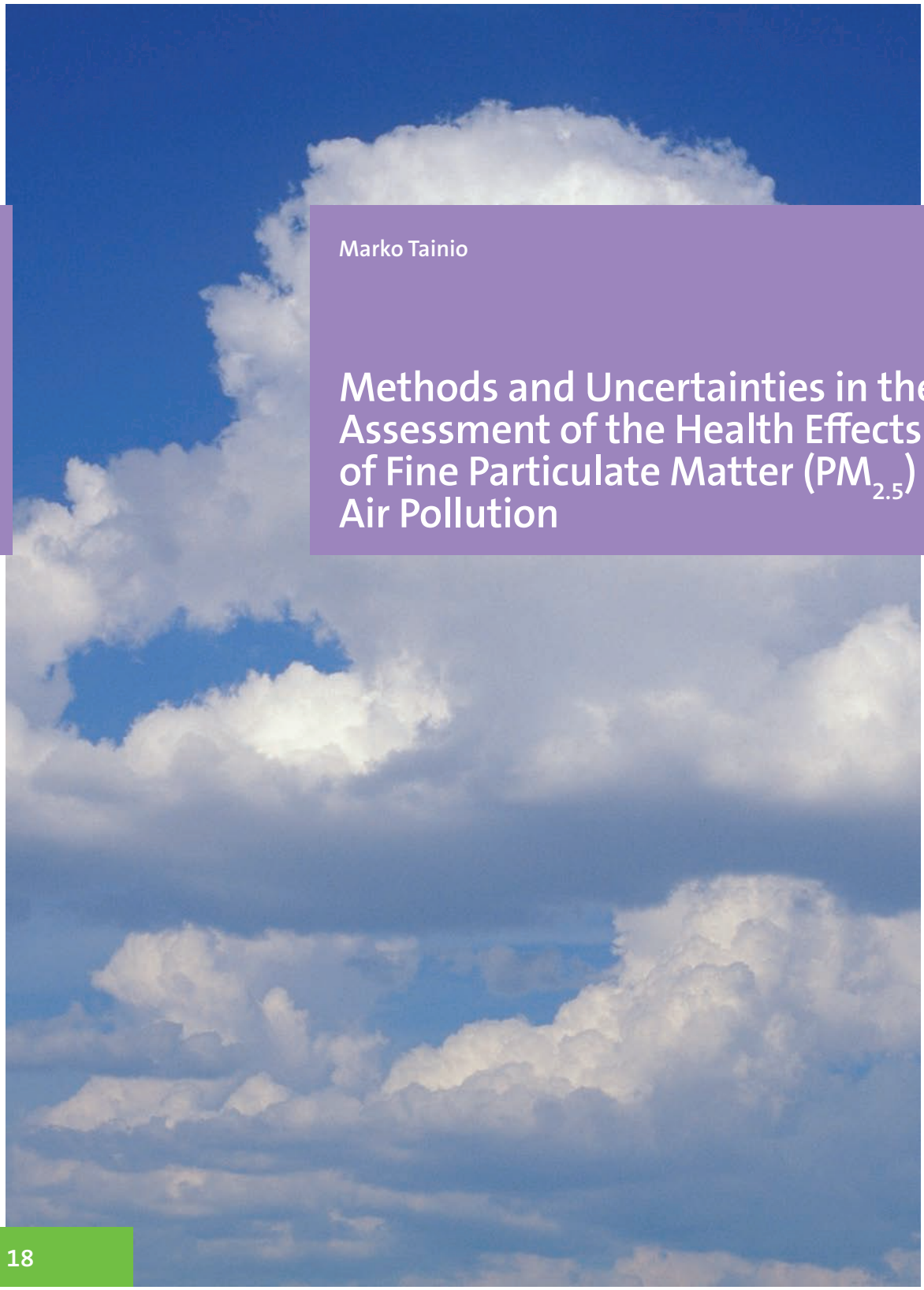

RESEARCH

Marko Tainio

Methods and Uncertainties in the Assessment of the Health Effects of Fine Particulate Matter ($PM_{2.5}$) Air Pollution

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ACADEMIC DISSERTATION

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Abstract

Fine particulate matter ($PM_{2.5}$) air pollution is a major environmental health problem in developed countries, causing several morbidity outcomes and decreasing the life expectancy of the population. National and international decisions, both current and proposed, are done to reduce the adverse health effects caused by $PM_{2.5}$. This decision making is supported by integrated assessment models. In this thesis, we compared how different methods estimate the adverse health effects caused by $PM_{2.5}$ air pollution. The main focus of the thesis was to identify and quantify uncertainties, and to estimate the importance of these uncertainties in the results of the integrated assessment.

The thesis is based on five studies, published or to be published in scientific peer reviewed journals. These studies have concerned the following topics:

- Estimation of premature deaths caused by local bus traffic related $PM_{2.5}$ air pollution in Helsinki Metropolitan Area, Finland.
- Development of a life-table model to estimate the change in life-expectancy due to local traffic related $PM_{2.5}$ air pollution in Helsinki Metropolitan Area, Finland.
- Comparison of the population densities near to traffic and domestic wood combustion emission sources in Finland.
- Estimation of emission-exposure relationship for primary $PM_{2.5}$ emissions from different countries and from different emission source categories in Europe.
- Estimation of premature death and change in life expectancy due to primary $PM_{2.5}$ air pollution in Finland.

In these studies, we have estimated exposure and health effects due to various primary $PM_{2.5}$ emissions sources. All the studies are based on computer models and the uncertainties have been propagated through the models using the Monte-Carlo method. We have concentrated on the anthropogenic primary $PM_{2.5}$ air pollution. Primary $PM_{2.5}$ means that particulate matter is in a particle format already when released from the source.

We observed that the uncertainty bounds of the premature death estimates are at least one order of magnitude around the mean estimate. The exposure-response

function (which quantifies the change in the population health due to a given exposure) was identified to be the main source of uncertainty in most of the individual studies. The increase in the number of premature deaths was mostly due to increased cardiopulmonary mortality. The toxicity differences between particles, due to differences in chemical and physical properties of $PM_{2.5}$, were identified to be the second most important source of uncertainty to be taken into account in the assessment.

The third most important uncertainty varied between emission source categories and studies. In general, the uncertainties in exposures were more important than uncertainties in emissions. Exposure assessment describes how and where people are exposed to $PM_{2.5}$ air pollution. Exposure to primary $PM_{2.5}$ varied between studies, and part of this variation is assumed to be due to methodological differences. In particular, the dispersion models with sparse spatial resolution may well underestimate the $PM_{2.5}$ concentrations near the emission sources, resulting in underestimation of exposure and the associated health effects.

The anthropogenic primary $PM_{2.5}$ air pollution was estimated to have caused a few hundred premature deaths in Finland in 2000. Over half of the premature deaths were estimated to be due to long-range transported $PM_{2.5}$ originating from other countries. With respect to the primary $PM_{2.5}$ emissions from Finland, approximately half of the premature deaths (~ 80 premature deaths per year) were due to traffic-related $PM_{2.5}$ emissions. The comparison of different study results suggests that the impact of traffic was underestimated, this being due to an underestimation of exposure.

The present study provided new information on the uncertainties and their impacts on integrated assessment of $PM_{2.5}$ air pollution. Based on data gathered in this thesis, the further development of $PM_{2.5}$ integrated assessments should focus on uncertainties in health effect estimation and developing suitable methods to estimate exposure for different source categories since these uncertainties have a major impact on the assessment results.

Keywords: Integrated assessment, risk assessment, fine particulate matter, air pollution, $PM_{2.5}$, exposure assessment, toxicity, sensitivity analysis, traffic.

Marko Tainio. Methods and Uncertainties in the Assessment of the Health Effects of Fine Particulate Matter ($PM_{2.5}$) Air Pollution [Menetelmät ja menetelmien epävarmuudet arvioitaessa pienhiukkasten ($PM_{2.5}$) terveysvaikutuksia]. Terveyden ja hyvinvoinnin laitos (THL), Tutkimus 18. 165 sivua. Helsinki, 2009. ISBN 978-952-245-101-9 (painettu), ISBN 978-952-245-102-6 (pdf).

Tiivistelmä

Ilman pienhiukkaset ($PM_{2.5}$) muodostavat merkittävimmän ympäristö-terveysongelman teollisuusmaissa. Altistuminen pienhiukkasille vähentää sekä väestön elinajanodotetta että lisää sairastavuutta. Kansallisia ja kansainvälisiä päätöksiä on tehty ja tehdään pienhiukkasten terveyshaittojen vähentämiseksi. Tätä päätöksentekoa voi tukea yhdennetyillä arviointimalleilla. Tässä väitöskirjatutkimuksessa vertailtiin erilaisia menetelmiä $PM_{2.5}$:n aiheuttamien terveysvaikutusten arvioimiseksi. Pääpaino väitöskirjassa oli tunnistaa ja määrittää epävarmuuksia ja arvioida näiden epävarmuuksien merkitystä yhdennettyjen arviointien tuloksiin.

Väitöskirja perustuu viiteen tutkimukseen, jotka on julkaistu tai julkaistaan tieteellisissä vertaisarvioiduissa lehdissä. Näissä tutkimuksissa on käsitelty seuraavia aiheita:

- Arvioitu paikallisen bussiliikenteen pienhiukkaspäästöjen aiheuttamia ennenaikaisia kuolemantapauksia Helsingin alueella.
- Kehitetty elinajanodotemalli pienhiukkasille ja arvioitu paikallisen liikenteen pienhiukkaspäästöjen vaikutusta väestön elinikään Helsingin alueella.
- Verrattu Suomessa väestön tiheyksiä liikenteen ja puun pienpolton päästölähteiden läheisyydessä
- Arvioitu pienhiukkaspäästöjen leviämistä ja niille altistumista eri Euroopan maissa ja eri päästölähteille.
- Arvioitu Suomessa primääristen pienhiukkasten aiheuttamia ennenaikaisia kuolemantapauksia sekä muutosta elinajanodotteessa.

Näissä tutkimuksissa olemme arvioineet altistumista ja terveysvaikutuksia useille pienhiukkasten päästölähteille. Kaikki tutkimukset perustuvat tietokonemalleihin ja useimmissa tutkimuksissa mallien epävarmuuksia on tarkasteltu Monte Carlo -menetelmällä. Tässä työssä on keskitytty ihmisen toiminnan aiheuttamiin primäärisiin $PM_{2.5}$-päästöihin. Primäärinen $PM_{2.5}$ tarkoittaa hiukkasia, jotka vapautuvat ilmaan hiukkasmuodossa ja ovat kooltaan alle 2,5 µm.

Tässä työssä havaittiin, että terveysvaikutusarvioiden epävarmuushaarukka on jopa kymmenkertainen. Annos-vastefunktio (joka kuvaa altistumisen aiheuttaman terveysmuutoksen suuruutta) havaittiin tärkeimmäksi epävarmuudeksi useimmissa yksittäisissä tutkimuksissa. Suurin osa pienhiukkasten aiheuttamasta ennenaikaisesta kuolleisuudesta johtui lisääntyneestä sydän- ja verisuonitauti-kuolleisuudesta. Hiukkasten kemiallisista ja fysikaalisista ominaisuuksista johtuvat toksisuuserot havaittiin toiseksi tärkeimmäksi epävarmuustekijäksi niissä tutkimuksissa, joissa toksisuuserot otettiin huomioon.

Altistus-vastefunktion ja toksisuuserojen jälkeen tärkeimmät epävarmuudet riippuivat tutkimuksesta ja pienhiukkasten päästölähteestä. Useimmille päästölähteille altistumisen arvioinnin epävarmuudet olivat tärkeämpiä kuin päästöepävarmuudet. Altistumisen arvioinnissa kuvataan, miten ja missä ihmiset altistuvat $PM_{2.5}$:lle. Tässä työssä havaittiin myös, että altistusarviot vaihtelivat eri tutkimusten ja menetelmien välillä. Erityisesti leviämismallit, joissa arvioidaan hiukkasten leviämistä harvalla resoluutiolla, voivat aliarvioida pitoisuuksia lähteiden lähellä, mikä johtaa myös terveysvaikutusten aliarviointiin.

Primäärisen $PM_{2.5}$:n arvioitiin aiheuttavan Suomessa muutama sata ennenaikaista kuolemantapausta vuonna 2000. Yli puolet ennenaikaisista kuolemantapauksista johtui muista maista kaukokulkeutuneista pienhiukkasista. Suomalaisten päästölähteiden Suomessa aiheuttamista terveysvaikutuksista noin puolet (arviolta 80 ennenaikaista kuolemantapausta vuodessa) johtui liikenneperäisistä pienhiukkasista. Vertailu eri tutkimusten välillä antaa viitteitä siitä, että liikenteen terveysvaikutukset on aliarvioitu tässä tutkimuksessa.

Tämä väitöskirjatyö lisäsi tietoa yhdennettyjen arviointimallien epävarmuuksista ja niiden vaikutuksista arviointien tuloksiin. Työn tulosten perusteella tulevien yhdennettyjen arviointien kannattaa keskittyä terveysvaikutusten arviointiin ja terveysvaikutuksen arvioinnin epävarmuuksiin sekä eri altistusmenetelmien kehittämiseen eri päästölähteille, sillä näillä epävarmuuksilla on suurin vaikutus arvioinnin lopputulokseen.

Avainsanat: Integroitu arviointi, riskinarviointi, pienhiukkaset, ilman saasteet, $PM_{2.5}$, altistuksen arviointi, toksisuus, herkkyysanalyysi, liikenne.

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Symbols and Abbreviations

Symbols and abbreviations used in this thesis summary. Original papers (chapters 4-8) use different symbols and abbreviations and those will be explained there.

Symbols in equations

β	Exposure-response coefficient.
BR	Breathing rate.
C	Concentration of pollutant.
E	Exposure.
Hb	Hazard rate.
M	Mortality rate.
OR	Odds ratio.
P0	Probability of health effect among those who were not exposed or were in lower exposed population.
P1	Probability of health effects among those that were exposed.
PM	Particulate matter.
PM_{10}	Coarse particulate matter, particulate matter with aerodynamic diameter less than 10 micrometer.
$PM_{2.5}$	Fine particulate matter, particulate matter with aerodynamic diameter less than 2.5 micrometer.
$PPM_{2.5}$	Primary fine particulate matter.
POP	Population.
Q	Emission strength.
RR	Relative risk.

Abbreviations

ACS	American Cancer Society. Epidemiological cohort study.
ANOVA	Analysis of variance.
APHEA	Air Pollution and Health - A European Approach. A research project.

BS	Black smoke.
CAFE	Clean Air for Europe –program.
CI	Confidence interval.
CO	Carbon monoxide.
COPD	Chronic obstructive pulmonary disease.
DALY	Disability-adjusted life-years.
DPSIR	Driving force - Pressure - State - Impact – Response. A framework for impact assessment.
EEA	European Environment Agency.
EMEP	Co-operative programme for monitoring and evaluation of the long range transmission of air pollutants in Europe.
EPA	United States Environmental Protection Agency.
EU	European Union.
EU15	15 member states of European Union (years 1995-2004).
EU25	25 member states of European Union (years 2004-2007).
EXPOLIS	Air pollution exposure in European cities. A research project.
ExternE	Externalities of Energy. A research project.
FMI	Finnish Meteorological Institute.
GAINS	Greenhouse gas – Air pollution Interactions and Synergies. A model.
GASBUS	Health Effects Caused by Primary Fine Particulate Matter Emitted from Buses. A research project.
HEI	Health Effects Institute.
HSC	Harvard Six Cities. Epidemiological cohort study.
IA	Integrated assessment.
iF	Intake fraction. A concept for emission-exposure relationship.
IIASA	International Institute for Applied Systems Analysis.
KOPRA	An integrated model for evaluating the emissions, atmospheric dispersion and risks caused by ambient air fine particulate matter. A research project.
KTL	National Public Health Institute, Finland.

OECD	Organisation for Economic Co-operation and Development.
NEEDS	New Energy Externalities Developments for Sustainability. A research project.
PILTTI	Health risks from nearby sources of fine particulate matter: domestic combustion and road traffic. A research project.
PSR	Pressure-State-Response. A framework for impact assessment.
PTAI	Population-based time-average inhalation –concept.
QALY	Quality-adjusted life-years.
RA	Risk assessment.
RAINS	Regional Air Pollution Information and Simulation. A model.
SIDS	Sudden Infant Death Syndrome.
SYKE	Finnish Environment Institute.
THL	National Institute for Health and Welfare.
UN	United Nations.
UNICE	Union of Industrial and Employers' Confederations of Europe.
VOI	Value of information.
WHO	World Health Organization.
YOLL	Years of life lost.

1 Introduction

The harmful impact of air pollution on human health has been known since ancient times (Makra and Brimblecombe, 2004). The air pollution episode known as the London Smog Episode in 1952 was by no means a unique event, it was preceded by similar episodes in the Meuse Valley, Belgium 1930 (Nemery et al., 2001), and in Donora, Pennsylvania 1948 (Bell and Davis, 2001). The significance of the London smog episode was on that it had political consequences. Mitigation actions decreased air pollution emissions and consequently the levels of harmful substances in the ambient air decreased to a fraction of its historical levels within a few decades. In the 1970's, a comprehensive review written by British scientist came to a conclusion that the (then) current ambient air pollution levels did not pose any significant threat to population (Holland et al., 1979).

Hundreds of new epidemiological studies in 1990's and 2000's have indicated that in fact the current air pollution levels are capable of harming public health. From the ambient air pollution mixture the attention has focused especially on solid and liquid parts of air, know as particulate matter (PM). Out from the entire PM mass, it is especially the smallest particles, known as fine particulate matter ($PM_{2.5}$) that has been associated with a number of adverse health effects (e.g. Pope and Dockery, 2006). The impact assessments have estimated that $PM_{2.5}$ causes annually over 800 000 premature deaths worldwide (Cohen et al., 2005); 350 000 in Europe alone (Watkiss et al., 2005). As a comparison, passive smoking (also known as second hand smoking) has been estimated to cause 79 000 premature deaths in EU25 (ERS, 2006) and ozone is believed to cause 21 400 premature deaths in Europe (Watkiss et al., 2005). $PM_{2.5}$ air pollution is one of the major environmental health problems in the developed world.

Much has been done to mitigate the adverse health effects of ambient air pollution. The change in legislation and the economical system in Eastern Europe has reduced PM precursors and primary PM emissions by approximately 45% in the 32 European Economic Area countries between years 1990-2004 (EEA, 2007). In particular precursor gas emissions have declined dramatically. However, the European Economic Area report concluded that apart from the reduction in emission volumes, the ambient PM concentrations have not decreased since 1997 (EEA, 2007). Thus, it seems that the mitigation actions have not been sufficient or effective to protect human health in the ambient environment.

The recent European Union (EU) Air Quality Directive (2008/50/EC) has been targeted to mitigate the adverse health effects of air pollution. The directive was

issued because of "*the need to reduce pollution to levels which minimise harmful effects on human health, paying particular attention to sensitive populations, and the environment as a whole, to improve the monitoring and assessment of air quality including the deposition of pollutants and to provide information to the public*". The directive also recommended that "*In order to protect human health and the environment as a whole, it is particularly important to combat emissions of pollutants at source and to identify and implement the most effective emission reduction measures at local, national and community level*". Assessment studies are required to meet the targets set by EU.

Several integrated assessment (IA), health impact assessment (HIA), risk assessment (RA) and other assessment studies have evaluated the health effects attributable to PM. These studies have assessed PM associated adverse health effects in urban environment (Deck et al., 2001), due to long-range transport (van Zelm et al., 2008), based on PM measurements (Forsberg et al., 2005), and based on dispersion models (Levy and Spengler, 2002). The adverse health effects have been estimated for adults (Levy et al., 2002) and for infants (Kaiser et al., 2004), and the adverse effects have been measured using premature death (Golub and Strukova, 2008), life-expectancy (Boldo et al., 2006) and quality-adjusted life-years (QALY) measures (Coyle et al., 2003). Thus, several research teams with a wide range of study objectives have developed methods to assess the health consequences induced by the PM air pollution and applied those methods in their own case studies.

Assessment methods for PM air pollution have been developed and recommended by several organizations. For example, the global update of the World Health Organization (WHO) air quality guidelines in 2005 provided values for different air pollutants, including PM, and reviewed the assessment methods for the use of risk assessment and policy analysis (WHO, 2006). The exposure-response functions for PM air pollution have been defined and discussed by e.g. WHO in their report concerning burden of disease caused by outdoor air pollution (Ostro, 2004) and in the European Externalities of Energy (ExternE) project (ExternE, 2005). The exposure-response function describes the relationship between exposure and related health effects. The ExternE -methodology was further updated in 2007 in a joint exercise of several European cost-benefit analysis projects (Torfs et al., 2007). Also the development of European Regional Air Pollution Information and Simulation model (RAINS) for the Clean Air for Europe (CAFE) program has involved a number of expert meetings and panels focusing on assessment methods (e.g. UN, 2004; WHO, 2003).

This thesis continues the development of the methods used to assess adverse health effects due to primary fine particulate matter ($PPM_{2.5}$) air pollution. This thesis has focused on uncertainties in assessments, and analysed the impacts of these

uncertainties with sensitivity analysis methods. The thesis has been undertaken by conducting integrated assessment studies for $PPM_{2.5}$ air pollution and by adapting and testing several methods in case studies. These case studies have been published, or will be published, in scientific peer reviewed journals. The case studies are also published in chapters 4-8 of this thesis.

The case studies are based on three research projects. The first project, Health Effects Caused by Primary Fine Particulate Matter Emitted from Buses (GASBUS), estimated the health effects of alternative bus technologies in the Helsinki Metropolitan Area, Finland. The second project, An integrated model for evaluating the emissions, atmospheric dispersion and risks caused by ambient air fine particulate matter (KOPRA), estimated the emission, dispersion and health effects of Finnish anthropogenic $PPM_{2.5}$ in Finland and elsewhere in Europe (Kukkonen et al., 2007). The third project, Health risks from nearby sources of fine particulate matter: domestic combustion and road traffic (PILTTI) evaluated the emission, dispersion and health effects of domestic wood combustion and traffic-related $PPM_{2.5}$ with a 1 km spatial resolution. The KOPRA and PILTTI projects have been undertaken in co-operation with the Finnish Environment Institute (SYKE) and Finnish Meteorological Institute (FMI).

A number of other studies have contributed to this thesis by testing ideas and methods or by focusing on parts of integrated assessment that are beyond the scope of this thesis. The decision analysis method value of information (VOI) was first adopted in a study concerning risks and benefits of eating farmed salmon (Tuomisto et al., 2004). This study was further developed and combined with work done by Tainio et al. (2005) to study the effect of two EU regulations in Helsinki Metropolitan Area, Finland (Leino et al., 2008). Several methods were tested in a study where we developed a theory of composite traffic that would change public transportation from a fixed-route service to a demand based service (Tuomisto and Tainio, 2005). The exposure-response functions for $PM_{2.5}$ were defined in an Expert Elicitation study (Cooke et al., 2007; Tuomisto et al., 2008) and the emission uncertainties in the Karvosenoja et al. (2008) study.

In addition to methodological development, these studies have also generated information for use by decision makers. Researchers from these projects have taken part in European and International legislation work and contributed to the development of the European Regional Air Pollution Information and Simulation (RAINS) model used in the CAFE program. In Finland, the results from these studies have raised awareness of the health effects of $PM_{2.5}$ originating from domestic wood combustion, and authorities are currently planning mitigation actions aimed at this emission source category.

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2 Review of literature

The review of literature is divided into general and specific literature reviews. The general literature review will concentrate on those issues that are outside the scope of this thesis but relevant in order to place the issues being considered into a larger context. These include defining $PM_{2.5}$ air pollution and integrated assessment method. The specific literature review will concentrate on those issues that are relevant for the objectives of this thesis.

2.1 General literature review

2.1.1 Particulate matter: Definition, sources and dispersion

Solid and liquid components in the air are defined with the common term, particulate matter (PM). The PM is commonly categorised based on aerodynamic size. An aerodynamic size of 5 micrometer means that the PM is behaving as if it were perfect sphere of 5 micrometer diameter. The fine particulate matter means PM with aerodynamic diameter less than 2.5 micrometer. The size of PM is described with the acronym PM followed by the maximum aerodynamic diameter (e.g. $PM_{1.0}$, particles that aerodynamic diameter is less than 1.0 µm). Other common size fractions are ultrafine particulate matter ($PM_{0.1}$) and coarse particulate matter (also known as thoracic particulate matter) (PM_{10}).

PM is formed into ambient air from gases in nucleation and condensation processes and directly through mechanical grinding (EPA, 2004; WHO, 2006). During nucleation, gases react with each other forming PM and during condensation, the existing PM react with gases increasing the size of PM. During coagulation, the particles become attached to each other, thus decreasing in number and increasing size. The size of a particle tends to increase with time through condensation and coagulation until the particle reaches the so called accumulation mode. The accumulation mode refers to PM with aerodynamic diameter approximately between 0.1 and 1.0 micrometer. A major part of $PM_{2.5}$ mass is in the accumulation mode. Due to the processes of coagulation and condensation, the PM inhaled by people has a different chemical composition, size and physical characteristics than the PM that were originally emitted into air.

The PM is divided into primary and secondary PM based on its formation time and place. The primary PM is emitted into air directly from sources, while secondary PM is formed outside the source through physical or chemical processes. However,

the borderline between primary and secondary PM is blurred. For example, exhaust gases react with each other forming PM in both the car's exhaust-pipe and in the air after the exhaust-pipe just seconds after release. Usually PM formed from so-called precursor gases is considered as secondary PM. These precursor gases include sulphur dioxide, nitrogen dioxide, ammonia, anthropogenic volatile organic compounds (VOC) and biogenic VOC (WHO, 2006).

The main anthropogenic emission sources of $PPM_{2.5}$ in EU15 are mobile sources (34%), industrial processes including energy production (20%) and domestic combustion (25%) (WHO, 2006). The main emission sources of precursor gases in EU25 countries are power generation, industry and transport. Almost all ammonia emissions are emitted from agriculture.

Typically $PM_{2.5}$ stays in atmosphere from about 1-2 days to 4-6 days (WHO, 2006). The time spent in the atmosphere depends mainly on the size of the PM i.e. $PPM_{2.5}$ remains longest in the atmosphere. During that time $PM_{2.5}$ can travel up to 2000 to 3000 kilometres from the release location. $PM_{2.5}$ is mainly removed from air by becoming attached to particles surface (dry deposition) or by forming cloud droplets and being rained out (wet deposition) (EPA, 2004).

People inhale PM emitted from outdoor sources both outdoors and indoors. Though they spend most of their time indoors. Ability of PM to penetrate indoors significantly determines population exposure to outdoor PM. For example, Hänninen et al., (2005) have assumed that the average infiltration factor for $PM_{2.5}$ in Helsinki Metropolitan Area, Finland, is 0.64 and 0.47 for residential and occupational buildings, respectively. Thus, around half of the PM in ambient air can penetrate from outdoors into indoor space.

$PM_{2.5}$ has been associated in epidemiology and toxicology with a number of adverse health effects (e.g. Pope and Dockery, 2006; Schwarze et al., 2006). The World Health Organization (WHO) concluded in 2003 that long-term exposure to $PM_{2.5}$ may reduce life-expectancy due to cardiopulmonary and lung cancer mortality. In addition, $PM_{2.5}$ can evoke lower respiratory symptoms and reduced lung function in children, and cause chronic obstructive pulmonary disease (COPD) and impaired lung function in adults (WHO, 2003). An association between $PM_{2.5}$ exposure and adverse health effects has been observed also in Finland (e.g. Halonen et al., 2008; Lanki et al., 2006; Pekkanen et al., 2002). The mechanisms causing adverse health effects are incompletely understood although several plausible mechanisms have been identified (Pope and Dockery, 2006).

2.1.2 Integrated assessment and $PPM_{2.5}$ air pollution

The European Environment Agency (EEA) defines an integrated assessment (IA) as "*an interdisciplinary process of structuring knowledge elements from various scientific disciplines in such a manner that all relevant aspects of a complex societal problem are considered in their mutual coherence for the benefit of decision-making*". Thus according to the definition integrated assessment is applied to complex problems that receive input from multi-disciplinary experts. The main goal of IA is to support decision making. Several other terms, including risk assessment, cost-benefit analysis, and environmental health impact assessment, are used to describe similar integrated procedures where scientific information is systematically collated and synthesised to permit decision making for the benefit of society. In this thesis, the term "integrated assessment" is used to refer to all different assessment types.

The integrated assessments are typically based on mathematical models. Mathematical models describe a part of the reality in mathematical terms providing quantitative estimates (e.g. the number of premature deaths due to air pollution emissions). The development of integrated assessment models involves several steps. The EPA Draft Guidance on the Development, Evaluation, and Application of Regulatory Environmental Models (EPA, 2003) divided an assessment into three steps: model development, model evaluation and model application steps. Model development involves the identification of the problem and the construction of a mathematical model. Model evaluation involves the determination of the quality of the model and running the model in a given situation (e.g. with sensitivity analysis). Model application covers the documentation and communication of the results.

The integrated assessment process aims to cover all the relevant interactions between society and the environment. Several causal frameworks have been developed to identify these interactions. These include e.g. PSR (Pressure-State-Response) by OECD (OECD, 1993) and DPSIR by EEA (Driving force - Pressure - State - Impact – Response). The integrated assessment process can use these frameworks in helping to identify the causal chain that leads to the current state of the environment. For example, people have a need to move (Driving force), which leads to air pollution emissions from traffic (Pressure), which causes increased concentrations of pollutants in the environment (State) leading to adverse health effects (Impact), which requires actions from society (Response). The society can intervene to mitigate the air pollution problem by influencing either the driving force, pressure, state or the impact.

An open assessment method, developed in the National Institute for Health and Welfare (THL), has its own framework for identifying the interactions between society and the environment (Tuomisto and Pohjola, 2007). The open assessment is a method to collect and synthesize scientific information in a coherent way. The open assessment process is divided into six phases (Tuomisto and Pohjola, 2007):

- Scoping (defining the purpose, question(s), intended use, boundaries, and the participatory width of the assessment)
- Applying (information and variables from existing assessments)
- Drawing a causal diagram (including decisions, outcomes, indicators and other variables)
- Designing variables (defining the attribute contents for individual variables)
- Executing variables and analyses (collecting the data needed, executing the models defining the results of variables and making assessment-specific analyses)
- Reporting the assessment (communicating the results and conclusions to the users)

The general integrated assessment framework for $PPM_{2.5}$ air pollution has been illustrated in Figure 2.1. The $PPM_{2.5}$ air pollution is emitted from a number of source categories of which the most important are traffic and energy production (WHO, 2006). These two source categories are also important for the formation of acidification and the greenhouse gas emissions. The $PPM_{2.5}$ air pollution is dispersed through the ambient air and causes adverse health to humans, damages vegetation, and has other effects. The integrated assessment model for $PPM_{2.5}$ combines information from these different steps taking into account the possible interactions with other environmental impacts (e.g. global warming, acidification). The sensitivity analysis, decision analysis and optimizing methods can be used to identify the sensitivity of the model and guide decision making. This thesis concentrates on exposure and health impact assessment, and sensitivity analysis methods.

The most comprehensive integrated assessment model for $PM_{2.5}$ air pollution in Europe is the Regional Air Pollution Information and Simulation (RAINS) model, developed by International Institute for Applied Systems Analysis (IIASA) (http://www.iiasa.ac.at/rains/). The RAINS model has been developed since the 1980's and e.g. it has been used to support acidification negotiations in Europe (Hordijk, 1991). In recent years, the RAINS-model has been updated to include both PM and precursor gas emissions. The RAINS-model was the main integrated assessment model used in the European Clean Air for Europe (CAFE) program that estimated the adverse health effects due to air pollution emissions in Europe. The current version of RAINS includes also greenhouse gas emissions. The new extended version of RAINS is called GAINS, Greenhouse gas – Air pollution

Interactions and Synergies (http://www.iiasa.ac.at/rains/). The GAINS is the main European level integrated assessment model used in estimating the impact of air pollution. Another European level integrated assessment model for air pollution is EcoSense that estimates health and other impacts due to classical air pollutants, including PM (http://ecosenseweb.ier.uni-stuttgart.de/).

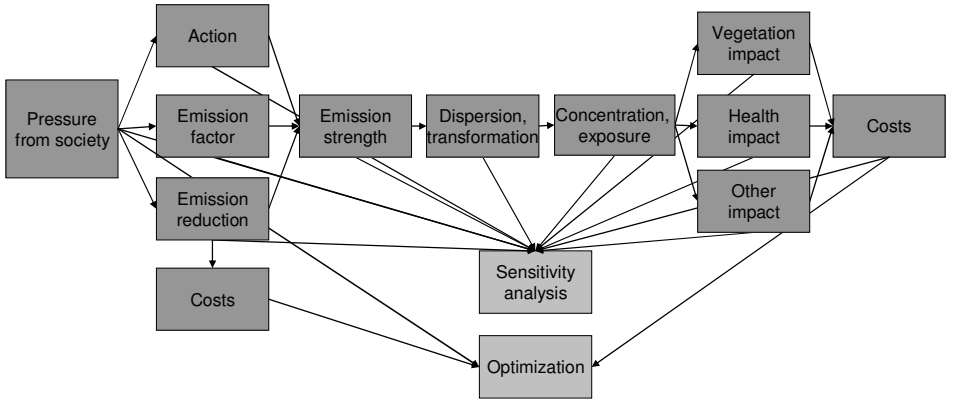

Figure 2.1: A general integrated assessment framework for $PM_{2.5}$ air pollution.

2.2 Specific literature review

2.2.1 Assessing exposure to anthropogenic $PPM_{2.5}$

In this thesis, exposure is defined as the concentration of the pollutant in the breathing zone. The breathing zone is the area where people inhale the air. The $PPM_{2.5}$ in breathing zone consists particles from different emission sources that can be located near or far away from the breathing zone.

The population exposure to $PPM_{2.5}$ can be calculated when the concentrations in different micro-environments (e.g. home, traffic, movie theatre) and the time spent in these micro-environments (time-activity) is known. The indoor concentrations can be estimated based on ambient air concentrations by calculating the penetration of PM from ambient air to indoors. There are few exposure models capable of estimating concentrations of $PPM_{2.5}$ indoors, or in different microenvironments, because the implementation of the model requires large amounts of measurement data. For example, the exposure model described by Kousa et al. (2002) uses data from the large EXPOLIS-study (Air pollution exposure in European cities), which

measured air pollutant concentration in indoor and ambient air, and with personal measurement devices (Jantunen et al., 1998).

In practise, most of the integrated assessment studies use ambient concentrations of $PPM_{2.5}$ at different home addresses as a proxy of exposure. This simplification has implications depending on the emission source or source category. For example, indoor $PPM_{2.5}$ emission sources have only a minor impact on ambient concentrations but a larger impact on indoor concentrations and exposures. In addition, different emission sources emit $PPM_{2.5}$ with different aerodynamic sizes and, as discussed earlier, the size is an important factor in determining the extent of penetration of PM to indoors. The importance of these differences to different $PPM_{2.5}$ emission source categories is unknown.

The exposure due to specific $PPM_{2.5}$ emission source categories can be estimated with a dispersion method or a receptor method. Dispersion methods use atmospheric dispersion models to estimate the dispersion of PM in ambient air after its release. For example, the study van Zelm et al. (2008) used dispersion models to evaluate PM_{10} concentrations over Europe. Receptor methods are based on PM measurements in the receptor location. The location can be a central monitor in city or a personal monitoring device. For example, exposure in the APHEA study was estimated based on $PM_{2.5}$ and PM_{10} measurements in a number of European cities (Boldo et al., 2006). A short description of these two methods is provided below.

The exposure-response functions set requirements for the exposure assessment in an integrated assessment. The exposure-response function describes the change in population health due to exposure. This will be discussed in more detailed later. With respect to $PPM_{2.5}$, the exposure-response functions are usually derived from epidemiological cohort studies that have studied correlations between $PM_{2.5}$ concentrations over a long time period (years) and health effects (e.g. Dockery et al., 1993; Pope et al., 2002). The integrated assessment studies that are based on exposure-response functions from these epidemiological cohort studies use typically annual $PM_{2.5}$ concentrations in their assessment.

Atmospheric dispersion models

Atmospheric dispersion models use dispersion algorithms to estimate the dispersion of pollutants in time and space. The atmospheric dispersion models require input data, for example about emission location and strength, meteorology, transformation of air pollutants in the air, and the removal of the air pollutants (deposition). The atmospheric dispersion model is a common term for a variety of modelling systems starting from a simple box model which assumes that there is constant concentration inside a given geographical area. For a review of different modelling systems see e.g. EPA Support Center for Regulatory Atmospheric Modeling

(http://www.epa.gov/scram001/). For European examples see e.g. EMEP (http://www.emep.int/) or Chimere (http://www.lmd.polytechnique.fr/chimere/).

The resolution of the dispersion model is important when one is evaluating the exposure to different $PPM_{2.5}$ emission source categories. The model resolution describes the area (grid) in which the concentration is assumed to be constant (e.g. 10 km x 10 km). The dispersion modelling systems are often divided into urban and regional (continental) scale systems based on spatial resolution.

The regional-scale dispersion models predict long-range dispersion of the PM on the national or continental scale (e.g. model used in Zhou et al., 2006 study). The strength of these models is their ability to predict air pollutant concentrations far away from release location (e.g. in a different country). However, the concentrations near the emission source (from a few meters to a few kilometres) may be underestimated, especially for low emission height sources, because the model assumes that the emissions are distributed evenly inside the grid cells, including the cell where the emission was released. For air pollutants, which have a high spatial variation in emissions and concentrations in short distances, this smoothing out of concentrations might underestimate the concentration near the emissions source. The underestimation can be assumed to increase when the grid size increases. For sources that have a high spatial correlation with the population, this underestimation of concentrations will also underestimate the population exposure.

The impact of spatial resolution was studied in the City-Delta modelling study in four European cities: Berlin, Milan, Paris and Prague (Thunis et al., 2007). That study predicted air pollutant concentration in these cities with both regional-scale and urban-scale dispersion models with 50 km and 5 km spatial resolutions, respectively. The study involved a number of air pollutants including PM_{10}. Thunis et al. (2007) concluded that the urban-scale dispersion models predicted higher PM_{10} concentrations in the urban areas due its better ability to capture the impact of urban sources. The results from Thunis et al. (2007) can be considered at best indicative for $PPM_{2.5}$ because the study included both primary and secondary PM, and the PM_{10} was used instead $PM_{2.5}$. In addition, the resolution of urban model was only 5 km.

The urban-scale dispersion models evaluate the dispersion of air pollutants in smaller geographical areas, such as one urban area, with a smaller grid size than the regional scale dispersion models. In this respect, urban-scale models can evaluate better the spatial variation over short distances. However, the large continental level integrated assessment involves sources in hundreds of cities and implementing an urban-scale dispersion model for all of these cities is unpractical. Urban-scale models alone are also unable to predict PM concentrations due to local and long-

range sources. Therefore many urban-scale studies utilize a variety of strategies to incorporate the long-range transported PM into the model results. For example, Stein et al. (2007) and Gariazzo et al. (2007) have combined the results of regional-scale and urban-scale models.

Receptor models

Receptor models rely on $PM_{2.5}$ measurements done in the receptor location. For example the receptor location could be a measurement station in the city or a personal measurement device. The source categories of measured PM can be traced by comparing the chemical properties of PM with information on emission sources using source apportionment methods (Hopke et al., 2006; Thurston et al., 2005). The receptor approach has been used especially in epidemiological studies to compare the toxicity differences between different types of PM (e.g. Lanki et al., 2006; Mar et al., 2000).

The strength of receptor methods is the reliable estimate of $PM_{2.5}$ concentrations in the receptor location. The main weakness is the possible misidentification of emission source categories in the source apportionment. The variation in results between different source apportionment methods was studied in U.S. in 2003 by comparing source apportionment methods between different research groups and between methods (Hopke et al., 2006; Thurston et al., 2005). The study concluded that the selection of the source apportionment method did not confer any significant uncertainty to the results (Thurston et al., 2005). With respect to the main source categories, emissions from traffic and burning vegetation had the greatest uncertainty (Thurston et al., 2005). On the other hand, the methodological review of Grahame and Hidy (2007) noted several disadvantages of the source apportionment method. Their main critique was that the source identification varies between the methods used so that the source categories cannot be identified with sufficient accuracy and the location of emissions is uncertain (for example, it is unclear from how far the long-range PM can be transported). Thus, with the receptor approach alone it is difficult to draw conclusions on what and where emission sources or source categories should be mitigated.

The estimation of exposure in geographically large integrated assessment studies is impractical with receptor methods. The measurements of PM are conducted mainly in cities and the estimation of $PM_{2.5}$ concentrations is rarely done in rural areas. Also, applying of source apportionment method so that it includes chemical analyses from hundreds of measurement stations is both time consuming and expensive. The receptor-based exposure assessment fits best to a geographically small area where there are large numbers of PM measurement stations.

2.2.2 The intake fraction concept

The intake fraction (iF) concept (Bennett et al., 2002b) is an application of the source-receptor relationship. The source-receptor relationship describes the change in the pollutant concentration (receptor) in relation to emission strength (source). For example, if we assume a linear source-receptor relationship, a 10% increase in emission strength from source x would increase the concentration of a pollutant (due to source x) by 10% in all receptor locations. The source-receptor relationship can also be nonlinear. The source-receptor relationship is used in integrated assessments to summarize and incorporate dispersion information into the model (Figure 2.2).

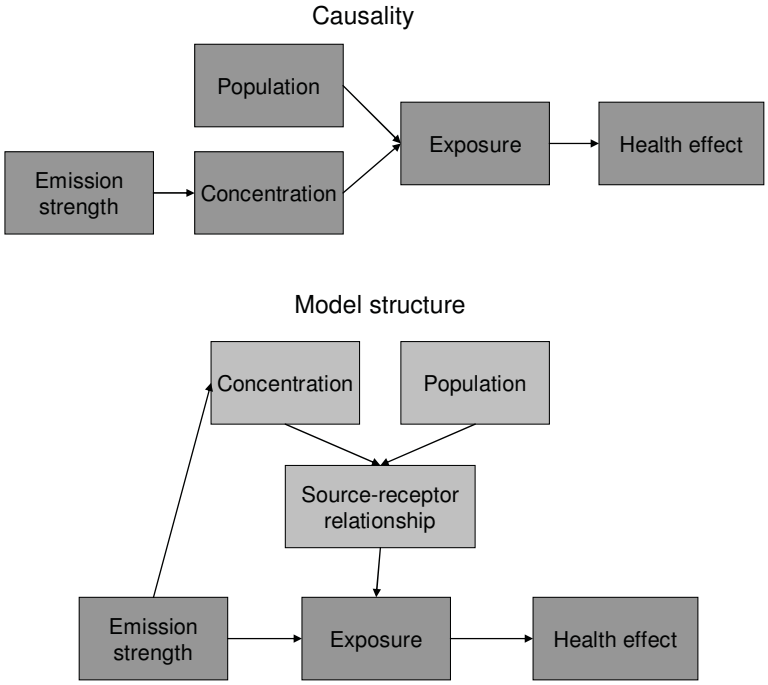

Figure 2.2: The difference in causal chain (upper part) and model structure (lower part) in $PPM_{2.5}$ integrated assessment. The iF concept (Bennett et al., 2002b) enables the combination and summarizing of concentration and exposure information into a single metric that can be used in the integrated assessment. The population location is usually assumed as the home addresses as described in the previous chapter.

The iF is defined as an "*integrated incremental intake of a pollutant released from a source category and summed over all exposed individuals*" (Bennett et al., 2002b). The exposure route can be inhalation, ingestion, or dermal. The concept of an iF is

based on a number of predecessor concepts with different names like exposure efficiency, exposure factor, and exposure effectiveness (see e.g. Evans et al., 2002; and Bennett et al., 2002b for details). The iF concept differs from concentration-based source-receptor relationship since it incorporating the population parameters (e.g. location, time-activity) with concentrations.

For $PPM_{2.5}$, iF can be calculated with the following equation (when using outdoor concentration of $PPM_{2.5}$ as a proxy of the population exposure):

$$iF = sum_i(C_i*Pop_i)*BR/Q$$

where iF is the intake fraction; C_i is the modelled concentration increase of $PM_{2.5}$ in a grid cell i (g/m^3); Popi is the population number in the grid cell i; BR is the average breathing rate; and Q is the emission strength (g/s). A breathing rate of $20m^3$/day/person is generally used in $PM_{2.5}$ iF studies (e.g. Wang et al., 2006) based on a past EPA recommendation (EPA, 1997). A number of grids cells (i) depend on the scale and the resolution of the assessment. Large integrated assessments may have hundreds of thousands of cells. In $PM_{2.5}$ integrated assessments, the exposure, and iF, is usually estimated for annual average concentrations as described in the previous chapter.

The exposure E (i.e. population average concentration in the study area) to $PPM_{2.5}$ can be calculated in the integrated assessment using equation:

$$E = (Q * iF)/(Pop*BR)$$

The iF concept has several benefits in integrated assessments (Evans et al., 2002). First, the iF concept allows the validation of results between exposure studies. The iFs for similar source categories should have fairly similar iFs; typical for outdoor air pollutants, like $PM_{2.5}$, between 10 per million to 0.1 per million (Bennett et al., 2002a). Second, the iF allows rapid screening-level integrated assessments since it permits the adoption and use of iF estimates from previous studies. This enables comparison of health risks from a number of sources in early assessment and then concentrating further assessment efforts on those sources, health effects, and uncertainties that have a major impact on assessment results.

The iF concept has been used in a number of $PM_{2.5}$ exposure studies. For example, Levy et al. (2002) have illustrated the exposure to both primary and secondary $PM_{2.5}$ emissions from individual power plants in the US using the iF concept. Zhou et al. (2003) have estimated iFs for power plants and Wang et al. (2006) for industrial processes in China. Marshall and Behrentz (2005) have used iF to estimate the

passengers` exposure to vehicle emission. Greco et al. (2007) have estimated spatial pattern of the iF of vehicle emissions in the city of Boston in the U.S.

2.2.3 Exposure-response function for $PPM_{2.5}$

The exposure information is combined with exposure-response functions to estimate adverse health effects caused by $PM_{2.5}$ (see Figure 2.1). Exposure-response function describes the change in the background health effect caused by the change in the exposure level.

The exposure to $PM_{2.5}$ has been associated with a number of health effects all over the world in hundreds of epidemiological and toxicological studies, (e.g. Schwarze et al., 2006; Pope and Dockery, 2006). In epidemiological studies the exposure-response is usually described with relative risk (RR) or odds ratio (OR). Relative risk is calculated with equation:

RR = P1/ P0

And odds ratio with equation:

OR = P1(1-P0)/ (P0(1-P1))

In these equations, P1 is the probability of health effects among those that were exposed (in this case exposed to $PM_{2.5}$) and P0 probability of health effect among those who were not exposed or were in a lower-exposed population group.

The integrated assessment on $PM_{2.5}$ has focused on long-term mortality impact because the major part of adverse health and economical impacts of PM are due to long-term mortality (e.g. EPA, 1999) in comparison to other adverse health effects (e.g. morbidity). The exposure-response functions used in these studies are based on epidemiological cohort studies.

The long-term epidemiological cohort studies
A number of epidemiological studies have examined the effect of long-term exposure and mortality for $PM_{2.5}$ (Pope and Dockery, 2006). Harvard Six Cities (HSC), American Cancer Society (ACS) and Dutch cohort, are discussed more detailed below. The main characteristics and results from these studies are described in Table 2.1.

Table 2.1: Comparison of different long-term epidemiological studies for $PM_{2.5}$. The results from different studies have been scaled to the same exposure level with Monte-Carlo methods. (ACS = American Cancer Society. HSC = Harvard Six Cities, CI = confidence interval)

Study	Percent change in all cause mortality per 1 μg/m^3 change in $PM_{2.5}$ concentration (mean and 95% CI)	$PM_{2.5}$ concentration range in the study (μg/m^3) (min-max)	Number of people in the analyses
ACS (Pope et al. 1995)	0.64 (0.33-0.93)	9.0-33.5	295 223
ACS reanalysis (Krewski et al. 2000)	0.68 (0.37-0.96)	9.0-33.5	295 223
ACS update (Pope et al. 2002)	0.58 (0.15-1.00)	5.0-30.0**	319 000
ACS Los Angeles (Jerrett et al. 2005)	2.17 (1.05-3.20)	6.0-30.0**	22 905
HSC (Dockery et al. 1993)	1.25 (0.34-2.04)	11.0-29.6	8111
HSC reanalysis (Krewski et al. 2000)	1.34 (0.42-2.13)	11.0-29.6	8111
HSC update (Laden et al. 2006)	1.50 (0.63-2.30)	10.2-29.0	8096
Dutch cohort (Hoek et al. 2002)*	2.74 (-1.21-5.66)*	9.6-35.8*	4 492
Dutch cohort update (Beelen et al. 2008)	0.58 (-0.36-1.45)	23.0-36.8	117 528

* The effect is for BS

** Visual inspection from the article

The Harvard Six City study consists of a cohort of adults in six different communities in US and was designed to study the health effects of air pollution. In the Dockery et al. (1993) article the $PM_{2.5}$ concentration was associated statistically significantly with total mortality and cardiopulmonary mortality. The analysis was based on $PM_{2.5}$ concentration taken from the years 1979-1985. The results were reanalyzed and replicated in 2000 by an expert team supported by the Health Effects Institute (HEI) (Krewski et al., 2000). The follow up extended the analysis period until the year 1998 (Laden et al., 2006). The follow up found a statistically significant association between $PM_{2.5}$ concentrations with total and cardiovascular mortality.

The American Cancer Society study is a large ongoing mortality study in which also the health effects of $PM_{2.5}$ air pollution have been estimated. The article of Pope et al. (1995) described the statistically significant association between total and cardiopulmonary mortality. The air pollution data was based on EPA measurements for the years 1982 and 1989. Furthermore, these results were confirmed by the reanalysis team in 2000 (Krewski et al., 2000). The follow up was published in 2002 and it extended the air pollution measurements with data from the years 1999-2000 (Pope et al., 2002). The study found statistically significant association between all-cause, cardiopulmonary and lung cancer mortality and $PM_{2.5}$ concentration. Jerrett et al. (2005) studied, based on the ACS cohort, within city variation in exposure and health effects in the Los Angeles, U.S. The study revealed a statistically significant correlation between $PM_{2.5}$ concentrations and all-cause mortality. The RR estimates in Jerrett et al. (2005) study were significantly higher than that reported in the previous ACS study (see Table 2.1).

The Dutch cohort study is based on The Netherlands Cohort study on Diet and Cancer. The pilot study from year 2002 included a random sample of 5000 people from the cohort (Hoek et al., 2002). In this study, the mortality impact was associated with black smoke and nitrogen dioxide concentrations. Black smoke is an indicator of the elemental carbon fraction of $PM_{2.5}$ and it has been common air pollution marker in air pollution measurements in Europe. The study founded a statistical association between cardiopulmonary mortality and black smoke concentrations near major roads. The full cohort, published in 2008, estimated $PM_{2.5}$ concentrations based on PM_{10} measurements (Beelen et al., 2008). All mortality estimates for $PM_{2.5}$ were statistically insignificant (Table 2.1.).

The implications of the evidence from these epidemiological studies have been reviewed and discussed in tens of publications (e.g. Pope and Dockery, 2006; Torfs et al. 2007), including critical publications (Moolgavkar, 2005). The exposure-response estimates differ substantially between different studies with the mean mortality increase due to 1 $\mu g/m^3$ $PM_{2.5}$ exposure varying from 0.58% to 2.74%. Pope and Dockery (2006) discussed two possible explanations for this phenomenon. First, as noticed in the reanalysis of HSC and ACS studies, education seems to modify the mortality impact so that those individuals with higher education have lower mortality risk (Krewski et al., 2000). The education level in ACS cohort is higher than in HSC cohort and the lower mortality increase in ACS study in comparison to HSC could be partly due to differences in the level of education of the cohort population. Second, the exposure estimates differ significantly between studies. In general, studies that have used finer spatial resolution to relate people to air pollution levels (HSC, ACS Los Angeles, and Dutch cohort) tend to report higher mortality impacts.

The HSC, ACS and Dutch cohort studies have concentrated on the adult population. Several epidemiological studies have also studied the association between PM and mortality in infants (age less than one year old) (see e.g. reviews Glinianaia et al., 2004; Sram et al., 2005; Tong and Colditz, 2004). These reviews have concluded that there are some evidence for an association between PM levels and different mortality outcomes but many methodological weaknesses may have modified the results.

Expert judgment studies

Expert judgment (elicitation of expert judgment) provides a method to assess and combine scientific information (Cooke, 1991). In an expert judgment study, several experts are asked formal questions about some particularly interesting questions (e.g. exposure-response function of $PM_{2.5}$). The experts then provide, based on their knowledge, the best guess and uncertainty intervals for their estimates. Two expert judgment studies have examined the relationship between $PM_{2.5}$ exposure and

mortality impact (Cooke et al., 2007; Industrial Economics Inc., 2004, 2006; Roman et al., 2008; Tuomisto et al., 2008).

The U.S. Environmental Protection Agency (EPA) has prepared a pilot and full study to characterize uncertainty in $PM_{2.5}$ exposure-response function for mortality (Industrial Economics Inc., 2004, 2006; Roman et al., 2008). The pilot study was performed with five experts from whom questions about both short-term and long-term mortality impact due to $PM_{2.5}$ exposure were asked. The five experts estimated that 1 $\mu g/m^3$ change in $PM_{2.5}$ exposure would change median non-accidental mortality in U.S. from 0% to 0.7% (Industrial Economics Inc., 2004). The uncertainty was recognised as being high.

After the pilot study, the EPA performed an expert judgment study with 12 experts (Roman et al., 2008). The study concentrated solely on long-term mortality and involved more detailed questions concerning the shape of the exposure-response function, confounding, threshold, and causality. In that study, the individual experts' median estimates for the change in non-accidental mortality due to 1 $\mu g/m^3$ change in $PM_{2.5}$ exposure varied from 0.4% to 2.0% (Industrial Economics Inc., 2006). In general, the experts in this study estimated a higher mortality response to $PM_{2.5}$ exposure than pilot study. This was explained as being due both to changes in the assessment protocol as well as new epidemiological evidence published after the pilot study (especially Jerrett et al. (2005) and Laden et al. (2006) studies). However, uncertainty was again recognised as being high

The second expert judgment study was performed for six European air pollution experts (Cooke et al. 2007, Tuomisto et al. 2008). In this expert elicitation study, the experts provided quantitative estimates of mortality impacts of hypothetical short- and long-term changes in $PM_{2.5}$ concentrations in the U.S. and Europe and for several other variables. The expert's estimates were then combined based on calibration questions. The median change in mortality due to 1 $\mu g/m^3$ change in $PM_{2.5}$ exposure was 0.60% or 0.97% in U.S. and 0.62% or 0.98% in Europe depending on the method of combining experts answers (Tuomisto et al., 2008). In general, experts were considering the uncertainties to be much higher than those reported in epidemiological studies. The experts also estimated that exposure-response function for $PPM_{2.5}$ is higher than that observed in cohort studies.

Causality

Epidemiology studies association between an environmental exposure, like $PPM_{2.5}$, and health outcomes. This association is assumed to be causal if the association between environmental stressor and health effects have been observed in several studies with varying methods (see Hill (1965)). If the evidence is not fully

convincing, the integrated assessment can describe this uncertainty as the plausibility of the association between the exposure and health effect.

At present, research has been unable to define a causal mechanism or mechanisms that are responsible for the increased mortality as noticed in a number of epidemiological studies (Pope and Dockery, 2006). However, as Pope and Dockery (2006) discussed, several biologically plausible mechanisms have been postulated and the plausibility of association has increased during the past decade. The critical review of Moolgavkar (2005) on the other hand did point out possible biases in the published studies and concluded that the evidence on causality is weak. The causality between $PM_{2.5}$ exposure and mortality was vigorously debated and analyzed in the EPA expert elicitation study (Roman et al., 2008). In that analysis, 10 out of 12 experts estimated that $PM_{2.5}$ exposure is causally linked to mortality with over 90% likelihood (Roman et al., 2008).

2.2.4 Toxicity differences between different $PM_{2.5}$ emission source categories

Toxicity describes the ability of a stressor to damage an organism. In this instance, we will examine the ability of $PM_{2.5}$ air pollution to cause adverse health effects in human population. For $PPM_{2.5}$, the dose dependency is expressed as exposure-response functions (percentage change in adverse health effect per change in $PM_{2.5}$ exposure).

Ambient $PPM_{2.5}$ is emitted from a number of sources, and it has different chemical (e.g. different elements and acidity) and physical (e.g. size, shape and number) characteristics depending on the source. It is most likely that these properties will modify toxicity. However, the research on toxicity differences of $PM_{2.5}$ is complicated because the chemical and physical characteristics change in the ambient air such that the inhaled PM are different from those PM that are emitted from a source. Thus, the toxicity of traffic emissions can be different in a location close to the source in comparison with one farther away. There is uncertainty related to the mechanisms that cause adverse health effects of PM. This complicates the comparison of PM properties to health outcomes. This has led to a situation where a number of PM properties are compared with a number of health outcomes. This does mean that even though there are hundreds of studies (e.g. reviews de Kok et al. (2006) and Schwarze et al. (2006)), the interpretation of this information to exposure-response functions is difficult.

The review of Schwarze et al. (2006) compared results from toxicological and epidemiological studies with different PM properties. They concluded that the

evidence is the most convincing for metals, especially for zinc, copper, vanadium, iron and nickel. They continued that the toxicological studies tend to place a higher importance for organic compounds but no such effect has been noticed in epidemiological studies. The situation for inorganic ions, especially for secondary sulphate, is less straightforward with conflicting results between toxicological and epidemiological studies. Another review by de Kok et al. (2006) considered the size fraction between 1.0 and 2.5 μm and the organic fraction in this size fraction to be the most important.

In epidemiology, toxicity differences have been studied by identifying the sources from measured PM with the source-apportionment method and then this information is incorporated into time-series studies. Time-series studies compare the daily air pollution levels to the same and the following day's health outcomes (e.g. mortality, hospital admissions). These studies face several methodological challenges. First, the identification of sources from the PM mass measures involves uncertainty (as discussed earlier in this thesis). Second, the observed exposure-response relationship differences could be due to differences either in exposure, toxicity or both. Third, PM from similar sources may have different chemical compositions and toxicities in different parts of the world, which complicates generalizing of the results.

Three time-series studies from the U.S. have studied the toxicity differences between PM exposure and their sources (Laden et al., 2000; Mar et al., 2000; Tsai et al., 2000). Laden et al. (2000) used the elemental composition of $PM_{2.5}$ to identify the sources of measured PM and then related the PM concentration to variation in daily mortality. They concluded that the combustion sources from both traffic and coal combustion were associated to mortality while crustal sources were not important sources. Mar et al. (2000) and Tsai et al. (2000) used factor analysis and Poisson regression to estimate source-specific risk ratio for $PM_{2.5}$. Mar et al. (2000) concluded that the combustion-related pollutants and secondary sulphate PM were associated with mortality. Tsai et al. (2000) detected a statistically significant association to PM from oil burning, industry, sulphate PM and traffic. The source apportionment in the studies of Laden et al. (2000) and Mar et al. (2000), especially for long-range transported PM, has been criticized by Grahame and Hidy (2007). They concluded that the identification of long-range transported sources was dependent on the source-apportionment method and therefore the identification of these source categories might lead to biased estimates.

In Europe, toxicity differences between sources have been studied in the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study (Pekkanen et al., 2002). In the ULTRA study, a panel of elderly subjects was visiting biweekly a clinic where a number of health indicators were measured and recorded. Lanki et al. (2006) compared the $PM_{2.5}$ exposure to an ischemic marker in

the electrocardiogram (ST-segment depression) in Helsinki, Finland. The $PM_{2.5}$ were apportioned to five source categories using absolute principal component analysis with multivariate linear regression based on both PM and gaseous air pollutant concentrations (Vallius et al., 2003). In the epidemiological analysis, the local traffic and long-range transported PM were associated to ST-segment depression (Lanki et al., 2006). In a recent article from the same study using data from all three cities (Amsterdam, the Netherlands, Erfurt, Germany, and Helsinki, Finland,) the researchers concluded that the traffic and long-range transported $PM_{2.5}$ were associated with health outcomes (de Hartog et al., 2009).

There are also epidemiological intervention studies where a change in legislation or some other intervention has rapidly decreased the PM concentration in a specific location. A study in Dublin, Ireland, noticed a reduction in mortality after banning of the sale of coal in the city area (Clancy et al., 2002). Another study compared the health effects and air pollution in Utah Valley, U.S., during a strike in a large steel mill and found that the all-cause mortality was correlated with PM_{10} concentrations (Pope et al., 1992).

The toxicity differences between different source categories were one of the main questions in the European elicitation study of expert judgment described in the previous chapter (Cooke et al., 2007; Tuomisto et al., 2008). As part of the study, experts were asked to give mortality impact estimates for the least and the most toxic component of PM mixture and to define those elements. All the experts identified combustion-related PM, especially from traffic, as being more toxic than the average PM mixture (Cooke et al., 2007). The experts were in less agreement about the least toxic component. In general, the experts assumed that secondary PM (sulphate, nitrate or both) and crustal material are less toxic than the average PM. The uncertainties were recognized to be high.

The toxicity difference between different PM characteristics was also discussed in the review of New Energy Externalities Developments for Sustainability (NEEDS) project that developed exposure-response functions for PM and ozone (Torfs et al. 2007). The review came to the conclusion that current evidence is not strong enough for quantification of toxicity differences between PM properties or sources.

In spring 2007 the WHO workshop in Bonn, Germany, discussed the evidence on exposure and toxicity differences between different PM sources (WHO, 2007). The workshop concluded that the current scientific knowledge does not provide sufficient information to separate the toxicities of different PM sources from one another. However, the workshop acknowledged that the evidence is strong for major combustion sources. In individual presentations, the experts recognized the need to

test toxicity differences in an integrated assessment and to evaluate the importance of these differences with sensitivity analysis methods.

2.2.5 Measures of public health

Several measures of public health have been developed to express the change in population health status due to exposure to stressors. For example, McAlearney et al. (1999) reviewed 13 different health measures including life-expectancy, quality-adjusted life-years (QALY), disability-adjusted life-years (DALY), health-adjusted life-expectancy, and healthy days gained. The review did not include the most common measure, premature death. Integrated assessments use these measures of public health in order to express the change in population health status due to exposure to environmental stressors. The selection of the measure depends on the environmental stressor, availability of data, computer resources, and skill. The premature death, life-expectancy, QALY, and DALY measures will be discussed in more detail below.

Premature death

The premature death (mortality) measures the change in mortality due to exposure to environmental stressor. Other terms for premature death are avoidable death (e.g. Kan et al., 2004) and attributable cases (e.g. Kunzli et al., 2000). The premature death due to $PM_{2.5}$ exposure can be estimated with the equation:

$$M = \exp(\beta \Delta E) \times Mb$$

where β is the exposure-response coefficient, ΔE change in $PM_{2.5}$ exposure, Mb background mortality, and M mortality after the exposure. The β can be estimated from the risk ratio (RR) with the equation:

$$\beta = \ln(RR)/\Delta Er$$

where RR is the risk ratio and the ΔEr is the change in $PM_{2.5}$ concentration to which RR has been related. The premature death can be estimated for all mortality outcomes combined or separately for different mortality outcomes (e.g. lung cancer and cardiopulmonary mortality).

The premature death measure has been criticized (Brunekreef and Hoek, 2000; Rabl, 2003). The authors of both reports argued that premature death is misleading because the measure does not provide any information on how premature is the actual death. Thus, the premature death does not distinguish between a case where death is advanced by one day from the situation of one year, or one decade.

Therefore the comparison of premature deaths due to $PM_{2.5}$ exposure with car accidents may give a false impression of the magnitude of health impact since the impact of air pollution affect predominantly at an older age while car accidents happen at a younger age. Rabl (2003) also concluded that the premature death is not meaningful because the number of deaths from different stressors would exceed the total observed mortality and because the number of people dying due to air pollution exposure can not be measured.

Despite these criticisms, the premature death is widely used in integrated assessments because of its easy intelligibility and the availability of data. Other requirements in integrated assessment also favour premature death, such as economical valuation, as discussed by the CAFE cost benefit analysis team in their response to "Union of Industrial and Employers' Confederations of Europe" (UNICE) concerns on the assessment methodology (Hurley et al., 2005).

Life-expectancy

The life-expectancy measure has been supported by most premature death critics (e.g. Rabl, 2003). Life-expectancy is a statistical measure of the average life span of a population and it takes into account the age when adverse effects occur. For example, one infant death due to exposure to $PM_{2.5}$ leads to a reduction of almost 80 years of life, while a heart attack at the age of 50 will lead to a reduction of 30 years. The life-expectancy can be estimated with life-tables that express the probability of surviving over the next age interval (Miller and Hurley, 2003). In addition to life-expectancy, a number of other health measures can be estimated from a life-table. Several life-table models have been developed to estimate the change in life-expectancy due to $PM_{2.5}$ exposure (Brunekreef, 1997; Leksell and Rabl, 2001; Mechler et al., 2002; Miller and Hurley, 2003; Nevalainen and Pekkanen, 1998; Rabl, 2003).

The life-tables are based on hazard rates which describe the probability of an event during a given time interval. The hazard rate is estimated with the equation (Miller and Hurley, 2003):

$$Hb = m/pop$$

where m is a number of deaths in a time interval (e.g. one year) and pop is the number of population in same time interval. Thus, 1 - Hb defines the probability to survive over the time interval. The hazard rates can be subdivided e.g. different time intervals, different mortality outcomes, or different sexes. The environmental stressors affect the life-expectancy estimates by multiplying hazard rates with the relative risks due to a given exposure.

The life-tables use these hazard rates to predict the survival of a population over the given time frame. Table 2.2 shows a life-table where the hazard rates have been estimated for one year age intervals and used to estimate the survival of population from one time interval (age) to another. The life-expectancy is then estimated by dividing the life years lived with the size of the starting population. The life years lived in each age group is calculated by assuming that those who did not survive the whole interval lived half a year.

Table 2.2: An illustration of life-table with one year time interval. The lives lived is the total number of person years lived during the time interval. The life-expectancy is calculated by dividing the lives lived over all time intervals with the size of the starting population. In the example, population life-expectancy is 79 785/1000 = 79.8 years.

Time interval (age)	Hazard rate (Hb)	Population at the beginning of time interval	Population at the end of time interval	Lives lived
0-1	0.00411	1000	996	997.9
1-2	0.00110	996	995	995.3
2-3	0.00110	995	994	994.2
3-4	0.00110	994	993	993.2
4-5	0.00110	993	992	992.1
5-6	0.00059	992	991	991.2
…	…	…	…	…
98-99	0.41590	50	29	39.6
99-100	0.70210	29	9	19.0
Sum	…	…	…	79785

The most common life-expectancy measure is the life-expectancy at birth. Life-expectancy at birth for year 2008 is estimate by calculating hazard rates based on population and mortality data from the year 2008 and assuming that the hazard rates remain constant over the lifespan of the population. More sophisticated methods take into account the change in hazard rates over the time e.g. by adopting the mortality projections from WHO (Mathers and Loncar, 2006). Conditional life-expectancy can be estimated for different age groups or taking into account population age structure.

The estimation of life-expectancy requires more time and data than the premature death measure. First, the life-table requires information on both population and mortality statistics at a more detailed level that premature death measure (e.g. mortality divided into one year intervals). Population and mortality statistics at the national level are readily available for example from WHO and UN databases but for smaller geographical areas (e.g. cities) the data may be inadequate.

Second, the life-table models require more computational efforts than the premature death measure. The practicality is important for integrated assessment because the integrated assessment models are created to support decision making and the complexity of model may hamper the usefulness of the model.

Adjusted health measures

Adjusted health measures (also known as weighted health indicators) measure the change in population health status by combining different health effects into one measure. The main benefit of adjusted health measure is the combination of mortality and morbidity effects. Two common adjusted health measures are the "quality-adjusted life-year" (QALY) and the "disability-adjusted life-year" (DALY) (McAlearney et al., 1999; Sassi, 2006).

The QALY measure combines the life-expectancy and the quality of the life. When calculating life-expectancy, the individual's health status is either 1 (life) or 0 (death). The QALY defines the quality of the life by using so called quality of the life weight factors. These weight factors are based on individual's feeling of their quality of life and can have a value between 1 (full health) and 0 (death) (Sassi, 2006). A number of QALY's gained in one year can be estimated simply by multiplying the 1 with the quality factor:

$$QALY = 1 \times Q$$

where Q is the quality weight based on the individual's health status. This equation can be combined with the life-table calculations so that both life-expectancy and the QALY are estimated for each time interval.

The DALY measure resembles QALY in many ways. The main difference between QALY and DALY is the interpretation of weighting factors. In QALY, the weighting factor is based on quality of life enjoyed by individuals, where as the DALY weighting factor represents the loss of functioning caused by a disease (Sassi, 2006). The DALY weights are scaled from 1 (death) to 0 (no disability). The DALY weights are usually based on expert valuations while QALY weights are based on measurement sampled from the population (Sassi, 2006). The DALY measure have been developed and applied especially in the Global Burden of Disease study (Murray and Lopez, 1997).

2.2.6 Sensitivity analysis

The sensitivity analyses determine how the uncertainty in the input variable affects the uncertainty of the model output. The definition of uncertainty may vary

depending on the fields. In this thesis, uncertainty means the lack of knowledge of an input variable or the mathematical formulation of the model. In some instances, these have been categorised to parameter and model uncertainty (e.g. Linkov and Burmistrov, 2003).

Sensitivity analysis resembles an experimental research where the impact of action or intervention (input variable) on the experiment result (model output) is measured. The sensitivity analysis can be used in the integrated assessment to identify the key sources of uncertainty and this can be used to guide model development and further research (Mokhtari et al., 2006). In order to be able to perform an importance analysis, a modeller needs (i) to define the causal system including consequences of actions and (ii) to define the uncertainties along the system.

The sensitivity analysis method can be divided to (i) mathematical, (ii) statistical, and (iii) graphical (Patil and Frey, 2004). More information on different sensitivity analysis methods is provided for example in the following papers (Frey and Patil, 2002; Mokhtari and Frey, 2005; Mokhtari et al., 2006; Patil and Frey, 2004). The performing of a sensitivity analysis is commonly recommended in the integrated assessment. For example, the WHO task force for health strongly recommended the use of sensitivity analysis methods in the development of the RAINS model in the CAFE project (UN, 2004).

Assessing uncertainties

The assessment of uncertainty to an individual input variable depends on the availability and the quality of the data. In an ideal case, both data and the results from statistical tests are available and the uncertainty can be expressed with normal, log-normal or other common probability distributions. For example, in the air pollution epidemiology the risk ratios (RR) are commonly expressed with a normal distribution (e.g. Dockery et al., 1993; Pope et al., 2002). However, as discussed earlier in the exposure-response chapter, this involves only statistical uncertainty and the true uncertainty might be higher, especially when using results obtained outside the target population.

The elicitation of expert judgment covers a variety of different methods that are applied to draw a so-called “educational guess” from the experts. In an expert elicitation study, experts are asked formal questions about some particular interesting question (e.g. exposure-response function for $PM_{2.5}$ as presented earlier in this thesis). The experts then provide, based on their knowledge, the best guess and uncertainty intervals for their estimates and these estimates can then be used in integrated assessments.

Unfortunately in many cases the data is incomplete, missing or unavailable and the modeller must define uncertainty estimates based on his/her own author judgement. This poses a serious risk of over- or underestimation of uncertainty.

The mathematical definition of model (model uncertainty) is another source of uncertainty in the integrated assessment. Linkov and Burmistrov (2003) have studied model uncertainty by comparing the results of different models developed to estimate the exposure to radionuclide by its presence in vegetation. They concluded that in a situation where limited data is available, the results of different models may differ from each other by several orders of magnitude. In comparison, the parameter uncertainty was recognized to contribute around one order of magnitude uncertainty to the modelling results.

Importance analysis

Importance analysis is a statistical sensitivity analysis method based on rank-order correlation (also known as Spearman's rank correlation coefficient). In importance analysis, the importance of an individual variable is defined as the absolute rank-order correlation between the input variable sample and the model output sample. The importance analysis has several benefits. First, the rank-order correlation function is available in many statistical programs. Second, the analysis can be assessed to non-linear models (unlike the sample correlation analysis). Third, the importance analysis can be integrated into both the assessment model or it can be assessed from outside the model with different statistical programs.

The importance analysis has also several limitations. First, the rank-order correlation assumes that the models are monotonic. Monotonic means that the function preserves the given order, i.e. the function either increases or decreases. Second, the estimation of correlation to binary values may provide misleading results. Thus, the reviews that have assessed the sensitivity analysis method (Mokhtari and Frey, 2005; Mokhtari et al., 2006) have concluded that the rank-order correlation is a good screening level sensitivity analysis method, especially useful at identifing those uncertainties that are not important for the given model output. In the statistical sensitivity analysis, analysis of variance (ANOVA) could be applied in situations where the importance analysis could be estimated to yield misleading results.

Importance analysis is limited to examining of the effect of uncertainty. Sometimes the input variable may contribute to a large part of uncertainty in the assessment, but it might have an insignificant effect on decision making. For example, in the $PM_{2.5}$ integrated assessment, the exposure-response function may have high correlation (uncertainty) with model output but this uncertainty may not have any impact on decision making in a situation where only the health effects of $PM_{2.5}$ are considered. The value of information (VOI) is a decision analysis method that examines the

effect of uncertainty on decision making (Yokota and Thompson, 2004a, b). Originally VOI analysis has been developed to estimate the benefits of collecting additional information.

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3 Aims of the study

This doctoral dissertation studies uncertainties and the impact of uncertainty in the integrated assessment of primary fine particulate matter ($PPM_{2.5}$) air pollution. The specific objectives of the thesis are:

1. To estimate emission-exposure relationship for different $PPM_{2.5}$ emission source categories, evaluate differences in emission-exposure relationship estimates between different exposure assessment methods, and evaluate the significance of these differences in an integrated assessment.
2. To test and apply the intake fraction (iF) concept as a way to summarize the emission-exposure relationship, and their uncertainties, in integrated assessment.
3. To define and estimate uncertainties in exposure-response functions for $PPM_{2.5}$ air pollution, and compare the importance of these uncertainties with respect to the other uncertainties in integrated assessments.
4. To study differences in toxicity between different $PPM_{2.5}$ emission source categories and to evaluate the significance of these differences in integrated assessments.
5. To apply premature death method and develop the life-expectancy method as ways to measure the public health impacts attributable to $PPM_{2.5}$ air pollution.
6. To perform sensitivity analyses for uncertainties in integrated assessments of $PPM_{2.5}$ air pollution and identify uncertainties that have a significant impact on the assessment results.

In additional to methodological aims, this thesis summarizes and discusses adverse health effects caused by $PPM_{2.5}$ air pollution in Finland.

4 Health effects caused by primary fine particulate matter ($PM_{2.5}$) emitted from busses in Helsinki Metropolitan Area, Finland

Published in Risk Analysis, 2005, 25 (1): 151-160.

5 Parameter and model uncertainty in a life-table model for fine particles ($PM_{2.5}$): a statistical modeling study

Published in Environmental Health, 2007, 6 (24).

6 A simple concept for GIS-based estimation of population exposure to primary fine particles from vehicular traffic and domestic wood combustion

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Note: Due to copyright reasons the article has not been attached to the pdf version of this thesis.

7 Evaluation of the European population intake fractions for European and Finnish anthropogenic primary fine particulate matter emissions

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8 The integrated modeling of the health effects caused by anthropogenic primary fine particulate matter, with special focus on various source categories

Submitted to Atmospheric Environment.

Note: Due to copyright reasons the article has not been attached to the pdf version of this thesis.

9 Discussion

The discussion is divided into seven chapters, conclusions and a final section on future recommendations. Each of six study objectives is discussed in chapters 9.1-9.6 and the magnitude of health effect attributable to $PPM_{2.5}$ in Finland in chapter 9.7. The conclusions summarize the main conclusions of this thesis and the future recommendation will integrate the separate findings and place them into a larger context. One aspect of the discussion is to benchmark the methods used in Articles I-V to those used by other research teams in previous integrated assessments. These other assessment studies are summarized in Table 9.1.

9.1 The emission-exposure relationship for different emission source categories

The emission-exposure relationship was evaluated in Article I for $PPM_{2.5}$ emissions due to bus traffic in urban area and in Article IV for six $PPM_{2.5}$ emission source categories in Finland. The iF concept was used in Article IV to summarize the emission-exposure relationship. In Article III, a new concept, called Population-based time-average inhalation (PTAI), was developed to relate how $PPM_{2.5}$ emissions impact on the nearby population. The method was applied for $PPM_{2.5}$ emission estimates for source categories "traffic" and "domestic wood combustion". The findings from these studies about emission source categories traffic, domestic combustion and power plant are discussed below.

9.1.1 Traffic

The spatial resolution of dispersion model was noted to impact on the emission-exposure relationship estimates for traffic. In Article I, the population exposure to bus traffic-related $PM_{2.5}$ in the urban area was based on personal and urban PM measurements. In the discussion of Article IV we estimated, based on exposure estimates in Article I, that the iF for busses $PPM_{2.5}$ emissions in study area were approximately 50 per million. In Article IV, the iF for Finnish anthropogenic $PPM_{2.5}$ emissions over Northern-Europe was estimated with 5 km spatial resolution. The iF for road traffic $PPM_{2.5}$ emissions was 0.68 per million.

The seventy -fold difference between iF estimates for traffic $PPM_{2.5}$ emissions in Articles I and IV (50 and 0.68 per million, respectively) is significant and is not explained by differences in the study areas alone. As discussed in the literature review of this thesis, the regional scale dispersion models may underestimate the

exposure to low emission height sources (Thunis et al., 2007). This probably decreased the iF estimate for traffic in article IV, because population density in the proximity of traffic emissions is high, as observed in Article III. The preliminary results from study where we used 1 km spatial resolution over Finland to estimate iF for traffic $PPM_{2.5}$ emissions indicate that the use of 1 km spatial resolution increased iF estimates for traffic-related $PPM_{2.5}$ emissions by one order of magnitude in comparison to a 5 km spatial resolution (Tainio et al., 2008, available only in Finnish).

Table 9.1: Integrated assessment (IA) and health impact assessment (HIA) studies used in benchmarking. Health impact assessment studies means in this case studies that have evaluated the PM health impact but not emissions nor emission-exposure relationship. The studies are in publication order starting from the oldest.

Study	Emission source or source category	Size fraction of PM[1]	Geographical location or area	Assessment type
Kunzli *et al.*, 2000	Traffic, other	PM_{10}	Austria, France and Switzerland	IA
Wolff, 2000[2]	Traffic, power plant	$PM_{2.5}$	US	IA
Deck *et al.*, 2001	-	PM_{10}, $PM_{2.5}$	Philadelphia and Los Angeles, US	HIA
Levy and Spengler, 2002	Power plant	Secondary $PM_{2.5}$	Massachusetts, US	IA
Levy *et al.*, 2002	Power plant	$PM_{2.5}$	Washington, US	IA
Coyle *et al.*, 2003	-	Sulphate particles	Canada	HIA
Kan *et al.*, 2004	Energy production	TSP, PM_{10}, $PM_{2.5}$	Shanghai, China	IA
Kaiser *et al.*, 2004	-	PM_{10}	23 Metropolitan area, US	HIA
Hutchinson and Pearson, 2004	Traffic	PM_{10}	United Kingdom	IA
Forsberg *et al.*, 2005	-	PM_{10}, $PM_{2.5}$	Sweden	HIA
Boldo *et al.*, 2006	-	PM_{10}, $PM_{2.5}$	23 Metropolitan Area, Europe	HIA
Norman *et al.*, 2007	-	PM_{10}, $PM_{2.5}$	6 Metropolitan Area, South Africa	HIA
Levy *et al.*, 2007	Power plant	$PM_{2.5}$	US	IA
Golub and Strukova, 2008	-	TSP, PM_{10}, $PM_{2.5}$	Russia	HIA

1. All the size fractions mentioned in the study. The health effect estimation was mainly based on $PM_{2.5}$ concentrations.
2. Case study 2 in Wolff (2000).

The integrated assessment model RAINS (Regional Air Pollution Information and Simulation, http://www.iiasa.ac.at/rains/) do take the near source exposure into account in the so called City-Delta equations (Amann et al., 2004). The City-Delta equations describe the functional relationship between regional (background) PM concentrations and PM concentration on the urban (local) scale. In practice, the background PM concentrations are enhanced based on information from (i) diameter of the city, (ii) annual mean wind speed, (iii) the number of winter days with low wind speed, and (iv) emission densities (Amann et al., 2007). The City-Delta equations have been estimated by comparing the results from an urban-scale dispersion model results and a regional-scale dispersion model results from number of European cities. The possible source of bias in City-Delta method is the estimation of equations for all low emission height sources combined. It is possible that this might underestimate the impact of traffic and overestimate the impact of other emission source categories.

Three integrated assessment studies mentioned in table 9.1 (Hutchinson and Pearson, 2004; Kunzli et al., 2000; Wolff, 2000) have evaluated the exposure to traffic-related PM with varying methods. The Wolff (2000) study calculated emission-exposure relationship using the iF concept for power plant and traffic emissions in US with a regional-scale dispersion model CALPUFF. The mean iF for traffic $PPM_{2.5}$ emissions was 9.1 per million (Wolff, 2000). Kunzli et al. (2000) evaluated the adverse health effects due to traffic-related PM air pollution in three central European countries. In Austria and Switzerland, the exposure assessment was based on emission inventory and dispersion modelling of PM, and in France the exposure was estimated based on statistical analyses between PM and land use parameters (Filliger et al., 1999). The sensitivity of the dispersion model was evaluated by comparing estimates between the two models. The dispersion models uncertainty had only a minor impact on the mortality estimates (Kunzli et al., 2000). Hutchinson and Pearson (2004) used the source apportionment method to identify exposure to traffic-related air pollutants in the United Kingdom. Details of the source apportionment method or the possible sensitivity of the mortality impact on exposure uncertainties were not reported.

The comparison of iF results between studies I and IV also raises the question of whether long-range transport is important at all when evaluating the exposure and health effects for low emission height sources in the urban environment. In Article IV, we estimated that 64% of Finnish iF for traffic emissions is due to exposure inside Finland. This means that 64% of all PM inhaled from traffic sources are inhaled in Finland. If the iF inside the city is an order of magnitude higher than outside the city, then exposure due to long-range transport would have only a minor impact on total iF. Similar conclusions were drawn from the results of exposure assessment study for benzene and CO in California U.S (Marshall et al., 2003). In

that study, the urban exposure was estimated to contribute 70 to 600 times more to iF than exposure outside the urban area (Marshall et al., 2003).

In conclusion, these results indicate that the calculation of emission-exposure relationship for $PPM_{2.5}$ emissions from traffic with dispersion models is sensitive to spatial resolution. The coarse resolution might underestimate the exposure because the $PPM_{2.5}$ emissions from traffic have a low emission height and the emissions are formed near the population breathing zones. A recent review concluded that the spatial extent of impact for traffic PM emissions is 100-400 meter around the road (Zhou and Levy, 2007). The Zhou and Levy (2007) study provides some hint of the kind of resolution that might be required to estimate the exposure to traffic-related $PPM_{2.5}$ emissions. Another possibility is to estimate exposure to traffic-related PM air pollution using receptor based models as has been successfully done in two integrated assessment studies (Article I; Hutchinson and Pearson, 2004).

9.1.2 Domestic combustion

Domestic combustion is another major $PPM_{2.5}$ emission source category in addition to traffic which has a low emissions height and a high correlation between emission strengths and population. Therefore the estimation of emission-exposure relationship for domestic combustion faces similar challenges as that for traffic emissions. Some specific characteristics of domestic combustion will be described below.

The emission-exposure relationship for domestic wood combustion source category has a high variation between sub-sectors. In Article IV, we estimated 0.54 per million iF for domestic wood combustion $PPM_{2.5}$ emissions, which is slightly lower than the average iF for all $PPM_{2.5}$ sources combined (0.57 per million). The comparison of $PPM_{2.5}$ emissions and population proximity in Article III showed that there are significant differences in population densities associated with different domestic wood combustion sub-sectors. This suggests that sub-sectors have different emission-exposure relationships and a separate exposure analyses for these sub-sectors might be required in any integrated assessment to estimate the impact of mitigation actions.

In this thesis, we estimate exposure to domestic combustion emission through the ambient air. The population is exposed to $PPM_{2.5}$ emissions from domestic combustion also in indoors during the combustion and this exposure could be higher at the population level than the exposure through ambient air. For example, a measurement study conducted in Sweden concluded that the domestic wood combustion increased exposure to specific PM components (K, Ca and Zn) (Molnar et al., 2005). However, here the exposure-response functions for $PM_{2.5}$ are based on

outdoor concentrations and they might not describe the changes in population health due to changes in indoor PM concentration.

9.1.3 Power plants

In Article IV we estimated iF for $PPM_{2.5}$ emissions from power plants in Finland and found an iF of 0.50 per million. This was the lowest iF from the modelled six emission source categories. The other integrated assessment studies (Table 9.1) have mainly used dispersion models to estimate the distribution of primary PM emissions from power plants. The power plant emissions are released high above the ground so in theory the resolution of dispersion model will have only a minor impact on emission-exposure estimates. The sensitivity analyses performed in Levy et al. (2002), Levy and Spengler (2002 and Levy et al. (2007) concluded that dispersion uncertainties had only a minor impact on health effect estimates in comparison to exposure-response uncertainties.

The different $PPM_{2.5}$ emission sources and source categories have different emission-exposure relationships. The estimation of these relationships in an integrated assessment consisting a number of emission source categories poses a challenge since the use of one dispersion model with the same parameters for all source categories might cause under- or overestimation of the exposure between the different source categories. The properties of emission sources or source categories, especially emission height and spatial correlation between emissions and population, need to be evaluated prior to the exposure assessment and the exposure method has to be chosen so that it captures exposure in the most important distances from the source. One possibility to estimate exposure to urban sources is to use combined receptor- and dispersion-models. The sensitivity analyses performed in this study and in previous integrated assessment studies revealed that dispersion and exposure uncertainties have a lower impact on model results than exposure-response uncertainties. However, dispersion and exposure uncertainties have been rarely analysed in integrated assessment and the possibility of underestimation of the uncertainty exists.

9.2 The use of the iF concept in integrated assessments

The iF concept was applied in the integrated assessment for $PPM_{2.5}$ and found to be a practical tool to incorporate emission-exposure relationship into the assessment model. The iF concept has several advantages. First, state of the art atmospheric dispersion models for air pollutants are computationally complex and incorporation of the dispersion model into integrated assessment would be unpractical, as was discussed in the documentation of RAINS integrated assessment model (Amann et

al., 2004). In addition to being unpractical, some analyses would be impossible if the dispersion model were to be incorporated with the integrated assessment model. In RAINS-model, the optimization (Amann et al., 2004) and in Article V, the sensitivity analysis based on Monte-Carlo simulation.

Second, the iF concept enabled direct comparison of emission-exposure relationships between similar emission source categories. The comparison of iF estimates for traffic-related $PPM_{2.5}$ emissions between Article I and IV would have been more complicated without the iF method and the seventy fold difference in emission-exposure relationship could have remained unnoticed.

Third, the dispersion model uncertainty was propagated to integrated assessment through iF and, thus, avoiding this way the simulation of uncertainty in the concentration data. As the sensitivity analysis in Article V showed, the dispersion model uncertainty was one of the most important uncertainties in the integrated assessment model. However, the propagating of uncertainty through iF has problems since the dispersion uncertainty does vary in relation to the distance from the source and after the distance has been summarized in the iF equation, it is impossible to evaluate the spatial uncertainty.

The iF concept, as with all source-receptor relationships, is a simplification of the atmospheric processes and it might increase bias when the true emission strengths are substantially different from those used in the dispersion model (especially in situations where emission-exposure relationship is non-linear). Therefore, if the integrated assessment is used to support decision making, the exposure to the most promising or likely scenarios should be estimated separately with dispersion models.

The previous integrated assessment studies have used various methods to incorporate the dispersion information into the integrated assessment models. The Wolff (2000) study was based on the iF concept, although during that time, the concept was known with the name exposure efficiency. Other dispersion model studies have reported the use of average concentration over the study area (Kunzli et al., 2000; Levy and Spengler, 2002; Levy et al., 2002; Kan et al., 2004) or the use of exposure-receptor matrix (Levy et al., 2007). The integrated assessment model RAINS uses the source-receptor matrices (Amann et al., 2004). The possible advantages or disadvantages of those methods in terms of practicality have not been explored.

9.3 Uncertainty in exposure-response function for $PPM_{2.5}$

The uncertainties related to the exposure-response functions for $PPM_{2.5}$ air pollution were the main uncertainties in Articles I, II and V of this thesis as well as in numerous previous assessment studies. In this thesis, the exposure-response uncertainty was divided into uncertainty in exposure-response function and in causality and these will be discussed separately below.

9.3.1 Exposure-response function uncertainty

The exposure-response functions for long-term mortality impact of $PPM_{2.5}$ were estimated with two different approaches. In Articles I and II, the exposure-response functions were based on combining the estimates from Harvard Six Cities (Dockery et al., 1993) and the American Cancer Society (Pope et al., 2002) studies. The mortality outcomes were divided into lung cancer, cardiopulmonary, other non-accidental, and accidental mortality. In Article II, infant mortality (age 0-1) was estimated separately based on study of Woodruff et al. (1997). The sensitivity analyses in Articles I and II showed that the cardiopulmonary mortality was the main health outcome and the uncertainties related to this mortality outcome had a major impact on integrated assessment model results. Other mortality outcomes and infant mortality had only a minor effect on the health effect estimations. The importance of cardiopulmonary mortality in relation to other health outcomes was due to the statistically significant association between $PM_{2.5}$ exposure and cardiopulmonary mortality, and due to high background prevalence.

In Article V, we used exposure-response functions derived from the expert elicitation study performed by six European air pollution health effect experts (Cooke et al., 2007; Tuomisto et al., 2008). The uncertainty of exposure-response function for $PM_{2.5}$ was higher in the expert elicitation study, reflecting the experts' views on uncertainty. The expert elicitation study related $PM_{2.5}$ exposure to non-accidental mortality and the mortality was divided into non-accidental and accidental mortality in Article V. The exposure-response function uncertainty was the main uncertainty in the integrated assessment study presented in Article V.

Almost all the PM integrated assessment studies referred to in this thesis (Table 9.1) have evaluated the exposure-response function uncertainties in the assessment. Forsberg et al. (2005) discussed uncertainties qualitatively and concluded that the mortality estimate for local sources were underestimated because the exposure-response functions, based on U.S. epidemiological cohort study, were not representative of local sources. Levy and Spengler (2002) estimated the impact on

Table 9.2: The source of exposure-response functions in different integrated assessment studies. (SIDS = Sudden Infant Death Syndrome).

Study	Mortality impact	Source of exposure-response function	Other health effects (morbidity)
Article I	Cardiopulmonary, lung cancer, other non-accidental	Dockery *et al.* , 1993; Pope *et al.* , 2002	-
Article II	Cardiopulmonary, lung cancer, other non-accidental	Dockery *et al.* , 1993; Pope *et al.* , 2002 ; Woodruff *et al.* , 1997	-
Article V	Non-accidental mortality	Tuomisto *et al.* , 2008	-
Kunzli *et al.* , 2000	Non-accidental mortality	Dockery *et al.* , 1993; Pope *et al.* , 1995	Hospital admission, bronchitis, restricted activity days, asthma attacks
Wolff, 2000[1]	Total mortality	Not reported	-
Deck *et al.* , 2001	Total mortality	Pope *et al.* , 1995	Short-term mortality, hospital admission, respiratory symptoms
Levy and Spengler, 2002	Total mortality	Krewski *et al.* , 2000	-
Levy *et al.* , 2002	Total mortality	Pope *et al.* , 2002	Hospital admissions, asthma
Coyle *et al.* , 2003	Total mortality	Krewski *et al.* , 2000	-
Kan *et al.* , 2004	Total mortality	Dockery *et al.* , 1993; Pope *et al.* , 1995	Bronchitis, hospital admissions, outpatient visits, asthma attacks
Kaiser *et al.* , 2004	All cause, SIDS, respiratory	Woodruff *et al.* , 1997	-
Hutchinson and Pearson, 2004	Total mortality	Not reported	Morbidity, hospital admissions
Forsberg *et al.* , 2005	Total mortality	Pope *et al.* , 2002	-
Boldo *et al.* , 2006	All-cause, cardiopulmonary, lung-cancer	Pope *et al.* , 2002	-
Norman *et al.* , 2007	Cardiopulmonary, lung cancer	Pope *et al.* , 2002	Respiratory infection
Levy *et al.* , 2007	All-cause	Pope *et al.* , 2002	-
Golub and Strukova, 2008	Non-accidental mortality, cardiopulmonary, lung-cancer	Pope *et al.* , 2002	-

1. Case study 2 in Wolff (2000).

mortality attributable to secondary $PM_{2.5}$ with a number of exposure-response functions. The mean premature deaths (97 per year) varied between 16 and 290 premature deaths with different exposure-response function assumptions being the main source of uncertainty in the assessment (Levy and Spengler, 2002). In the study of Kunzli et al. (2000) the exposure-response uncertainty changed the mean mortality estimates in Switzerland +/- 40%. The uncertainties related to exposure had less impact and uncertainties related to the threshold value had more impact on the integrated assessment model results than the uncertainty of the exposure-response function (Kunzli et al., 2000).

The exposure-response functions used in previous integrated assessment studies have been based mainly on the update of the ACS study (Pope et al., 2002) (Table 9.2). The reasons for using the exposure-response function from the ACS update have been e.g. (i) large and geographically diverse population (Levy et al., 2002; Levy et al., 2007), or (ii) the example of previous assessments (Levy et al., 2007; Golub and Strukova, 2008). The ACS update was also the main source of information for most respondents in the expert elicitation study conducted in the U.S. (Roman et al., 2008) and the recommended source of exposure-response function in the WHO guidebook for assessing the burden of disease due to outdoor air pollution (Ostro, 2004). As revealed in the sensitivity analysis of Levy and Spengler (2002), the selection of exposure-response function has a substantial effect on the integrated assessment results. Boldo et al. (2006) also discussed the adoption of emission-exposure functions from Pope et al. (2002) and assumed that this probably underestimated the mortality impact in their study.

9.3.2 Causality

Uncertainty of causality was the main uncertainty in Articles I and II. The uncertainty in causality between $PM_{2.5}$ and mortality was modelled in these Articles as plausibility. Plausibility was defined as the probability that the observed exposure-response association actually represents a causal association (i.e. the cause is not some other factor that happens to correlate with $PM_{2.5}$). The plausibility was defined separately for different mortality outcomes so that plausibility was highest for lung cancer and lowest for other non-accidental mortality. The high plausibility of lung cancer was selected because PM mass contains known carcinogens (Cohen, 2000). In Article V, the plausibility was incorporated into the exposure-response functions.

In conclusion, the uncertainty of the exposure-response function for $PPM_{2.5}$ air pollution has been the main uncertainty in the integrated assessments considering

the health effects of $PPM_{2.5}$ air pollution. The increased mortality has been mainly due to increased cardiopulmonary mortality. The uncertainty in the exposure-response function for infant mortality had only a minor impact on integrated assessment. This could indicate that the estimation of infant mortality has a minor impact on the results of the assessment. Most of the previous integrated assessment studies have adopted the exposure-response function from the update of ACS cohort study (Pope et al., 2002). Expert elicitation studies considering exposure-response functions for $PM_{2.5}$ air pollution have estimated that the uncertainty in the exposure-response function is higher than that reported in epidemiological studies. This uncertainty may be significant in integrated assessments where the $PM_{2.5}$ induced health effects are compared to other health effects.

9.4 The toxicity difference between $PPM_{2.5}$ from different sources categories

The toxicity of PM depends on the chemical (e.g. different elements and acidity) and physical (e.g. size, shape and number) characteristics of the particles. The chemical and physical characteristics of PM vary between emission sources and source categories which could lead to toxicity differences between emission sources. The toxicity difference between $PPM_{2.5}$ emission source categories is one of the main uncertainties encountering when trying to make decisions on emission mitigation actions. Toxicity differences, together with emission strength and emission-exposure relationship differences, can modify the benefits obtained from different mitigation actions from the health effects point of view. Together with the costs of these strategies, these determine how $PPM_{2.5}$ emissions can be mitigated effectively and economically.

The sensitivity analysis in Article V revealed that toxicity differences were one of the main uncertainties in the integrated assessment model. In the assessment, the $PPM_{2.5}$ emissions due to road traffic were responsible for approximately 40% of exposure and 50% of health effects in Finland due to $PPM_{2.5}$ emissions from Finland. Thus, the toxicity difference increased the relative importance of traffic-related exhaust gas emissions and decreased the cost per life saved for mitigation actions with respect this source category.

The previous integrated assessment studies have considered toxicity differences mainly in qualitative terms. In Article I, we concluded that the toxicity difference between $PM_{2.5}$ emissions from different busses was one of the main uncertainties in the assessment. This uncertainty was not propagated into the model due to lack of data. Forsberg et al. (2005) assumed that the mortality impact in their study was

underestimated because they assumed that local sources have a higher toxicity than long-range transported PM. In the European ExternE project (http://www.externe.info/) methodology update from 2005, the traffic was assumed to have 1.5 times the toxicity of the average $PM_{2.5}$ mass, while secondary sulphate and nitrate possessed lower toxicity than the average $PM_{2.5}$ (ExternE, 2005). The importance of these assumptions on the integrated assessment was not tested.

The current knowledge on toxicity differences between PM, or characteristics modifying PM toxicity, is inadequate. The WHO workshop in 2007 concluded that the evidence supporting the different toxicities for $PM_{2.5}$ is as strong as that assuming that they have equal toxicity (WHO, 2007). The workshop recommended the use of sensitivity analysis methods to quantify the impact of differential toxicity for integrated assessment models. In Article V we present a method which can be used to perform sensitivity analysis taking into account toxicity differences in the $PPM_{2.5}$ integrated assessment and concluded that toxicity differences have a significant impact on model results.

9.5 Measures of public health

The adverse health effects are indicated in the integrated assessment with measures of public health. The estimation of these measures varies from a simple premature death to more complicated, dynamic, life-table models that are used to estimate life-expectancy, QALY, DALY or other measures. The more complicated the measure, the more likely the uncertainty in the public health measure will affect the results of the integrated assessment model. In this thesis we performed a sensitivity analysis to test how uncertainties in the $PM_{2.5}$ life-table could affect the results of the integrated assessment. The results from this study were published in Article II and the model was updated in Article V based on a sensitivity analysis.

The life-table model in Article II used a sensitivity analysis to test the impact of model and parameter uncertainty on the life-table model results. The study focused on lag, defined as the time elapsing between a change in exposure and the ensuing change in the hazard rate. Thus, lag was used to estimate the time that exposure to $PPM_{2.5}$ will increase different mortality outcomes. The lag was estimated separately for the different mortality outcomes. In addition to lag, the life-table model had several other model and parameter uncertainties. The results of the sensitivity analysis showed that lag had only a minor impact on the results obtained with the model. This result has been supported by two PM life-table studies which reached similar conclusions (Leksell and Rabl, 2001; Roosli et al., 2005). The main

uncertainties in Article II were the exposure-response function for $PM_{2.5}$ and the discount rate.

Table 9.3: Measures of public health used in different integrated assessment studies. YOLL=Years-of-life-lost, QALY=Quality-adjusted life-years)

Study	Premature death	Morbidity outcomes	Life expectancy	YOLL	QALY	Monetary valuation
Article I	x					
Article II	x		x			x
Article V	x		x			
Kunzli et al., 2000	x	x				
Wolff, 2000	x					x
Deck et al., 2001	x	x				
Levy and Spengler, 2002	x					x
Levy et al., 2002	x	x				
Coyle et al. 2003			x	x	x	
Kan et al., 2004	x	x				
Kaiser et al., 2004	x					
Hutchinson and Pearson, 2004	x	x				x
Forsberg et al., 2005	x		x			
Boldo et al., 2006	x		x			
Norman et al., 2007	x	x		x		
Levy et al., 2007	x					
Golub and Strukova, 2008	x					

Previous PM integrated assessment studies have mainly used the premature death measure in their analyses (Table 9.3). The model or parameter uncertainties related to public health measures, and their impact on integrated assessments has been estimated mainly in qualitative terms. The Coyle et al. (2003) used the Monte-Carlo simulation to estimate the change in both QALY and life-expectancy due to sulphate PM in Canada. The background hazard rates were estimated separately for both gender and for different socioeconomic status. The sensitivity of life-expectancy and QALY measured for different discount estimates were assumed to be small (Coyle et al., 2003). Other integrated assessment studies reviewed here have concerned mainly uncertainties related to the exposure-response function.

9.6 Sensitivity analyses

The importance of uncertainty was defined in Articles I, II and V with a sensitivity analysis method called importance analysis. Importance analysis is based on a rank-order correlation between samples of an input variable and the model output.

The uncertainties were propagated through the models with a Monte-Carlo simulation. The parameter uncertainties were modelled with continuous uncertainty

distributions (e.g. normal, log-normal, uniform) and the model uncertainties with Bernoulli (binary) function. The sensitivity analyses faced several challenges of which two, the definition of uncertainty and choice of model result for sensitivity analyses, will be discussed below.

The identification of uncertainties in model structures and in input variables was a substantial task in all case assessments. In a few cases, the input variable uncertainty could be based on the literature. For most of the input variables, the uncertainty was assessed using expert estimates. Formal expert elicitation method was applied to investigate the uncertainty in the exposure-response function for $PM_{2.5}$. For other variables, the uncertainty was estimated based on sparse data, single expert judgment, or modeller's judgment (so called author judgment). Thus, different uncertainties were defined with different methods ranging from a full study to a crude guess of the modeller. This raises the doubt about how comparable are the different uncertainty estimates. However, the purpose of this thesis was to demonstrate how the sensitivity analysis methods based on a Monte-Carlo simulation can be used in integrated assessment and to identify those input variables that are having a major impact on model results.

The identification of model results for the sensitivity analysis was another challenge. For example, in Article II the sensitivity was estimated in the mortality estimates of different scenarios and in the comparison of different scenarios. These yielded different results showing different input variables important. For the scenarios, the result of sensitivity analysis was dependent on the discount rate while the comparison of scenarios revealed those input variables that were influencing the ranking of different scenarios.

Previous integrated assessment studies have used a variety of sensitivity analysis methods. Coyle et al. (2003) and Golub and Strukova (2008) used a Monte-Carlo simulation to propagate exposure-response function uncertainties in the integrated assessment. The other studies have compared the mean estimates of mortality impact between different model and parameter assumptions. For example, Kunzli et al. (2000) compared premature death estimates between different model assumptions and published their data in a table where the results were compared to the main point estimate. A similar sensitivity analysis method has been applied by others e.g. Deck et al. (2001) and Levy and Spengler (2002).

In this thesis we applied only a sensitivity analysis method. For the policy purposes, the sensitivity analysis could be supported with value of information analysis (VOI) (Yokota and Thompson, 2004a, b). Value of information examines the effect of uncertainty on decision and reveals those uncertainties that impact on the decision making. For example, the toxicity difference between $PPM_{2.5}$ emissions from

different buses in Article I would have had significant effects on the choice about which was the best bus option. This kind of information can be assumed to be more practical for the decision maker than information about different input variable uncertainties.

9.7 The magnitude of health effects caused by $PPM_{2.5}$ in Finland

In Finland the $PPM_{2.5}$ emissions from Finnish and non-Finnish sources were estimated to cause between 179 (mean, 90% confidence interval 8.85-515) and 306 (mean, 90% CI 9.39-1040) premature deaths, in 2000 and to lower population life-expectancy of 0.10 (mean, 90% CI 0.00-0.34) years (Article V). The main source countries of long-range transported primary $PM_{2.5}$ were Russia, Ukraine, Sweden and Estonia. The source categories responsible for the emissions from these countries were not evaluated in the assessment.

The $PPM_{2.5}$ emissions from Finland were estimated to cause 179 premature deaths in Finland in 2000. Of these 179 premature deaths, 78 were estimated to be due to traffic related $PPM_{2.5}$ emissions. In this sense traffic refers to tail-pipe and non-tail-pipe emissions from traffic and $PPM_{2.5}$ emissions from off-road, air traffic, and maritime sources. It was predicted that the local bus traffic and local traffic-related $PPM_{2.5}$ emissions would cause 20 premature deaths in 2020 if emission remain at 1997 level and 31 premature deaths due to local traffic in 2000, respectively, in Helsinki Metropolitan Area.

In Article IV and in this thesis we have assumed that the exposure, and the resulting health effects, for traffic-related $PPM_{2.5}$ was underestimated in Article IV. The preliminary results from a new study using a 1 km spatial resolution in dispersion model have indicated that traffic-related $PPM_{2.5}$ emissions could be responsible for several hundreds of premature deaths in Finland (Tainio et al., 2008). The exhaust gas $PPM_{2.5}$ emissions from traffic are expected to decline in the future and the importance of traffic-related $PPM_{2.5}$ emissions are also in the wane.

The second most important $PPM_{2.5}$ emission source category in Finland is domestic combustion estimated as being responsible for 41 premature deaths per year. In this case, domestic combustion refers to primary and secondary wood and non-wood heating in residential and recreational buildings. On the average, domestic combustion has a low emission height and high emission strengths near where people live which increase the importance of this source category. However, as demonstrated in Article III, different domestic combustion sub-categories may have completely different emission-exposure relationships. The $PPM_{2.5}$ emissions from

domestic combustion are assumed to remain constant in the future but if viewed in conjunction with declining $PPM_{2.5}$ emissions from traffic, then it could be anticipated to increase the relative importance of these domestic combustion-related $PPM_{2.5}$ emissions.

The $PPM_{2.5}$ emissions from other sources were responsible for 60 premature deaths per year in Finland. The power plants (both major and small power plants) were responsible for approximately half of this total. The power plants do emit high amounts of secondary PM precursor gases and the importance of this source category would increase if the secondary PM were to be included in the assessment.

In 2000, the European CAFE program estimated that primary $PM_{2.5}$ air pollution caused approximately 1300 premature deaths In Finland every year (Watkiss et al., 2005). The CAFE study included both primary and secondary $PM_{2.5}$. Forsberg et al. (2005) concluded, based on measurements in Sweden, that the long-range transported sulphate PM dominated the health effects in Scandinavian countries. This thesis did not evaluate secondary $PM_{2.5}$. Based on the relative importance of local emission source categories (traffic, domestic combustion) and the fact that secondary was estimated to have lower toxicity in the expert elicitation study, it can be speculated that local sources are also significant for population health. This indicates that both local actions and international agreements are both required to mitigate the $PPM_{2.5}$ induced health effects in Finland.

9.8 Conclusions

The main methodological conclusions from the present study are:

- The emission-exposure relationship variation between emission source categories has a significant impact on the results obtained with the integrated assessment model – especially for traffic emissions.
- The sparse spatial resolution of dispersion model may underestimate the exposure to low-emission height source categories. With respect to $PPM_{2.5}$ emissions from these sources, the exposure needs to be evaluated with a finer resolution to capture the true exposure to these sources.
- The intake fraction concept provided a practical way to describe the emission-exposure relationship and to incorporate this information into the integrated assessment. The intake fraction concept also enables the propagation of the dispersion model uncertainty into an integrated assessment through intake fraction estimates
- The exposure-response uncertainty was identified as being the main quantified uncertainty in integrated assessment model for $PM_{2.5}$ air pollution
- The difference in toxicity between $PPM_{2.5}$ emission sources has a major importance for the health effect estimation and this variation should be taken into account in large integrated assessments – at least in sensitivity analyses, if the evidence about toxicity differences is considered to be insufficient.
- The change in life-expectancy due to $PPM_{2.5}$ exposure can be assessed with reasonable accuracy without the need to incorporate the most complex and time-consuming parts of the model. The premature death indicator is easier to estimate and it has its own relevance when publishing results for the general public although interpretation of the results might be misleading. Therefore both indicators should be estimated when assessing health effects of $PPM_{2.5}$
- The sensitivity analysis method provides practical information about model uncertainties and their effect during the assessment and when reporting findings of the integrated assessment.

The main conclusions related to health effects are:

- Traffic is the most important $PPM_{2.5}$ emission source category in Finland causing tens of premature deaths annually. The current legislation is aimed at reducing the tail-pipe emissions from traffic and in the future other

emission sources, especially domestic combustion, might become more important.

- The health effects caused by long-range transported $PPM_{2.5}$ from other countries are higher in Finland than the health effects of domestic $PPM_{2.5}$. Different $PPM_{2.5}$ emissions source categories have different emission-exposure relationships and for some sources, the local exposure is the dominant factor impacting on the health. Therefore in particular international negotiations, in addition to national and local actions, are required to mitigate long-range transported $PPM_{2.5}$ emissions.

9.9 Future recommendations

The main recommendations for future $PPM_{2.5}$ air pollution integrated assessment studies are as follows:

- This thesis focused on $PPM_{2.5}$ air pollution. The most important primary and secondary $PM_{2.5}$ emission source categories and greenhouse gas emission source categories are same and therefore combined assessment of these air pollutants is required in order to create effective air pollution mitigation policies.
- Different $PPM_{2.5}$ emission source categories have different emission-exposure and exposure-response relationships. Therefore the exposure and health effects of these sectors should be assessed separately. This is especially important in studies which are using optimizing methods to estimate economically feasible mitigation strategies
- The resolution of the dispersion model seems to have a significant impact on exposure estimates for those emission sources that have low emission height and high emissions strengths near to population hotspots. More research is required to study optimal resolution and methods to fully capture the exposure to these emissions sources
- The main uncertainties in the integrated assessment were related to exposure-response functions and to toxicity differences between different PM sources. However, the current epidemiological and toxicological literature is of limited use in assessing toxicity differences between various emission sources. An integrated assessment needs to take even greater account of the toxicity differences, for example based on different hypotheses, and test how these hypotheses will affect the results obtained with the models.
- The identification of model and parameter uncertainties and propagating them through integrated assessment models with the Monte-Carlo simulation provide practical tools for the modeller to utilize in guiding the assessment process. An early assessment of uncertainties makes it possible to focus on those uncertainties which have a major impact on results obtained with the models.

9.10 References

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