aspects of Air Pollution, REVIHAAP (WHO 2013b).
411 These projects aim to provide advice in support of the comprehensive review of European
412 Union’s air quality policies scheduled for 2013. A consensus document reflecting this effort
413 was published end of 2013 (WHO 2013a). Whereas the GBD 2010 effort focuses on cause-
414 specific mortality, the HRAPIE/REVIHAAP projects recommend all-cause analysis as
415 primary choice and cause-specific analysis as alternative method based on similarity of the
416 frequency of the causes of death linked with exposure between considered cohorts and
417 countries. It can be argued that a cause-specific assessment is particularly important in global
418 assessments because of the large geographical variability in the relative importance of various
419 causes of death. This view is supported by several studies (Lipsett et al. 2011, Miller et al.
420 2007, Puett et al. 2011, Puett et al. 2009).

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

426

427 6.3 Shape of exposure-response functions

428 In current LCIA practice, the shape of population ERF is usually assumed to be linear
429 with no threshold. This approach is supported by several studies which find no evidence of a
430 departure from linearity (Chen et al. 2013, Schwartz et al. 2008, Stafoggia et al. 2013, WHO
431 2006) and no evidence suggesting a threshold at the population level (COMEAP 2009, 2010).
432 Despite this, when these linear functions are applied to the very high PM_{2.5} levels often found
433 in developing countries, the estimates of risk are so high as to be implausible (Abrahamowicz
434 et al. 2003, EC 2010b). Recently, several research groups have suggested non-linear ERF that
435 could be applied across a large range of PM_{2.5} concentrations, from very low to very high
436 PM_{2.5} concentrations. These are typically steep at low concentration levels and relatively flat

³ <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>

437 at high levels (Abrahamowicz et al. 2003, Burnett et al. 2014, Ostro 2004, Pope III et al.
438 2009). Whether, and if so, how this approach can be adapted for use in LCIA needs to be
439 further discussed, acknowledging that LCA aims to support decisions in regions with low
440 concentration levels and also in regions with high concentration levels. From a sustainability
441 point of view, intervention in highly polluted areas may be a priority despite the lower
442 response per unit exposure. Significant departures from linearity would imply that iF would
443 need to be reconstructed in a manner that is stratified by PM_{2.5} concentration or other relevant
444 factors. In making such a change, it is also important to realize that the shape of the ERF
445 might be effect-specific – for example nearly linear for lung cancer, but substantially non-
446 linear for cardiovascular mortality (Pope III et al. 2011). In GBD 2010, effect-specific,
447 integrated ERF are proposed for PM_{2.5} (Lim et al. 2012). These ERF express relative risk as
448 an exponential function (or a power function) of PM_{2.5} concentration (Burnett et al. 2014). In
449 order to apply such non-linear ERF in LCIA, non-linear models can either be directly applied
450 as e.g. in van Zelm et al. (2008) for ozone formation or be decomposed into piecewise linear
451 functions. Workshop participants explained that methods for applying this approach are
452 currently being developed.

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

466

467 6.4 Particle characteristics and differential toxicity

468 PM_{2.5} mass is commonly used as an indicator of the risk associated with exposure to a
469 mixture of particle-related pollutants (of different sizes below 2.5 µm diameter) from diverse
470 (primary or secondary) sources and in different environments (COMEAP 2009, Lim et al.

471 2012, Pope III et al. 2009, Pope III et al. 2011). This approach, which implicitly assumes
472 equal toxicity of PM_{2.5} constituents per mass unit, is commonly used in LCIA (Potting et al.
473 2007). There is currently no scientific consensus on the relative toxicity of various
474 constituents of PM_{2.5}. This, however, does not suggest that all particle constituents are in fact
475 equally toxic, but instead that the toxicological and epidemiological evidence of differential
476 toxicity is inconclusive (Hurley et al. 2005). One study found differential toxicity of multiple
477 particle constituents for short-term exposure effects on hospital admissions (Levy et al. 2012),
478 but further research is required to address other health outcomes, long-term exposure, and
479 other geographical settings (Rohr & Wyzga 2012).

480 In view of this it was agreed to use PM_{2.5} mass as an indicator of exposure without
481 differentiating between and among primary and secondary PM_{2.5} and without differentiating
482 between different PM_{2.5} constituents in terms of toxicity for cause-specific chronic mortality
483 effects. However, the workshop participants understood that given the current state of
484 scientific uncertainty about this matter it would be important to develop an approach for
485 characterizing the uncertainty of the toxicity of various constituents of PM_{2.5} which reflects
486 the lack of knowledge about which constituents of PM_{2.5} are in fact responsible for the
487 toxicity of the mixture.

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

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

502

503 7 Conclusions and next steps

504 7.1 Conclusions

505 Workshop participants discussed the questions shown in Table 1 in an effort to find ways
506 to refine and improve the overall framework and to suggest data and models that could
507 harmonize the analysis of health impacts from exposure to ambient particulate matter. This
508 discussion constituted a first step towards developing recommendations for addressing the
509 health effects from exposure associated with emissions of primary PM_{2.5} and secondary PM_{2.5}
510 precursors in LCIA. A set of 10 recommendations reflecting the consensus of workshop
511 participants are summarized as follows:

- 512 ■ The intake fraction framework proposed by Humbert et al. (2011) provides a useful
513 starting point for assessing health effects of ambient PM in LCIA with a focus on PM_{2.5}.
- 514 ■ Human intake fractions can be used to estimate emission-related population exposure. In
515 conjunction with population-averaged breathing rates, intake fractions can be used to
516 estimate intake from air concentrations.
- 517 ■ Disability-adjusted life years without age-weighting or discounting, which aggregate
518 mortality and morbidity, can be used as a summary health metric.
- 519 ■ For most cases, where emission locations are unknown, exposure scenario archetypes
520 provide a useful approach to account for factors, such as population density, emission
521 height, and exposure to PM_{2.5} from indoor sources, which influence human intake
522 fractions. The decision whether additional archetypes are necessary should be based on a
523 sensitivity analysis that considers the importance of these additional factors in reducing
524 uncertainty in exposure estimates. When the exact emission location is known, spatially
525 explicit fate and transport models should be used.
- 526 ■ Geographical archetypes of intake fractions should be established for indoor, near-field,
527 neighborhood, urban, regional, and continental scales. Geographical differentiation
528 should be further discussed and analyzed with respect to scale and non-linear chemical
529 processes in the formation of secondary PM_{2.5}.
- 530 ■ Emission-weighted average intake fractions should be used in cases where the nature of
531 the emission sources and/or exposure conditions is unclear.
- 532 ■ The Global Burden of Disease Study 2010 is considered to provide a useful starting point
533 for developing exposure-response functions for assessing PM_{2.5}-related health effects in
534 LCIA.

- 535 ▪ Cause-specific mortality can provide a more informative basis for developing LCIA
536 characterization factors than all-cause mortality. Assumptions for age- and cause-specific
537 disability weights should be further discussed and analyzed.
- 538 ▪ Non-linear exposure-response functions are recommended in the Global Burden of
539 Disease Study 2010, whereas linear functions are used in the consensus document of the
540 Health Risks of Air Pollution in Europe projects. There remains a need for discussion
541 about whether, and if so, how to integrate non-linear (or piecewise linear) exposure-
542 response functions into LCIA.
- 543 ▪ PM_{2.5} mass can be used as the indicator of the health risk associated with PM inhalation
544 exposure in LCIA. There is no justification at this time to differentiate between different
545 primary/secondary PM_{2.5} sources or between different PM_{2.5} particle sizes regarding
546 toxicity. However, analyses should report the uncertainties inherent in any assumptions
547 made about the relative toxicity of various types of particles.

548

549 7.2 Next steps

550 Within the next two years, the goals of the task force on human health impacts are to
551 build a global guidance framework and to determine characterization factors for incorporating
552 the health effects from exposure to PM_{2.5} in LCIA and for including both indoor and outdoor
553 releases. As next steps towards these goals, the first set of recommendations from the Basel
554 Guidance Workshop will be taken. Open questions and unsolved problems will be further
555 studied that were pointed out by workshop participants and the proposed framework will be
556 refined based on best available data and methods. The harmonized framework and related
557 results will finally be presented at a Pellston Technical Workshop⁴ in 2015.

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.

558 **References**

- 559 Abrahamowicz M, Schopflocher T, Leffondré K, du Berger R, Krewski D (2003): Flexible
560 modeling of exposure-response relationship between long-term average levels of particulate
561 air pollution and mortality in the American Cancer Society Study. *J Toxicol Env Health* 66,
562 1625-1654
- 563 Apte JS, Bombrun E, Marshall JD, Nazaroff WW (2012): Global intraurban intake fractions
564 for primary air pollutants from vehicles and other distributed sources. *Environ Sci Technol*
565 46, 3415-3423
- 566 Beelen R, Raaschou-Nielsen O, Stafoggia M, Andersen ZJ, Weinmayr G et al. (2014): Effects
567 of long-term exposure to air pollution on natural-cause mortality: An analysis of 22 European
568 cohorts within the multicentre ESCAPE project. *Lancet* 383, 785-795
- 569 Brauer M, Hoek G, Van Vliet P, Meliefste K, Fischer PH, Wijga A, Koopman LP, Neijens
570 HJ, Gerritsen J, Kerkhof M, Heinrich J, Bellander T, Brunekreef B (2002): Air pollution from
571 traffic and the development of respiratory infections and asthmatic and allergic symptoms in
572 children. *Am J Resp Crit Care* 166, 1092-1098
- 573 Brook RD, Rajagopalan S, Pope III CA, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F,
574 Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith Jr SC, Whitsel L,
575 Kaufman JD (2010): Particulate matter air pollution and cardiovascular disease: An update to
576 the scientific statement from the American Heart Association. *Circulation* 121, 2331-2378
- 577 Burnett RT, Pope III CA, Ezzati M, Olives C, Lim SS et al. (2014): An integrated risk
578 function for estimating the global burden of disease attributable to ambient fine particulate
579 matter exposure. *Environ Health Persp* 122, 397-403
- 580 Chen H, Goldberg MS, Villeneuve PJ (2008): A systematic review of the relation between
581 long-term exposure to ambient air pollution and chronic diseases. *Rev Environ Health* 23,
582 243-297
- 583 Chen H, Burnett RT, Kwong JC, Villeneuve PJ, Goldberg MS, Brook RD, van Donkelaar A,
584 Jerrett M, Martin RV, Brook JR, Copes R (2013): Risk of incident diabetes in relation to
585 long-term exposure to fine particulate matter in Ontario, Canada. *Environ Health Persp* 121,
586 804-810
- 587 COMEAP, 2009: Long-Term Exposure to Air Pollution: Effect on Mortality, Health
588 Protection Agency for the Committee on the Medical Effects of Air Pollutants, London, UK
- 589 COMEAP, 2010: The Mortality Effects of Long-Term Exposure to Particulate Air Pollution
590 in the United Kingdom, Health Protection Agency for the Committee on the Medical Effects
591 of Air Pollutants, London, UK
- 592 Dadvand P, Parker J, Bell ML, Bonzini M, Brauer M et al. (2013): Maternal exposure to
593 particulate air pollution and term birth weight: A multi-country evaluation of effect and
594 heterogeneity. *Environ Health Persp* 121, 367-373
- 595 Delfino RJ, Sioutas C, Malik S (2005): Potential role of ultrafine particles in associations
596 between airborne particle mass and cardiovascular health. *Environ Health Persp* 113, 934-946
- 597 Donaldson K, Gilmour MI, MacNee W (2000): Asthma and PM₁₀. *Resp Res* 1, 12-15

- 598 EC, 2010a: International Reference Life Cycle Data System (ILCD) Handbook: General
599 guide for Life Cycle Assessment - Detailed guidance, 1st Ed. European Commission, Brussels
- 600 EC, 2010b: International Reference Life Cycle Data System (ILCD) Handbook: Framework
601 and requirements for LCIA models and indicators, 1st Ed. European Commission, Brussels
- 602 EC, 2010c: International Reference Life Cycle Data System (ILCD) Handbook: Analysis of
603 existing Environmental Impact Assessment methodologies for use in Life Cycle Assessment,
604 1st Ed. European Commission, Brussels
- 605 Finnveden G, Hauschild MZ, Ekvall T, Guinée J, Heijungs R, Hellweg S, Koehler A,
606 Pennington D, Suh S (2009): Recent developments in life cycle assessment. *J Environ*
607 *Manage* 91, 1-21
- 608 Gavett SH, Koren HS (2001): The role of particulate matter in exacerbation of atopic asthma.
609 *Int Arch Allergy Imm* 124, 109-112
- 610 Greco SL, Wilson AM, Spengler JD, Levy JI (2007): Spatial patterns of mobile source
611 particulate matter emissions-to-exposure relationships across the United States. *Atmos*
612 *Environ* 41, 1011-1025
- 613 Gronlund CJ, Humbert S, Shaked S, O'Neill MS, Jolliet O (2014): Characterizing the burden
614 of disease of particulate matter for life cycle impact assessment. *Air Qual Atmos Health*,
615 doi:10.1007/s11869-014-0283-6
- 616 Hänninen O, Knol AB, Jantunen M, Lim T-A, Conrad A, Rappolder M, Carrer P, Fanetti A-
617 C, Kim R, Buekers J, Torfs R, Iavarone I, Classen T, Hornberg C, Mekel OCL (2014):
618 Environmental burden of disease in Europe: Estimates for nine stressors in six countries.
619 *Environ Health Persp* 122, 439-446
- 620 Harrison RM, Yin J (2000): Particulate matter in the atmosphere: Which particle properties
621 are important for its effects on health? *Sci Total Environ* 249, 85-101
- 622 Hauschild MZ (2005): Assessing environmental impacts in a life-cycle perspective. *Environ*
623 *Sci Technol* 39, 81A-88A
- 624 Hauschild MZ, Goedkoop M, Guinée J, Heijungs R, Huijbregts M, Jolliet O, Margni M, De
625 Schryver A, Humbert S, Laurent A, Sala S, Pant R (2013): Identifying best existing practice
626 for characterization modeling in life cycle impact assessment. *Int J Life Cycle Assess* 18, 683-
627 697
- 628 Heath GA, Granvold PW, Hoats AS, W Nazaroff W (2006): Intake fraction assessment of the
629 air pollutant exposure implications of a shift toward distributed electricity generation. *Atmos*
630 *Environ* 40, 7164-7177
- 631 HEI, 2013: Understanding the Health Effects of Ambient Ultrafine Particles, Health Effects
632 Institute, Boston
- 633 Hellweg S, Demou E, Bruzzi R, Meijer A, Rosenbaum RK, Huijbregts MAJ, McKone TE
634 (2009): Integrating human indoor air pollutant exposure within life cycle impact assessment.
635 *Environ Sci Technol* 43, 1670-1679

636 Hill J, Polasky S, Nelson E, Tilman D, Huo H, Ludwig L, Neumann J, Zheng H, Bonta D
637 (2009): Climate change and health costs of air emissions from biofuels and gasoline. *P Natl*
638 *Acad Sci* 106, 2077-2082

639 Hoek G, Krishnan RM, Beelen R, Peters A, Ostro B, Brunekreef B, Kaufman JD (2013):
640 Long-term air pollution exposure and cardio-respiratory mortality: A review. *Environ Health*
641 12, 43-57

642 Humbert S, Manneh R, Shaked S, Wannaz C, Horvath A, Deschênes L, Jolliet O, Margni M
643 (2009): Assessing regional intake fractions in North America. *Sci Total Environ* 407, 4812-
644 4820

645 Humbert S, Marshall JD, Shaked S, Spadaro JV, Nishioka Y, Preiss P, McKone TE, Horvath
646 A, Jolliet O (2011): Intake fraction for particulate matter: Recommendations for life cycle
647 impact assessment. *Environ Sci Technol* 45, 4808-4816

648 Hurley F, Alistair Hunt, Cowie H, Holland M, Miller B, Pye S, Watkiss P 2005: Methodology
649 for the Cost-Benefit analysis for CAFE: Vol. 2: Health Impact Assessment, AEA Technology
650 Assessment, Oxon, UK

651 ISO, 2006: ISO 14040 International Standard. Environmental Management - Life Cycle
652 Assessment - Principles and Framework. International Organization for Standardization,
653 Geneva, Switzerland

654 Jolliet O, Frischknecht R, Bare J, Boulay A-M, Bulle C et al. (2014): Global guidance on
655 environmental life cycle impact assessment indicators: Findings of the scoping phase. *Int J*
656 *Life Cycle Assess* 19, 962-967

657 Kelly FJ, Fussell JC (2012): Size, source and chemical composition as determinants of
658 toxicity attributable to ambient particulate matter. *Atmos Environ* 60, 504-526

659 Kheirbek I, Wheeler K, Walters S, Kass D, Matte T (2013): PM_{2.5} and ozone health impacts
660 and disparities in New York City: Sensitivity to spatial and temporal resolution. *Air Qual*
661 *Atmos Health* 6, 473-486

662 Krewski D, Burnett RT, Goldberg MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz
663 M, White WH 2000: Reanalysis of the Harvard Six Cities Study and the American Cancer
664 Society Study of particulate air pollution and mortality, Health Effects Institute, Boston, MA

665 Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, Herry M, Horak F,
666 Puybonnieux-Texier V, Quénel P, Schneider J, Seethaler R, Vergnaud J-C, Sommer H
667 (2000): Public-health impact of outdoor and traffic-related air pollution: A European
668 assessment. *Lancet* 356, 795-801

669 Laden F, Schwartz J, Speizer FE, Dockery DW (2006): Reduction in fine particulate air
670 pollution and mortality. *Am J Resp Crit Care* 173, 667-672

671 Leksell I, Rabl A (2001): Air pollution and mortality: Quantification and valuation of years of
672 life lost. *Risk Anal* 21, 843-857

673 Levy JI, Wolff SK, Evans JS (2002): A regression-based approach for estimating primary and
674 secondary particulate matter intake fractions. *Risk Anal* 22, 895-904

675 Levy JI, Wilson AM, Evans JS, Spengler JD (2003): Estimation of primary and secondary
676 particulate matter intake fractions for power plants in Georgia. *Environ Sci Technol* 37, 5528-
677 5536

678 Levy JI, Diez D, Dou Y, Barr CD, Dominici F (2012): A meta-analysis and multisite time-
679 series analysis of the differential toxicity of major fine particulate matter constituents.
680 *Epidemiology* 175, 1091-1099

681 Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K et al. (2012): A comparative risk
682 assessment of burden of disease and injury attributable to 67 risk factors and risk factor
683 clusters in 21 regions, 1990 - 2010: A systematic analysis for the Global Burden of Disease
684 Study 2010. *Lancet* 380, 2224-2260

685 Lippmann M, Chen L-C (2009): Health effects of concentrated ambient air particulate matter
686 (CAPs) and its components. *Cr Rev Toxicol* 39, 865-913

687 Lipsett MJ, Ostro BD, Reynolds P, Goldberg D, Hertz A, Jerrett M, Smith DF, Garcia C,
688 Chang ET, Bernstein L (2011): Long-term exposure to air pollution and cardiorespiratory
689 disease in the California Teachers Study cohort. *Am J Resp Crit Care* 184, 828-835

690 Lobscheid AB, Nazaroff WW, Spears M, Horvath A, McKone TE (2012): Intake fractions of
691 primary conserved air pollutants emitted from on-road vehicles in the United States. *Atmos*
692 *Environ* 63, 298-305

693 Loomis D, Grosse Y, Lauby-Secretan B, Ghissassi FE, Bouvard V, Benbrahim-Tallaa L,
694 Guha N, Baan R, Mattock H, Straif K (2013): The carcinogenicity of outdoor air pollution.
695 *Lancet Oncol* 14, 1262-1263

696 Marshall JD, Riley WJ, McKone TE, Nazaroff WW (2003): Intake fraction of primary
697 pollutants: motor vehicle emissions in the South Coast Air Basin. *Atmos Environ* 37, 3455-
698 3468

699 Marshall JD, Teoh S-K, W. Nazaroff W (2005): Intake fraction of nonreactive vehicle
700 emissions in US urban areas. *Atmos Environ* 39, 1363-1371

701 Mehta S, Shin H, Burnett R, North T, Cohen AJ (2013): Ambient particulate air pollution and
702 acute lower respiratory infections: A systematic review and implications for estimating the
703 global burden of disease. *Air Qual Atmos Health* 6, 69-83

704 Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD
705 (2007): Long-term exposure to air pollution and incidence of cardiovascular events in women.
706 *New Engl J Med* 356, 447-458

707 Murray CJL, Lopez AD 1996a: *The Global Burden of Disease: A comprehensive assessment*
708 *of mortality and disability from diseases, injuries and risk factors in 1990 and projected to*
709 *2020*, Harvard University Press, Cambridge, MA

710 Murray CJL, Lopez AD (1996b): Evidence-based health policy - Lessons from the Global
711 Burden of Disease Study. *Science* 274, 740-743

712 Murray CJL, Ezzati M, Flaxman AD, Lim S, Lozano R, Michaud C, Naghavi M, Salomon
713 JA, Shibuya K, Vos T, Wikler D, Lopez AD (2012): GBD 2010: Design, definitions, and
714 metrics. *Lancet* 380, 2063-2066

- 715 Oberdörster G, Oberdörster E, Oberdörster J (2005): Nanotoxicology: An emerging discipline
716 evolving from studies of ultrafine particles. *Environ Health Persp* 113, 823-839
- 717 Ostro B 2004: Environmental Burden of Disease Series, No. 5. Outdoor air pollution:
718 Assessing the environmental burden of disease at national and local levels, World Health
719 Organization, Geneva, Switzerland
- 720 Paulot F, Jacob DJ (2014): Hidden cost of U.S. agricultural exports: Particulate matter from
721 ammonia emissions. *Environ Sci Technol* 48, 903-908
- 722 Pelucchi C, Negri E, Gallus S, Boffetta P, Tramacere I, La Vecchia C (2009): Long-term
723 particulate matter exposure and mortality: A review of European epidemiological studies.
724 *BMC Public Health* 9, 453-460
- 725 Pope III CA, Dockery DW, Schwartz J (1995): Review of epidemiological evidence of health
726 effects of particulate air pollution. *Inhal Toxicol* 7, 1-18
- 727 Pope III CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD (2002): Lung
728 cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *J*
729 *Am Med Assoc* 287, 1132-1141
- 730 Pope III CA, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ (2009):
731 Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke:
732 Shape of the exposure-response relationship. *Circulation* 120, 941-948
- 733 Pope III CA, Burnett RT, Turner MC, Cohen A, Krewski D, Jerrett M, Gapstur SM, Thun MJ
734 (2011): Lung cancer and cardiovascular disease mortality Associated with ambient air
735 pollution and cigarette smoke: Shape of the exposure-response relationships. *Environ Health*
736 *Persp* 119, 1616-1621
- 737 Potting J, Preiss P, Seppälä J, Struijs J, Wiertz J, Blazek M, Heijungs R, Itsubo N, Masanet E,
738 Nebel B, Nishioka Y, Payet J, Becaert V, Basset-Mens C, Joliet O 2007: Current Practice in
739 LCIA of Transboundary Impact Categories. Report of Task Force 4 on Transboundary
740 Impacts. UNEP/SETAC Life Cycle Initiative
- 741 Pregger T, Friedrich R (2009): Effective pollutant emission heights for atmospheric transport
742 modelling based on real-world information. *Environ Pollut* 157, 552-560
- 743 Puett RC, Hart JE, Yanosky JD, Paciorek C, Schwartz J, Suh H, Speizer FE, Laden F (2009):
744 Chronic fine and coarse particulate exposure, mortality, and coronary heart disease in the
745 Nurses' Health Study. *Environ Health Persp* 117, 1702-1706
- 746 Puett RC, Hart JE, Suh H, Mittleman M, Laden F (2011): Particulate matter exposures,
747 mortality, and cardiovascular disease in the Health Professionals Follow-up Study. *Environ*
748 *Health Persp* 119, 1130-1135
- 749 Rabl A (2005): Air pollution mortality: Harvesting and loss of life expectancy. *J Toxicol Env*
750 *Health*, 68, 1175-1180
- 751 Ries FJ, Marshall JD, Brauer M (2009): Intake fraction of urban wood smoke. *Environ Sci*
752 *Technol* 43, 4701-4706

- 753 Riley WJ, McKone TE, Lai ACK, Nazaroff WW (2002): Indoor particulate matter of outdoor
754 origin: Importance of size-dependent removal mechanisms. *Environ Sci Technol* 36, 200-207
- 755 Rohr AC, Wyzga RE (2012): Attributing health effects to individual particulate matter
756 constituents. *Atmos Environ* 62, 130-152
- 757 R ckerl R, Schneider A, Breitner S, Cyrys J, Peters A (2011): Health effects of particulate air
758 pollution: A review of epidemiological evidence. *Inhal Toxicol* 23, 555-592
- 759 Salomon JA, Vos T, Hogan DR, Gagnon M, Naghavi M et al. (2012): Common values in
760 assessing health outcomes from disease and injury: Disability weights measurement study for
761 the Global Burden of Disease Study 2010. *Lancet* 380, 2129-2143
- 762 Schwartz J, Coull B, Laden F, Ryan L (2008): The effect of dose and timing of dose on the
763 association between airborne particles and survival. *Environ Health Persp* 116, 64-69
- 764 Squizzato S, Masiol M, Brunelli A, Pistollato S, Tarabotti E, Rampazzo G, Pavoni B (2013):
765 Factors determining the formation of secondary inorganic aerosol: A case study in the Po
766 Valley (Italy). *Atmos Chem Phys* 13, 1927-1939
- 767 Stafoggia M, Samoli E, Alessandrini E, Cadum E, Ostro B, Berti G, Faustini A, Jacquemin B,
768 Linares C, Pascal M, Randi G, Ranzi A, Stivanello E, Forastiere F (2013): Short-term
769 associations between fine and coarse particulate matter and hospitalizations in Southern
770 Europe: Results from the MED-PARTICLES project. *Environ Health Persp* 121, 1026-1033
- 771 Stanek LW, Sacks JD, Dutton SJ, Dubois J-JB (2011): Attributing health effects to
772 apportioned components and sources of particulate matter: An evaluation of collective results.
773 *Atmos Environ* 45, 5655-5663
- 774 Straif K, Cohen A, Samet J (2013): *Air Pollution and Cancer*. IARC Scientific Publication
775 No. 161. International Agency for Research on Cancer, Lyon Cedex, France
- 776 Tainio M, Sofiev M, Hujo M, Tuomisto JT, Loh M, Jantunen MJ, Karppinen A, Kangas L,
777 Karvosenoja N, Kupiainen K, Porvari P, Kukkonen J (2009): Evaluation of the European
778 population intake fractions for European and Finnish anthropogenic primary fine particulate
779 matter emissions. *Atmos Environ* 43, 3052-3059
- 780 Tessum CW, Marshall JD, Hill JD (2012): A spatially and temporally explicit life cycle
781 inventory of air pollutants from gasoline and ethanol in the United States. *Environ Sci*
782 *Technol* 46, 11408-11417
- 783 Udo de Haes HA, Finnveden G, Goedkoop M, Hauschild MZ, Hertwich E, Hofstetter P,
784 Jolliet O, Kl pffer W, Krewitt W, Lindeijer E, M ller-Wenk R, Olsen S, Pennington DW,
785 Potting J, Steen B (2002): *Life-cycle impact assessment: Striving towards best practice*.
786 SETAC Press, Pensacola, Florida, USA
- 787 van Zelm R, Huijbregts MAJ, den Hollander HA, van Jaarsveld HA, Sauter FJ, Struijs J, van
788 Wijnen HJ, van de Meent D (2008): European characterization factors for human health
789 damage of PM₁₀ and ozone in life cycle impact assessment. *Atmos Environ* 42, 441-453
- 790 Wang H, Dwyer-Lindgren L, Lofgren KT, Rajaratnam JK, Marcus JR, Levin-Rector A,
791 Levitz CE, Lopez AD, Murray CJL (2012): Age-specific and sex-specific mortality in 187

792 countries, 1970-2010: A systematic analysis for the Global Burden of Disease Study 2010.
793 Lancet 380, 2071-2094

794 Weichenthal S, Dufresne A, Infante-Rivard C (2007): Indoor ultrafine particles and childhood
795 asthma: exploring a potential public health concern. Indoor Air 17, 81-91

796 WHO, 2006: Health risks of particulate matter from long-range transboundary air pollution,
797 World Health Organization, European Centre for Environment and Health, Bonn, Germany

798 WHO, 2013a: Health risks of air pollution in Europe - HRAPIE project Recommendations for
799 concentration-response functions for cost-benefit analysis of particulate matter, ozone and
800 nitrogen dioxide, World Health Organization, Geneva, Switzerland

801 WHO, 2013b: Review of evidence on health aspects of air pollution - REVIHAAP Project,
802 World Health Organization, Geneva, Switzerland

803 Xu L, Penner JE (2012): Global simulations of nitrate and ammonium aerosols and their
804 radiative effects. Atmos Chem Phys 12, 9479-9504

805 Zhou Y, Levy JI, Evans JS, Hammitt JK (2006): The influence of geographic location on
806 population exposure to emissions from power plants throughout China. Environ Int 32, 365-
807 373
808
809