

Molecular and Integrative Toxicology

Jozef M. Pacyna  
Elisabeth G. Pacyna *Editors*

# Environmental Determinants of Human Health

 Humana Press

# Molecular and Integrative Toxicology

Series Editor

Rodney R. Dietert

Department Microbiology and Immunology

Cornell University College of Veterinary Medicine

Ithaca, NY, USA

More information about this series at <http://www.springer.com/series/8792>

Jozef M. Pacyna • Elisabeth G. Pacyna  
Editors

# Environmental Determinants of Human Health

 Springer

*Editors*

Jozef M. Pacyna  
Norwegian Institute for Air Research  
Kjeller, Norway

Elisabeth G. Pacyna  
Norwegian Institute for Air Research  
Kjeller, Norway

ISSN 2168-4219

ISSN 2168-4235 (electronic)

Molecular and Integrative Toxicology

ISBN 978-3-319-43140-6

ISBN 978-3-319-43142-0 (eBook)

DOI 10.1007/978-3-319-43142-0

Library of Congress Control Number: 2016954421

© Springer International Publishing Switzerland 2016

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made.

Printed on acid-free paper

This Springer imprint is published by Springer Nature

The registered company is Springer International Publishing AG

The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

# Preface

Polluted air and contaminated food and water are major causes of human health deterioration. Over the years, well-established relationships between human health effects and environmental pollution (focusing on air pollutants and chemicals) have formed. A population can be exposed to environmental pollution throughout their whole life span, which may increase during periods of particular vulnerability.

The relationship between health deterioration and environmental contamination has been studied for several decades. “Losses” to human health have been estimated in economic terms, as well as in terms of years of life lost. In addition, benefits due to the reduction of human exposure to pollution have also been assessed. In this context, various assessment methodologies have been developed and applied to quantify dose–response relationships for many pollutants. From these relationships, exposure research frameworks have been developed from: the identification of human exposure; the emissions derived from anthropogenic and natural sources; the development of policies and the assessment of cost of reduction of this exposure. This enables the evaluation of human health benefits from the reduction in the context of human welfare improvement. For this purpose, various models have been developed to evaluate the role of public health externalities on society.

During the last decade, several research programs and individual projects have been carried out on the aforementioned topics, including various EU Framework Programmes along the EU Environment and Health Action Plan to support the development of cost-effective policy measures against pollution-related diseases and their wider impacts. The purpose of this book is to summarize the research results from these programs, as well as projects carried out outside the EU region. The knowledge regarding environmentally induced human health problems is presented in a coherent and easy to understand way for the wider community. At present, an important task is to properly communicate the great risk for human health deterioration due to increasing environmental pollution on a local, regional, and even global scale. The book describes an ideal potential, the enhanced participation of the general public in current and future studies of environmental determinants of human health.

The scope of the book is to provide the reader with an assessment of emission sources and releases of various pollutants to the main environmental media and a discussion of exposure pathways and health end points for these pollutants. Dose–response relationships for selected pollutants (exposure–early effects relationship) are then assessed. Technological and non-technological solutions to reduce human exposure to pollutants are discussed, including the assessment of monetary and non-monetary benefits from exposure reduction. Macroeconomic impacts of human health deterioration are presented. Finally, risk communication and awareness are described, including public participatory approaches.

The book provides information about various pollutants, both inorganic and organic, in the context of their human health impacts. Methodological approaches are reviewed, such as various types of environmental models, life cycle assessment analysis, and human health scenarios in relation to environmental change at various scales. Challenges on how to translate contaminant concentrations measured in blood into the information useful for risk characterization are discussed. Monetary and nonmonetary approaches are presented for assessing the human health benefits in the context of human welfare improvement. The monitoring of environmental pollution in the context of human health is also discussed. Climate change and a future change in pollutant exposure will further complicate the assessment of human health deterioration due to the contamination of the environment, providing a good rationale to continue this monitoring, as concluded in the book. To this end, the transition to a low-carbon economy (and society) and its impacts on the health of the environment and humans are also discussed. A trend analysis of funding for environment and human health research may be of particular interest to regulatory authorities and policy makers.

Major features of the book include an assessment of environmental parameters (such as air quality, environmental emissions, etc.) in the context of human health deterioration and an improvement of knowledge on public health status as a component of human welfare. A presentation of solutions for a reduction of human exposure to environmental pollution is given in the context of environmental policy improvement. A presentation of the role of the public in monitoring the human exposure change due to environmental pollution is assessed in the context of risk communication. Hazard and risk assessment is approached in the context of consumer safety.

As the subject of health effects due to environmental pollution is extremely broad and multidimensional, it is not feasible in the frame of this book to provide a comprehensive picture of this burgeoning field of research, policy, and public health action. Therefore, where feasible, references are made to recent reviews of evidence, outcome documents of relevant professional conferences, and statements from expert communities, as well as major reports from the organizations and institutions involved in public policy on environment and population health.

This book is a crucial text for policy makers requiring scientific justification for the development of new environmental regulations and exposure reduction strategies;

scientists researching public health and environmental contamination; and members of the public interested in human health issues.

The editors thank all of the contributing authors contributing to this volume and are grateful to Norwegian Institute for Air Research (NILU) for the financial support during the preparation of this work.

Kjeller, Norway

Jozef M. Pacyna  
Elisabeth G. Pacyna

# Contents

<b>1 Sources and Fluxes of Harmful Metals</b> .....	1
Jozef M. Pacyna, Kyrre Sundseth, and Elisabeth G. Pacyna	
<b>2 Exposure to Environmental Hazards and Effects on Chronic Disease</b> .....	27
Miranda Loh	
<b>3 Human Exposure to Pollutants and Their Health Endpoints: The Arctic Perspective</b> .....	51
Jon Øyvind Odland and Shawn Donaldson	
<b>4 Effects of Pollutant Exposure on Human Health as Studied with Selected EU Projects</b> .....	83
Arja Rautio	
<b>5 Organic Metal Species as Risk Factor for Neurological Diseases</b> .....	97
Sören Meyer, Till Weber, Robert Haferkorn-Starke, Tanja Schwerdtle, and Julia Bornhorst	
<b>6 Integrated Assessment of Policies for Reducing Health Impacts Caused by Air Pollution</b> .....	117
Rainer Friedrich	
<b>7 Monetary and Non-Monetary Measures of Health Benefits from Exposure Reduction</b> .....	133
Milan Ščasný and Vojtěch Máca	
<b>8 Capturing the Health Effects of Environmental and Industrial Policy in a Macroeconomic Model</b> .....	151
Hector Pollitt and Ben Gardiner	
<b>9 Transition to a Low Carbon Economy; Impacts to Health and the Environment</b> .....	169
Rebecca J. Thorne	



**10 Unmasking Environmental Health Zorros: The Need for Involvement of Real Risk Communication Experts for Two-Way and Problem-Solving Communication Approaches ..... 203**  
Hans Keune, Peter Van Den Hazel, and Frederic Boudier

**11 Citizen Participation Approaches in Environmental Health ..... 225**  
Sonja Grossberndt and Hai-Ying Liu

**12 Environment and Health Research Funded by the European Union (EU) Research Framework Programmes: Increasing Scientific Knowledge and Building a Solid Evidence-Base for Policy Making ..... 249**  
Tuomo K. Karjalainen

**Index..... 265**

# Contributors

**Julia Bornhorst** Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam, Nuthetal, Germany

**Frederic Boudier** Department of Technology and Society Studies, Maastricht University, Maastricht, Netherlands

**Shawn Donaldson** Population Studies Division, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, ON, Canada

**Rainer Friedrich** University of Stuttgart, Stuttgart, Germany

**Ben Gardiner** Cambridge Econometrics, Covent Garden, Cambridge, UK

**Sonja Grossberndt** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Robert Haferkorn-Starke** Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam, Nuthetal, Germany

**Tuomo K. Karjalainen** DG Research and Innovation, Health Directorate, European Commission, Brussels, Belgium

**Hans Keune** Research Institute for Nature and Forest, Brussels, Belgium

Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium

**Hai-Ying Liu** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Miranda Loh** The Institute of Occupational Medicine, Research Avenue North, Riccarton, Edinburgh, UK

**Vojtěch Máca** Environment Center, Charles University, Prague, Czech Republic

**Sören Meyer** Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam, Nuthetal, Germany

**Jon Øyvind Odland** Department of Community Medicine, Faculty of Health Sciences, The Arctic University of Norway, Tromsø, Norway

**Elisabeth G. Pacyna** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Jozef M. Pacyna** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Hector Pollitt** Cambridge Econometrics, Covent Garden, Cambridge, UK

**Arja Rautio** Centre for Arctic Medicine, Thule Institute, University of Oulu, Oulu, Finland

**Milan Ščasný** Environment Center, Charles University, Prague, Czech Republic

**Tanja Schwerdtle** Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam, Nuthetal, Germany

**Kyrre Sundseth** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Rebecca J. Thorne** Department of Environmental Impacts and Economics (IMPEC), NILU – Norwegian Institute for Air Research, Kjeller, Norway

**Peter Van Den Hazel** Public Health Services Gelderland-Midden, Arnhem, The Netherlands

International Network for Children's Health, Environment and Safety (INCHES), Dieren, The Netherlands

Health and Environment Alliance (HEAL), Brussels, Belgium

**Till Weber** Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam, Nuthetal, Germany

## About the Editors

**Jozef M. Pacyna** is a research director at the Norwegian Institute for Air Research (Kjeller, Norway) and is also professor of chemistry at the Gdansk University of Technology (Gdansk, Poland). He has been a visiting professor at the University of Michigan, Chalmers University of Technology (Gothenburg, Sweden), and Yale University.

Prof. Pacyna's expertise is on biogeochemical cycling and fluxes of mercury, other heavy metals, persistent organic pollutants, and radionuclides in the environment. Another field of his expertise is the assessment of environmental and socio-economic impacts of energy production and other anthropogenic sources on climate change and human welfare. He has carried out more than 30 EU projects within these topics.

Prof. Pacyna is the author of more than 450 scientific publications, including more than 120 papers in peer-reviewed journals and more than 30 books and book chapters. His works have been cited more than 7500 times, bringing his citation index H to 40. In his career, he has been awarded the Life Achievement Award, the Bene Merito honorable decoration by the minister of foreign affairs in Poland, and the "White Tiger" award, which is the highest award in the Polish energy sector for outstanding achievements in energy and environment research.

**Elisabeth G. Pacyna** is a senior scientist at the Norwegian Institute for Air Research (Kjeller, Norway). She received her M.Sc. in industrial economics and Ph.D. in environmental engineering.

Dr. Pacyna's scientific experience is in the field of energy, resources, and environmental interactions. Her research focuses mainly on the development and application of tools for the assessment of socioeconomic impacts of energy production on climate change and human welfare. These tools include life cycle assessment (LCA) analysis and various monetary valuation methods used to value environmental benefits from the implementation of various environmental policies. She has carried out 16 EU projects within these topics. Dr. Pacyna has also participated in several projects for the UNEP Chemicals and Nordic Council of Ministers on

socioeconomic and ecological impacts of energy production and other anthropogenic sources on the contamination of the environment. She has also participated in projects on development of new measures for reduction of greenhouse gas emissions from coal-fired power plants and improvement of coal combustion efficiency in power plants.

Dr. Pacyna is an author/co-author of 4 books and book chapters, 20 articles in peer-reviewed scientific journals, more than 60 presentations at international conferences, and several technical reports. Her works have been cited more than 2000 times, bringing her citation index H to 13.

# Chapter 1

## Sources and Fluxes of Harmful Metals

Jozef M. Pacyna, Kyrre Sundseth, and Elisabeth G. Pacyna

**Abstract** Metals are important chemicals occurring in various ecosystems. Some of them are toxic, some are toxic when appearing in excess and some are essential for the environment and human health. They are ubiquitous constituents of various natural materials in the lithosphere, the hydrosphere and the biosphere.

High-temperature processes in the primary non-ferrous metal industries are the major source of atmospheric arsenic (As), cadmium (Cd), copper (Cu), indium (In), antimony (Sb), and zinc (Zn), and an important source of lead (Pb) and selenium (Se). Combustion of coal in electric power plants and industrial, commercial, and residential burners is the major source of anthropogenic mercury (Hg), molybdenum (Mo), and selenium and a significant source of arsenic, chromium (Cr), manganese (Mn), antimony, and thallium (Tl). Combustion of oil for the same purpose is the most important source of vanadium (V) and nickel (Ni). Combustion of leaded gasoline is estimated to be the major source of lead. Atmospheric chromium and manganese derive primarily from the iron and steel industry. The largest discharges of these metals are estimated for soils followed by discharges to water.

The degree of human alteration of metals biogeochemical cycles caused by their anthropogenic emissions affects human health impacts of these chemicals. The information on alteration degree on global and regional scale is discussed.

Emissions of metals from various anthropogenic sources can be reduced using technological (e.g. Best Available Technology (BAT) solutions) and non-technological measures (e.g. Best Environment Practice (BEP) option). Various emission reduction measures are presented.

**Keywords** Emission source • Emission inventory • Biogeochemical cycling • Emission control • BAT • BEP

---

J.M. Pacyna (✉) • K. Sundseth • E.G. Pacyna  
Department of Environmental Impacts and Economics (IMPEC),  
NILU–Norwegian Institute for Air Research, PO Box 100, 2027 Kjeller, Norway  
e-mail: [jp@nilu.no](mailto:jp@nilu.no)

© Springer International Publishing Switzerland 2016  
J.M. Pacyna, E.G. Pacyna (eds.), *Environmental Determinants of Human Health*,  
Molecular and Integrative Toxicology, DOI 10.1007/978-3-319-43142-0\_1

## 1.1 Metals as Harmful Pollutants

During the last few decades human society has dramatically altered biogeochemical cycles of various chemicals. Increasing human population requires larger amounts of energy, other industrial goods and food. Provision of these goods has generated increasing amounts of various pollutants emitted to the atmosphere, as well as aquatic and terrestrial ecosystems (e.g. Rauch and Pacyna 2009; Pacyna et al. 2010a; UNEP 2013a, b). Metals are among the most important pollutants emitted to the environment. On one side, metals are an important category of resources for various uses and technologies. For many applications metals are critical resources that enable modern technologies, e.g. the application of Rare Earth Elements (e.g. Pacyna et al. 2015a).

On the other side, many metals have an adverse impact on the environment and human health (e.g. UNEP 2013a). A number of studies have been carried out to assess the impact of metals in terms of: (1) carcinogenesis and genotoxicity, (2) neurotoxicology, (3) renal toxicology, and (4) reproductive and developmental toxicology. In addition, research has been carried out on bioresponses and reactivities in metal toxicity, immunomodulation by metals, and effects of metals on other organ systems. Information on clinical aspects of metals toxicity, as well as on environmental and human risk assessment has been reported (e.g. an overview in Chang 1996). Metals in the environment have been regarded as a risk factor for infectious diseases (e.g. Ackland et al. 2015).

Knowledge about public health concern associated with metals has brought a question of sources and fluxes of these pollutants. In this context various criteria have been examined including the following issues:

- Has the biogeochemical cycle of a given metal been substantially altered by human activities, and on what scale?
- What are the critical pathways by which the most toxic species of a metal can reach the organ in man which is the most sensitive to its effect?, and
- What is the degree of public health concern associated with the metal?

The above mentioned issues were first discussed by an inter-disciplinary group of scientists at the beginning of the 1980s (Andrea et al. 1984). Since then a large body of information has been collected and discussed, including the latest analysis presented in Ackland et al. (2015). As a result of these discussions, information on selected metals as hazardous pollutants in the context of human health has been prepared and presented in Table 1.1. Only some metals were selected, including those with documented health effects and information on anthropogenic origin, transport and migration through different environmental media, and exposure pathways. A major review of this information has been carried out by UNEP (2013a).

The information in Table 1.1 includes data on scale of perturbations of biogeochemical cycles on global, regional and local scale, exposure pathways, and health concern. Metals enter the environment through various natural and geological processes, as well as human activities. After being emitted to the atmosphere, metals

**Table 1.1** Information on perturbations of geochemical cycles, exposure pathways and health concern for selected metals

Metal	Scale of perturbation			Exposure pathway	Health concern
	Global	Regional	Local		
Mercury (Hg)	+	+	+	A, F, W	+ <sup>a</sup>
Lead (Pb)	+	+	+	A, F, W, M, T	+
Arsenic (As)	–	+	+	A, F, W	+ <sup>b</sup>
Cadmium (Cd)	–	+	+	A, F, W, T	+
Zinc (Zn)	–	+	+	A, F, W	E
Copper (Cu)	–	+	+	A, F	E
Selenium (Se)	+	+	+	A, F	E
Antimony (Sb)	–	+	+	A, F	+
Tin (Sn)	+	+	+	A, F, W	+ <sup>a</sup>
Chromium (Cr)	–	+, c	+	A, F, W	+ <sup>c</sup>
Manganese (Mn)	–	+, c	+	A, F, W	E
Nickel (Ni)	–	+	+	A, F, W	E
Vanadium (V)	–	+, c	+	A, F	+
Molybdenum (Mo)	–	+	+	A, F	E

A air, F food, W drinking water, T tobacco, smoking, M hand to mouth pathway

Regarding perturbations of geochemical cycles: + means significant perturbations, – means no perturbations, c means enhanced due to mobilization of crustal materials

Regarding health concern: + means toxic in excess, E means essential

<sup>a</sup>Toxic but organometallic forms only

<sup>b</sup>Trivalent forms toxic, pentavalent forms essential

<sup>c</sup>Hexavalent form toxic, trivalent form essential

can be transported with air masses and water currents at various distances before being deposited to the aquatic and terrestrial surfaces. Most of the metals are transported within the air masses on particles or with sediments in water. Some metals, however, such as mercury (Hg) and to some extent selenium (Se), can be transported in gaseous phase, resulting in their long range transport within air masses. The geographical scale defined in Table 1.1 as “global” means that metals can be transported at very long distances, causing metal perturbations of biogeochemical cycles on a continental scale, e.g. in large parts of the Northern Hemisphere. “Regional” effects refer to a scale of 100–1000 km, while “local” effects cover the range of less than 100 km. Significant health effects of metals are usually caused by very strong perturbations at a local scale. All metals presented in Table 1.1 do have their biogeochemical cycles altered by various drivers of environmental change on regional and local scale. Mercury, lead (Pb), selenium and tin (Sn) can be regarded as global pollutants in the context of alteration of their biogeochemical cycles.

It should also be noted, that some processes such as land use change and agriculture may mobilize very large amounts of soil material as atmospheric dust and suspended material in aquatic ecosystems. Some metals, such as chromium (Cr), manganese (Mn) and vanadium (V) can be mobilized by these processes and transported on at least regional scale. This has been indicated in Table 1.1.



Most of metals enter the human body with contaminated food and drinking water or through inhalation of polluted air. Many metals are essential for life but their intake has an optimum below that deficiency symptoms occur, and on the other side, adverse impacts of too high intakes can be detected. Impacts on human health occur mostly at the local scale, however, for some metals, such as mercury, these affects are detected also on regional and global scale. Another pathway is also defined for lead, namely hand - to - mouth pathway. This is the case mainly for children. Inhalation of lead and cadmium (Cd) through tobacco smoking is also indicated in Table 1.1.

A group of four metals: mercury, lead, arsenic (As) and cadmium has been separated in Table 1.1. These metals are considered as the most important in the context of documented effects on human health, and anthropogenic perturbations of their biogeochemical cycles. Historically, lead was the most often studied metal due to proven evidence of human health impacts and large amounts of anthropogenic emissions of this pollutant (e.g. UNEP 2013a). As a result, a number of policies were introduced to reduce anthropogenic emissions of lead, including phase out of leaded additives to gasoline (e.g. van Storch et al. 2002, 2003). Recently, mercury has become the most studied metal in the context of its adverse impacts in environment and human health, persistent character and transport with air masses on global scale (Pacyna et al. 2010a; UNEP 2013b; Sundseth et al. 2015). In 2013, the Minamata Convention has been agreed to reduce emissions and human exposure to mercury on a global scale ([www.mercuryconvention.org](http://www.mercuryconvention.org)).

## 1.2 Emissions of Metals

Metals listed in Table 1.1 can be emitted from natural and anthropogenic sources.

### 1.2.1 *Natural Sources of Metals*

Metals are ubiquitous constituents of various natural materials in lithosphere, hydrosphere and biosphere. Global mean metal concentrations of metals in bulk continental, upper continental and ocean crusts, as well as in continental and oceanic sediments, loess and soil are presented in Rauch and Pacyna (2009). These concentrations are rather low for most metals. In some places metals are enriched in geological formations, such as ore deposits or rock types, including naturally anomalous abundances of metals. Metals can be liberated from rocks and soil through weathering when the lattice structures of rocks, to which metals are tightly bound, break and the metal ions are emitted. In soils, the soil forming processes are the major factors to move tightly in mineral lattices bound metal ions into soil water solution (UNEP 2013a). Windblown dust is considered as the most important natural source of metals (e.g., Pacyna 1986; Rauch and Pacyna 2009; UNEP 2013a).

Agricultural and residential patterns are changing the rates of continental weathering and erosion. The input of hydrogen ions from precipitation changes the chemical characteristics of the weathering process. The acidification of soils enhances the rate of podsolization and consequently the release of many metals which are otherwise either rather immobile or locked into tightly closed loops within the ecosystems.

Volcano explosive eruptions and quiet degassing are also important natural sources of several metals in gaseous and particulate forms, including mercury, cadmium and arsenic. Emission rates for various metals vary substantially from one region to another. Major historic eruptions have had both regional and global effects in increasing deposition of metals from the atmosphere for periods of up to several years, e.g. eruptions in Tambora in 1815 and Krakatou in 1883. Emissions of pollutants from volcanoes has been studied quite widely, particularly emission of mercury (Hinkley 2003; Rytuba 2005).

In certain parts of the world forest fires are very important sources of metals and an intense turbulence of the atmosphere in desert regions can result in their very high concentrations in soil dust and air. Biomass burning is not a phenomenon restricted to the tropics as initially been thought. Large fires have become a common feature of the world's boreal forest. The type and quantity of gaseous and particulate emissions resulting from burning are a strong function of both the ecosystem and the phase of burning, namely flaming versus smoldering (e.g. Levine 1991). The bulk of biomass burning is human indicated and appears to be increasing with time. Gaseous and particulate emissions resulting from biomass burning may have increased by as much as 50% since 1850, as indicated on the basis of satellite data.

Other possible natural processes which can be important for some metals include biological mobilization (including methylation) and airborne sea-salt. Two sea-salt production processes have been considered as natural sources of metals: (1) "bubble bursting" (cadmium, copper, nickel (Ni), lead, and zinc (Zn)) and (2) sea-air gas exchange (arsenic and mercury). Emissions of metals from bubble bursting are often estimates on the basis of metal concentrations in surface ocean waters and the enrichment of metals in the atmospheric sea-salt particles. The potential significance of arsenic and mercury vapour emissions is usually assessed from ambient measurements of arsenic and mercury in marine aerosols and gaseous samples.

### ***1.2.2 Anthropogenic Sources of Metals***

Metals are ubiquitous in various raw materials as impurities, such as fossil fuels and metal ores, as well as in industrial products. Some of them evaporate entirely or partially from raw materials during the high-temperature production of industrial goods, combustion of fuels, and incineration of municipal and industrial wastes, entering the ambient air with exhaust gases. Releases to other environmental compartments (e.g. spills to water bodies, landfills, sewage lagoons, holding ponds) may also result in volatilization and entrainment of several trace metals.

### 1.2.2.1 Atmospheric Emissions

Major anthropogenic sources of metals to the atmosphere can be grouped as follows:

- stationary fuel combustion for production of electricity and heat,
- internal combustion engines (lead emissions from gasoline combustion),
- nonferrous metal manufacturing,
- iron, steel and ferroalloy plants and foundries,
- cement production, waste incineration, and
- various uses of metals.

There are four major groups of parameters affecting the amount of metal emissions from these sources:

- concentration of metals as impurities in raw materials, such as coal, crude oil, and various ores,
- physical and chemical properties of metals affecting their behaviour during the production process,
- the technology employed in industrial processes, and
- the type and efficiency of emission control equipment.

Concentrations of metals in raw materials are associated with the affinity of metals in pure coal, mineral fraction of raw material, etc. For example, there are more than 60 chemicals in coal that shall be considered as coal impurities, including the metals in Table 1.1. Some of them are associated with organic fraction of coal, some with inorganic fraction and some with both fractions. The affinity of metals for pure coal or mineral matter would affect metal emissions during coal combustion by being responsible for a chemical form of a given metal emitted and metal association with various types of coals, such as, bituminous, subbituminous coals or lignites. Particularly, the chemical form of emitted metal is important, making impact on transport length with air masses (solubility of metals), chemical behaviour during this transport, as well as methylation processes. It should be noted that methylated form of metals decide on toxicity of many of them, thus on adverse impacts on human health. The best example is mercury. Mercury is not emitted as toxic element but when it undergoes methylation after emission to the atmosphere and deposition to aquatic and terrestrial ecosystems, very toxic methylmercury forms are formed.

The fate of metals during coal combustion or other high temperature industrial processes with non-ferrous and ferrous metal production depends not only on the affinity, concentration and distribution of each metal within the coal or ore matrix but also on industrial process conditions, such as temperature, heating rate, exposure time and the surrounding environment of either oxidizing or reducing conditions. The volatile species in the coal or ores are evaporated in the boiler or roaster and then re-condensed as submicron fine particles when the flue gas cools in the convective sections of industrial installations. This is the reason that concentrations of chalcophiled lead, antimony (Sb), cadmium, selenium, arsenic, zinc and molyb-

denum (Mo) increase markedly with decreasing particle size. About 90 % of mercury is emitted in gaseous forms under such conditions. The physical form of metal emissions is then of great importance when discussing the efficiency of metal emission control, their transport within air masses and atmospheric deposition, and metal exposure route characteristics such as, inhalation rate and the metal uptake and migration within the food chain.

The type of fly ash control system and its efficiency also influence metal emissions to the atmosphere. From among several types of control devices, electrostatic precipitators (ESPs) and wet scrubbers are mainly installed in electric power plants, major smelters and cement kilns. Average control efficiency for fine particles is higher than 99.9 %. Generally, a venture wet scrubber system is more efficient than ESP system for removing arsenic, cadmium, manganese, nickel, lead, and zinc. Flue gas desulphurization (FGD) installations are also efficient control systems, not only to remove sulphur compounds but also gaseous portion of metals, such as mercury and selenium.

The above mentioned parameters affecting the amount of metal emission to the atmosphere are widely discussed in Pacyna and Pacyna (2001) and Pacyna et al. (2010b).

### Aquatic Emissions

The major sources of metal pollution in aquatic ecosystems, including the ocean, are:

- domestic wastewater effluents (especially for arsenic, chromium, copper, manganese, and nickel),
- coal-burning power plants (arsenic, mercury, and selenium in particular),
- non-ferrous metal smelters employing the hydrometallurgical process (mercury, cadmium, nickel, lead and selenium),
- iron and steel plants (chromium, molybdenum, antimony, and zinc), and
- the damping of sewage sludge (arsenic, manganese and lead in particular).

The atmosphere is the major route of lead entry in natural waters. This source accounts also for 40 % V loading and substantial loadings of other metals. There are also emissions of metals from products in use. They refer to corrosion from metal surfaces exposed to the environment: roofs, fences, gutters, etc. The use of metals in paint, sprays and pesticides also leads to emissions to the environment. They are often considered as dissipative applications, where the use of a product equals the emission to the environment.

### Discharges into Soils

Soils are receiving large quantities of metals from a wide range of industrial wastes. However, the two principal sources of metals in soils are:

- the disposal of ash residues from coal combustions, and
- the general wastage of commercial products on land.

Urban refuse represents an important source of copper, mercury, lead and zinc with significant contributions of cadmium, lead and vanadium also coming through the atmospheric deposition. Chemical composition of urban refuse is the main factor affecting the amount of discharge of metals into soils. The large volume of wastes associated with animal husbandry, logging, as well as agricultural and food production form also an important contribution to the metal budget of many soils.

Municipal sewage sludge may not be a particularly important source of metals on a global scale. However, municipal sewage represents one of the most important sources of metal contamination in soils on a local scale.

### 1.2.2.2 Global Emission of Metals to the Environment

A first assessment of metal emissions from natural sources on a global scale has been presented by Nriagu (1989). The largest emissions were estimated for metals emitted during weathering of rocks and windblown dust, including copper, chromium, manganese, nickel, lead, selenium, vanadium and zinc.

The first quantitative worldwide estimate of the annual industrial input of metals into the air, soil, and water at the beginning of the 1980s was published by Nriagu and Pacyna (1988). Pyrometallurgical processes in the primary non-ferrous metal industries were found to be the major source of atmospheric arsenic, cadmium, copper, In, antimony, and zinc, and an important source of lead and selenium. Combustion of coal in electric power plants and industrial, commercial, and residential burners was the major source of anthropogenic mercury, molybdenum, and selenium and a significant source of arsenic, chromium, manganese, antimony, and thallium. Combustion of oil for the same purpose was the most important source of vanadium and nickel. Combustion of leaded gasoline was estimated to be the major source of lead. Atmospheric chromium and manganese were derived primarily from the iron and steel industry. The largest discharges of these metals were estimated for soils followed by discharges to water. The atmospheric emissions are the lowest for all metals assessed.

The updates of the above mentioned global emission inventory have been prepared by Pacyna and Pacyna (2001) with emission data calculated for the conditions at the beginning of the 1990s and very recently for this work. The outcome of these assessments is presented in Table 1.2. The data in table confirm the earlier conclusion that emissions of metals from anthropogenic sources to soil are the highest compared to anthropogenic emissions of these contaminants to the aquatic ecosystems and the atmosphere and to emissions from natural sources.

The assessment in Table 1.2 confirms an earlier conclusion by Nriagu and Pacyna (1988) that mankind has become the most important element in the global biogeochemical cycling of metals. The man-kind mobilization of metals into biosphere, estimated as the difference between the terrestrial and aquatic inputs minus atmospheric emissions comes to about 13 tonnes for mercury, 934 tonnes for lead, 123 tonnes for arsenic, 31 tonnes for cadmium, 1600 tonnes for zinc, 1066 tonnes for copper, 82 tonnes for selenium, 44 tonnes for antimony, 1038 tonnes for chromium,

**Table 1.2** Current global emissions of metals to the environment (in 10<sup>3</sup> tonnes/year)

Metal	Natural	Anthropogenic				Ratio atmospheric/ natural
		Atmospheric	Water	Soil	Total	
Hg	2.5	2.0	4.6	8.3	14.9	0.8
Pb	12.0	119.3	138.0	796.0	1053.3	10.0
As	12.0	5.0	41.0	82.0	128.0	0.4
Cd	1.3	3.0	9.4	22.0	34.4	2.3
Zn	45.8	57.0	226.0	1372.0	1655.0	1.2
Cu	28.0	25.9	112.0	955.0	1092.9	0.9
Se	9.3	4.6	41.0	41.0	86.6	0.5
Sb	2.4	1.6	18.0	26.0	45.6	0.7
Cr	44.0	14.7	142.0	896.0	1053.7	0.3
Mn	317.0	11.0	262.0	1670.0	1943.0	0.03
Ni	30.0	95.3	113.0	325.0	533.3	3.2
V	26.0	240.0	12.0	132.0	384.0	9.2
Mo	3.0	2.6	11.0	88.0	101.6	0.9

1932 tonnes for manganese, 438 tonnes for nickel, 144 tonnes for vanadium and 99 tonnes for molybdenum. This man-kind mobilization of metals is much higher than their emission from natural sources.

The largest anthropogenic emissions of metals were estimated in Asia. This can be explained by growing demands for energy in the region and increasing industrial production. As a result, the Asian emissions are not only larger than the emissions on other continents, but also showing an increasing trend. Another factor contributing to high emissions in Asia is the efficiency of emission control which is lower than in Europe and North America. Concerning the two latter continents, emissions of metals show a decreasing tendency over the last two decades.

The annual total toxicity of all the metals mobilized exceeds the combined total toxicity of all the radioactive and organic wastes generated each year, as measured by the quantity of water needed to dilute such wastes to drinking water standard. The circulation of toxic metals through the soil, water and air and their inevitable transfer to the human food chain has become a very serious health risk. Better understanding of metal cycling is clearly needed to quantify this risk.

### 1.3 Biogeochemical Cycling of Metals on Global Scale

Metals undergo a natural biogeochemical cycling in the Earth system. This cycling starts with the processes within the Earth's crust and on its surface. On a global scale the earth's inner magma, containing various metals is brought to the surface as rocks. Weathering of these rocks releases the metals into the surface environment. Then the metals undergo cycling within and between various environmental

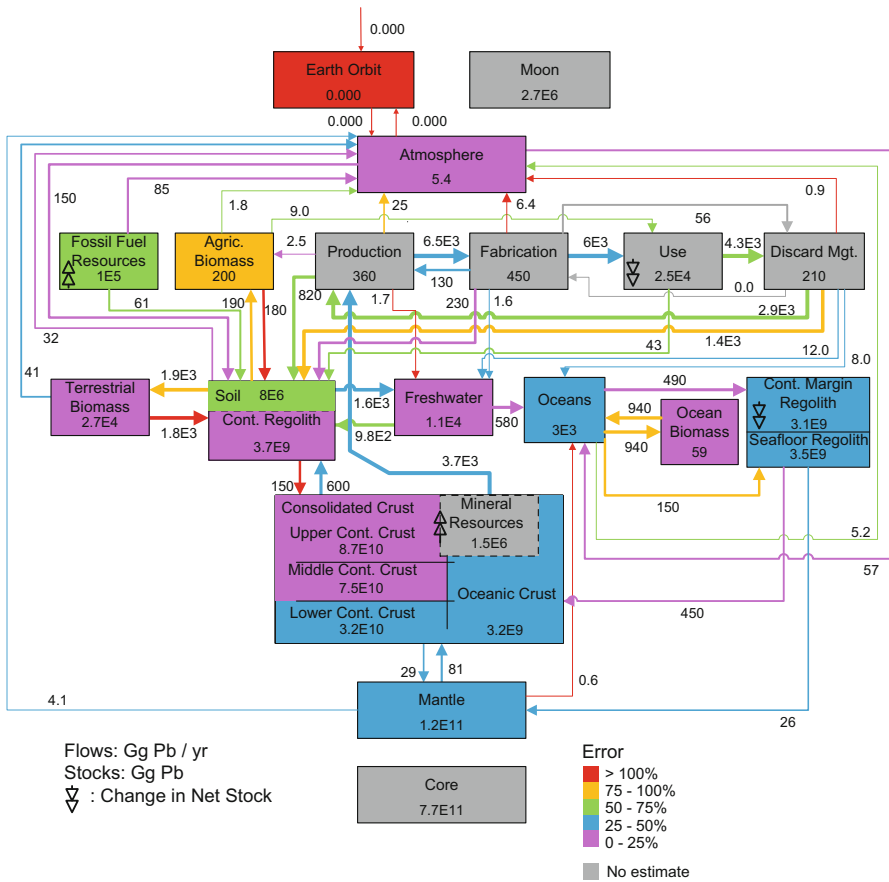
compartments, such as the ocean, the atmosphere, land and groundwater. Metals are deposited during this cycling and buried again, mostly in sediments. Subsidence of these sediments to the Earth's inner sphere completes the cycle. This geological part of the biogeochemical cycling of metals has been described by various authors, including De Vos and Tarvainen (2006) and in the UNEP (2013a) report.

Metals can be mobilized as a result of various chemical, mechanical and biological processes occurring in the Earth's systems. These processes cause metals to be dissolved from rocks exposed to acid rainwater. Metals can be liberated during the mechanically breaking rocks, e.g. by plants. Another natural way of mobilizing metals to the atmosphere is through volcanic activities.

Upon being mobilized, metals can undergo chemical processes, such as oxidation and reduction, complexation and sorption. These processes have a direct impact on fate and biological significance of metals. Understanding of metal speciation is extremely important for assessing the metal impact on biotic ecosystems. Metals adsorbed or complexed are generally not available for biological uptake. However, oxidation (e.g. for sixvalent chromium) and methylation (e.g. for mercury) do have great influence on bioavailability of these metals. Changing the redox state of metals, e.g. by various organisms, leads to changing the speciation of metals, thus their biological uptake.

Discharges of metals to the atmosphere and aquatic and terrestrial ecosystems during various anthropogenic processes described above change the natural biogeochemical cycling of metals. Though metals serve as "technological nutrients", our understanding of perturbations of natural biogeochemical cycles of these chemicals is much less understood than the anthropogenic impacts in biogeochemical cycling of "grand nutrients" (Rauch and Pacyna 2009). This is particularly true for assessment of human made perturbations of metal biogeochemical cycles on a global scale. Most of the studies do address only stocks and flows of metals between the environmental compartments. The first assessment of Earth's global biogeochemical cycles of metals, such as Ag, chromium, copper, nickel, lead and zinc has been prepared by Rauch and Pacyna (2009). This assessment provided an understanding of the comparable magnitudes of metals appropriated by humans and by the natural environment. An assessment tool was developed to quantify the changes to the environment caused by any change of anthropogenic drivers, such as industry production, population growth, or increased demand for energy and food production.

An example of biogeochemical cycling of lead is presented in Fig. 1.1 after Rauch and Pacyna (2009). In the first part of the assessment, natural reservoirs and metal stocks have been quantified using an information on the mass totals of the subdivided continental crust and oceanic crust and lead concentrations in this crust. Global living biomass has been disaggregated between terrestrial phytomass and marine phytomass, assuming that 80% of the world's biomass is constituted by trees. The Pb concentrations were estimated for foliage, twigs and branches, bark, wood and roots, separately. Average Pb concentrations in marine organisms were applied separately. Details on the above mentioned assessment are available in Rauch and Pacyna (2009) together with the information on anthropogenic reservoirs and flows of lead.



**Fig. 1.1** Earth’s global cycle of lead, anthropogenic data are for year 2000. The colors indicate the percent uncertainty (from Rauch and Pacyna 2009, reprinted with permission from John Wiley and Sons)

The results of lead cycling in Fig. 1.1 show the amounts of tonnes of the metal in various reservoirs and flows in the year 2000, coloured to reflect the estimated uncertainty. These results indicate that despite successful reduction in hydrologic and atmospheric emissions, society’s increasing consumption of metals has increased both in-use stocks and waste stream discards of lead. It was pointed out that the process of translocating metal from below ground to human built infrastructure has seen an overall reduction over the past few decades in the wasteful and potentially harmful dissipative emissions to environmental media. However, the continued increase in mass movement from ore to in-use stocks has repercussions for the future of environmental emissions. Though dissipative emissions from in-use stocks have traditionally been minor relative to upstream emissions, the chances of increased emissions from in-use stocks is greater with the increasing size of this reservoir. This will have consequences for the future sources of lead



resources, as more metal will likely be sourced from the discards of in-use stocks through recycling. This recovery will not necessarily reduce the burden on raw ore extraction, for most raw ores remain highly concentrated relative to in-use stocks, and ore is required to provide for increasing levels of consumption and compensate for dissipative losses.

Overall results of analysis carried out by Rauch and Pacyna (2009) indicate that human action has become a significant geomorphic agent in lead and other metal mobilization. Total natural and anthropogenic flows presently exist at approximately the same order of magnitude across most metals investigated. The largest total mass movements in the natural system (crustal production and subduction, sediment erosion and denudation, and ocean deposition) exceed the largest anthropogenic mass movements (mining extraction, metal production, and metal consumption). Copper is the only metal in which anthropogenic flows exceed natural flows by an order of magnitude or more. This order of magnitude equivalency compares with the grand nutrients, where the mobilization of C by human activity is less than an order of magnitude of natural flows, N and S are approximately the same, and P mobilization exceeds that of nature by half an order of magnitude (Falkowski et al. 2000). As concluded by Rauch and Pacyna (2009), such perturbation of Earth's natural global metal cycles is resulting in the reallocation of metal mass from surface sediments to continental margins and from ore bodies to in-use stocks.

## 1.4 Regional Scale Flows of Metals

Anthropogenic emissions alter substantially metal cycling on regional, e.g. continental scale. The degree of this alteration depends mainly on the amount of emissions to the atmosphere, water and land and their flows through these environmental compartments. Recently there were a few EU projects aiming at the improvement of metal emission estimates to the environment in Europe, including the SOCOPSE project on Source control of priority substances in Europe ([www.socopse.se](http://www.socopse.se)) and the ESPREME project on Estimation of willingness-to-pay to reduce risks of exposure to heavy metals and cost-benefit analysis for reducing heavy metal occurrence in Europe (<http://espreme.ier.uni-stuttgart.de>).

Emission inventories were used to study metal impacts on the environment in the form of Substance Flow Analysis (SFA). A stock-flow model approach, originally developed to simultaneously forecast resource demand (inputs) and waste generation (outputs), was applied for developing future projections on emissions, discharges and releases from metals used intentionally in products, whilst at the same time accounting for the metals accumulated in society (i.e. in the use phase). The basic model assumptions are driven by physical determinants, such as regulative changes or substitution effects which in turn, affect the demand of metals embodied in products. This in turn, induces a push of emissions or waste over a certain lifetime whilst metals stocks are accumulated and generated in the system. Exchange of the

metals between different environmental reservoirs (atmosphere, water and land) is based on a greatly simplified mass balance technique based on fixed exchange rates between the environmental compartments. An example of the SFA analysis for cadmium in Europe is presented below.

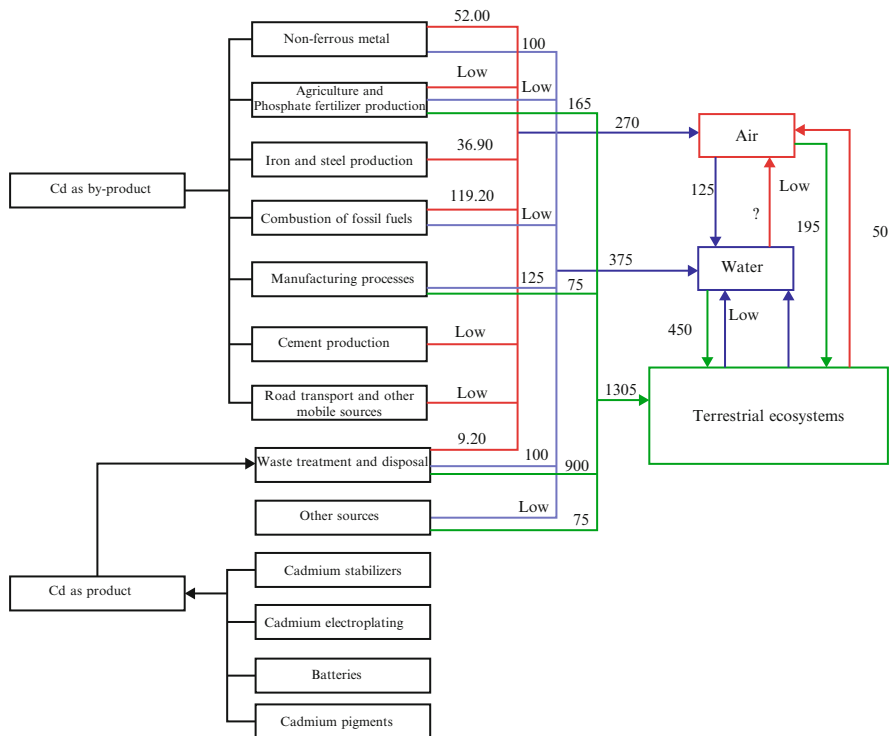
The emission sources for the SFA application for cadmium in Europe were identified as:

- By-product emission sources, including:
  - Phosphate fertilizer production
  - Non-ferrous metals
  - Iron and steel production
  - Combustion installations
  - Manufacturing processes
  - Cement production
  - Road transport and other mobile sources
  - Waste treatment and disposal, and
- Cadmium intentional uses, including:
  - Cadmium electroplating
  - Secondary battery manufacture
  - Cadmium stabilizers for plastics
  - Cadmium pigments in plastics

The most common industry source of cadmium is the by-product obtained when treating zinc, copper, lead and iron ores. The combustion of fossil fuels in combustion installations (especially coal and oil burning power plants) and waste treatment and disposal are also large sources for emitted cadmium. In addition there are emissions from phosphate fertilizer production and usage, cement productions, road transport and other mobile sources. Description of the above mentioned sources is presented in Pacyna (2009).

Material Flow Analysis for cadmium in Europe in 2000 in tonnes/year is presented in Fig. 1.2. The estimated emissions into the atmosphere are about 584 tons cadmium per year. Cadmium emissions from fuel combustion installations contribute alone to about 63 % to the total emissions. For fuel combustion, the main part of the emissions are emerging from oil boilers (26 % of total emissions to air) and coal boilers (17 % of total emissions to air). Large quantities are also related from cement production, non-ferrous metal industry and iron and steel production being responsible for 11, 9 and 9 % of the total emissions to air. Agriculture and road transport are assumed to be very low. Large parts of the atmospheric deposition are deposited into the aquatic and terrestrial surfaces in Europe. Atmospheric emissions with an average residence time of 7 days are deposited into the oceans (30 %) while about 70 % is deposited on land.

Cadmium emissions to the aquatic environment is for Europe estimated to be about 500 tons per year where the half of the discharge basically is caused by



**Fig. 1.2** MFA diagram for cadmium in Europe in 2000 (numbers in tonnes/year, unless indicated otherwise)

manufacturing processes (including metals, chemicals and petroleum products) and atmospheric deposition (25 % of the total emissions to water each). Both, primary non-ferrous metal production and domestic waste treatment plants are counting for about 20 % of the total emissions to water.

The compartment for soil and terrestrial ecosystems is the largest receiver of cadmium and it is estimated to receive about 1500 tons per year. The source of this emission is first of all occurring from waste treatment and disposal. The contribution of this source is estimated to be about 30 % and it is basically related to disposal of fly ash and bottom ash from power plants and waste incineration. Land filling of urban refuse is responsible for about 25 % and wastage of commercial products are responsible for about 5 % of the cadmium emissions to the terrestrial ecosystems in Europe. The atmospheric deposition to terrestrial ecosystems is responsible for about 13 %. Land filling of various foods and agriculture wastes counts for about 11 % of the total emissions received by the compartment for soil. Except food, the disposal of wastes from various manufacturing processes and wastage of commercial products on land both are responsible for about 5 % of the total. Municipal sewage sludge application is estimated to give low contributions.

### 1.5 Local Scale Flows of Metals

Emissions of metals may occur at different stages of a given installation or production technology. In order to assess the environmental and human health impact of emissions from a given technology or point source, a metal mass balance is needed. This assessment is aiming at:

- quantification of emissions from various emission generation points within a given installation or technology,
- selection of appropriate emission control device in order to reduce emissions at these points
- definition of best available practice in order to reduce/avoid emissions from these points.

An example of mercury balance in a 2000 MW coal-fired power plant is presented in Fig. 1.3 on the basis of a project on integrated environment assessment of new boilers employed at a 2000 MW coal-fired power plant, carried out by the authors of this Chapter recently. As much as 912 kg of mercury are being introduced to the boilers of this plant each year. Atmospheric emissions with a flue gases account for 85 kg, thus about 9.3 % of mercury entering the power plan. The largest amounts of mercury are estimated for the products after dry type of desulphurization (dry FGD) of flue gases from six existing blocks, accounting for 499 kg. Therefore, it is of utmost importance to determine an appropriate measure for dealing with these products or even using them, as they can be very much contaminated by mercury.

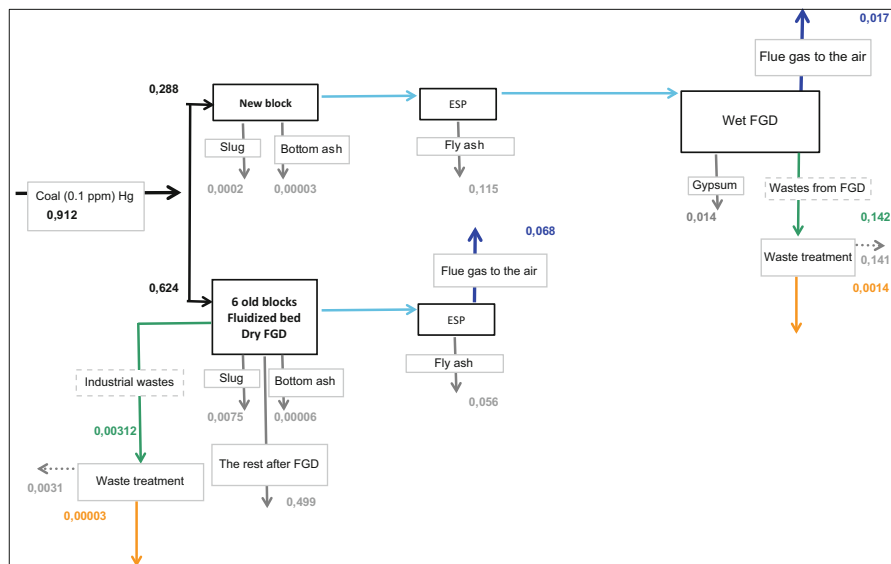


Fig. 1.3 Mercury balance in a 2000 MW coal-fired power plant (in tonnes/year)

Quite often the dry FGD products end as wastes and they are deposited on landfills, or used to fill up the space in open pit mines after coal exploitation. In this way mercury enters the environment and it can be transported with ground waters to enter the food chain. Mercury can be also re-emitted to the atmosphere, depending on the air temperature, humidity and other meteorological conditions.

Mercury in the stream leaving the wet type of FGD often enters gypsum. In this way mercury can be re-emitted to the atmosphere from building materials using gypsum. Eventually, mercury can be deposited on landfills with the used building materials based on gypsum.

Similar analysis on the basis of chemical mass balance for various installations, technologies, or point sources of emissions can be carried out for other metals. The main goal of such study is the assessment of human alterations of environmental cycles of metals on a local scale.

## **1.6 Measures to Reduce Anthropogenic Emissions of Metals**

Emissions of metals from various anthropogenic sources can be reduced using technological (e.g. Best Available Technology—BAT concept) and non-technological measures (e.g. Best Environment Practice—BEP concept).

### ***1.6.1 Technological Measures***

In general, metal emissions can be reduced through the application of the following methods (Pacyna and Ahmadzai 1997):

- pre-treatment methods, such as fuel substitution and fuel washing,
- primary reduction methods, when emission reduction occurs at the emission generating point, e.g. through the application of various combustion modification methods in electricity production boilers, and
- secondary methods, when emission reduction occurs in the flue gas.

Fuel substitution and fuel washing are the major pre-treatment measures for reduction of metal emissions. The following options of fuel substitution are often considered:

- switching from high- to low-ash and sulphur content coals burnt in coal-fired power plants, including direct switching from one kind of coal supply to another and blending various kinds of coal,
- increasing the use of natural gas, which has inherently lower content of metals than coal and oil, and
- increasing the use of alternative fuels and wider application of renewable sources to produce energy.

The decision to switch fuels is based on the following considerations: (1) availability and cost of new fuels, (2) the assessment of several engineering factors, such as fuel handling, coal preheating, internal boiler configurations, ash handling system, etc., and (3) the characteristics of the generating unit.

The cleaning of coal takes place in water, a dense medium or a dry medium. Physical cleaning processes are based on either the specific gravity or surface property differences between the fuel and its metal impurities. Chemical cleaning depends of chemical reactivity of metals as fuel impurities. An estimate reduction of metal content in various coals by fuel cleaning is between 5 and 10% (Pacyna and Ahmadzai 1997). The cleaning of crude oil occurs mostly through residue desulphurization (RDS). However, this process is less important than cleaning of coals, as crude oil contains less metals as impurities than most coals, except for nickel and vanadium for some heavy contaminated crude oils.

Regarding primary methods of emission reduction, it should be noted that various technologies within the same industry may emit different amounts of metals to the air or aquatic environment (Pacyna and Pacyna 2005). It can be generalized for conventional thermal power plants that the plant design, particularly the burner configuration has an impact on the metal emission quantities. Wet bottom boilers produce the highest emissions among the coal-fired utility boilers, as they need to operate at the temperature above the ash -melting temperature. The load of the burner affects also the emissions of metals in such a way that for low load and full load the emissions are the largest. For a 50% load the emission rates can be lower by a factor of two. The influence of plant design or its size on atmospheric emissions of metals from oil-fired boilers is not as clear as for the coal-fired boilers. Under similar conditions the emission rates for the two major types of oil-fired boilers: tangential and horizontal units are comparable.

Non-conventional methods of combustion, such as fluidized bed combustion (FBC) were found to generate comparable or slightly lower emissions of metals than the conventional power plants (e.g. Pacyna and Pacyna 2005). However, a long residence time of the bed material may result in increased fine particle production and thus more efficient condensation of gaseous metals, primarily mercury.

Among various steel making technologies the electric arc (EA) process produces the largest amounts of metals and their emission factors are about one order of magnitude higher than those for other techniques, e.g., basic oxygen (BO) and open hearth (OH) processes. The EA furnaces are used primarily to produce special alloy steels or to melt large amounts of scrap for the reuse. The scrap which often contains metals is processed in electric furnaces at very high temperatures resulting in volatilization of metals.

Quantities of atmospheric emissions from waste incineration depend greatly on the type of combustor and its operating characteristics. The mass burn/waterwall (MB/WW) type of combustor is often used. In this design the waste bed is exposed to fairly uniform high combustion temperatures resulting in high emissions of gaseous portions of metals. Other types of combustors seem to emit lesser amounts of metals.

The type and efficiency of control equipment is the major parameter affecting the amount of metals released to the atmosphere. Major fraction of metals emitted from high-temperature sources, such as coal-fired power plants, non-ferrous metal smelters, ferrous foundries, cement kilns and waste incinerators, occurs on fine particles with a diameter of less than 2  $\mu\text{m}$ . At present, ESPs and Fabric Filters (FFs) are the major types of particle control devices used in major industrial and electric power plants. Current removal efficiency of ESPs and FFs is more than 99.5 % for particles carrying metals mentioned in Table 1.1, except mercury.

Unlike other metals, mercury enters the atmosphere from various industrial processes mostly (around 90 %) in a gas form. The application of FGD has a very important impact on removal of mercury. A number studies have been carried out to assess the extent of this removal and parameters having major impact on this removal. These studies were reviewed in connection with the preparation for the Minamata Convention (Pacyna et al. 2010b). It was concluded that the relatively low temperatures found in wet scrubber systems allow many of the more volatile metals to condense from the vapour phase and thus to be removed from the flue gases. In general, removal efficiency for mercury ranges from 30 to 50 %. It was also concluded that the overall removal of mercury in various spray dry systems varies from about 35 to 85 %. The highest removal efficiencies are achieved from spray dry systems fitted with downstream fabric filters. Even higher mercury emission control efficiency is obtained with the application of mercury specific sorbents. As much as 90–95 % of mercury in the flue gas can be reduced by such installations (Pacyna et al. 2015b).

## 1.6.2 *Non-technological Measures*

The main objective of the Best Environmental Practices (BEP) concept is to identify and promote recommended practices as requirements in the environment protection context for new facilities and as goals for continual improvements for existing facilities. The BEP is formed along the Code of Environmental Practices for a given sector of economy. In fact, such Code also includes the recommended measures to prevent or mitigate adverse effects on the environment. In most of the cases these measures are non-technological, as the technological measures are mostly within the BAT concept.

There are various means and levels of implementation of BEPs (e.g., EC 2006), including:

- voluntary adoption by a corporation and/or facility;
- use as performance benchmarks for environmental audits;
- use as a benchmark for public corporate commitments and performance reporting;
- inclusion as a commitment in an environmental performance agreement between a corporation and/or facility and government bodies responsible for environmental protection or pollution prevention;

- inclusion of some or all of the recommendations as regulatory requirements at various government levels.

There are two major steps before the BEP concept is to be developed, including the description of: (1) activities and processes within a given industrial sector or a facility, that may result in possible adverse environmental and human health impacts, and (2) environmental concerns related to these activities and processes. These two steps and the whole concept of BEP can be implemented on the whole industrial sector at national scale or at the level of a given facility.

The literature information on developing and employing the BEP concept with regard to metal contamination concern at an industrial sector scale is quite limited. On the whole sector scale the most comprehensive document available is the Environmental Code of Practice for Base Metals Smelters and Refineries document (EC 2006). This document describes smelter and refinery operations and environmental concerns in the zinc production sector. Environmental protection practices are recommended to reduce or eliminate the adverse environmental impacts associated with smelters and refineries. Recommendations are defined separately for: (1) environmental management systems, (2) atmospheric release management, (3) water and wastewater management, and (4) waste management.

Environmental management systems are defined as an organized set of activities, actions, and procedures that go beyond legal requirements in the process of ensuring the minimal adverse impacts on the environment from various processes within the zinc production (EC 2006). In this way the zinc production sector would become more environment friendly industry. The recommendations identified with environmental management systems take into account various policies, principles, and commitments defined by Environment Canada, various provinces, and organizations. They include environment management plans, pollution prevention, emergency and decommissioning planning, various inspections, auditing, and training, as well as reporting for public.

Atmospheric release management is dealing with recommendations leading to the minimization of emissions of pollutants to the atmosphere. Mercury is treated separately in these recommendations and mercury emission guidelines are described in the Recommendation R 205 (EC 2006). The following targets for the release of mercury from base metal smelters are defined under the Canada-wide Standards (EC 2006):

- For existing facilities: application by all primary zinc, lead, and copper smelters of best available pollution prevention and control techniques economically achievable to achieve an environmental source performance (atmospheric emission) guideline of 2 g of mercury per tonne total production of finished metals.
- For new and expanding facilities: application of best available pollution prevention and control techniques to minimize mercury emissions throughout the life cycle of the minerals in question to achieve an environmental source performance (atmospheric emission) guideline of 0.2 g of mercury per tonne production of finished zinc, nickel, and lead and 1 g of mercury per tonne production of finished copper, and consideration of a mercury offset program to ensure that no “net” emission increases occur.



Water and wastewater management deals with recommendations for water use/reuse in various processes within the metal base production, wastewater collection and containment sizing, as well as water effluent guidelines and reporting. These guidelines are metal specific. For example, mercury appears in the ambient water quality guideline Recommendation R 306. The ambient water guideline for inorganic mercury is defined as 0.026 ug/l and 0.004 ug/l for methylmercury. This is guideline proposed by the Canadian Council of Ministers of the Environment, Canadian Environmental Quality Guidelines for the Protection of Freshwater Aquatic Life (in EC 2006).

Environmental Code of Practice or BEP concept on a whole sector scale often serves as a basis/guidance for development of the BEP concept at a given facility. On one side facilities are often requested to report on BEP using the sector forms, as mentioned above, on the other side the performance of a given facility along a detailed BEP form is often used as a benchmark for public corporate commitments, as well as for the improvement of the environmental image of the facility.

One of the first steps in the implementation of BEP concept at a facility level is the development of a control plan for environmental aspect assessment. This plan takes into account recommendations defined in the environmental code of practice for a given industrial sector in the country where facility is located. This is an important issue because the plan at a facility level should be compatible with and useful for reporting of a facility performance along the national or sector-based code of practice. Having this issue in mind, the control plan would need to include the elements related to:

- Identification of key production processes, production equipment, emission control systems, procedures for dealing with storage of wastes and monitoring.
- Establishment of environmental standards, recommendations for environmental friendly production processes (limits of atmospheric emissions, releases to aquatic and terrestrial ecosystems), procedures for handling raw materials, product and wastes, procedures for testing the implementation of these recommendations and procedures, responsibilities regarding the environmental risks.
- Identification of personal responsibilities for implementation of control plan, training programs resulted from the plan, as well as expectations arriving from the plan.

The above mentioned elements are presented in the control plan along the issues of safety and health, site specificity (including site chemistry), environmental charge through emissions and wastes, and environmental problem management.

## **1.7 Decision Support System (DSS) for Metal Emission Control**

The degree of necessary reduction of emissions and fluxes of metals to the environment is assessed using various tools (models, databases, measurement data) integrated into a Decision Support System (DSS). The DSS is a framework established

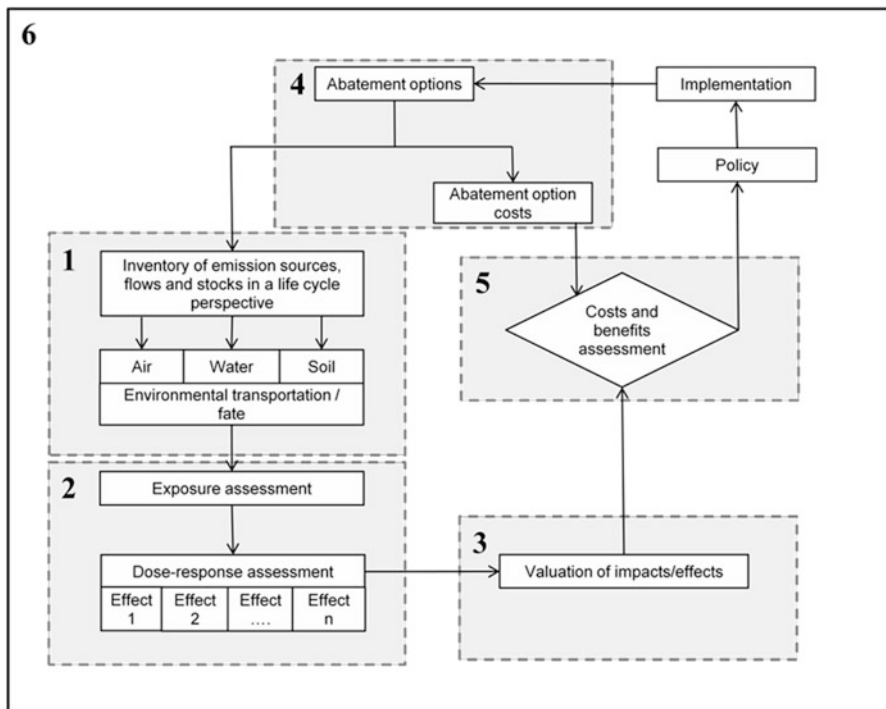


Fig. 1.4 Structural approach that forms a framework for Decision Support System

for policy makers responsible for development of environmental policies in various regions, countries or on a global scale (e.g. within UN Environment Programme – UNEP). In formation on emissions and fluxes of pollutants is the key information for the development and use of DSS. The structural approach that forms a framework for decision support is presented in Fig. 1.4.

There are various steps in the development of DSS presented in Fig. 1.4. These steps include:

- problem definition,
- system definition,
- inventory of sources and emissions,
- definition of emission scenarios,
- inventory of possible emission reduction measures,
- assessment of the effects of emission reduction measures, and
- selection of the best measures for emission reduction.

The problem definition sets the aim of the assessment. The result of this step is an overview of existing legislations, directives, or other commitments towards of emission reductions of metals, as well as provides the reasoning of the required emission reductions.

The boundaries of the assessed system are then defined. The geographical, temporal, physical and societal characteristics of the system are decided upon. These characteristics may be adjusted when the impacts are analysed in more detail in the next steps.

The next step involves an inventory of sources and emissions, which eventually affects the environmental concentrations of metals. The inventory phase should contain data collection for all relevant source categories emitting or consuming metals according to the system boundaries decided upon in the problem and system definition (in previous steps). Emissions and flows should be calculated, resulting in the construction of a substance flow diagrams for metals.

The following step seeks to answer whether additional measures are necessary to reduce emissions of metals and to investigate if there are reasons to assume that the present emission quantities will change in the future. The SFA diagrams are constructed with future emissions and flows. The baseline scenario in such case considers any relevant legislation/directives and/or modifications of them during the time scale undertaken in the analysis. Some emission sources may already or in the near future be eliminated by existing regulations and policies. This fact should be reflected when building the future emission scenarios. The analysis within this step can be used for identification of the most important emission sources, as well as for description of cross-media environmental effects under the baseline scenario.

Possible additional measures and their emission removal efficiencies (including co-control of other substances) are identified in the next step of the DSS. The result is a SFA scheme that illustrates likely future emissions based on assumptions on implementation degrees of technical measures (e.g. within BAT) as well as other management options, e.g. within BEP. The options include process-oriented options, end-of-pipe techniques, product substitution, as well as community level options (e.g. waste disposal, sediment or soil removal etc.). The investment and operation costs for these measures are also taken into account.

The environmental and human health effects of the measures identified in the previous step are assessed in the following step. Potential impacts are identified through data on emissions and exposures (source–receptor relationships) and on human health and environmental related effects (dose–response functions for various metals). The assessment of impacts should focus on the difference between the baseline scenario and the alternative (restricted) scenario. In this way, the additional costs or savings compared to the baseline scenario are assessed. The impacts should as far as possible be described quantitatively where suitable data exists. The results of the step are presented in a form of a table of effects, costs and benefits for a given emission scenario for metals, compared to the baseline scenario.

In the final step of the DSS, a selection of a set of best emission reduction measures is prepared for their implementation. The ranking of the measures is based on the results of cost–benefit analysis associated with these measures.

The DSS for metals in Europe has been developed by the authors of this Chapter together with other scientists within the EU project SOCOPSE (e.g. Baartmans et al. 2008). The application of this DSS for mercury in Europe has been made by Sundseth (2012). He concluded that large economic benefits can be achieved globally with the reduction of mercury emissions using the measures applied in the EU

region. The investigated Baseline scenario highlighted the importance of full implementation of existing measures, and the importance of making further progress in reducing mercury emissions from European sources. However, the application of cost–benefit analysis indicated that the economic cost of alternative technological options for coal combustion and industrial boilers in the EU region, which is the main emission source for mercury in Europe, exceeds the economic benefits. Sundseth (2012) pointed out that reducing emissions in developing countries may be most effective which reconfirms the importance of the Minamata convention plans for emission reductions worldwide.

## 1.8 Final Remarks

Metals are important chemicals occurring in various ecosystems. Some of them are toxic, some are toxic when appearing in excess and some are essential for the environment and human health. Environmental and human health impacts of metals depend of various factors, however, the key parameters are emission quantity to the air, water and land, and their transport through various ecosystems resulting in the food chain and air contamination, which are the main pathways of metals for entering human body. Thus the quantity of metal emissions and the degree of human alteration of metals biogeochemical cycles are the key information for the human health impacts of these chemicals.

Complete and accurate emission inventories and future emission scenarios for metals are needed in order to assess the current and future exposure of the ecosystems and human to these pollutants. Major sources of metals are already well defined, particularly when assessing the emissions on local and regional scale. Most of these emissions inventories are prepared on the basis of emission factors for various sources and source categories. More emission measurements are needed for verification of emission factors and improving them with respect to their accuracy and appropriate applicability (for a given technology, source category or even a geographical region). Information is often lacking on chemical and physical forms of metal emissions, as well as on metal organic forms, so important when discussing the toxicity of metals.

Anthropogenic sources of metal emissions are better evaluated than natural sources, including the re-emission of volatile species, such as mercury from land and aquatic ecosystems to the atmosphere. More focus is needed on the assessment of metal emissions from biogenic sources, crustal weathering and volcanic activities. Improved information on sources and emissions of metals will have the main effect on the improvement of our knowledge on biogeochemical cycling of these pollutants. Improvement of information on emissions of metals is particularly needed for assessment of water and land releases. More accurate information on biogeochemical cycling of metals will result in the improvement of our knowledge of metal exposure pathways, and consequently of metal dose to human. Quality of information on dose–response functions for metals will benefit from the above mentioned improvement. Accurate assessment of dose–response functions is the

most important research task when studying the impacts of environmental pollutants on human health.

There are various technological and non-technological measures that can be applied to reduce emissions and fluxes of metals in the case of their excess. The selection of the most appropriate measure in the context of largest environmental and human health benefits and the optimal investment and operational costs can be made using the framework of the decision support system. The DSS structure has been developed, however, its successful application depends on the quality of various models and data bases brought into the DSS. While environmental transport and distribution models undergo continuous improvement, particularly the improvement of chemical and physical schemes for metal transport, more research emphasis is needed to improve the quality of food chain modelling and the uptake models to various human organs, particularly to critical organs for specific metals.

In general, major focus of environmental research on metals has been on lead and mercury, resulting in legal actions to reduce emissions and exposure to these contaminants. Lead additives to gasoline are phased out worldwide. In 2013, the Minamata convention has been established with the aim to reduce the levels of mercury in the environment. However, there are also other metals which need more attention because of their biogeochemical cycling is altered significantly by human activities causing a risk to human health. These metals are mentioned in Table 1.1, with focus on arsenic, cadmium, nickel, and vanadium. However, there are also other metals, such as Rare Earth Elements (REE), which need to be studied in the context of increasing demand by the market and industry for these chemicals on one side and a very limited knowledge on their toxicity and other environmental impacts on the other side.

## References

- Ackland ML, Silbergeld EK, Bornhorst J, Dedoussis G, Dietert RR, Nriagu JO, Pacyna JM, Pettifor JM (2015) Metals in the environment as risk factor for infectious diseases. In: Nriagu, Skaar ES (eds) Heavy metals and infectious diseases. Ernst Strungmann Forum reports (Lupp J, series ed.), vol 16. MIT Press, Cambridge, MA. ISBN: 978-0-262-02919-3
- Andrea MO, Asami T, Bertine KK, Buat-Ménard PE, Duce RA, Filip ZK, Förstner U, Goldberg ED, Heinrichs H, Jernelöv AB, Pacyna JM, Thornton J, Tobschall JJ, Zoller WH (1984) Perturbations of biogeochemical cycles of metals. In: Nriagu JO (ed) Changing metal cycles and human health. Dahlem Konferenzen and Springer, Berlin, Heidelberg, New York, and Tokyo
- Baartmans R, van Tongeren W, van der Vlies J, Ullrich S, Mattila T, Cousins AP, Belhaj M, Munthe J, Pacyna JM, Sundseth K (2008) Deliverable D4. DSS handbook SOCOPSE, draft version. Source Control of Priority Substances in Europe (SOCOPSE). TNO, The Netherlands
- Chang LW (ed) (1996) Toxicology of metals, vol 1. CRC Press, Boca Raton, FL
- De Vos W, Tarvainen T (eds) (2006) Geochemical atlas of Europe. Part 2: Interpretation of geochemical maps, additional table, figures, maps and related publications. Geological Survey of Finland, Espoo
- EC (2006) Environmental Code of Practice. Canadian Environmental Protection Act 1999, Base metals smelters and refineries. EPS1/MM/11E, Environment Canada, Gatineau, Canada, Mar 2006

- Falkowski P et al (2000) The global carbon cycle: a test of our knowledge of Earth as a system. *Science* 290(5490):291–296
- Hinkley TK (2003) Volcanic emissions of mercury. *U S Geol Surv Circ* 1248:35–37
- Levine JS (ed) (1991) Global biomass burning: atmospheric, climatic and biospheric implications. MIT Press, Cambridge, MA
- Nriagu JO (1989) A global assessment of natural sources of atmospheric trace metals. *Nature* 338:47–49
- Nriagu JD, Pacyna JM (1988) Quantitative assessment of worldwide contamination of air, water, and soils by trace elements. *Nature* 333:134–139
- Pacyna JM (1986) Atmospheric trace elements from natural and anthropogenic sources. In: Nriagu JO, Davidson CI (eds) *Toxic metals in the atmosphere*, Advances in environmental science and technology. Wiley, New York
- Pacyna JM (2009) Material flow analysis for selected priority substances. The SOCOPSE project, Report WP2–D2. Norwegian Institute for Air Research, Kjeller
- Pacyna JM, Ahmadzai H (1997) Air pollution abatement. In: Brune D, Chapman D, Gwynne M, Pacyna JM (eds) *The global environment*. Wiley and Scandinavian, Weinheim, pp 724–748
- Pacyna JM, Pacyna EG (2001) Assessment of global and regional emissions of trace metals to the atmosphere from anthropogenic sources worldwide. *Can J Environ Rev* 9:269–298
- Pacyna JM, Pacyna EG (2005) Anthropogenic sources and global emissions of mercury. In: MB Parsons, JB Percival (eds) *Mercury: sources, measurements, cycles and effects*. Short course series, vol 34. Mineralogical Association of Canada, pp 21–41
- Pacyna EG, Pacyna JM, Sundseth K, Munthe J, Kindbom K, Wilson S, Steenhausen F, Maxson P (2010a) Global emission of mercury to the atmosphere from anthropogenic sources in 2005 and projections to 2020. *Atmos Environ* 44:2487–2499
- Pacyna JM, Sundseth K, Pacyna EG, Munthe J, Belhaj M, Astrom S (2010b) An assessment of costs and benefits associated with mercury emission reductions from major anthropogenic sources. *J Air Waste Manag Assoc* 60:302–315
- Pacyna JM, Sundseth K, Pacyna EG (2015a) New technologies using trace metals of concern. In: Nriagu JO, Skaar ES (eds) *Heavy metals and infectious diseases*. Ernst Strungmann Forum reports (Lupp J, series ed), vol. 16. MIT Press, Cambridge, MA. ISBN: 978-0-262-02919-3
- Pacyna JM, Sundseth K, Pacyna EG (2015b) Technological and non-technological measures to reduce mercury emissions from industrial sources. The 2014 international conference on mercury as a global pollutant, Jeju, 15–19 June
- Rauch JN, Pacyna JM (2009) Earth's global anthropogenic Ag, Al, Cr, Cu, Fe, Ni, Pb, and Zn cycles. *Global Biogeochem Cycles* 23, GB2001. doi:[10.1029/2008GB003376](https://doi.org/10.1029/2008GB003376)
- Rytuba JJ (2005) Geogenic and mining sources of mercury to the environment. In: Parsons MB, Percival JB (eds) *Mercury: sources, measurements, cycles and effects*. Short course series, vol 34. Mineralogical Association of Canada, pp 21–41
- Sundseth K (2012) A novel combination of methods developed for decision support on abatement of mercury in Europe. PhD Dissertation, Gdansk University of Technology, Gdansk
- Sundseth K, Pacyna JM, Banel A, Pacyna EG, Rautio A (2015) Climate change impacts on environmental and human exposure to mercury in the Arctic. *Int J Environ Res Public Health* 12:3579–3599. doi:[10.3390/ijerph120403579](https://doi.org/10.3390/ijerph120403579)
- UNEP (2013a) Environmental challenges of metals cycles. UNEP Resource Panel, United Nations Environment Programme, Geneva
- UNEP (2013b) Global mercury assessment 2013: sources, emissions, releases and environmental transport. UNEP Division of Technology, Industry and Economics, United Nations Environment Programme, Geneva
- van Storch H, Hagner C, Costa-Cabral M, Feser F, Pacyna JM, Pacyna E, Kolb S (2002) Curbing the omnipresence of lead in the European environment since the 1970's – a successful example of efficient environmental policy. *EOS* 83(36):393–399
- van Storch H, Costa-Cabral M, Hagner C, Feser F, Pacyna JM, Pacyna EG (2003) Four decades of gasoline lead emissions and control policies in Europe: a retrospective assessment. *Sci Total Environ* 311:151–176

## Chapter 2

# Exposure to Environmental Hazards and Effects on Chronic Disease

Miranda Loh

**Abstract** There are numerous hazards that people are exposed to in everyday life: at home, at work, and in other locations (microenvironments). Between exposure and health effect there are a large number of modifying risk factors, as embodied in the concept of the exposome. Exposures to other hazards, genetics, and society all play a role in whether and how an exposure results in an adverse effect. The timing of exposure is also an important factor. In utero and early life exposures may be of particular importance for initiating some types of diseases that manifest later in life.

Chronic diseases such as cardiovascular and respiratory disease are important contributors to the global burden of disease. These diseases are a result of the interplay of several risk factors, which include environmental exposures. The exposomic approach is therefore particularly applicable to the study of environmental causes of chronic disease. Rather than take a pollutant-by-pollutant focus, this chapter will examine two common chronic diseases which research has shown to have multiple contributing environmental risk factors. The proposed diseases are cardiovascular disease and non-malignant respiratory disease (particularly asthma). The chapter will cover both acute and chronic effects, including a discussion of the evidence for some hazards where exposures in childhood or before birth can potentiate future disease.

**Keywords** Exposome • Exposure • Cardiovascular disease • Asthma

## 2.1 Introduction

There are numerous hazards that people are exposed to in everyday life: at home, at work, and in other locations (microenvironments). Health impacts of environmental hazards are a product of many different factors. These factors can be co-exposures, epigenetics, genetics, psychosocial stress, the physical environment, cultural factors,

---

M. Loh (✉)

The Institute of Occupational Medicine Research Avenue North, Riccarton,  
Edinburgh EH14 4AP, Edinburgh, UK

e-mail: [loathue@tal.upv.es](mailto:loathue@tal.upv.es)

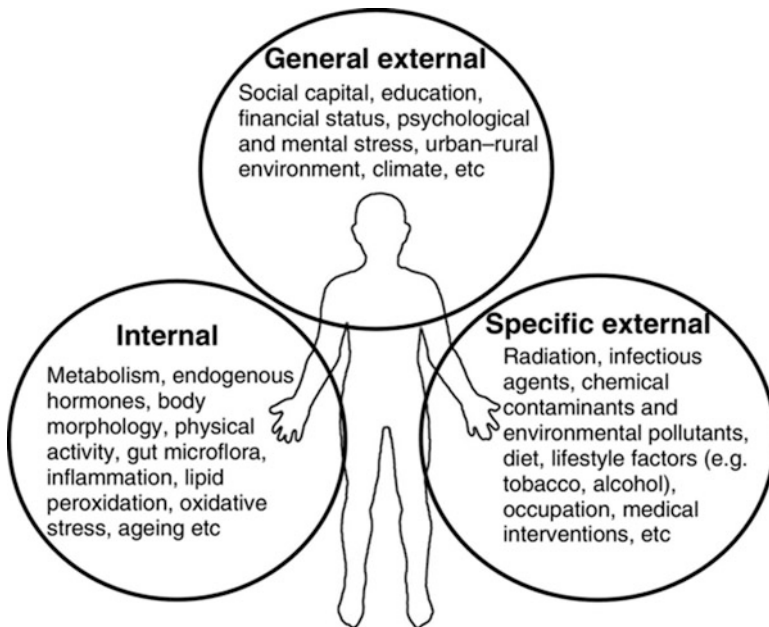
© Springer International Publishing Switzerland 2016

J.M. Pacyna, E.G. Pacyna (eds.), *Environmental Determinants of Human Health*,  
Molecular and Integrative Toxicology, DOI 10.1007/978-3-319-43142-0\_2

etc. Whereas in the past, research into health impacts of environmental exposures tended to focus on eliciting the effect of a single hazard, while controlling for potential confounding factors, the research paradigm is moving towards a multi-factorial approach. The idea of “-omics” embodies this approach, where “-ome” refers to the totality of something, the genome, the transcriptome, the microbiome, etc. Methods of analysis—chemical, biological, and statistical—are now more focused on evaluating multiple risks as a whole, examining risk profiles, rather than single predictors.

Exposure science is now faced with the “exposome”, or all exposures that a person experiences from conception to death (Wild 2012). It is thought that only a small percentage of disease is explained by genetic factors (Thomas 2010). Gene-environment interactions likely play an important, but not well understood role in disease development. These include a wide range of factors, which can be grouped as general external (e.g. urban environment, psychosocial stress), specific external (e.g. air pollutants, radiation), and internal (e.g. genetics, inflammation) as reflected in Fig. 2.1. The interaction of these spheres results in disease manifestation. The challenge for the future will be developing frameworks and methodologies for assessing the exposome. Although it may be impossible to assess all aspects of the exposome, at least in the near future, it is important to begin taking this approach for understanding and preventing disease.

This chapter will examine two common chronic diseases which research has shown to have multiple contributing environmental risk factors. The proposed diseases are cardiovascular disease and asthma. Chronic diseases such as these



**Fig. 2.1** The exposome (Wild 2012) by permission of Oxford University Press



are important contributors to the global burden of disease, and are a result of the interplay of several risk factors, which include environmental exposures. The exposomic approach is therefore particularly applicable to the study of environmental causes of chronic disease. Although the pathways through which the environment influences the development and manifestation of these diseases differ, inflammation is a key common mechanism of action. There are a number of hazards and risk factors that affect both heart and lung disease, including exposure to air pollution, arsenic, cigarette smoke (and smoking), diet, obesity, and stress. Exposures *in utero* and in early life are also thought to be an important influence on the later development of disease. This chapter will not discuss exposures to parents pre-conception, but includes a brief discussion of exposures via the mother *in utero*. In the fetal origins of disease theory, exposures *in utero* may make a person more susceptible to disease later on in life. Exposures and lifestyle patterns after birth and into adulthood also affect the propensity of the person to develop disease as an adult.

The chapter will cover both acute and chronic effects, including a discussion of the evidence for some hazards where exposures in childhood or before birth can potentiate future disease. The focus will be primarily on the “specific external” exposome, with some discussion of how “general external” factors may contribute to disease effects. Internal aspects will not be examined. The factors described here are meant to cover many of the non-occupational risk factors for Cardiovascular disease (CVD) and asthma, but are not a complete list.

## 2.2 Cardiovascular Disease and Asthma

### 2.2.1 Cardiovascular Disease

Cardiovascular diseases (CVD) are the leading cause of death globally, and ischemic heart disease is estimated to be responsible for about half of all CVD deaths. Genetics and lifestyle play a key role in development of disease, but environmental risk factors are also an important contributor. Risk prediction based on the Framingham Heart Study for heart disease outcomes such as myocardial infarction, coronary heart disease, and death, include age, gender, total serum cholesterol, high density lipoproteins (HDL), blood pressure, diabetes, and smoking (D’Agostino et al. 2001). Ethnic and racial groups exhibit differences in CVD risk. Tobacco, low HDL and high low density lipoproteins (LDL), and elevated glucose are accepted as causal risk factors for CVD, while external risk factors that predispose individuals to CVD include physical inactivity, socioeconomics, stress, and diet (Yusuf et al. 2001). Exposure to several pollutants has also been found to increase risk of developing cardiovascular disease, and contribute to inducing acute cardiovascular events. Inflammation is a key mechanism by which many non-genetic risk factors, including environmental ones, are thought to influence development of CVD.

### **2.2.2 Asthma**

Asthma is a complex disease of the airways, with several phenotypes (observable characteristics) and endotypes (disease entity defined by a specific biological mechanism) (Lötvall et al. 2011). Allergic phenotypes tend to occur in youth, and are more likely in males, although they can still manifest in adulthood. The onset of asthma in childhood is greater in males than females, whereas in adulthood the pattern is switched. Non-allergic asthma tends to occur more often in adulthood. A number of environmental agents have been found to play a role in impacting the development of the lung, and hence susceptibility to lung disease in childhood or adulthood (Miller and Marty 2010). Asthma can be characterized by chronic inflammation, which leads to airway hyper-responsiveness and remodeling (Bousquet et al. 2000), although there have been observations that not all asthma patients have a strong inflammatory component (Lötvall et al. 2011).

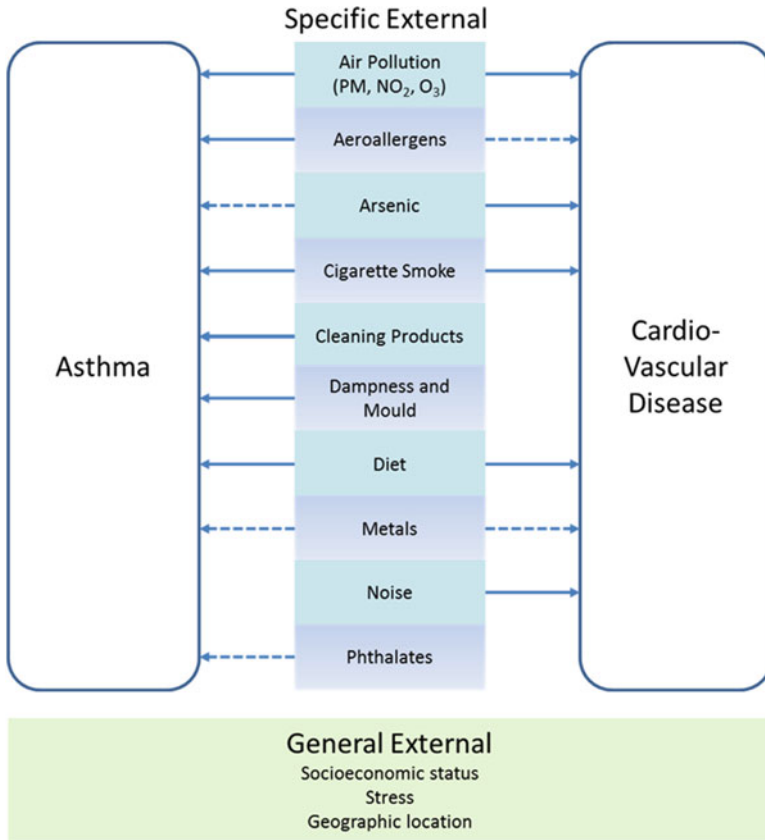
### **2.2.3 Common Exposures and Risk Factors for Cardiovascular Disease and Asthma**

Major “specific external” exposome factors that play a role in development and symptom manifestation of both CVD and asthma include exposures to air pollution, cigarette smoke, arsenic, and diet (Fig. 2.2). Psychosocial stress and socio-economic status are also “general external” exposome factors for both sets of diseases. Specific occupational exposures can also play an important role for development and exacerbation of CVD and asthma, although these are not described in detail in this chapter. Other exposures related to CVD include noise and metals, while those related to asthma include cleaning product chemicals, phthalates, and dampness and mould.

#### **2.2.3.1 Air Pollution**

Much of the evidence around air pollution risks relates to combustion-source pollutants. Exposure to these pollutants can take place outdoors, in transit, at work, and indoors. Outdoor sources include power plants, industry, residential wood burning, vehicles, and ships. Indoor sources include smoking, cooking, candle and incense burning, and fireplaces. Outdoor sources are the most easily controlled by regulatory authorities and policies, but people spend relatively little time outdoors (~ less than 10 % of time, on average) (Klepeis et al. 2001; Schweizer et al. 2007).

Air pollution is a complex mixture of gases and particulate matter, which is determined by sources and atmospheric or microenvironmental climate and conditions. The mixture of pollutants a person breathes in depends on the location of the person



**Fig. 2.2** Aspects of the external exposome that influence asthma and cardiovascular disease. The *solid arrows* indicate relatively strong evidence for either symptoms or induction of disease and the *dashed arrows* indicate suggestive but not conclusive evidence

and the sources that contribute to the pollution in that location. Outdoor sources include power plants, industry, residential wood burning, vehicles, and ships along with natural sources such as desert dust (in some regions) and wildfires.

Of outdoor sources, traffic pollution is of particular concern because this source tends to be closer to the population as a whole, especially in urban areas. People therefore have a greater risk of exposure to traffic pollution. Epidemiology studies have also found associations between exposure to traffic and cardiovascular disease and asthma. Traffic pollution includes particulate matter (PM), nitrogen dioxide, carbon monoxide, aldehydes, volatile organic compounds (VOCs), and semi-volatile compounds (SVOCs), such as polycyclic aromatic hydrocarbons (PAHs). One difficulty with assessing the health impacts for pollutants from a common source, such as traffic, is that the concentrations tend to be correlated, thus making it difficult to separate the effects of one pollutant from another. Controlled exposures or laboratory studies provide some possibility of distinguishing the individual effects of pollutants.

Particulate matter has been strongly related to cardiovascular disease in epidemiology, toxicology, and controlled exposure studies (Brook et al. 2010; Miller et al. 2012). Evidence for the effects of ozone and nitrogen dioxide on cardiovascular disease is not as clear as for particulate matter (Review of evidence on health aspects of air pollution—REVIHAAP project 2013). In chamber studies, exposures to concentrated ambient particles or dilute diesel exhaust led to acute vasoconstriction in volunteers, but exposures to NO<sub>2</sub> and filtered air showed no effects on vascular dysfunction (Langrish et al. 2010). Exposure to NO<sub>2</sub> may increase the effects of particulate matter, although this has not been demonstrated in all areas (Brook et al. 2004).

Nitrogen dioxide is often used as a marker of traffic exhaust, and there is some evidence in the time series literature for respiratory related mortality (Review of evidence on health aspects of air pollution—REVIHAAP project 2013). Nitrogen dioxide levels in homes with gas appliances has been found to range from 180 to 2500 µg/m<sup>3</sup>, which are many times higher than typical outdoor levels (Heinrich 2011). In controlled exposure studies NO<sub>2</sub> leads to sustained increases in neutrophil response and decreases in lung function in healthy subjects, but only when exposure is >1 ppm, which is about two orders of magnitude greater than typical outdoor exposures (Parnia et al. 2002). Evidence is less clear for lower concentrations, which are what people are exposed to in daily life. Nitrogen dioxide may play a role in exacerbating asthma, and has been associated with incident wheeze, but it is uncertain whether or how nitrogen dioxide influences the development of asthma. A modest association between NO<sub>2</sub> and incidence of asthma (OR 1.09, 0.96–1.23 per 10 µg/m<sup>3</sup> increase) was found in a meta-analysis of five birth cohorts for longitudinal exposure and childhood asthma, but with substantial variability between studies (Bowatte et al. 2015). Co-exposures of more than one air pollutant have been found to increase the airway response of the allergic asthmatic response to triggers (Parnia et al. 2002).

Ozone exposure is also associated with asthma symptoms, exacerbations, and decreased lung function in adults. It has been shown to induce inflammation in the lung, and animal studies indicate that ozone exposure leads to airway remodeling (Review of evidence on health aspects of air pollution—REVIHAAP project 2013). Experimental exposure of humans to ozone causes irritation and cough, with decrements in FVC and FEV<sub>1</sub>, but at high levels (0.4 ppm) (Koren et al. 1989). Inflammatory biomarkers were also shown to increase, along with indicators of increased vascular permeability. Such effects may allow allergens to penetrate deeper into airway mucosa, increasing or altering the immune response (Parnia et al. 2002).

Particulate matter itself is a mixture of particles of different sizes and composition. PM is a trigger for short-term cardiovascular events, and for biological responses that can raise long-term CV risk (Brook et al. 2010). Several characteristics of PM are thought to play a role in its toxicity: size, surface area, and composition. These characteristics are linked to the sources that emit PM and the physio-chemical transformations that the particles undergo in the atmosphere. Although particles of various sources have been linked with CVD, combustion-produced particles are of particular interest, due to the small size of these particles, and the potential for

numerous toxins to be sorbed onto these particles. One mechanistic explanation for the effect of PM on CVD is through the inflammatory response. Inhalation of particles leads to inflammation in the airways, with fine and ultrafine particles reaching the alveolar region. Particles less than a nanometer in size may cross the alveolar membrane and enter systemic circulation.

In occupational settings, particles are classified into three sizes. Inhalable particles enter the nose and mouth through breathing, and are 100  $\mu\text{m}$  in diameter or less. These may include dusts and other mechanically generated particles. Thoracic particles ( $\leq 20 \mu\text{m}$  diameter) penetrate further into the bronchi, and respirable particles ( $\leq 4 \mu\text{m}$  diameter) yet further into the alveoli. Non-occupational regulations distinguish between  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ , which are particles with an aerodynamic diameter  $\leq 10 \mu\text{m}$  and  $\leq 2.5 \mu\text{m}$ , respectively.  $\text{PM}_{2.5}$  is also known as fine particulate matter, while particles between 2.5 and 10  $\mu\text{m}$  are termed coarse particles. Fine particles are generally emitted from combustion sources, such as fires, power plants, and vehicles, and from secondary formation from gases or liquid droplets. Coarse particles in the environment can include dust, such as re-suspended road dust and desert dust. Biological aerosols such as pollen, mould, allergens from insects and pets, bacteria, and viruses can be found throughout the range of particle sizes. Freshly emitted particles from traffic are in the ultrafine range ( $< 100 \text{ nm}$ ). Ultrafine particle (UFP) concentrations tend to drop off relatively quickly from the road, with levels reaching background around 300 m, with a 50% reduction by about 150 m (HEI Review Panel on Ultrafine Particles 2013).

In addition to size, particulate matter can differ widely in composition from source to source. Healthy volunteers exposed to diesel exhaust particulate in controlled exposure studies have been found to induce responses such as inflammation, endothelial dysfunction, and thrombosis (Lucking et al. 2008; Lundbäck et al. 2009; Törnqvist et al. 2007). Exposure to particles of different composition in controlled exposure studies (i.e. concentrated ambient particulates, CAPS, and diesel exhaust) showed differing effects on heart rate variability, with reductions in variability seen with CAPS but not with diesel (Miller et al. 2012). Individual constituents of particles such as certain metals may affect risk of CVD development.

Although combustion source particles are thought to be most dangerous for health, time series studies have also found that desert dust can impact mortality and hospital admissions (Middleton et al. 2008; Perez et al. 2008). Particles from indoor sources may also be from combustion sources, dust, biological material, or secondary organic aerosols, generated from reactions between volatile organic compounds and ozone. Health effects of indoor source particles are less well studied, and it is assumed that combustion source indoor particles would have the same effect as ambient particles. However, the inflammatory response to particles in the respiratory tract may be a key mechanism, in the development and exacerbation of CVD, thus implying that all particles may be of concern.

Air pollutants have been clearly shown to increase the risk of asthma exacerbations and symptoms, and may also increase risk of developing asthma, especially in children (Gowers et al. 2012). Traffic exhaust exposure is associated with higher risk of asthma symptom development. Ozone, nitrogen dioxide, and particulate matter, all

also related to traffic, have been associated with asthma exacerbation. A multi-cohort study of more than 23,000 adults followed over 10 years was suggestive of an effect from  $\text{NO}_2$  and  $\text{PM}_{10}$  on asthma development, but results were not conclusive (Jacquemin et al. 2015). A review by the British Committee on the Medical Effects of Air Pollutants (COMEAP) determined that evidence for individual pollutants in asthma induction is still inconclusive, but studies indicate that living close to major roads (<150 m) is linked to wheeze and asthma, with increasing effects with proximity (Gowers et al. 2012). In addition, truck traffic has been identified as a risk for asthma, which may be considered a proxy for increased exposure to diesel exhaust.

Particulate matter elicits an inflammatory response and contains aeroallergens, metals, PAH, and other substances that may trigger both allergic and non-allergic airway responses. A meta-analysis of four birth cohort studies found an increased incidence of childhood asthma (OR 1.14, 1.00–1.30 per  $2 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ ) but also with substantial heterogeneity between studies (Bowatte et al. 2015). Diesel exhaust particulate (DEP) has been identified as a possible carrier of allergens on surface, therefore increasing exposure during pollution episodes, or near roads with diesel traffic (Parnia et al. 2002). DEP have been shown to enhance IgE production and activation of basophils, particularly due to the polycyclic aromatic hydrocarbons (PAH) on the particle surface rather than carbon core (Lubitz et al. 2010; Parnia et al. 2002; Takahashi et al. 2010), but do not induce IgE production. Also, DEP causes inflammation of the airways, which makes them more susceptible to allergen challenge. Damage to epithelium and cilia reduces the effectiveness of the biological barrier against allergens (Parnia et al. 2002). In general, PM has been associated with asthma symptoms, but evidence for development of asthma is limited, although *in utero* exposure to  $\text{PM}_{2.5}$  has been associated with early childhood asthma (Hsu et al. 2015).

An important source of exposure for many air pollutants is cigarette smoke. Smoking is a known risk factor for cardiovascular and respiratory diseases, including asthma. In addition, non-smokers chronically exposed to cigarette smoke are similarly at risk (Barnoya and Glantz 2005; Öberg et al. 2011; Pope et al. 2009). Exposure to second hand smoke (SHS) is of particular concern indoors, where due to a smaller volume for dilution and, in some homes, lower ventilation rates, the accumulation of particles and other pollutants from cigarette smoking can lead to quite high levels of pollutants (Callinan et al. 2010; Loh et al. 2006; Nazaroff and Singer 2004; Semple et al. 2012; Waring and Siegel 2007). For example, the concentration of  $\text{PM}_{2.5}$  and benzene in Irish pubs decreased approximately 80 % from pre-ban levels after the Irish ban on workplace smoking (Goodman et al. 2007). These bans have also been found to decrease hospital admissions for cardiovascular and asthma outcomes (Cesaroni et al. 2008; Goodman et al. 2007; Sims et al. 2010). While smoking bans in workplaces and public buildings have become more prevalent in many countries, smoking may still occur inside homes and private vehicles.

*In vivo* studies of exposure to diesel exhaust and cigarette smoke show that *in utero* and in early life exposures can induce physiological changes that increase the risk of cardiovascular related risk factors (e.g. blood pressure, weight gain) in adult mice (Weldy et al. 2014). Some epidemiological studies have found that

prenatal exposure to air pollution results in birth outcomes indicating reductions in fetal growth (e.g. small for gestational age, low birthweight, low intra-uterine growth rate), which may be associated with CVD later in life (Ballester et al. 2010; Šrám et al. 2005).

### 2.2.3.2 Noise

Noise exposure may contribute to the development of CVD due to stress or activation of the sympathetic nervous system, and has been associated with increased blood pressure, changes in heart rate, and release of stress hormones (Babisch 2011; Basner et al. 2014). Overall, road traffic and aircraft noise seems to have an independent effect on CVD and hypertension from air pollution, although effects and significance across epidemiology studies are mixed (Davies and Kamp 2012). Nocturnal noise is considered more important than daytime noise, especially with respect to sleep disturbance.

The HYENA study of noise near Heathrow Airport in London found significant increases in blood pressure with night noise, but not with daytime noise. Cohort studies, which investigate long-term exposure to noise, have found increased hypertension with road traffic noise, with a meta-analysis showing an odds ratio of 1.08 (1.04–1.13) per 10 dB increase in  $L_{DN}$  (day-night equivalent level, over 24 h, with an additional penalty for night-time noise), where noise levels were in the range of 50–75 dB (Münzel et al. 2014). Sleep restriction or fragmentation is another factor that affects the development of various CVD risk factors, and has been associated with inadequate pancreatic insulin secretion, decreased insulin sensitivity, changes in appetite regulating hormones, increased sympathetic tone, and venous endothelial dysfunction (Münzel et al. 2014). Habitual sleep of less than 6 h per night has been related to obesity, diabetes, hypertension, CVD-related and all-cause mortality (Münzel et al. 2014). Vulnerable groups such as the elderly, shift workers, and those with illness may be more susceptible to noise related sleep disturbance (WHO 2009). These groups are also more vulnerable to other environmental risks for CVD, particularly air pollution.

In non-occupational settings, noise exposure from transportation-related sources has been most studied, especially with road and aircraft noise. Road noise is often correlated with air pollutants released by vehicles, and therefore these must both be considered in analyses. Noise exposure has two components: a measure of loudness (sound pressure level) and frequency. Loudness is generally what is used to define noise exposure in epidemiology studies, but frequency is also an important characteristic. The frequency of a noise can affect a person's perception of the annoyance of noise, even if the noise is not loud. High frequency noises are also easier to dampen than low frequency noise, due to the much shorter wavelength of high frequency noises. Masking of one noise source with another also depends on whether the noise sources' frequency domains overlap (WHO 2009). The intervals at which a noise occurs can also affect the health impact of noise.

Noise can be monitored using sound level meters for area measurements, or dosimeters, for personal measurements. In many epidemiology studies, however, noise exposure is modeled at the outer façade of a building, based on inputs such as traffic flow and road type. Noise exposure is generally reported as  $L_{Aeq,T}$ , which refers to A-weighted sound pressure level in decibels averaged over an amount of time,  $T$ . The A-weighting refers to a means of correcting sound pressure levels for the frequencies to which the human ear is most sensitive. Day ( $L_{day}$ ) and night ( $L_{night}$ ) noise where sound pressure levels are averaged over specified daytime and nighttime hours may be used as metrics of environmental noise exposure. Additionally, the  $L_{den}$  may be used, where noise levels in the daytime, evening, and night are differentially weighted, with the greatest penalty for night-time noise. While the sound pressure levels only refer to loudness, it is assumed that they are specific to noise of a particular source and frequency domain.

Transport-related noise levels are relatively lower than levels at which occupational noise is regulated. Low noise exposure assignment is <50 dBA, while high exposures are >60, however noise below 40 dBA may disturb sleep (Münzel et al. 2014; WHO 2009). The World Health Organisation (WHO) recommends that noise be assessed for relevant locations in the home at different times of the day, such as the bedroom for nighttime noise. However, this resolution of data is not generally available. Sound dampening of buildings can account for from 24 to over 45 dB of reduction of outdoor source noise. Noise from other sources in the home, or noise at non-home locations that people spend time in are not included when examining the health impacts of noise.

### 2.2.3.3 Metals

Certain elements have been found to influence the development of CVD and asthma, generally at high levels of exposure. Much of the evidence, especially for asthma, is seen in occupational exposures, for example in smelter workers, welders, and similar jobs working with high metal exposures. Cigarettes are also a significant source for smokers and those with frequent exposure to environmental tobacco smoke. There is support for a link between lead exposures, especially at high levels as seen in occupational settings, and hypertension, although the evidence for the effect at low environmental exposures is not clear (Bhatnagar 2006; Hu et al. 1996; Navas-Acien et al. 2007). The mechanism of action is still unknown, but may involve changes in renal function and oxidative stress (Vaziri 2008). There has been some suggestion that cadmium and mercury exposure may also influence the development of CVD, but these effects are not yet well supported by the literature. Exposure to metals tends to be via ingestion (dietary and non-dietary) or inhalation, with varying absorption efficiencies depending on the route of exposure and medium of exposure (e.g. water, food, dust, or soil) (Solenkova et al. 2014). For most people, cadmium and mercury exposure would come from food. Leafy vegetables, potatoes, grains, some nuts and legumes, and organ meats have high levels of cadmium (EFSA (European Food Safety Authority) 2012; Solenkova et al. 2014). Mercury



intake can be particularly high for those who eat certain types of fish, or marine mammal meat. Other sources include certain cosmetics, dental amalgams, and medicines (Solenkova et al. 2014).

Lead exposures have declined greatly in many countries, with the removal of lead from gasoline, paint, and common products. Occupational groups, those living in homes with lead paint, or those living near industries such as smelters are vulnerable to high exposures. In the United States, the Centers for Disease Control (CDC) reduced the guideline for blood lead from 10 to 5  $\mu\text{g}/\text{dL}$ . Ingestion of lead is of most concern for children, although in areas where the particle load of lead is high, inhalation may be an additional exposure route. Exposures can exceed the guidelines even for non-occupational groups for people who live near lead-emitting industries (Gulson et al. 1994; Wilson et al. 1986). In some countries, lead may still be present in pottery glazes or glassware used for food, and this may be an additional pathway of exposure. Occupational exposures most likely occur from inhalation of lead fumes or lead dust, and from ingestion of lead dust. Occupations at greater risk for lead exposure include those that manufacture or use ammunition, recycling of electronics, metal, or batteries, welders, and workers in lead mining, refining, or smelting. Occupational limits are 50  $\mu\text{g}/\text{m}^3$  in the US and UK.

#### 2.2.3.4 Arsenic

Arsenic exposure is near ubiquitous, as it is found in many foods, both naturally and as a contaminant. Arsenic is a metalloid that people are commonly exposed to either as inorganic arsenic, As(III) or As(V), which are thought to be of most concern, and also as organic species. In addition to chronic lifetime exposures, *in utero* exposure to arsenic has been associated with increased risk of CVD. Inhalation exposure is a relatively small contributor to total arsenic exposure, except in occupational settings. Arsenic may be found bound to particles in the air and in soil, and can be present in high concentrations in certain regions due to a region's geological characteristics, or due to contamination, such as in areas near copper smelters or mines. Arsenic in soil or indoor dust may be inadvertently ingested, a pathway of exposure that infants and toddlers are particularly susceptible to. In areas with high arsenic in soil or dust, arsenic in urine in young children has been found to be correlated with soil or dust concentrations. The major pathways of exposure to arsenic, however, are ingestion of contaminated drinking water and food.

Arsenic exposure has been linked to increased risk of developing cardiovascular diseases and related risk factors of hypertension and diabetes and decreased lung function, bronchiectasis, and increased susceptibility to respiratory infections. Although arsenic is associated with reduced lung function and respiratory infection, it has not yet been found to be related to asthma. It was, interestingly, considered an anti-asthmatic in earlier times. Health effects have primarily been observed in populations with relatively high exposures via contaminated drinking water. Effects at lower levels, which many more people are exposed to, are not conclusive.

Chronic exposure to high levels of arsenic in drinking water (>100 ppb) is related to hypertension and CVD in various populations around the world. In a meta-analysis, Moon et al. found pooled relative risks comparing the highest to lowest exposure groups of 1.32 (95 % CI 1.05–1.67) for CVD, 1.89 (1.33–2.69) for coronary heart disease, 1.08 (0.98–1.19), and 2.17 (1.47–3.20) for peripheral artery disease (Moon et al. 2012). Exposure at low to moderate levels (<100 ppb) shows mixed results for an effect on CVD and related disease. The WHO recommends a limit of 10 ppb of arsenic in drinking water. Arsenic in drinking water is of most concern in areas where it is present in the mineralogy of an area and leaches into the ground water drinking supply. Well known high contamination cases have occurred in Bangladesh, China, Chile, among others. Anthropogenic activities, such as mining, can also lead to contamination of ground water if not controlled.

A study in Chile of adults who were exposed *in utero* to high levels of arsenic in drinking water (>800 ppb) were at higher risk of bronchiectasis, a chronic obstructive lung disease (Dauphine et al. 2013). Studies in mice have found that exposures *in utero* and in early life around the current and past drinking water standards (10 ppb and 50 ppb, respectively), can induce airway hyper-responsiveness and changes in gene regulation for collagen and elastin, indicating that structural changes occur early on that may lead to lung disease (Lantz et al. 2009). Although high arsenic exposures from water ingestion are only prevalent in certain parts of the world, the role of exposures through dietary ingestion on health outcomes is less well known. Vitamin and mineral deficiencies have been found to decrease methylation capacity for inorganic arsenic, which may therefore increase susceptibility to the toxic effects of arsenic. Deficiencies in protein, folate, iron, zinc, niacin, vitamin E and B<sub>12</sub> have been associated with decreases in arsenic methylation (Gamble et al. 2006; Steinmaus et al. 2005).

Dietary ingestion may also be a significant source of exposure to arsenic. In areas where there are no sources of water or soil/dust contamination, diet is the main source of exposure (Kurzius-Spencer et al. 2014). Some foods naturally have arsenic, such as seafood and some types of seaweed (Moreda-Pineiro et al. 2012; Navas-Acien et al. 2011; Sirot et al. 2009). Seafood tends to be high in arsenobetaine, an organic arsenic species that is thought to be non-toxic. Certain plant species are known to effectively uptake arsenic if it is present in the growing medium (Ramirez-Andreotta et al. 2013a). Rice is a staple crop, grown, exported, and used around the world. In recent years, high levels of arsenic have been found in rice grown in various areas, including Asia and the United States (Adomako et al. 2009; Juhasz et al. 2006; Rahman et al. 2009). Vegetables grown in home gardens in areas where the soil has high arsenic content may also uptake arsenic and can be an additional pathway for arsenic intake for residents in areas where arsenic is naturally high in the soil or where the ground may be contaminated by nearby industry or industrial waste (Ramirez-Andreotta et al. 2013a, b). The role of food-related exposure to arsenic on the development of health effects has not been extensively studied.

Occupational exposure to arsenic mainly occurs in copper or lead smelting industries, those working with arsenic-containing antifungal wood preservatives, anti-fouling paints, pigments, pesticides, glass manufacturing, or coal-fired power plants that use coal with high arsenic content. Occupational air exposure limits are 10 µg/m<sup>3</sup> (US OSHA).

### 2.2.3.5 Diet and Physical Activity

Diet and physical activity are key protective factors against many types of disease. Physical inactivity and poor diet lead to obesity and diabetes, dyslipidemia, and can increase inflammation in the body, which are important risk factors for CVD. Obesity can also affect the development of asthma. A healthy diet and at least moderate activity can be protective against the negative effects of exposure to the environmental risk factors described in this chapter.

Characteristics of a healthy diet include low in saturated fat, low-moderate sodium, and high in fiber, fruit, and vegetables. It appears that focusing on changing only one or a couple of these factors is not enough to protect from CVD development and events, but rather the whole diet needs to be adjusted. The Mediterranean diet has been identified as particularly beneficial for preventing both CVD and asthma (Antó 2012; Dalen and Devries 2014; Garcia-Marcos et al. 2013; Nagel et al. 2010). The Mediterranean diet includes nuts, fruits, vegetables, green leafy vegetables, legumes, whole grain, fish, moderate alcohol, poultry, and olive oil. Many of these food groups have been found to decrease risk of cardiovascular and other diseases. Whole grains, fruit and vegetables contain fiber, vitamins, minerals, phenolic compounds, and other phytochemicals that may support the antioxidant response and reduce inflammation (Bhupathiraju and Tucker 2011; Lock et al. 2005; Tang et al. 2015). A high fiber diet may reduce insulin secretion, thus reducing the development of CVD related risk factors such as diabetes, dyslipidemia, and obesity (Bernstein et al. 2013; Ludwig et al. 1999). Fish, particularly oily fish, are high in omega-3 fatty acids (or *n*-fatty acids), a type of polyunsaturated fatty acid (PUFA) which have been found to be protective against CVD. There is some suggestive evidence that omega-3 fatty acids may also protect against decreased lung function and asthma (Romieu and Trenga 2001). Proposed mechanisms include the prevention of fatal arrhythmias, reduction of blood pressure, and reduction of inflammation (Breslow 2006).

Studies found that reducing total dietary fat was less effective at preventing CVD than reduction in specific types of fats, particularly saturated and *trans*-fatty acids. Reducing total fat intake may be beneficial for reducing serum cholesterol, but not for preventing cardiovascular events or death (Dalen and Devries 2014). On the other hand, mono- and poly-unsaturated fats have been associated with reduced risk of coronary events and deaths (Bhupathiraju and Tucker 2011). In a meta-analysis of 32 cohort studies examining the effects of mono-unsaturated fats on CVD, olive oil consumption showed the most consistent and significant protective effects, compared to total mono-unsaturated fats or the mono-unsaturated fat to saturated fat ratio (Schwingshackl and Hoffmann 2014). Other analyses found that replacing saturated fats with poly-unsaturated fats was better associated with reduced risk of coronary events and deaths than replacement with mono-unsaturated fats (Bhupathiraju and Tucker 2011). Mono-unsaturated fats can also include animal fats, therefore it is possible that plant-derived fats, olive oil in particular, may be more protective, or that olive oil use is a better indicator of a Mediterranean-style diet.

Interestingly, while the vitamins, minerals, beneficial fatty acids found in various foods are thought to be the bioactive compounds that have protective effects, supplementation trials have not shown as clear a benefit as dietary intake, with some trials showing protective effects, some with null results, and even some trials showing harmful effects at high doses of some supplements (Bhupathiraju and Tucker 2011; Myung et al. 2013). Therefore, interventions based on change in whole diet rather than supplementation are likely to be more protective.

Reducing sodium intake is also associated with a reduction in risk of high blood pressure and CVD, although the effect appears to follow a U-shape, where very low intake and high intake have been related to cardiovascular death and hospitalization (O'Donnell et al. 2015). An increase in potassium intake has also been associated with improved cardiovascular outcomes, with reductions in high sodium intake diets plus increase in potassium intake showing benefits.

The nutritional environment of the fetus has also been found to influence risk of disease, including CVD and asthma, in later life. Observations of surveys of men in England born in the early 1900s found that risk of death increased with lower birthweight, head circumference, and ponderal index. Low growth rates and body weights up to the age of 1 year were associated with greater risk of development of CVD risk factors (e.g. blood pressure, plasma glucose and insulin, inflammatory biomarkers) and a higher risk of death from coronary heart disease. (Barker et al. 1993). Animal studies showed that poor fetal nutrition may affect structure and physiology of organs and tissues, including endocrine pancreas, liver, and blood vessels, thus leading to the effects seen later in life. Similar outcomes in terms of fetal nutrition and development of obesity, diabetes, and CVD in adulthood were also observed in children born during the Dutch famine (Painter et al. 2005; Roseboom et al. 2006).

### 2.2.3.6 Phthalates

Phthalates have been found to be asthma and allergy adjuvants, particularly DEHP and longer chain phthalates in *in vivo* studies. *In vitro* tests have found that eight-carbon phthalates, especially DEHP or MEHP induced rapid histamine production in the presence of a co-allergen (Jaakkola and Knight 2008). DEHP's interaction with PPAR receptors can modulate gene expression in tissues, and in mice, appears to increase lung air space and decrease the gas exchange surface (Miller and Marty 2010). The levels of exposure *in vivo* at which these effects were found, however, are much higher than typical human exposures.

Occupational studies where workers are exposed to heated PVC fumes, such as meat wrappers, have found an increased risk of reported asthma symptoms and decreases in lung function metrics (Jaakkola and Knight 2008). Studies of children have found a relationship between PVC in floors and wall materials with bronchial obstruction and wheeze, respiratory symptoms and infections. Dampness seems to increase the degradation of PVC flooring, and this degradation effect has been associated with asthma related symptoms in occupational settings and asthma and

rhinitis in children. These studies, however, did not measure phthalate exposure levels directly, either via environmental or biomarker samples. Several studies in Bulgaria, Sweden, and Taiwan did find an association between DEHP in house dust samples and allergy, asthma, rhinitis, and wheezing.

Exposure to higher molecular weight phthalates, such as DEHP, tend to be via ingestion food or dust, rather than inhalation (Wormuth et al. 2006). Dermal exposure is possible, but more likely to be via application of personal care products or wearing of gloves or textiles containing phthalates. Phthalate exposure is often multi-pathway, and measurement of urinary metabolites is a useful quantification of a marker of total exposure. It can be difficult to determine the contribution of various pathways and routes of exposure in the real world, as dust ingestion rates are highly uncertain for children and adults, and exposure via diet, dermal, and air are not well quantified. The most comprehensive examination of multi-pathway exposures was done by modelling exposures using a scenario based risk assessment approach (Wormuth et al. 2006), which demonstrated the potential contribution of various pathways to exposure for different age groups.

### 2.2.3.7 Cleaning Products

Occupational exposure to cleaning agents and disinfectants has been associated with asthma onset. The European Community Respiratory Health Survey II (ECRHS II) found the most significant risk of asthma onset in occupational settings was with use of cleaning products, particularly for nurses (Kogevinas et al. 2007). In Finland, a case-control study found a 42% increase in risk for cleaning women compared to those in administrative types of jobs (Jaakkola and Jaakkola 2006; Zock et al. 2007). Asthma onset was also linked to use of spray cleaning products, including air fresheners, glass cleaners, and furniture cleaners, at least once a week in the general population. This risk was higher with use of multiple products. Quaternary ammonium compounds have been identified as sensitizers, and have been most strongly associated with occupational asthma, particularly in hospital settings (Heederik 2014).

Cleaning products contain a number of potentially irritating or sensitizing chemicals, which, when applied, lead to exposure, generally through inhalation, which would be the route of most relevance for asthma (Nazaroff and Weschler 2004). Another way in which cleaning products can lead to exposures to pollutants is via secondary reactions. Unsaturated organic compounds such as terpenes can form volatile organic compounds that may affect asthma in the presence of reactive substances such as ozone and the hydroxyl radical, or surfaces of walls or furniture (Nazaroff and Weschler 2004; Singer et al. 2006). Terpenes, for example, react with ozone and various surfaces to produce formaldehyde and other potentially airway irritating chemicals. Secondary organic aerosols can also form from reactions of volatile compounds released by cleaning products. These aerosols tend to be less than 2.5  $\mu\text{m}$  in diameter and can persist at high levels for several hours post-production (Singer et al. 2006).

### 2.2.3.8 Dampness and Mould

In Europe, the proportion of the population living in homes with self-assessed problems of damp ranges from 10 to 50 % in the period of time between 1995 and 2001 (Heinrich 2011; WHO guidelines for indoor air quality n.d.). Dampness has been related to respiratory symptoms in various studies, although the means by which dampness has been measured has been quite variable (Kennedy and Grimes 2013). A meta-analysis of people living in damp or mouldy homes found a 30–50 % increase in risk of asthma (Fisk et al. 2007). In a meta-analysis of 8 European cohorts, exposure to dampness/mould at home in first 2 years of life resulted in an adjusted OR of early asthma symptoms of 1.39 (1.06–1.84) (Tischer et al. 2011).

Many studies use qualitative methods of assessing dampness and mould, such as questionnaire, visual assessment by trained assessor, dampness or mould index, or mould odour, especially near skirting of boards, which is area where moisture is most likely to accumulate over time and promote growth of micro-organisms, insects, rodents, release of chemicals from damp materials. Temperature and relative humidity have also been used as measures of indoor dampness. In recent times, however, quantitative measures of dampness and mould did not necessarily find any associations with health outcomes (Kennedy and Grimes 2013). Duration and period of exposure may also determine the health response (Heinrich 2011; Kennedy and Grimes 2013). Finally, it is difficult to know what particular hazard(s) from dampness is the explanatory variable, as many biological and chemical agents are released due to dampness or water damage.

### 2.2.3.9 Allergens

Allergic asthma phenotypes may be the largest group of asthma phenotypes, especially in childhood (Wenzel 2006). Common environmental triggers in homes include dust mite, cockroach, cat (and other pets) and mouse (Ahluwalia and Matsui 2011). Grass and tree pollens can also be triggers. Many biological and chemical agents have been demonstrated to be sensitizers in the workplace, leading to occupational asthma (List of substances that can cause occupational asthma—HSE n.d.). Removal of exposure usually stops symptoms, although for non-occupational triggers, it may be difficult to completely remove the sensitizing agent from an individual's environment. As asthmatics may be sensitized to more than one allergen, cleaning and pest management solutions in homes need to be multi-pronged and sustained in order to have an effect (Morgan et al. 2004).

### 2.2.3.10 Other Exposure Factors

Exposures are a product of environmental sources, conditions, and people's locations and activities. Reduction of exposures by modifying any of these factors improves health by improving symptoms and reducing risk of developing disease. Additionally, the “general external” sphere of the exposome has an impact on the “specific external”

factors described in this chapter. Improvements in “general external” factors not only directly impact health, but also affect “specific external” factors. For example, reduction of psychosocial stress can reduce the body’s general inflammation and also reduce risk of asthma exacerbations or heart attacks. Improvement of one’s home environment can not only reduce stress, but also reduce exposures to various pollutants.

Most exposure occurs indoors, and this consists of pollutants generated indoors and those that infiltrate indoors. Building materials, tightness, and ventilation, products used inside, and people’s activities are all predictors of exposure. Indoor microenvironments (including both buildings and vehicles) are sources of multiple chemicals and other environmental agents. Not including work, indoor exposures occur primarily at home. Time spent in other establishments, such as stores and restaurants, is generally of low duration and frequency (Loh et al. 2008). The indoor environment can be an important modifier of air pollutant exposures and related health effects. Air cleaners have been shown to be an effective reducer of some indoor pollutants (Barn et al. 2008) and the use of air conditioning, which tends to indicate newer buildings and lower infiltration of outdoor pollutants, was associated with a reduction in hospital admissions related to particulate matter (Janssen et al. 2002). The quality of the indoor environment can also have an effect. For example, poor housing stock can mean poor insulation, thus leading to poor indoor climate conditions such as low temperature and drafts, greater infiltration from air pollutants, noise, and dampness. Conversely, an overly well-insulated but poorly ventilated building can have a build-up of pollutants indoors and humidity, leading also to dampness indoors.

In transit exposures typically consist of pollution entering the vehicle from outdoors, or self-pollution from the vehicle. Although time spent in-transit is not necessarily a large part of a person’s day (~6%), this microenvironment may contribute a much larger amount to a person’s exposure than the percentage of time spent in transit (~24%) (de Nazelle et al. 2013). Mode of transport not only modifies a person’s air pollution exposure (McNabola et al. 2008; Zuurbier et al. 2010), but can also be a source of physical activity, thus reducing risk of obesity, an asthma and CVD risk factor.

## 2.3 Conclusion

Two of the most common types of diseases world-wide, cardiovascular diseases and asthma, are a complex interplay of genetics and environment. The environmental aspect of disease is itself a complex set of factors. Thinking holistically about disease using the concept of the exposome, developing methods for analysing the contribution of multiple factors, and understanding individuals’ circumstances broadly, can help us better intervene to reduce disease risk. This chapter has addressed several aspects of the “specific external” sphere of the exposome, as originally defined by Wild (2012). Although it is not an exhaustive review of environmental causes of cardiovascular disease and asthma, it already shows how many factors contribute to the development and manifestation of disease.

**Acknowledgments** This work has received funding from the European Union Seventh Programme for research, technological development, and demonstration under grant agreement No 603946.

## References

- Adomako EE, Solaiman AR, Williams PN, Deacon C, Rahman GK, Meharg AA (2009) Enhanced transfer of arsenic to grain for Bangladesh grown rice compared to US and EU. *Environ Int* 35:476–479. doi:[10.1016/j.envint.2008.07.010](https://doi.org/10.1016/j.envint.2008.07.010)
- Ahluwalia SK, Matsui EC (2011) The indoor environment and its effects on childhood asthma. *Curr Opin Allergy Clin Immunol* 11:137–143. doi:[10.1097/ACI.0b013e3283445921](https://doi.org/10.1097/ACI.0b013e3283445921)
- Antó JM (2012) Recent advances in the epidemiologic investigation of risk factors for asthma: a review of the 2011 literature. *Curr Allergy Asthma Rep* 12:192–200. doi:[10.1007/s11882-012-0254-7](https://doi.org/10.1007/s11882-012-0254-7)
- Babisch W (2011) Cardiovascular effects of noise. *Noise Health* 13:201
- Ballester F, Estarlich M, Iñiguez C, Llop S, Ramón R, Esplugues A, Lacasaña M, Rebagliato M (2010) Air pollution exposure during pregnancy and reduced birth size: a prospective birth cohort study in Valencia, Spain. *Environ Health* 9:6. doi:[10.1186/1476-069X-9-6](https://doi.org/10.1186/1476-069X-9-6)
- Barker DJP, Godfrey KM, Gluckman PD, Harding JE, Owens JA, Robinson JS (1993) Fetal nutrition and cardiovascular disease in adult life. *Lancet* 1(8850):341, 938–941. doi:[10.1016/0140-6736\(93\)91224-A](https://doi.org/10.1016/0140-6736(93)91224-A)
- Barn P, Larson T, Noullett M, Kennedy S, Copes R, Brauer M (2008) Infiltration of forest fire and residential wood smoke: an evaluation of air cleaner effectiveness. *J Expo Sci Environ Epidemiol* 18:503–511. doi:[10.1038/sj.jes.7500640](https://doi.org/10.1038/sj.jes.7500640)
- Barnoya J, Glantz SA (2005) Cardiovascular effects of secondhand smoke nearly as large as Smoking. *Circulation* 111:2684–2698. doi:[10.1161/CIRCULATIONAHA.104.492215](https://doi.org/10.1161/CIRCULATIONAHA.104.492215)
- Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, Stansfeld S (2014) Auditory and non-auditory effects of noise on health. *Lancet* 383:1325–1332. doi:[10.1016/S0140-6736\(13\)61613-X](https://doi.org/10.1016/S0140-6736(13)61613-X)
- Bernstein AM, Titgemeier B, Kirkpatrick K, Golubic M, Roizen MF (2013) Major cereal grain fibers and psyllium in relation to cardiovascular health. *Nutrients* 5:1471–1487. doi:[10.3390/nu5051471](https://doi.org/10.3390/nu5051471)
- Bhatnagar A (2006) Environmental cardiology studying mechanistic links between pollution and heart disease. *Circ Res* 99:692–705. doi:[10.1161/01.RES.0000243586.99701.cf](https://doi.org/10.1161/01.RES.0000243586.99701.cf)
- Bhupathiraju SN, Tucker KL (2011) Coronary heart disease prevention: nutrients, foods, and dietary patterns. *Clin Chim Acta* 412:1493–1514. doi:[10.1016/j.cca.2011.04.038](https://doi.org/10.1016/j.cca.2011.04.038)
- Bousquet J, Jeffery PK, Busse WW, Johnson M, Vignola AM (2000) Asthma. *Am J Respir Crit Care Med* 161:1720–1745. doi:[10.1164/ajrccm.161.5.9903102](https://doi.org/10.1164/ajrccm.161.5.9903102)
- Bowatte G, Lodge C, Lowe AJ, Erbas B, Perret J, Abramson MJ, Matheson M, Dharmage SC (2015) The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies. *Allergy* 70:245–256. doi:[10.1111/all.12561](https://doi.org/10.1111/all.12561)
- Breslow JL (2006) n–3 Fatty acids and cardiovascular disease. *Am J Clin Nutr* 83:S1477–S1482
- Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC, Tager I (2004) Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation* 109:2655–2671. doi:[10.1161/01.CIR.0000128587.30041.C8](https://doi.org/10.1161/01.CIR.0000128587.30041.C8)
- Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC, Whitsel L, Kaufman JD (2010) Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 121:2331–2378. doi:[10.1161/CIR.0b013e3181d8bec1](https://doi.org/10.1161/CIR.0b013e3181d8bec1)



- Callinan JE, Clarke A, Doherty K, Kelleher C (2010) Legislative smoking bans for reducing secondhand smoke exposure, smoking prevalence and tobacco consumption. *Cochrane Database Syst Rev*. Wiley.
- Cesaroni G, Forastiere F, Agabiti N, Valente P, Zuccaro P, Perucci CA (2008) Effect of the Italian smoking ban on population rates of acute coronary events. *Circulation* 117:1183–1188. doi:[10.1161/CIRCULATIONAHA.107.729889](https://doi.org/10.1161/CIRCULATIONAHA.107.729889)
- D'Agostino RB Sr, Grundy S, Sullivan LM, Wilson P (2001) Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *JAMA* 286:180–187
- Dalen JE, Devries S (2014) Diets to prevent coronary heart disease 1957–2013: what have we learned? *Am J Med* 127:364–369. doi:[10.1016/j.amjmed.2013.12.014](https://doi.org/10.1016/j.amjmed.2013.12.014)
- Dauphine DC, Smith AH, Yuan Y, Balmes JR, Bates MN, Steinmaus C (2013) Case–control study of arsenic in drinking water and lung cancer in California and Nevada. *Int J Environ Res Public Health* 10:3310–3324. doi:[10.3390/ijerph10083310](https://doi.org/10.3390/ijerph10083310)
- Davies H, Kamp I (2012) Noise and cardiovascular disease: a review of the literature 2008–2011. *Noise Health* 14:287. doi:[10.4103/1463-1741.104895](https://doi.org/10.4103/1463-1741.104895)
- de Nazelle A, Seto E, Donaire-Gonzalez D, Mendez M, Matamala J, Nieuwenhuijsen MJ, Jerrett M (2013) Improving estimates of air pollution exposure through ubiquitous sensing technologies. *Environ Pollut* 176:92–99. doi:[10.1016/j.envpol.2012.12.032](https://doi.org/10.1016/j.envpol.2012.12.032)
- EFSA (European Food Safety Authority) (2012) Cadmium dietary exposure in the European population. *EFSA J* 10:2551
- Fisk WJ, Lei-Gomez Q, Mendell MJ (2007) Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. *Indoor Air* 17:284–296
- Gamble MV, Liu X, Ahsan H, Pilsner JR, Ilievski V, Slavkovich V, Parvez F, Chen Y, Levy D, Factor-Litvak P, Graziano JH (2006) Folate and arsenic metabolism: a double-blind, placebo-controlled folic acid-supplementation trial in Bangladesh. *Am J Clin Nutr* 84:1093–1101
- Garcia-Marcos L, Castro-Rodriguez JA, Weinmayr G, Panagiotakos DB, Priftis KN, Nagel G (2013) Influence of Mediterranean diet on asthma in children: a systematic review and meta-analysis. *Pediatr Allergy Immunol* 24:330–338. doi:[10.1111/pai.12071](https://doi.org/10.1111/pai.12071)
- Goodman P, Agnew M, McCaffrey M, Paul G, Clancy L (2007) Effects of the Irish smoking ban on respiratory health of bar workers and air quality in Dublin pubs. *Am J Respir Crit Care Med* 175:840–845. doi:[10.1164/rccm.200608-1085OC](https://doi.org/10.1164/rccm.200608-1085OC)
- Gowers AM, Cullinan P, Ayres JG, Anderson HR, Strachan DP, Holgate ST, Mills IC, Maynard RL (2012) Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. *Respirology* 17:887–898. doi:[10.1111/j.1440-1843.2012.02195.x](https://doi.org/10.1111/j.1440-1843.2012.02195.x)
- Gulson BL, Howarth D, Mizon KJ, Law AJ, Korsch MJ, Davis JJ (1994) Source of lead in humans from Broken Hill mining community. *Environ Geochem Health* 16:19–25. doi:[10.1007/BF00149589](https://doi.org/10.1007/BF00149589)
- Heederik D (2014) Cleaning agents and disinfectants: moving from recognition to action and prevention. *Clin Exp Allergy* 44:472–474. doi:[10.1111/cea.12286](https://doi.org/10.1111/cea.12286)
- Heinrich J (2011) Influence of indoor factors in dwellings on the development of childhood asthma. *Int J Hyg Environ Health* 214:1–25. doi:[10.1016/j.ijheh.2010.08.009](https://doi.org/10.1016/j.ijheh.2010.08.009)
- HEI Review Panel on Ultrafine Particles (2013) HEI perspectives 3: understanding the health effects of ambient ultrafine particles
- Hsu H-HL, Chiu Y-HM, Coull BA, Kloog I, Schwartz J, Lee A, Wright RO, Wright RJ (2015) Prenatal particulate air pollution and asthma onset in urban children: identifying sensitive windows and sex differences. *Am J Respir Crit Care Med*. doi:[10.1164/rccm.201504-0658OC](https://doi.org/10.1164/rccm.201504-0658OC)
- Hu H, Aro A, Payton M et al (1996) The relationship of bone and blood lead to hypertension: the normative aging study. *JAMA* 275:1171–1176. doi:[10.1001/jama.1996.03530390037031](https://doi.org/10.1001/jama.1996.03530390037031)
- Jaakkola JJK, Jaakkola MS (2006) Professional cleaning and asthma. *Curr Opin Allergy Clin Immunol* 6:85–90. doi:[10.1097/01.all.00000216849.64828.55](https://doi.org/10.1097/01.all.00000216849.64828.55)
- Jaakkola JJK, Knight TL (2008) The role of exposure to phthalates from polyvinyl chloride products in the development of asthma and allergies: a systematic review and meta-analysis. *Environ Health Perspect* 116:845–853. doi:[10.1289/ehp.10846](https://doi.org/10.1289/ehp.10846)
- Jacquemin B, Siroux V, Sanchez M, Carsin A-E, Schikowski T, Adam M, Bellisario V, Buschka A, Bono R, Brunekreef B, Cai Y, Cirach M, Clavel-Chapelon F, Declercq C, de Marco R, de

- Nazelle A, Ducret-Stich RE, Ferreti VV, Gerbase M, Hardy R, Heinrich J, Janson C, Jarvis D, Kanaani ZA, Keidel D, Kuh D, Le Moual N, Nieuwenhuijsen MJ, Marcon A, Modig L, Pin I, Rochat T, Schindler C, Sugiri D, Stempfelet M, Temam S, Tsai M-Y, Varraso R, Vienneau D, Vierkötter A, Hansell AL, Krämer U, Probst-Hensch NM, Sunyer J, Künzli N, Kauffmann F (2015) Ambient air pollution and adult asthma incidence in six European cohorts (ESCAPE). *Environ Health Perspect*. doi:[10.1289/ehp.1408206](https://doi.org/10.1289/ehp.1408206)
- Janssen NAH, Schwartz J, Zanobetti A, Suh HH (2002) Air conditioning and source-specific particles as modifiers of the effect of PM10 on hospital admissions for heart and lung disease. *Environ Health Perspect* 110:43–49
- Juhász AL, Smith E, Weber J, Rees M, Rofe A, Kuchel T, Sansom L, Naidu R (2006) In vivo assessment of arsenic bioavailability in rice and its significance for human health risk assessment. *Environ Health Perspect* 114:1826–1831
- Kennedy K, Grimes C (2013) Indoor water and dampness and the health effects on children: a review. *Curr Allergy Asthma Rep* 13:672–680. doi:[10.1007/s11882-013-0393-5](https://doi.org/10.1007/s11882-013-0393-5)
- Kleipeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P, Behar JV, Hern SC, Engelmann WH (2001) The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *J Expo Anal Environ Epidemiol* 11:231–252
- Kogevinas M, Zock JP, Jarvis D, Kromhout H, Lillienberg L, Plana E, Radon K, Toren K, Alliksoo A, Benke G, Blanc PD, Dahlman-Hoglund A, D'Errico A, Hery M, Kennedy S, Kunzli N, Leynaert B, Mirabelli MC, Muniozguen N, Norback D, Olivieri M, Payo F, Villani S, van Sprundel M, Urrutia I, Wieslander G, Sunyer J, Anto JM (2007) Exposure to substances in the workplace and new-onset asthma: an international prospective population-based study (ECRHS-II). *Lancet* 370:336–341. doi:[10.1016/S0140-6736\(07\)61164-7](https://doi.org/10.1016/S0140-6736(07)61164-7)
- Koren HS, Devlin RB, Graham DE, Mann R, McGee MP, Horstman DH, Kozumbo WJ, Becker S, House DE, McDonnell WF, Bromberg PA (1989) Ozone-induced inflammation in the lower airways of human subjects. *Am Rev Respir Dis* 139:407–415. doi:[10.1164/ajrccm/139.2.407](https://doi.org/10.1164/ajrccm/139.2.407)
- Kurzius-Spencer M, Burgess JL, Harris RB, Hartz V, Roberge J, Huang S, Hsu C-H, O'Rourke MK (2014) Contribution of diet to aggregate arsenic exposures—an analysis across populations. *J Expo Sci Environ Epidemiol* 24:156–162. doi:[10.1038/jes.2013.37](https://doi.org/10.1038/jes.2013.37)
- Langrish JP, Lundbäck M, Barath S, Söderberg S, Mills NL, Newby DE, Sandström T, Blomberg A (2010) Exposure to nitrogen dioxide is not associated with vascular dysfunction in man. *Inhal Toxicol* 22:192–198. doi:[10.3109/08958370903144105](https://doi.org/10.3109/08958370903144105)
- Lantz RC, Chau B, Sarihan P, Witten ML, Pivniouk VI, Chen GJ (2009) In utero and postnatal exposure to arsenic alters pulmonary structure and function. *Toxicol Appl Pharmacol* 235:105–113. doi:[10.1016/j.taap.2008.11.012](https://doi.org/10.1016/j.taap.2008.11.012)
- List of substances that can cause occupational asthma—HSE (n.d.) <http://www.hse.gov.uk/asthma/substances.htm>. Accessed 20 Sept 2015
- Lock K, Pomerleau J, Causer L, Altmann DR, McKee M (2005) The global burden of disease attributable to low consumption of fruit and vegetables: implications for the global strategy on diet. *Bull World Health Organ* 83:100–108. doi:[10.1590/S0042-96862005000200010](https://doi.org/10.1590/S0042-96862005000200010)
- Loh MM, Houseman EA, Gray GM, Levy JI, Spengler JD, Bennett DH (2006) Measured concentrations of VOCs in several non-residential microenvironments in the United States. *Environ Sci Technol* 40:6903–6911
- Loh MM, Houseman EA, Levy JI, Spengler JD, Bennett DH (2008) Contribution to volatile organic compound exposures from time spent in stores and restaurants and bars. *J Expo Sci Environ Epidemiol*
- Lötvall J, Akdis CA, Bacharier LB, Bjermer L, Casale TB, Custovic A, Lemanske RF Jr, Wardlaw AJ, Wenzel SE, Greenberger PA (2011) Asthma endotypes: a new approach to classification of disease entities within the asthma syndrome. *J Allergy Clin Immunol* 127:355–360. doi:[10.1016/j.jaci.2010.11.037](https://doi.org/10.1016/j.jaci.2010.11.037)
- Lubitz S, Schober W, Pusch G, Effner R, Klopp N, Behrendt H, Buters JTM (2010) Polycyclic aromatic hydrocarbons from diesel emissions exert proallergic effects in birch pollen allergic individuals through enhanced mediator release from basophils. *Environ Toxicol* 25:188–197. doi:[10.1002/tox.20490](https://doi.org/10.1002/tox.20490)

- Lucking AJ, Lundback M, Mills NL, Faratian D, Barath SL, Pourazar J, Cassee FR, Donaldson K, Boon NA, Badimon JJ, Sandstrom T, Blomberg A, Newby DE (2008) Diesel exhaust inhalation increases thrombus formation in man. *Eur Heart J*. doi:[10.1093/eurheartj/ehn464](https://doi.org/10.1093/eurheartj/ehn464)
- Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattey ML, Jacobs DR (1999) Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. *JAMA* 282:1539–1546
- Lundbäck M, Mills NL, Lucking A, Barath S, Donaldson K, Newby DE, Sandström T, Blomberg A (2009) Experimental exposure to diesel exhaust increases arterial stiffness in man. *Part Fibre Toxicol* 6:7. doi:[10.1186/1743-8977-6-7](https://doi.org/10.1186/1743-8977-6-7)
- McNabola A, Broderick BM, Gill LW (2008) Relative exposure to fine particulate matter and VOCs between transport microenvironments in Dublin: Personal exposure and uptake. *Atmos Environ* 42:6496–6512. doi:[10.1016/j.atmosenv.2008.04.015](https://doi.org/10.1016/j.atmosenv.2008.04.015)
- Middleton N, Yiallourou P, Kleanthous S, Kolokotroni O, Schwartz J, Dockery DW, Demokritou P, Koutrakis P (2008) A 10-year time-series analysis of respiratory and cardiovascular morbidity in Nicosia, Cyprus: the effect of short-term changes in air pollution and dust storms. *Environ Health* 7. doi:[10.1186/1476-069x-7-39](https://doi.org/10.1186/1476-069x-7-39)
- Miller MD, Marty MA (2010) Impact of environmental chemicals on lung development. *Environ Health Perspect* 118:1155–1164. doi:[10.1289/ehp.0901856](https://doi.org/10.1289/ehp.0901856)
- Miller MR, Shaw CA, Langrish JP (2012) From particles to patients: oxidative stress and the cardiovascular effects of air pollution. *Future Cardiol* 8:577–602. doi:[10.2217/fca.12.43](https://doi.org/10.2217/fca.12.43)
- Moon K, Guallar E, Navas-Acien A (2012) Arsenic exposure and cardiovascular disease: an updated systematic review. *Curr Atheroscler Rep* 14:542–555. doi:[10.1007/s11883-012-0280-x](https://doi.org/10.1007/s11883-012-0280-x)
- Moreda-Pineiro J, Alonso-Rodriguez E, Romaris-Hortas V, Moreda-Pineiro A, Lopez-Mahia P, Muniategui-Lorenzo S, Prada-Rodriguez D, Bermejo-Barrera P (2012) Assessment of the bioavailability of toxic and non-toxic arsenic species in seafood samples. *Food Chem* 130:552–560. doi:[10.1016/j.foodchem.2011.07.071](https://doi.org/10.1016/j.foodchem.2011.07.071)
- Morgan WJ, Crain EF, Gruchalla RS, O'Connor GT, Kattan M, Evans R, Stout J, Malindzak G, Smartt E, Plaut M, Walter M, Vaughn B, Mitchell H (2004) Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med* 351:1068–1080. doi:[10.1056/NEJMoa032097](https://doi.org/10.1056/NEJMoa032097)
- Münzel T, Gori T, Babich W, Basner M (2014) Cardiovascular effects of environmental noise exposure. *Eur Heart J* 35:829–836. doi:[10.1093/eurheartj/ehu030](https://doi.org/10.1093/eurheartj/ehu030)
- Myung S-K, Ju W, Cho B, Oh S-W, Park SM, Koo B-K, Park B-J (2013) Efficacy of vitamin and antioxidant supplements in prevention of cardiovascular disease: systematic review and meta-analysis of randomised controlled trials. *BMJ* 346:f10. doi:[10.1136/bmj.f10](https://doi.org/10.1136/bmj.f10)
- Nagel G, Weinmayr G, Kleiner A, Garcia-Marcos L, Strachan DP, The IPTS Group (2010) Effect of diet on asthma and allergic sensitisation in the International Study on Allergies and Asthma in Childhood (ISAAC) phase two. *Thorax* 65:516–522. doi:[10.1136/thx.2009.128256](https://doi.org/10.1136/thx.2009.128256)
- Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ (2007) Lead exposure and cardiovascular disease: a systematic review. *Environ Health Perspect* 115:472–482
- Navas-Acien A, Francesconi KA, Silbergeld EK, Guallar E (2011) Seafood intake and urine concentrations of total arsenic, dimethylarsinate and arsenobetaine in the US population. *Environ Res* 111:110–108. doi:[10.1016/j.envres.2010.10.009](https://doi.org/10.1016/j.envres.2010.10.009)
- Nazaroff WW, Singer BC (2004) Inhalation of hazardous air pollutants from environmental tobacco smoke in US residences. *J Expo Anal Environ Epidemiol* 14:S71–S77
- Nazaroff WW, Weschler CJ (2004) Cleaning products and air fresheners: exposure to primary and secondary air pollutants. *Atmos Environ* 38:2841–2865. doi:[10.1016/j.atmosenv.2004.02.040](https://doi.org/10.1016/j.atmosenv.2004.02.040)
- O'Donnell M, Mente A, Yusuf S (2015) Sodium intake and cardiovascular health. *Circ Res* 116:1046–1057. doi:[10.1161/CIRCRESAHA.116.303771](https://doi.org/10.1161/CIRCRESAHA.116.303771)
- Öberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A (2011) Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet* 377:139–146. doi:[10.1016/S0140-6736\(10\)61388-8](https://doi.org/10.1016/S0140-6736(10)61388-8)

- Painter RC, Roseboom TJ, Bleker OP (2005) Prenatal exposure to the Dutch famine and disease in later life: an overview. *Reprod Toxicol* 20:345–352. doi:10.1016/j.reprotox.2005.04.005
- Parnia S, Brown JL, Frew AJ (2002) The role of pollutants in allergic sensitization and the development of asthma. *Allergy* 57:1111–1117. doi:10.1034/j.1398-9995.2002.02167.x
- Perez L, Tobias A, Querol X, Kunzli N, Pey J, Alastuey A, Viana M, Valero N, Gonzalez-Cabre M, Sunyer J (2008) Coarse particles from Saharan dust and daily mortality. *Epidemiology* 19:800–807. doi:10.1097/Ede.0b013e31818131cf
- Pope CA 3rd, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ (2009) Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure-response relationship. *Circulation* 120:941–948. doi:10.1161/CIRCULATIONAHA.109.857888
- Rahman MM, Owens G, Naidu R (2009) Arsenic levels in rice grain and assessment of daily dietary intake of arsenic from rice in arsenic-contaminated regions of Bangladesh—implications to ground-water irrigation. *Environ Geochem Health* 31(Suppl 1):179–187. doi:10.1007/s10653-008-9238-x
- Ramirez-Andreotta MD, Brusseau ML, Artiola JF, Maier RM (2013a) A greenhouse and field-based study to determine the accumulation of arsenic in common homegrown vegetables grown in mining-affected soils. *Sci Total Environ* 443:299–306. doi:10.1016/j.scitotenv.2012.10.095
- Ramirez-Andreotta MD, Brusseau ML, Beamer P, Maier RM (2013b) Home gardening near a mining site in an arsenic-endemic region of Arizona: assessing arsenic exposure dose and risk via ingestion of home garden vegetables, soils, and water. *Sci Total Environ* 454:373–382. doi:10.1016/j.scitotenv.2013.02.063
- Review of evidence on health aspects of air pollution—REVIHAAP project (n.d.) Final technical report. <http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/review-of-evidence-on-health-aspects-of-air-pollution-revihaap-project-final-technical-report>. Accessed 12 Sept 2015
- Romieu I, Trenga C (2001) Diet and obstructive lung diseases. *Epidemiol Rev* 23:268–287
- Roseboom T, de Rooij S, Painter R (2006) The Dutch famine and its long-term consequences for adult health. *Early Hum Dev* 82:485–491. doi:10.1016/j.earlhumdev.2006.07.001
- Schweizer C, Edwards RD, Bayer-Oglesby L, Gauderman WJ, Ilacqua V, Jantunen MJ, Lai HK, Nieuwenhuijsen M, Kunzli N (2007) Indoor time-microenvironment-activity patterns in seven regions of Europe. *J Expo Sci Environ Epidemiol* 17:170–181
- Schwingshackl L, Hoffmann G (2014) Monounsaturated fatty acids, olive oil and health status: a systematic review and meta-analysis of cohort studies. *Lipids Health Dis* 13:154. doi:10.1186/1476-511X-13-154
- Semple S, Garden C, Coggins M, Galea KS, Whelan P, Cowie H, Sánchez-Jiménez A, Thorne PS, Hurley JF, Ayres JG (2012) Contribution of solid fuel, gas combustion, or tobacco smoke to indoor air pollutant concentrations in Irish and Scottish homes. *Indoor Air* 22:212–223. doi:10.1111/j.1600-0668.2011.00755.x
- Sims M, Maxwell R, Bauld L, Gilmore A (2010) Short term impact of smoke-free legislation in England: retrospective analysis of hospital admissions for myocardial infarction. *Br Med J* 340. doi:10.1136/Bmj.C2161
- Singer BC, Coleman BK, Destailhats H, Hodgson AT, Lunden MM, Weschler CJ, Nazaroff WW (2006) Indoor secondary pollutants from cleaning product and air freshener use in the presence of ozone. *Atmos Environ* 40:6696–6710
- Sirot V, Guérin T, Volatier J-L, Leblanc J-C (2009) Dietary exposure and biomarkers of arsenic in consumers of fish and shellfish from France. *Sci Total Environ* 407:1875–1885. doi:10.1016/j.scitotenv.2008.11.050
- Solenkova NV, Newman JD, Berger JS, Thurston G, Hochman JS, Lamas GA (2014) Metal pollutants and cardiovascular disease: mechanisms and consequences of exposure. *Am Heart J* 168:812–822. doi:10.1016/j.ahj.2014.07.007
- Šrám RJ, Binková B, Dejmeš J, Bobak M (2005) Ambient air pollution and pregnancy outcomes: a review of the literature. *Environ Health Perspect* 113:375–382
- Steinmaus C, Carrigan K, Kalman D, Atallah R, Yuan Y, Smith AH (2005) Dietary intake and arsenic methylation in a U.S. population. *Environ Health Perspect* 113:1153–1159

- Takahashi G, Tanaka H, Wakahara K, Nasu R, Hashimoto M, Miyoshi K, Takano H, Yamashita H, Inagaki N, Nagai H (2010) Effect of diesel exhaust particles on house dust mite-induced airway eosinophilic inflammation and remodeling in mice. *J Pharmacol Sci* 112:192–202
- Tang G, Wang D, Long J, Yang F, Si L (2015) Meta-analysis of the association between whole grain intake and coronary heart disease risk. *Am J Cardiol* 115:625–629. doi:10.1016/j.amjcard.2014.12.015
- Thomas D (2010) Gene–environment-wide association studies: emerging approaches. *Nat Rev Genet* 11:259–272. doi:10.1038/nrg2764
- Tischer CG, Hohmann C, Thiering E, Herbarth O, Müller A, Henderson J, Granell R, Fantini MP, Luciano L, Bergström A, Kull I, Link E, von Berg A, Kuehni CE, Strippoli M-PF, Gehring U, Wijga A, Eller E, Bindslev-Jensen C, Keil T, Heinrich J, As Part of the ENRIECO Consortium (2011) Meta-analysis of mould and dampness exposure on asthma and allergy in eight European birth cohorts: an ENRIECO initiative. *Allergy* 66:1570–1579. doi:10.1111/j.1398-9995.2011.02712.x
- Törnqvist H, Mills NL, Gonzalez M, Miller MR, Robinson SD, Megson IL, MacNee W, Donaldson K, Söderberg S, Newby DE, Sandström T, Blomberg A (2007) Persistent endothelial dysfunction in humans after diesel exhaust inhalation. *Am J Respir Crit Care Med* 176:395–400. doi:10.1164/rccm.200606-872OC
- Vaziri ND (2008) Mechanisms of lead-induced hypertension and cardiovascular disease. *Am J Phys Heart Circ Phys* 295:H454–H465. doi:10.1152/ajpheart.00158.2008
- Waring MS, Siegel JA (2007) An evaluation of the indoor air quality in bars before and after a smoking ban in Austin, Texas. *J Expo Sci Environ Epidemiol* 17:260–268. doi:10.1038/sj.jes.7500513
- Weldy CS, Liu Y, Liggitt HD, Chin MT (2014) In utero exposure to diesel exhaust air pollution promotes adverse intrauterine conditions, resulting in weight gain, altered blood pressure, and increased susceptibility to heart failure in adult mice. *PLoS One* 9, e88582. doi:10.1371/journal.pone.0088582
- Wenzel SE (2006) Asthma: defining of the persistent adult phenotypes. *Lancet* 368:804–813. doi:10.1016/S0140-6736(06)69290-8
- WHO (2009) Night noise guidelines for Europe. <http://www.euro.who.int/en/health-topics/environment-and-health/noise/publications/2009/night-noise-guidelines-for-europe>. Accessed 11 Aug 2015
- Wild CP (2012) The exposome: from concept to utility. *Int J Epidemiol* 41:24–32. doi:10.1093/ije/dyr236
- Wilson D, Esterman A, Lewis M, Roder D, Calder I (1986) Children’s blood lead levels in the lead smelting town of Port Pirie, South Australia. *Arch Environ Health Int J* 41:245–250. doi:10.1080/00039896.1986.9938340
- Wormuth M, Scheringer M, Vollenweider M, Hungerbühler K (2006) What are the sources of exposure to eight frequently used phthalic acid esters in Europeans? *Risk Anal* 26:803–824. doi:10.1111/j.1539-6924.2006.00770.x
- Yusuf S, Reddy S, Ôunpuu S, Anand S (2001) Global burden of cardiovascular diseases. Part I: General considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation* 104:2746–2753. doi:10.1161/hc4601.099487
- Zock JP, Plana E, Jarvis D, Anto JM, Kromhout H, Kennedy SM, Kunzli N, Villani S, Olivieri M, Toren K, Radon K, Sunyer J, Dahlman-Hoglund A, Norback D, Kogevinas M (2007) The use of household cleaning sprays and adult asthma: an international longitudinal study. *Am J Respir Crit Care Med* 176:735–741. doi:10.1164/rccm.200612-1793OC
- Zuurbier M, Hoek G, Oldenwening M, Lenters V, Meliefste K, van den Hazel P, Brunekreef B (2010) Commuters’ exposure to particulate matter air pollution is affected by mode of transport, fuel type, and route. *Environ Health Perspect* 118:783–789. doi:10.1289/ehp.0901622

## Chapter 3

# Human Exposure to Pollutants and Their Health Endpoints: The Arctic Perspective

Jon Øyvind Odland and Shawn Donaldson

**Abstract** Studies of longitudinal design are ongoing in all Arctic countries, as well as many countries in the southern hemisphere. For both ethical and scientific reasons many studies have a cohort design, with a long term follow up of mothers and their respective children.

The most vulnerable period in a human life is before you are born and in early childhood. A number of reports and publications are now coming out of these studies and this chapter will go into details of some of the most important studies and their defined health endpoints. Different epidemiological studies in the circumpolar area have shown associations between contaminants and different health outcomes. It is important to note that associations are not the same as causal relationships between exposure to a single substance (or a mix of substances) and an effect. When finding an association between a contaminant and an effect it is important to bear in mind the possibility that the studied substance are proxies for other substances in the mixture of contaminants to which the study population has been exposed—both harmful and beneficial. The effects reported are findings from different circumpolar communities. However, due to differences in genetic composition, socio-cultural practices, local food consumption patterns and exposure mixtures, a finding in one population should not be extrapolated to another population without careful investigation and comparative information.

Health outcomes to be discussed in the chapter from the different studies are: neurotoxic effects, immunotoxic effects, reproductive effects, cardiovascular effects, endocrine effects, and carcinogenic effects. Genetical and epigenetical aspects are also briefly discussed.

---

J.Ø. Odland (✉)

Department of Community Medicine, Faculty of Health Sciences,  
The Arctic University of Norway, 9037 Tromsø, Norway  
e-mail: [jon.oyvind.odland@uit.no](mailto:jon.oyvind.odland@uit.no)

S. Donaldson

Population Studies Division, Healthy Environments and Consumer  
Safety Branch, Health Canada, Ottawa, ON, Canada

© Springer International Publishing Switzerland 2016

J.M. Pacyna, E.G. Pacyna (eds.), *Environmental Determinants of Human Health*,  
Molecular and Integrative Toxicology, DOI 10.1007/978-3-319-43142-0\_3

Risk assessment of the effects of environmental pollutants is an essential tool in the overall health protection of Arctic residents. Different methods are available. One of the biggest challenges is how to translate contaminant concentrations measured in blood into information useful for risk characterization (the likelihood that specific effects will occur at these concentrations). The next challenge is to communicate this risk into policy making for populations and information to the individuals for preventive reasons. The Precautionary principle should be the basis for all approaches to the acquired knowledge, as well as providing good arguments for reduction of exposures to humans. Climate change and future change of exposure will further complicate the assessment, providing a good rationale to continue monitoring and assessment of the exposure and related health effects. Risks and benefits of breast feeding are not discussed in this chapter. A recent Norwegian report on risks and benefits of breast feeding concluded strongly that breast feeding is healthy and beneficial for both mother and child, and the nutritional value of the milk is far more important than the impact of documented levels of contaminants in the milk ([www.vkm.no](http://www.vkm.no) 2014).

**Keywords** Persistent toxic substances • Human health effects • Arctic populations • Indigenous people

### 3.1 The Monitoring Programs

The most comprehensive information we have about human exposure to contaminants in the Arctic has been collected, systematized, and presented in the different AMAP reports briefly described below.

Exposure to environmental contaminants through the traditional diet remains one of the risks to human health in the Arctic. Due to unique geographic and climatic characteristics, the Arctic has become a repository for contaminants transported long distances through the atmosphere and via ocean currents. These chemicals bioaccumulate and biomagnify through Arctic food chains into the species that make up traditional food sources for many Arctic peoples. These Arctic populations traditionally rely on fish, terrestrial and marine mammals, as market foods are difficult to access or are not as nutrient-rich as traditional foods (Donaldson et al. 2010). Many of the marine mammals, some of which are top predators in the Arctic marine food web, and some freshwater fish species tend to be the most highly contaminated with persistent, bioaccumulative chemicals. Biomonitoring studies are essential elements of the management of these risks, including the ability to analyze the risks and benefits for human populations which consume traditional food.

The AMAP 1998 report marked the first international, circumpolar report from the Arctic Monitoring and Assessment Programme (AMAP). Seven of the eight circumpolar countries (Canada, Denmark/Greenland/Faroe Islands, Iceland, Finland, Sweden, Norway, and Russia) contributed data to this report. Several polychlorinated biphenyls (PCBs), OCs, and metals were reported in different Arctic populations, both indigenous and non-indigenous.

The preliminary data identified in AMAP (1998) suggested that levels of POPs and methylmercury (MeHg) in breast milk and cord blood were two- to ten-fold higher than levels found south of the Arctic region. Despite worldwide risk management put in place for several POPs during the 1970s and 1980s, there seemed to be no evidence that levels in Arctic populations had decreased. Persistence, ubiquity and continued use in different parts of the globe contributed to continuous exposure levels for Arctic populations. This report was the first thorough report of human biomonitoring data for Arctic populations. While it could not provide temporal trends, it did provide the first Arctic-wide perspective (AMAP 2015).

Levels of hexachlorobenzene (HCB), mirex and three chlordane metabolites were markedly higher in Greenland and eastern Canadian Inuit maternal blood samples than in corresponding samples from the other participating countries. PCBs were highest in maternal blood samples from Greenland. Organochlorines such as  $\beta$ -hexachlorocyclohexane ( $\beta$ -HCH) and *p,p'*-dichlorodiphenyltrichloroethane (*p,p'*-DDT) were found to be higher in Russian populations than in populations examined from other countries, indicating potential uses in Russia or that these pesticides are being used on the food products consumed by these Arctic populations. Levels of *p,p'*-dichlorodiphenyldichloroethylene (*p,p'*-DDE) in Greenland and Russian populations were 3–5 times higher than those in other Arctic countries.

Greenland and Canada emerged as hotspots for metals, particularly mercury (Hg), lead (Pb) and cadmium (Cd). This exposure to Hg and Pb was attributed to specific traditional food consumption in these two populations in comparison with the food species consumed by populations in other parts of the Arctic; and smoking behavior was linked with levels of Cd.

Exposure levels found in the Arctic in this report were identified as being unacceptable. While temporal trends for Hg and Cd could not be determined from the data sets available, it was evident that Pb levels were decreasing in the Arctic in a similar way as levels in the south, probably in response to the risk management action taken on Pb emissions, and the partial ban on lead shot.

The second AMAP human health assessment (AMAP 2003) provided the first opportunity to compare contaminant levels over time, with the potential of multi-year data sets, especially in the Canadian and Greenlandic data sets for the maternal blood contaminant study. Data were submitted by all eight circumpolar countries. Fewer contaminants were reported in this report, but core contaminants of concern were consistently tested and reported in the Arctic countries, allowing for geographical comparison.

Levels of contaminants in blood samples from the populations living in the Arctic region again showed that certain POPs and Hg were high in Arctic people who consume certain traditional foods. Greenland Inuit mothers, in particular, were found to have levels of PCBs, HCB, total chlordanes and Hg higher than any other Arctic population. Russian non-indigenous populations had the highest levels of DDT and  $\beta$ -HCH; and elevated levels were also observed in Iceland and in the non-Caucasian, non-Dene/Métis, non-Inuit 'Others' group for the Canadian Arctic.

The third AMAP human health assessment report (AMAP 2009) contained the first comparison of all Arctic regions in Russia as a part of the overall assessment. It provided some of the first trend data for particular OCs and metals. Data from Canada, Greenland,



Iceland, Sweden, Finland and Russia showed that levels of contaminants such as PCBs, oxychlorodane, and Hg were decreasing in a broad range of Arctic populations. It was apparent that the consumption of marine mammals as a part of a healthy traditional diet seemed to result in elevated contaminant levels in Inuit regions of Greenland and Canada.

A new set of contaminants were measured in the Arctic for this report. It was possible to report the levels of brominated flame retardants (BFRs) and perfluorinated compounds (PFCs) due to advances in detection techniques and methodologies. Levels of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) were found throughout the Arctic and were elevated compared to other POPs. Polybrominated diphenyl ethers (PBDEs) were found at low levels across the Arctic, with the exception of high levels found in Alaska. Tetrabromobisphenol A (TBBPA) and hexabromocyclo-decane (HBCD) had limited data but showed low blood levels. Concentrations of PFOS were significantly higher in consumers of fish and marine mammal meat in Nunavik, Canada. PBDEs were found to require more data before a source or trend could be identified. In general, more monitoring data were needed for the emerging contaminants, as some were very data-limited but anticipated to be of potential concern in the future.

Since the 2009 AMAP human health assessment, biomonitoring activities have continued in the eight Arctic countries. POPs and metals are still undergoing long-range transport to the Arctic, and are bioaccumulating and bioconcentrating within the Arctic food chains relied upon for a socially, economically, culturally and nutritionally beneficial traditional food supply. However, declines are beginning to be detected in certain Arctic populations, and public health interventions have been instituted based on some of the biomonitoring results presented. Different contaminants are also being detected in the Arctic, indicating that new international risk management may be necessary. The use and production of PFCs started in the 1950s, increased during the 1970s, and continues today with PFCs still in demand in industrial and consumer applications (Nøst et al. 2014). Although PFCs do not bioaccumulate in fatty tissues, they are exhibiting similar behavior in terms of most other POPs, however the patterns for BFRs are slightly different. Further monitoring of BFRs is being pursued to identify their potential threat to Arctic populations.

## 3.2 Methodological Challenges

Since the first AMAP Human Health expert meeting in 1991, considerable efforts have been made to ensure the measurement of high quality human biomonitoring data for POPs and metals of concern to AMAP. The AMAP Human Health Assessment Group recommended in 2000 that a quality assessment (QA) program be established for POPs in human biological fluids (the AMAP Ring Test). The 2009 AMAP Human Health Assessment (AMAP 2009) described in detail the implementation and evolution of this external quality assessment scheme (EQAS), which has provided a means of comparing the quality of data produced by laboratories involved in the measurement of POPs in samples of human origin from Arctic countries (Weber 2002).

Since its inception in 2001, the scope of the AMAP Ring Test has changed to add or remove different POPs, with a substantial revision occurring in 2007 to add more POPs and also to modify the scoring system. The scheme now considers 37 analytes (11 pesticides, eight PBDEs, nine PCBs, six PFCs, total lipids, cholesterol and triglycerides). Several international EQAS already exist for metals in biological fluids, as well as a number of national schemes. Participation in the AMAP Ring Test by both AMAP and non-AMAP countries has fluctuated over the years. Currently, 32 laboratories are registered participants.

It is important for AMAP to present spatial and temporal trends of exposure with confidence and to demonstrate that the trends are not influenced unduly by analytical uncertainty. The AMAP Ring Test has thus established criteria for good (within 20 % of target) and acceptable (within 40 % of target) EQAS performance. Although fluctuations in individual performance are expected, the proportion of laboratories showing excellent or good performance generally increased between 2001 and 2007, often with pronounced improvements for some analytes (AMAP 2009). Ongoing participation in the AMAP Ring Test (or another suitable intercomparison program) is therefore highly encouraged, as this seems to have provided impetus for participating laboratories to examine and refine analytical procedures and organizational protocols necessary for meeting performance targets. All laboratories submitting data to this assessment were active participants in a QA/QC program.

We can now explore how contaminant levels are changing with time, with indications of differences between populations, geographical areas, and traditional practices. In doing so, insight is obtained about how international risk management efforts, dietary shifts, and risk communication interventions from public health officers and other officials may be affecting Arctic residents' exposure. For several Arctic countries, there are now almost 20 years of biomonitoring data available to assess changes in contaminant concentrations.

### 3.3 The Studies

This section will describe some of the most important ongoing trend and effect studies in the Arctic. More details are available in the recently released AMAP Human Health Assessment Report (AMAP 2015). The Arctic studies have the overall aim to provide compatible data independent of geographical or socioeconomic/cultural differences, which is still only partly fulfilled.

#### 3.3.1 *The MISA Study of Northern Norway*

The Northern Norway mother-and-child contaminant cohort study—the MISA study—is a cross-sectional study with longitudinal aspects aimed at establishing a new northern Norway mother-and-child contaminant cohort study. The MISA database is considered suitable for exploring associations between contaminant

exposure and diet, enhancing understanding of the interplay between physiological changes that occur in mothers and contaminant pharmacokinetics (including transfer to the infant before and after birth), and conducting prospective health studies of the children (Hansen et al. 2010, 2011; Veyhe et al. 2012).

### **3.3.2 *The Tromsø Study***

The Tromsø Study is a population-based health survey initiated in 1974 to investigate the reasons for high mortality due to cardiovascular disease in northern Norway. Six surveys have been undertaken since 1974 and the health research topics included have increased. A total of 40,051 people have participated in at least one survey and 15,157 have participated in three or more surveys. The Tromsø Study was also used to explore changes in POP concentrations from 1979 to 2007 on an individual basis with a repeat measurement design. Serum samples were obtained from the freezer archive for 54 men who participated in all of the survey points: 1979, 1986, 1994, 2001, and 2007. The archived serum samples were analysed for PCBs, chlorinated pesticides, and per-and polyfluoroalkyl substances (PFASs).

The trends observed between 1979 and 2007 probably reflect the overall trends in the use and emission of the different POPs, together with compound-specific persistency, bioaccumulation potential and long-range transport. Study design and population characteristics must also be considered in monitoring studies. The Tromsø Study has increased knowledge of intra-individual variation in POP concentrations with respect to time and exposure history, which is essential for understanding past exposure and predicting future exposure. These findings have important implications for future studies of exposure and vulnerable groups (Nøst et al. 2013, 2014).

### **3.3.3 *Northern Finland 1966 Birth Cohort***

There is information on the individuals born into the Northern Finland 1966 Birth Cohort in the provinces of Oulu and Lapland since the 24th gestational week as well as on their mothers and, to a lesser extent, fathers. A total of 12,058 live-born children were born into the cohort (around 96 % of all eligible) and 11,665 were alive in 1997. Data were collected by questionnaire, from hospital records and various registers and databases (social benefits, medication reimbursement, hospital discharges and deaths, community wealth), as well as by interview and clinical examination at birth, age 1 year and 14 years, and at age 31 years when a comprehensive follow-up was conducted on each subject's well-being, social standing and health. In 1997, all members of the cohort who lived in the provinces of Oulu and Lapland (n=7191) or who had moved to the capital region (n=1272) completed a questionnaire and underwent a health

examination. Those living in other parts of the country (n=2164) or abroad (n=695) were also sent the questionnaire. In total, 8676 people returned the questionnaire, making a response percentage of about 77 %. About 71 % attended the health examination. Selected blood samples (n=250) from the 1997 sampling were analyzed for toxic and essential elements to establish levels in persons born and living for the last 5 years in the eastern and western part of Lapland (AMAP 2015).

### ***3.3.4 The Chukotka Studies***

Data were collected in 2001–2003 in Chukotka (Russia) on PCB and DDT contamination of different local foods (and indoor materials). Exposure of indigenous people was evaluated by comparing pollutant levels in foods with levels in human blood in people living in coastal and inland regions (Dudarev et al. 2012).

Levels of persistent toxic substances (PTS) in blood from 17 mothers and cord blood from the corresponding 17 babies born in the Chukotka coastal area in 2001–2002 were compared with PTS levels in blood sampled from the same women and their 5-year old children in 2007 with the aim of examining the influence of breastfeeding on maternal POPs serum levels and the link between children's POPs blood levels and the frequency of infectious diseases (Dudarev et al. 2010, 2012).

In the period 2001–2002 126 indigenous pregnant women (68 coastal and 58 inland) were interviewed and gave blood samples in the Chukotka delivery departments. Births with adverse outcomes were observed in almost every fourth woman (23 %): premature births (22 cases) partially accompanied by low birth weight (13 cases), stillbirths (3 cases), and congenital malformations (3 cases—a heart defect and two multiple defects, one stillborn).

### ***3.3.5 The Kola Lapland Study 2001–2006***

Interviews and blood samples of 4359 residents of Kola Lapland in Murmansk Oblast in 2006 (2736 rural and 1623 urban, including Sami—694, Komi—910, Nenets—80) showed that risk of Type 2 diabetes (overweight/obesity, enhanced blood pressure, sedentary lifestyle, malnutrition, alcohol abuse) was 3–7 times lower in indigenous residents than non-indigenous residents. Among Sami people of the remote villages signs of diabetes were absent. Elevated levels of blood glucose were found mainly in large settlements. By all criteria of predisposition to diabetes mellitus, indigenous residents in remote villages demonstrate minimum risk levels, and this appears to reflect their traditional diet based on local foods, a physically active lifestyle, and minimal consumption of high carbohydrate foods (Dudarev et al. 2012a).

### **3.3.6 *The Nunavik Child Development Study***

The primary objective of the Nunavik Child Development Study (NCDS) was to document the growth, neurobehavioral and cardiac effects of pre- and postnatal exposure to OCs with endocrine-disruptive properties, as well as to Hg and Pb. A secondary aim was to test whether nutritional variables, such as prenatal polyunsaturated fatty acid (PUFA) intake, breastfeeding, and childhood vitamin deficiency, mediate and/or mitigate the effects of environmental contaminants on health and development. The NCDS was made possible by the Nunavik Cord Blood Monitoring Program, through which exposure to environmental contaminants was determined from cord blood samples obtained from Nunavik infants born between 1994 and 2001 (Muckle et al. 1998; Dallaire et al. 2003).

### **3.3.7 *The Inuit Cohort: Canada***

The Nunavik Health Survey Qanuippitaa was conducted between 30 August and 1 October 2004 on the scientific research vessel CCGS *Amundsen*. The research group visited the 14 communities of Nunavik ([www.qanuippitaa.com](http://www.qanuippitaa.com)) and recruited 917 participants. Samples were analyzed for POPs that had also been determined during the 1992 Santé Québec Health Survey. Plasma concentrations of new halogenated hydrocarbons such as PBDEs, PFOS, hydroxy-PCBs, methyl-sulfone PCBs and chlorophenols were also determined.

The Inuit Health Survey (2007–2008) was a comprehensive study that included the measurement of dietary intake of contaminants, contaminant body burden, as well as other determinants of health and their relationship with health outcomes of the participants. It was the first time that such a complete set of data was collected in the Arctic. Of the 2595 individuals that participated in IHS, 2172 provided blood samples. The body burden of several metals (e.g. Cd, Pb) and POPs (e.g. PCBs, DDT, DDE, toxaphene, chlordane, PBDEs) were measured for Inuit participants (n=2172) from 36 communities in Nunavut, Nunatsiavut, and the Inuvialuit Settlement Region, in Canada (Laird et al. 2013).

### **3.3.8 *INUENDO***

The INUENDO project: Biopersistent OCs in diet and human fertility was supported by the European Union FP5 ([www.inuendo.dk](http://www.inuendo.dk)) (Bonde et al. 2008). The cohort was established in 2002–2004 and involved about 1400 pregnant women from Greenland, Poland and Ukraine, as well as studies on about 600 previous pregnant fishermen's wives and fishermen.

The study included measurement of PCB153 and *p,p'*-DDE and bioeffect markers of serum legacy POP-induced estrogen- and androgen-receptor transactivity and epidemiological cross-sectional studies on male and female reproductive health on these individuals in relation to the measured exposures.

### 3.3.9 CLEAR

The CLEAR project: Climate Change, Environmental Contaminants and Reproductive health is supported by the European Union FP7 ([www.inuendo.dk/clear](http://www.inuendo.dk/clear)). In addition to modelling the effects of climate change on contaminant distribution to the environment, the project includes a series of cross-sectional studies on male and female reproductive health combined with a follow-up study on childhood growth and development at 6–9 years of age in a cohort of about 1400 mothers, fathers and offspring from Greenland, Poland and Ukraine. The original INUENDO cohort was established in 2002–2004 and a follow-up on the children was undertaken in 2010–2012.

### 3.3.10 Cohorts in the Faroe Island

A cohort of 1022 singleton births was assembled in the Faroe Islands during a 21-month period in 1986 and 1987 (Grandjean et al. 1992). Frequent whale meat dinners during pregnancy, frequent consumption of fish (to a much lesser degree) and increased parity or age were associated with high Hg concentrations in cord blood and maternal hair. Mercury concentrations in cord blood correlated moderately with blood-selenium. Lead concentrations in cord blood were low (median, 82 nmol/L). Because the effects of fetal childhood exposure to MeHg are persistent, detailed examination of children with prenatal exposure to this neurotoxicant were performed at age 7 years (1993–1994). A total of 917 of the children (90.3%) completed the examinations. Past medical history, current health status and social factors were recorded on a self-administered form. The physical examination included a functional neurological examination with emphasis on motor coordination and perceptual-motor performance. The main emphasis was placed on detailed neurophysiological and neuropsychological tests that had been selected on the basis of a range of considerations (Grandjean et al. 1997, 2012a).

The findings from the first cohort suggested that exposure assessment should encompass several lipophilic pollutants in addition to MeHg. A follow-up cohort was therefore established during a 12-month period in 1994–1995 and included 182 singleton term births from consecutive births at the National Hospital in Tórshavn, Faroe Islands. Relevant obstetric data were obtained by standardized procedures and supplemented by a brief nutrition questionnaire.

New insight into health risks caused by environmental pollutants and changing exposure patterns in the Faroes lead to the formation of Cohort 3 from 656

consecutive births in Tórshavn between November 1997 and March 2000. Following dietary recommendations from the Faroese health authorities, MeHg exposure had now decreased thus allowing better characterization of possible effects of PCBs and other persistent contaminants. Additional attention was turned to the perfluorinated compounds (PFCs), as they have been documented as contaminants of marine food chains with possible toxicity to the immune system. The most recent cohort was born during an 18-month period between October 2007 and April 2009. The total of mother-child pairs included was 475 (73 % of the eligible population). Blood was taken from the cord, and blood, hair, and milk were obtained from the mother (AMAP 2015).

### 3.4 Effects Discussed in the Different Studies, Single Compounds or Combined Effects

#### 3.4.1 Neurological Effects

##### 3.4.1.1 Mercury

The cohort studies in the Faroe Islands have demonstrated that children exposed to methylmercury (MeHg) *in utero* exhibit decreased motor function, attention span, verbal abilities, memory, and other mental functions (Grandjean et al. 1997). The effects are dose-dependent: the greater the mercury (Hg) exposure, the greater the effect. A follow-up of these children at ages 14 and 22 years showed the deficits appear to be permanent (Debes et al. 2006). Overall, a doubling of the prenatal Hg exposure of a child resulted in a developmental delay of 1–2 months at the age of 7 years; the age when the child is expected to enter school. This delay corresponds to about 1.5 IQ points (Grandjean and Herz 2011).

In the Nunavik Child Development Study (NCDS) the children were followed up at age 11 years. The results show that Hg was associated with: poorer early processing of visual information; alteration of attentional mechanisms modulating processing of sensory information; lower estimated IQ; poorer comprehension and perceptual reasoning; poorer immediate memory and recollection of information stored into memory; and increased risk of attention problems and attention deficit hyperactivity disorder (ADHD) behavior.

In the Nunavik study, prenatal Hg was related to poorer IQ after adjustment for exposure to the other contaminants assessed, to docosahexaenoic acid (DHA) and selenium, and to the other potentially confounding variables. The Hg effect became stronger when cord DHA was also considered, indicating that the beneficial effect of prenatal DHA statistically suppresses the adverse effects of prenatal Hg exposure. Verbal comprehension and perceptual reasoning were indices most sensitive to prenatal Hg exposure.

Some of the adverse effects of MeHg on neurodevelopment may be masked by beneficial effects of seafood nutrients (Budtz-Jørgensen et al. 2007). On the other hand, the benefits from seafood nutrients may be consumed or cancelled by the Hg

toxicity. Thus, full benefit from fish and seafood diets requires that MeHg exposure is minimized. Given the continued development of the nervous system after birth, postnatal exposure to MeHg is also likely to cause adverse effects. Neurophysiological assessment of brain function supports the notion that postnatal exposure up to the teenage years can cause harm (Murata et al. 2004). Thus, both pregnant women and children should be considered populations at increased risk (Grandjean 2013).

Adverse effects may also include aspects of brain function other than cognitive achievement. In the NCDS (Nunavik 2005–2010), exposure to contaminants was measured at birth and at school age. An assessment of child behavior ( $n = 279$ ; mean age = 11.3 years) was obtained from the child's classroom teacher on the Teacher Report Form (TRF) from the Child Behavior Checklist, and the Disruptive Behavior Disorders Rating Scale (DBD). Cord blood Hg concentrations were associated with higher TRF symptom scores for attention problems and DBD scores consistent with ADHD. This appears to be the first study to identify an association between prenatal MeHg and ADHD symptomatology in childhood (Boucher et al. 2012). Likewise, cross-sectional evidence links MeHg exposure to autism spectrum disorder (Geier et al. 2012). However, the evidence available is limited and so conclusions regarding autism or ADHD must be drawn with caution.

Mercury may also negatively affect other aspects of human neurological health. Some research suggests that MeHg neurotoxicity may spur the development of degenerative diseases of the nervous system, such as Parkinson's disease (Petersen et al. 2008).

Effects associated with MeHg exposure have been documented in humans at lower and lower exposures. This tendency reflects the use of better study design, larger groups of subjects examined, more sensitive methodology and better control of confounding factors that may influence study outcome. From the evidence available, it is clear that the developing brain is the most vulnerable organ system. Given the complexity of brain development and the difficulties in determining detailed functions, especially in small children, it is likely that future studies will continue to identify effects at lower exposures than those considered safe today.

#### **3.4.1.2 Lead and Mixed Metal Exposures**

Few studies have examined the effects of mixed metal exposures in humans. In one of the Faroese birth cohorts the effect of prenatal lead (Pb) exposure in the presence of similar molar-level exposure to MeHg was evaluated (Yorifuji et al. 2011). A cohort of 1022 singleton births was assembled during 1986–1987 and Pb was measured in cord blood. A total of 896 cohort subjects participated in a clinical examination at age 7 years and 808 subjects in a follow up at age 14 years. The association between cord-blood Pb concentration and cognitive deficits (attention/working memory, language, visuospatial, and memory) using multiple regression models was evaluated. Overall, the Pb concentration showed no clear pattern of association. However, in subjects with a low MeHg exposure, Pb-associated adverse effects on cognitive function were observed. Some interaction terms between Pb and MeHg suggested that the combined effect of the exposures was less than additive.



### 3.4.1.3 Persistent Organic Pollutants

Grandjean et al. (2012a) analyzed banked cord blood from a Faroese birth cohort to determine the possible neurotoxic impact of prenatal exposure to polychlorinated biphenyls (PCBs). The subjects were born in 1986–1987, and 917 cohort members completed a series of neuropsychological tests at age 7 years. Major PCB congeners (PCB118, PCB138, PCB153, PCB180), the calculated total PCB concentration, and the PCB exposure estimated in a structural equation model showed weak associations with test deficits, with statistically significant negative associations only with the Boston Naming test. Likewise, neither hexachlorobenzene (HCB) nor *p,p'*-dichlorodiphenyldichloroethylene (*p,p'*-DDE) showed clear links with neurobehavioral deficits. Thus, these associations were much weaker than those associated with cord-blood Hg concentration, and adjustment for Hg substantially attenuated the regression coefficients for PCB exposure. When the outcomes were merged into motor and verbally mediated functions in a structural equation model, the PCB effects remained weak and virtually disappeared after adjusting for MeHg exposure, while Hg remained statistically significant. Thus, in the presence of elevated MeHg exposure, PCB neurotoxicity may be difficult to detect, and PCB exposure does not explain the MeHg neurotoxicity previously reported in this cohort (Grandjean et al. 2012a).

### 3.4.1.4 Immunological Effects

Certain environmental pollutants can adversely affect the development of the immune system (Dewailly et al. 1993; Weisglas-Kuperus et al. 1995, 2000; Chao et al. 1997; Dewailly et al. 2000; Vine et al. 2001; ten Tusscher et al. 2003; Jusko et al. 2010).

The high incidence of infectious diseases—particularly meningitis, broncopulmonary infections, and middle ear infections—in young children from Nunavik has been known for many years (Dufour 1988). In view of the immunotoxic properties displayed by some organochlorines (OCs), in particular following perinatal exposure, it has been hypothesized that part of the high infection incidence among Inuit infants could be related to the relatively high maternal body burden of these contaminants, and their partial transfer to newborns during breastfeeding. To test this hypothesis, three epidemiological studies have been conducted during the past 20 years in Arctic Quebec to investigate the relationship between pre- or postnatal OC exposure, immune status, and the occurrence of infectious diseases among Inuit infants. Results in three different groups of Inuit children indicated that prenatal exposure to OCs increases susceptibility to infectious diseases, and in particular to otitis media (Dewailly et al. 2000; Dallaire et al. 2004, 2006). Although potential confounding factors were considered in the statistical analyses, residual confounding is still a possibility.

The Faroese studies are the first to provide epidemiological data on human immunotoxicity—as reflected by a reduction in serum antibody production after routine childhood immunizations—in relation to developmental exposures to

environmental chemicals (Heilmann et al. 2006, 2010; Grandjean et al. 2012b). The studies showed that developmental and perinatal exposure to PCBs and PFCs from marine food and other sources may inhibit immune function, as indicated by deficient serum concentrations of antibodies against childhood vaccines. Results from the Faroe Islands show that the risk of having an antibody concentration below 0.1 IU/mL at age 7 years increased at higher levels of exposure to PCBs and PFCs. The results suggest that PFCs have an even stronger negative effect than PCBs on serum-antibody concentrations (Heilmann et al. 2006, 2010; Grandjean et al. 2012b). For PCBs, a doubling of the serum concentration at age 18 months was associated with a decline of 20% in the antibody level at age 7 years. After the completion of breastfeeding and associated transfer of PCBs, the child at age 18 months has an average serum-PCB concentration similar to that of the mother (Heilmann et al. 2010), after which the concentration declines as the body lipid compartment continues to expand. For PFCs, the recent accumulation was found to be the most important predictor of immunotoxicity: A doubling in serum-PFC concentration measured at age 5 years was linked to a decrease of up to 50% in the antibody concentration at age 7 years ( $p < 0.001$ ). Due to the long half-life of PFCs (Olsen et al. 2007), serum concentrations at early school age are expected to be relatively stable.

In conclusion, elevated exposure to PCBs and PFCs in Faroese children was associated with reduced humoral immune response to routine childhood immunizations (Heilmann et al. 2006, 2010; Grandjean et al. 2012b). These findings suggest a decreased effect of childhood vaccines and may reflect a more general immune system deficit. The clinical implications of insufficient antibody production emphasize the need to prevent immunotoxicant exposure and assessment of risk related to exposure to these contaminants.

#### 3.4.1.5 Reproductive Effects

In 1992, Carlsen and co-workers published a combined analysis of results from 61 papers published between 1939 and 1991 and showed a significant decline in sperm count over the 50-year period. A detailed reanalysis of the results found that this conclusion was supported by the underlying studies (Swan et al. 1997, 2000). Following the 1992 publication, many researchers retrospectively analyzed their historical data for temporal trends, some finding a decline and others not.

The causes of decreased semen quality are not clear, but it is feasible that many cases may have been caused by exposure to environmental factors *in utero*, during adolescence or in adulthood (Joensen et al. 2008); probably also influenced by different genetic susceptibility.

The median sperm concentration of fertile men in a semen quality study conducted in Greenland in 2004 was 53 million/mL, with a median sperm cell volume of 3.2 mL, a total sperm count of 186 million and a median motility of 60% (Toft et al. 2004). No regional difference was found in sperm count, but sperm cell motility differed among regions. In a following study, Toft et al. (2006)

found that sperm concentration was not impaired by increasing serum PCB153 or *p,p'*-DDE levels in Greenlanders. Also, that there was no association between the proportion of morphologically normal sperm and either PCB153 or *p,p'*-DDE concentration in blood. However, sperm motility was inversely related to PCB153 concentration in this population.

In a recent study on testicular function in the Faroe Islands, Halling et al. (2013) found sperm concentrations for Faroese men to be lower than for Danish men (crude median 40 vs 48 million/mL,  $p < 0.0005$ ). However, because semen volume was higher in the Faroese men, the total sperm counts did not differ (159 vs 151 million,  $p = 0.2$ ). Similarly, there was no overall difference between the two populations in terms of sperm motility or morphology. Recent data have shown sperm count to be low in young men from several European countries, but slightly higher than among the Danes (Jorgensen et al. 2002; Punab et al. 2002; Richthoff et al. 2002). This indicates that semen quality for both Danish and Faroese men seems to be low compared to men from other European countries.

The reason for low testicular function in the Faroese young men is unclear, but could be due to high exposure to persistent organic pollutants (POPs). Studies have shown associations between high PCB levels and low semen quality, and since PCBs and *p,p'*-DDE have the potential to interfere with sex hormone functions, it could be assumed that these compounds can affect the function of the testicles (Elzanaty et al. 2006). Some reports on the effect of POPs on male reproduction in humans indicate weak negative effects on sperm motility (Hauser et al. 2002; Richthoff et al. 2003; Elzanaty et al. 2006). Among the Faroese men the present study found the percentage of motile cells to be significantly lower compared to Danish men, indicating that increased exposure to endocrine disruptors may be one explanation for the difference.

Serum steroid hormone-binding globulin (SHBG) levels for the Faroese men were much higher than for the Danes. One explanation could be the high PCB levels among the Faroese. Grandjean et al. (2012c) reported that SHBG increased at higher PCB exposure, both prenatally and later in life. Because PCBs are known to affect a number of liver functions it may be that PCB-induced hepatic SHBG synthesis could play a role, although this remains to be confirmed (Grandjean et al. 2012c).

### **3.4.2 Cardiovascular Effects**

#### **3.4.2.1 Mercury**

Possible cardiovascular effects of Hg have recently emerged in the scientific literature (Roman et al. 2011). A growing body of evidence suggests that MeHg exposure can increase risk of adverse cardiovascular impacts in exposed populations. The link between MeHg and acute myocardial infarction or sudden cardiac death is still debated in low Hg exposed populations (Mozaffarian et al. 2011; Virtanen et al. 2012).

Contradictory results have been reported on Hg exposure and the risk of hypertension (Mozaffarian et al. 2012). In Nunavik adults, a retrospective analysis of the 1992 survey reported no association between Hg and high blood pressure (Valera et al. 2013). Based on the 2004 data, however, Hg was associated with increased blood pressure and pulse pressure (Valera et al. 2008, 2009). In the Faroe Islands, high blood pressure was found to be associated with Hg exposure among male whale hunters (Choi et al. 2009). In Greenland, no association was found between Hg exposure and high blood pressure (Nielsen et al. 2012). Associations between Hg exposure and blood pressure were also studied in children. Associations were reported between prenatal Hg exposure and lower systolic blood pressure in 7-year-old Faroese children (Sorensen et al. 1999) and for lower diastolic blood pressure in the Seychelles (Thurston et al. 2007). In Nunavik children, no association was found between blood pressure and either cord blood or contemporary Hg exposure at age 11 years (Valera et al. 2012).

Heart rate variability has also been studied in Arctic populations. An association was reported between Hg exposure and decreased heart rate variability in adults from Nunavik (Valera et al. 2008). Similar results were reported among James Bay Cree adults (Valera et al. 2011). In children from Nunavik, cord blood Hg concentrations were not related to heart rate variability parameters at age 11 years, but child blood Hg levels were associated with decreased overall heart rate variability parameters, and these associations remained significant after adjusting for cord blood Hg, *n*-3 polyunsaturated fatty acids (PUFA) and selenium. In Faroese children, cord Hg concentrations were related to heart rate variability at age 7 years as well as 14 years, and hair Hg at age 7 years was associated with a low frequency variation coefficient (Grandjean et al. 2004).

The predictive value of heart rate variability parameters in healthy children and risk of chronic diseases is unknown. Nevertheless, results from the Faroese and Nunavik cohorts provide evidence that Hg exposure during childhood is related to changes in cardiac autonomic activity at school age.

#### 3.4.2.2 Endocrine Effects

Environmental chemicals have significant impacts on biological systems. Exposure during early stages of fetal and neonatal development is especially critical and can disrupt the normal pattern of development and thus dramatically alter disease susceptibility in later life. Endocrine-disrupting chemicals are those that interfere with the body's endocrine system and so result in adverse developmental, reproductive, neurological, cardiovascular, metabolic and immune effects in humans. An endocrine-disrupting chemical is defined as 'an exogenous substance or mixture, that alters the function(s) of the endocrine system, and consequently causes adverse health effects in an intact organism or its progeny or (sub)-population' (Schug et al. 2011). Thus they are compounds that can mimic, interfere with or block the function of endogenous hormones and thereby disrupt the normal hormone homeostasis of the body. Growing evidence shows that endocrine-disrupting chemicals may also

modulate the activity and/or expression of steroidogenic enzymes, having the ability to convert circulating precursors into active hormones as well as to affect hormone metabolism and transport through the body (Yang et al. 2006; Schug et al. 2011).

### 3.4.2.3 Impact on the Hypothalamo-Pituitary-Gonadal Axis

Exposure to POPs may have a negative impact on reproductive function via impact on the hypothalamo-pituitary-gonadal axis. A study of reproductive hormones in men from Greenland and three European cohorts (Swedish fishermen, Warsaw Poland, Kharkiv Ukraine) (Bonde et al. 2008) reported significant variations in associations between exposure to PCB153 and *p,p'*-DDE and the outcomes. For the Kharkiv group, statistically significant positive associations were found between levels of both PCB153 and *p,p'*-DDE and steroid hormone-binding globulin, as well as luteinizing hormone, while for the Greenlandic Inuit men there was a positive association between PCB153 exposure and luteinizing hormone. In the pooled data set from all four centers, there was a positive association between *p,p'*-DDE and FSH levels ( $\beta=1.1$  IU/L; 95 % CI: 1.0–1.1 IU/L), whereas the association between PCB153 and SHBG was of borderline statistical significance ( $\beta=0.90$  nmol/L; 95 % CI:  $-0.04$ – $1.9$  nmol/L). The authors concluded that gonadotropin levels and SHBG seem to be affected by POPs exposure, but that the pattern of endocrine response is the subject of considerable geographic variation (Giwercman et al. 2006).

Studying differences between men living south and north of the Arctic Circle in Norway, Haugen et al. (2011) found no geographical differences in either mean levels of PCB153 (50 vs 59 ng/g lipid;  $p=0.27$ ) or sperm parameters. However, mean levels of *p,p'*-DDE were higher in the south than the north (81 vs. 66 ng/g lipid;  $p=0.02$ ), as were levels of total and free testosterone. Moreover, FSH levels were lowest in the south. A strong relationship was observed between PCB153 and SHBG levels. The regional differences observed for *p,p'*-DDE, testosterone and FSH were not reflected in semen quality.

In the Faroe Islands, where PCB exposure is elevated, Grandjean et al. (2012c) studied the possible endocrine disruption of PCBs on 438 adolescent boys from a birth cohort by measuring PCB and *p,p'*-DDE levels in cord blood and serum from clinical examination at age 14 years. Higher prenatal PCB exposure was associated with lower serum concentrations of both luteinizing hormone and testosterone. In addition, SHBG was positively associated with both prenatal and concurrent PCB exposure. The PCB–SHBG association was robust to covariate adjustment. In a structural equation model, a doubling in prenatal PCB exposure was associated with a decrease in luteinizing hormone of 6 % ( $p=0.03$ ). Prenatal exposure to PCB and DDE showed weak, non-significant inverse associations with testicular size and Tanner stage. DDE was highly correlated with PCB and showed slightly weaker associations with the hormone profile. The findings suggest that delayed puberty with low serum-LH concentrations associated with developmental exposure to non-dioxin-like PCBs may be due to a central hypothalamo-pituitary mechanism.

### 3.4.2.4 Impact on the Hypothalamo-Pituitary-Thyroid Axis

Dallaire et al (2008) investigated the potential impact of transplacental exposure to PCBs and HCB on thyroid hormone concentrations in neonates from two remote coastal populations in Canada; one in Nunavik (n=410) and one on the Lower North Shore of the St. Lawrence River (n=260). Both populations were exposed to OCs through seafood consumption. Cord blood samples were analyzed for thyroid parameters (TSH; free T4, fT4; total T3, tT3) and contaminants. PCB153 was not associated with thyroid hormone and TSH levels in either population. Prenatal exposure to HCB was positively associated with fT4 levels at birth in both populations (Nunavik,  $\beta=0.12$ ,  $p=0.04$ ; St. Lawrence,  $\beta=0.19$ ,  $p<0.01$ ), whereas thyroxine-binding globulin concentrations were negatively associated with PCB153 concentrations ( $\beta=-0.13$ ,  $p=0.05$ ) in the St. Lawrence cohort. Thus OC levels were not associated with a reduction in thyroid hormones in neonates from the two populations. Essential nutrients derived from seafood such as iodine may have prevented the negative effects of OCs on thyroid function during fetal development. Dallaire et al. (2009) studied the relationship between exposure to potential thyroid hormone-disrupting toxicants and thyroid hormone status in pregnant Inuit women from Nunavik and their infants within the first year of life. In pregnant women, they found a positive association between hydroxylated metabolites of PCBs and total triiodothyronine (T(3)) concentrations ( $\beta=0.57$ ,  $p=0.02$ ). In umbilical cord blood, PCB153 concentrations were negatively associated with thyroxine-binding globulin levels ( $\beta=-0.26$ ,  $p=0.01$ ). In a subsample analysis, a negative relationship was also found between maternal pentachlorophenol levels and cord free thyroxine [fT(4)] concentrations in neonates ( $\beta=-0.59$ ,  $p=0.02$ ). No association was observed between contaminants and thyroid hormones at 7 months of age. The authors concluded that there is little evidence that the environmental contaminants analyzed in their study affect thyroid hormone status in Inuit mothers and their infants. The possibility that pentachlorophenol may decrease thyroxine levels in neonates requires further investigation.

Audet-Delage et al. (2013) studied whether exposure to POPs might decrease the circulating concentrations of T4 bound to transthyretin (TTR) in Inuit women of reproductive age in Nunavik Canada. Hydroxylated PCBs, pentachlorophenol and PFOS compete with T4 binding sites on TTR. The data suggested that circulating levels of TTR-binding compounds were not high enough to affect TTR-mediated thyroid hormone transport. However, the authors suggested that the possibility of increased delivery of these compounds to the developing brain requires further investigation.

In a study on the relationship between exposure to POPs and anti-thyroid peroxidase antibody (TPOAb), Schell et al. (2009) assessed thyroid dysfunction of 115 young adults of the Akwesasne Mohawk Nation tribe. Overall, 18 participants (15.4%) had TPOAb levels above the normal laboratory reference range (23% of females, 9% of males). Among participants who were breast fed (n=47), those with an elevated TPOAb level had significantly higher levels of all PCB groupings, with the exception of levels of non-persistent PCBs which did not differ significantly. Levels of *p,p'*-DDE were also significantly elevated, while HCB and Mirex were not higher among those with elevated TPOAb. Also, after stratifying by

breast-feeding status, participants who were breast fed showed significant, positive relationships between TPOAb levels and all PCB groupings, except groups comprising non-persistent PCBs, and with *p,p'*-DDE, HCB, and Mirex. No effects were evident among non-breast-fed young adults.

Bloom et al. (2014) observed an association between POPs and thyroid hormones in aging residents of upper Hudson River communities (48 women, 66 men, aged 55–74). The POPs included 39 PCBs, DDT and DDE and nine PBDEs. Among women, DDT+DDE increased T4 by 0.34 µg/dL ( $p=0.04$ ) and T3 by 2.78 ng/dL ( $p=0.05$ ). Also in women, ΣPCBs in conjunction with PBDEs elicited increases of 24.39–80.85 ng/dL T3 ( $p<0.05$ ), and ΣPCBs in conjunction with DDT+DDE elicited increases of 0.18–0.31 µg/dL T4 ( $p<0.05$ ). For men, estrogenic PCBs were associated with a 19.82 ng/dL T3 decrease ( $p=0.003$ ), and the sum of estrogenic PCBs in conjunction with DDT+DDE elicited an 18.02 ng/dL T3 decrease ( $p=0.04$ ). The authors suggested that the influence of POPs on thyroid hormones in aging populations may have clinical implications and merits further investigation.

### 3.4.2.5 Persistent Organic Pollutants and Type 2 Diabetes

Several descriptive epidemiology studies suggest that certain POPs can contribute to the development of Type 2 diabetes (Carpenter et al. 2002; Patel et al. 2010). Although many POPs have now been banned, exposure continues for the most persistent, such as the PCBs and the pesticide metabolite *p,p'*-DDE. Other persistent environmental chemicals still in current use are suspected to be diabetogenic, for example the brominated flame retardants and perfluorinated compounds. Most of the recent epidemiological evidence is from cross-sectional case–control studies, where increased serum POP concentrations were found to be a major determinant of diabetes (Everett and Matheson 2010) and metabolic syndrome (Lee et al. 2007). Genetic predisposition to Type 2 diabetes seems to play a role, but most genetic variants so far identified are associated with β-cell function and account for no more than about 10% of the risk. Thus it is very likely that POP exposure may trigger gene–environment interactions with effects on insulin resistance and/or secretion.

In the Faroe Islands, 713 septuagenarians with a high POP exposure owing to a traditional diet (including pilot whale blubber), were examined for indicators of glucose metabolism in subjects free of Type 2 diabetes and pre-diabetes (impaired fasting glycaemia) (Grandjean et al. 2011). Results showed that the fasting insulin concentration decreased by about 8% for each doubling of the serum concentration of PCBs, and that a similar increase occurred in the fasting glucose level. Along with higher PCB exposure in subjects with Type 2 diabetes and impaired fasting glycaemia, the results suggest that PCB-induced β-cell deficiency may be involved in the disease pathogenesis. Impaired insulin secretion appears to constitute an important part of the Type 2 diabetes pathogenesis associated with exposure to persistent lipophilic food contaminants).

Individuals with vitamin D levels below 50 nmol/L doubled their risk of newly diagnosed Type 2 diabetes. Thus, in elderly subjects, vitamin D may provide protection

against Type 2 diabetes (Dalgard et al. 2011). The high prevalence of low vitamin D levels among the elderly Faroese population (19% with <25 nmol/L) reflects the low skin synthesis during most months of the year, which is caused by limited sun exposure and insufficient intake of marine diet (Dalgard et al. 2010).

### 3.4.2.6 Carcinogenic Effects

Throughout the twentieth century, the cancer patterns of the Inuit population have been characterized by a high risk of Epstein-Barr virus (EBV)-associated carcinomas of the nasopharynx and salivary glands, and a lower risk of tumors common in Caucasian populations, including cancer of the breast, prostate, testis, and hemopoietic system. Both genetic and environmental factors seem to be responsible for this pattern. Over the past 50 years, Inuit societies have undergone major changes in lifestyle and living conditions. The incidence of traditional Inuit cancers (nasopharynx and salivary glands cancer) has remained relatively constant, whereas the incidence of lifestyle-associated cancers, especially cancer of the lung, breast, stomach and colorectal has increased considerably following changes in lifestyle (smoking, alcohol), diet, and reproductive factors (Friborg and Melbye 2008).

A comparison of cancer incidence patterns between residents of Inuit Nunangat and the rest of Canada showed the age-standardized incidence rate for all cancer sites (1998–2007) to be 14% lower for the Inuit Nunangat male population and 29% higher for the female population compared to the rest of Canada (Carriere et al. 2012). Cancers of the nasopharynx, lung and bronchus, colorectal, stomach (males), and kidney and renal pelvis (females) were elevated in the Inuit Nunangat population compared to the rest of Canada, whereas prostate and female breast cancers were lower. As well as higher smoking prevalence within Inuit Nunangat, distinct socio-economic characteristics between the respective populations (including housing and income) may have contributed to the incidence differentials (Carriere et al. 2012).

Some cancer incidence in Greenland is several times higher compared to Denmark, such as nasopharyngeal, esophagus, biliary, ventricle, cervical, lung, liver, pancreas and colorectal cancer, respectively (AMAP 2015).

A study showed that the indigenous coastal Chukchi and Inuit living in Chukotka (Russia) were at higher risk of death from cancer during 1961–1990 than the Russian population nationally, with age-standardized cancer mortality among men twice that of Russia in general, and among women 3.5 times higher. The difference is due to the particularly high mortality from esophageal cancer and lung cancer in the indigenous people of coastal Chukotka. The mortality data from this study correspond to the pattern of incidence reported among other indigenous people of the Russian Arctic (Dudarev et al. 2012a). The incidence of colorectal cancer is currently higher in Alaskan Inuit than in Caucasians living in the United States (Friborg and Melbye 2008). Cancer is now the leading cause of death among Alaska Native people, and cancer mortality rates in Alaska are significantly higher than in the mainland United States (Lanier et al. 2008).



### 3.4.2.7 Lung Cancer

The incidence of lung cancer has increased remarkably in all Inuit populations over the past 40 years. Lung cancer now constitutes about 20% of all cancers in Inuit (Friborg and Melbye 2008). In fact, lung cancer incidence in circumpolar Inuit is among the highest in the world, for men and women. The age-standardized incidence rate of lung and bronchus cancer during 1998–2007 of male Inuit from Nunangat was 113 per 100,000 which is double that for the rest of Canada (50.6 per 100,000) (Carriere et al. 2012). Greenland Inuit have double the standardized incidence rate of lung cancer in Denmark. The smoking pattern among Inuit, possibly combined with co-factors related to environment and diet, are believed to be the relevant causal factors (AMAP 2015). Although modern housing conditions have decreased exposure to fumes from lamps and open fires for cooking, many Inuit still spend substantial periods out on the land, cooking on open stoves inside tents. Marijuana smoking in 85% of adults (of Nunavik, Canada) might also play a role in the high incidence of lung cancer (AMAP 2015).

A cross-sectional study of Alaska Natives has demonstrated the importance of genetic variation in *CYP2A6* on the regulation of tobacco consumption behavior, procarcinogen exposure and metabolism in both light smokers and smokeless tobacco users (Zhu et al. 2013).

### 3.4.2.8 Breast Cancer

Breast cancer is the most common cancer for women in the western world. The established risk factors include genetic inheritance, for example mutations in the *BRCA-1* and *BRCA-2* genes (Ferla et al. 2007), lifelong exposure to estrogens (early menarche and late menopause increases risk), obesity after menopause, alcohol, smoking and high fat intake. Although breast cancer risk is influenced by genetics and reproductive history, the known risk factors explain less than a third of all cases and more than 70% of women diagnosed with breast cancer have no inherited or sporadic cancer. Risk is thought to be modified by lifestyle and environmental exposure (Madigan et al. 1995). Although still lower, incidence is now approaching incidences recorded in Western populations (Friborg et al. 2003) and today about 12–15 women are diagnosed every year in Greenland. From 1988 to 1997, the age-adjusted incidence rate for women in Greenland was 46.4 per 100,000. For comparison, the rate in the United States was 124 per 100,000 for 2001–2008 and in Denmark about 100 per 100,000 in 2010 (Fredslund and Bonefeld-Jørgensen 2012). The age-adjusted incidence rate for breast cancer in Inuit Nunangat was lower than for the rest of Canada (45 vs 81 per 100,000) (Carriere et al. 2012). Although there is no significant difference in breast cancer rate between Alaska Native women and U.S. white women, a significant increase in Alaska Native women was reported during 1974–2003 (Day et al. 2010).

Breast cancer incidence in the Arctic increased between 1969 and 2003 (Fredslund and Bonefeld-Jørgensen 2012). The enormous transition in health conditions and lifestyle in the Arctic might be contributing to the known risk factors. Previous data suggest that exposure to POPs including PFCs might contribute to breast cancer risk. PCBs have been associated with effects relevant breast cancer development such as

estrogenic, tumor promotion (Moysich et al. 2002). Rat studies showed that PFCs cause a significant increase in mammary fibroadenomas (Sibinski 1987). Bonefeld-Jorgensen et al. (2011) evaluated the association between serum levels of POPs/PFCs in 31 cases of breast cancer in Greenlandic Inuit and 115 controls during 2000–2003 to establish whether the combined POP-related effect on nuclear hormone receptors affects risk. Serum levels of POPs, PFCs, some metals and the combined serum POP-related effect on ER-, AR- and AhR-transactivity were determined. For the very first time a significant association between serum PFC levels and the risk of BC was observed. The breast cancer cases also showed a significantly higher concentration of PCBs at the highest quartile. Also, for the combined serum legacy POP-induced agonistic AR transactivity a significant association with breast cancer risk was found, and cases elicited a higher frequency of samples with significant POP-related hormone-like agonistic ER transactivity. The level of serum POPs, particularly PFCs, might be a risk factor in the development of breast cancer in Inuit. Hormone disruption by the combined serum POP-related xenoestrogenic and xenoandrogenic activities may contribute to the risk of developing breast cancer in Inuit (Bonefeld-Jorgensen et al. 2011).

### 3.4.2.9 Ovarian Cancer and Prostate Cancer

The etiology of ovarian cancer is not fully understood. Previous results support the hypothesis of long-term elevated estrogen concentrations as etiologically important for this disease (Spillman et al. 2010). For Arctic populations, the age-standard incidence rate of ovarian cancer among Alaska Native women was significantly lower than for U.S. white women (5.2 vs. 10.5 per 100,000) in 1999–2003.

Risk of prostate cancer in Inuit is 10–20% of the risk in the respective national white population (Friborg et al. 2003; Snyder et al. 2006). A recent study showed that the age-standard incidence rate for prostate cancer during 1998–2007 was lower in the Inuit Nunangat population than in the rest of Canada (17 vs. 85 per 100,000) (Carriere et al. 2012).

### 3.4.2.10 Pancreatic Cancer

A comprehensive meta-analysis has suggested that tobacco smoking, obesity, Type 2 diabetes mellitus and chronic pancreatitis are risk factors for pancreatic cancer. Kirkegaard (2012) reported that the age-standardized incidence rate for pancreatic cancer is 138% higher in Greenland Inuit than in Denmark. This could be partly explained by a higher prevalence of smoking and Type 2 diabetes.

### 3.4.2.11 Genetic Polymorphisms and Contaminants in the Arctic

The Indigenous Arctic population is of Asian descent, and their genetic background is different from that of the Caucasian populations. Relatively little is known about the specific genetic polymorphisms in genes involved in the activation and detoxification

mechanisms of environmental contaminants in Inuit and their relation to health risk. The Greenlandic Inuit are highly exposed to legacy POPs such as PCBs and OC pesticides, and an elucidation of gene-environment interactions in relation to health risks is needed.

Ghisari et al. (2013) compared the genotype and allele frequencies of the cytochrome P450 *CYP1A1* Ile462Val (rs1048943), *CYP1B1* Leu432Val (rs1056836) and catechol-*O*-methyltransferase *COMT* Val158Met (rs4680) in Greenlandic Inuit (n=254) and Europeans (n=262) and explored the possible relation between the genotypes and serum levels of POPs. The genotype and allele frequency distributions of the three genetic polymorphisms differed significantly between the Inuit and Europeans. For Inuit, the genotype distribution was more similar to those reported for Asian populations. A significant difference in serum PCB153 and *p,p'*-DDE levels between Inuit and Europeans was found, and for Inuit associations were also found between POP levels and genotypes for *CYP1A1*, *CYP1B1* and *COMT*. Thus, the data provide new information on gene polymorphisms in Greenlandic Inuit that might support evaluation of susceptibility to environmental contaminants and warrant further studies (Ghisari et al. 2013).

Ghisari et al. (2014) investigated the main effect of polymorphisms in genes involved in xenobiotic metabolism and estrogen biosynthesis, *CYP1A1*, *CYP1B1*, *COMT* and *CYP17*, *CYP19* and the *BRCA1* founder mutation in relation to breast cancer risk, and possible interactions between the gene polymorphisms and serum POP levels on breast cancer risk in Greenlandic Inuit women. They found that the *BRCA1* founder mutation and polymorphisms in *CYP1A1* and *CYP17* can increase breast cancer risk among Inuit women and that risk increases with higher serum levels of PFOS and PFOA. Serum PFAS levels were a consistent risk factor for breast cancer, but inter-individual polymorphic differences might cause variations in sensitivity to the PFAS/POP exposure.

#### 3.4.2.12 Genetics in Relation to Lifestyle Factors in Arctic Populations

Nicotine, the psychoactive ingredient in tobacco, is metabolically inactivated by *CYP2A6* to cotinine. *CYP2A6* also activates procarcinogenic tobacco-specific nitrosamines (TSNA). Genetic variation in *CYP2A6* is known to alter smoking quantity and lung cancer risk in heavy smokers. A study by Zhu et al. (2013) aimed to investigate how *CYP2A6* activity influences tobacco consumption and procarcinogen levels in light smokers and smokeless tobacco users. Cigarette smokers (n=141), commercial smokeless tobacco users (n=73) and iqmik (mixture of tobacco and ash) users (n=20) were recruited in a cross-sectional study of Alaska Native people. The participants' *CYP2A6* activity was measured by both endophenotype and genotype, and their tobacco and procarcinogen exposure biomarker levels were also measured. Smokers, smokeless tobacco users and iqmik users with lower *CYP2A6* activity had lower urinary total nicotine equivalents and (methylnitrosamino)-1-(3)pyridyl-1-butanol (NNAL) levels (a biomarker of TSNA exposure). Levels of N-nitrosonornicotine (NNN), a TSNA metabolically bioactivated by *CYP2A6*, were higher in smokers with lower *CYP2A6* activities. Light smokers and smokeless tobacco users with lower *CYP2A6* activity reduce their tobacco consumption in ways (such as inhaling less

deeply) that are not reflected by self-report indicators. Tobacco users with lower *CYP2A6* activity are exposed to lower procarcinogen levels (lower NNAL levels) and have lower procarcinogen bioactivation (as indicated by the higher urinary NNN levels suggesting reduced clearance), which is consistent with a lower risk of developing smoking-related cancers. This study demonstrates the importance of *CYP2A6* in the regulation of tobacco consumption behaviors, procarcinogen exposure and metabolism in light smokers and smokeless tobacco users (Zhu et al. 2013).

#### 3.4.2.13 Epigenetics

The placenta is central to successful reproductive outcomes. Its functions can be influenced by the environment encountered throughout pregnancy, thereby altering the genetic programming needed to allow sustained pregnancy and appropriate fetal development. This altered programming may result from epigenetic alterations related to environmental contaminant exposure. Epigenetic alterations are now being linked to several important reproductive outcomes, including early pregnancy loss, intrauterine growth restriction, congenital syndromes, preterm birth, and pre-eclampsia. As research continues to enhance understanding of the molecular processes including epigenetic regulation that influence pregnancy, it will be critical to specifically examine how the environment, broadly defined, may play a role in altering these critical functions (Robins et al. 2011).

Rusiecki et al. (2008) analyzed the relationship between plasma POP concentrations and global DNA methylation (percent 5-methylcytosine) in DNA extracted from blood samples from 70 Greenlandic Inuit and used pyrosequencing to estimate global DNA methylation via Alu and LINE-1 assays of bisulfite-treated DNA to evaluate the correlations between plasma POP concentrations and global DNA methylation. This first study investigating environmental exposure to POPs and DNA methylation levels in an Arctic population has shown that global methylation levels are inversely associated with blood plasma levels for several POPs and merit further investigation.

#### 3.4.2.14 Genetic Predisposition and Methylmercury Neurotoxicity

Cognitive consequences at school age associated with prenatal MeHg exposure may need to take into account nutritional and socio-demographic cofactors as well as relevant genetic polymorphisms. Julvez et al. (2013) selected a subsample (n = 1311) of the Avon Longitudinal Study of Parents and Children (Bristol, UK) and measured Hg concentrations in freeze-dried umbilical cord tissue as a measure of MeHg exposure. In this population with low MeHg exposure, associations between MeHg exposure and adverse neuropsychological outcomes were equivocal. Assessment of several relevant genes suggests possible genetic predisposition to MeHg neurotoxicity in a substantial proportion of the population.

### 3.4.2.15 Effect Modifiers

Most environmental research on the effects of chemicals focuses on single exposures. However, exposure to mixtures of chemicals is ubiquitous in real life (Bellinger 2009). Certain chemical substances may target the same organ and induce similar effects in an additive or non-additive way (Carpenter et al. 2002). Recent studies suggest a synergistic effect of metal mixtures with neuropsychological outcomes (Kim et al. 2009) or kidney disease (Navas-Acien et al. 2009). However, studies that examine the effects of chemical mixtures remain limited in humans, and even in experimental animal studies (Carpenter et al. 2002).

Methylmercury, a worldwide contaminant of seafood, can cause adverse effects on the developing nervous system. However, long-chain n-3 PUFAs in seafood provide beneficial effects on brain development. Negative confounding is likely to result in underestimation of both Hg toxicity and nutrient benefits unless mutual adjustment is included in the analysis. In the Faroe Islands cohort studies, associations between prenatal exposure to MeHg and neurobehavioral deficits at school age were strengthened after fatty acid adjustment, thus suggesting that n-3 fatty acids should be included in similar studies to avoid underestimating associations with MeHg exposure (Choi et al. 2014).

Yorifuji et al. (2011) examined the effects of prenatal Pb exposure on cognitive deficits in the presence of a similar molar concentration of a neurotoxic co-pollutant (MeHg) in 7- and 14-year-olds born in the Faroe Islands. Their analyses of the total cohort and those of cohort members without interaction terms among lower co-pollutant exposure categories showed equivocal results. When the subjects were restricted to a lower co-pollutant category, and statistical interaction terms were entered within the category, adverse effects of prenatal Pb exposure on cognitive function in childhood were observed, especially on attention, learning and memory. In the Faroe Islands it has been suggested that another co-pollutant, the PCBs, may affect human brain development through maternal ingestion of contaminated pilot whale blubber.

## 3.5 The Challenge of Climate Change

### 3.5.1 *Combined Effects of Climate Warming, Anthropogenic Contaminants and Zoonotic Disease*

A change in temperature may directly affect community infrastructure and community food and water security. The combined effects of climate warming, anthropogenic contaminants, and zoonotic diseases on rural food and water security also represent a significant risk to the safety of rural subsistence food and water supplies. Many circumpolar communities, particularly remote and small communities, will need to develop monitoring and adaptation strategies to deal with these interacting risks.

- The warming climate has resulted in changes in ocean and atmospheric contaminant transport, which may have increased the movement of anthropogenic contaminants

and Hg from lower latitudes to the Arctic (Kallenborn et al. 2012). Warmer water in freshwater lakes, tundra ponds and streams may result in greater bacterial methylation of Hg, and Hg released from thawing permafrost.

- Climate-mediated changes in the transport of anthropogenic contaminants to the circumpolar region may result in higher exposure in subsistence wildlife which could increase the risk of immunosuppression, and active zoonotic infection in these animals. This could result in risk to human consumers of infection and toxic effects from contaminant exposure (Fisk et al. 2005).
- The warming climate is enabling southern plant, insect and animal species to expand their ranges further north, in some cases, into Arctic regions. These species may bring new zoonotic diseases with them. Higher winter temperatures in the Arctic may increase the winter survival of infected animals, raising the risk of hunter/consumer exposure. The warming climate may drive changes in the forage resources of subsistence species, and thus in the range, health and abundance of those subsistence species. The ecosystem changes could also impact on existing and newly emerging zoonotic pathogens of subsistence species ([www.IPCC.CH/report](http://www.IPCC.CH/report)).

### 3.6 Final Remarks

A summary of known or predicted health endpoints for persistent toxic substances is shown in Box 1. Contaminants are still being transported to and recycled in the Arctic, and levels in certain populations demonstrate that biomonitoring of contaminants of concern should continue. Looking towards the future, it is possible that climate change may affect contaminant exposure in the Arctic (Carrie et al. 2010), and may release contaminants currently held in soil, permafrost or ice. Even so, few differences have yet been seen in the food chain. Changes in food-web structure, feeding habits and diet can affect concentrations of contaminants in biota and humans.

Despite global action through the Stockholm Convention to reduce the production and use of POPs, contaminants are still being transported to and recycled within the Arctic environment ([www.IPCC.CH/report](http://www.IPCC.CH/report); <http://chm.pops.int/>). In addition to long-range transport as a source of contaminants to the Arctic, there is evidence that global warming may affect the cycling of contaminants within the Arctic environment (Carrie et al. 2010), with the potential for release of contaminants currently held in soil, permafrost or ice, although few changes have been seen in the Arctic food chain to date. Nevertheless, changes in the structure and dynamics of the Arctic food web, especially in relation to species forming part of the traditional diet, could have implications for contaminant levels in Arctic populations, with associated impacts on human health. Although time series data sets indicate a current decline in concentration for most POPs in Arctic biota, particularly PCBs and DDT, and contaminants such as PBDEs and PFOS that, prior to 2000 appeared to be increasing now appear to show either no trends or a decrease (AMAP 2015). The potential implications for human health highlight the clear need to continue biomonitoring contaminants of concern. Further biomonitoring

will also aid in measuring the success of international risk management strategies for reducing risk for all Arctic and vulnerable populations. The ongoing cohort studies will continuously add more evidence and knowledge about environmental contaminants and effects on human health and development in the coming years. The precautionary principle must be the central issue to protect the health of the coming generations in the Arctic.

**Acknowledgments** All coauthors of the AMAP Human Health Assessment Report 2015 are to be heartily acknowledged, especially the co-editors Anders Carlsen and Alexey Dudarev, as well as the chapter leads Jennifer Gibson, Pal Weihe, Arja Rautio, Eva Kruemmel, Jim Berner, and Bryan Adlard. All studies mentioned are hopefully properly cited. Possible mistakes are, however, the authors' responsibility.

## References

- AMAP (1998) AMAP assessment report: arctic pollution issues. In: Hansen JC, Gilman A, Klopov V, Odland J (eds) *Pollution and human health. Arctic Monitoring and Assessment Programme (AMAP)*, Oslo, pp 775–844
- AMAP (2003) AMAP assessment 2002: human health in the arctic. In: Hansen JC, Gilman A, Klopov V, Odland JO (eds). *Arctic Monitoring and Assessment Programme (AMAP)*, Oslo
- AMAP (2009) AMAP assessment 2009: human health in the arctic. In: Hansen JC, van Oostdam J, Gilman A, Odland JO, Vaktskjold A, Dudarev A (eds). *Arctic Monitoring and Assessment Programme (AMAP)*, Oslo
- AMAP (2015) AMAP assessment 2015: human health in the arctic. In: Odland JO, Donaldson S, Adlard B (eds) (in press)
- Audet-Delage Y, Ouellet N, Dallaire R, Dewailly E, Ayotte P (2013) Persistent organic pollutants and transthyretin-bound thyroxin in plasma of Inuit women of childbearing age. *Environ Sci Technol* 47(22):13086–13092
- Bellinger DC (2009) Interpreting epidemiologic studies of developmental neurotoxicity: conceptual and analytic issues. *Neurotoxicol Teratol* 31(5):267–274
- Bloom MS, Jansing RL, Kannan K, Rej R, Fitzgerald EF (2014) Thyroid hormones are associated with exposure to persistent organic pollutants in aging residents of upper Hudson River communities. *Int J Hyg Environ Health* 217(4–5):473–482
- Bonde JP, Toft G, Rylander L, Rignell-Hydbom A, Giwercman A, Spano M, Manicardi GC, Bizzaro D, Ludwicki JK, Zvyezday V, Bonefeld-Jorgensen EC, Pedersen HS, Jonsson BA, Thulstrup AM (2008) Fertility and markers of male reproductive function in Inuit and European populations spanning large contrasts in blood levels of persistent organochlorines. *Environ Health Perspect* 116(3):269–277
- Bonefeld-Jorgensen EC, Long M, Bossi R, Ayotte P, Asmund G, Kruger T, Ghisari M, Mulvad G, Kern P, Nzulumiki P, Dewailly E (2011) Perfluorinated compounds are related to breast cancer risk in Greenlandic Inuit: a case control study. *Environ Health* 10:88
- Boucher O, Jacobson SW, Plusquellec P, Dewailly E, Ayotte P, Forget-Dubois N, Jacobson JL, Muckle G (2012) Prenatal methylmercury and postnatal lead exposure: Risk factors for attention deficit hyperactivity disorder among Inuit children in Arctic Quebec. *Environ Health Perspect* 120(10):1456–1461
- Budtz-Jorgensen E, Grandjean P, Weihe P (2007) Separation of risks and benefits of seafood intake. *Environ Health Perspect* 115(3):323–327
- Carpenter DO, Arcaro K, Spink DC (2002) Understanding the human health effects of chemical mixtures. *Environ Health Perspect* 110(Suppl 1):25–42

- Carrie J, Wang F, Sanei H, Macdonald RW, Outridge PM, Stern GA (2010) Increasing contaminant burdens in an Arctic fish, burbot (*Lota lota*) in a warming climate. *Environ Sci Technol* 44:316–322
- Carriere GM, Tjepkema M, Pennock J, Goedhuis N (2012) Cancer patterns in Inuit Nunangat: 1998–2007. *Int J Circumpolar Health* 71:18581
- Chao WY, Hsu CC, Guo YL (1997) Middle-ear disease in children exposed prenatally to polychlorinated biphenyls and polychlorinated dibenzofurans. *Arch Environ Health* 52(4):257–262
- Choi AL, Weihe P, Budtz-Jorgensen E, Jorgensen PJ, Salonen JT, Tuomainen TP, Murata K, Nielsen HP, Petersen MS, Askham J, Grandjean P (2009) Methylmercury exposure and adverse cardiovascular effects in Faroese whaling men. *Environ Health Perspect* 117(3):367–372
- Choi AL, Mogensen UB, Bjerne KS, Debes F, Weihe P, Grandjean P, Budtz-Jorgensen E (2014) Negative confounding by essential fatty acids in methylmercury neurotoxicity associations. *Neurotoxicol Teratol* 42:85–92
- Dalgard C, Petersen MS, Schmedes AV, Brandslund I, Weihe P, Grandjean P (2010) High latitude and marine diet: vitamin D status in elderly Faroese. *Br J Nutr* 104(6):914–918
- Dalgard C, Petersen MS, Weihe P, Grandjean P (2011) Vitamin D status in relation to glucose metabolism and type 2 diabetes in septuagenarians. *Diabetes Care* 34(6):1284–1288
- Dallaire F, Dewailly E, Muckle G, Ayotte P (2003) Time trends of persistent organic pollutants and heavy metals in umbilical cord blood of Inuit infants born in Nunavik (Quebec, Canada) between 1994 and 2001. *Environ Health Perspect* 111(13):1660–1664
- Dallaire F, Dewailly E, Muckle G, Vezina C, Jacobson SW, Jacobson JL, Ayotte P (2004) Acute infections and environmental exposure to organochlorines in Inuit infants from Nunavik. *Environ Health Perspect* 112(14):1359–1365
- Dallaire F, Dewailly E, Vezina C, Muckle G, Weber JP, Bruneau S, Ayotte P (2006) Effect of prenatal exposure to polychlorinated biphenyls on incidence of acute respiratory infections in preschool Inuit children. *Environ Health Perspect* 114(8):1301–1305
- Dallaire R, Dewailly E, Ayotte P, Muckle G, Laliberte C, Bruneau S (2008) Effects of prenatal exposure to organochlorines on thyroid hormone status in newborns from two remote coastal regions in Quebec, Canada. *Environ Res* 108(3):387–392
- Dallaire R, Muckle G, Dewailly E, Jacobson SW, Jacobson JL, Sandanger TM, Sandau CD, Ayotte P (2009) Thyroid hormone levels of pregnant Inuit women and their infants exposed to environmental contaminants. *Environ Health Perspect* 117(6):1014–1020
- Day GE, Lanier AP, Bulkow L, Kelly JJ, Murphy N (2010) Cancers of the breast, uterus, ovary and cervix among Alaska Native women, 1974–2003. *Int J Circumpolar Health* 69(1):72–86
- Debes F, Budtz-Jorgensen E, Weihe P, White RF, Grandjean P (2006) Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. *Neurotoxicol Teratol* 28(5):536–547
- Dewailly E, Bruneau S, Laliberte C, Belles-Isles M, Weber JP, Ayotte P, Roy R (1993) Breast milk contamination by PCBs and PCDDs/PCDFs in arctic Quebec: preliminary results on the immune status of Inuit infants. *Organohalogen Compd* 13:403–406
- Dewailly É, Ayotte P, Bruneau S, Gingras S, Belles-Isles M, Roy R (2000) Susceptibility to infections and immune status in Inuit infants exposed to organochlorines. *Environ Health Perspect* 108:205–211
- Donaldson SG, van Oostdam J, Tikhonov C, Feeley M, Armstrong B, Ayotte P, Boucher O, Bowers W, Chan L, Dallaire F, Dallaire R, Dewailly É, Edwards J, Egeland GM, Fontaine J, Furgal C, Leech T, Loring E, Muckle G, Nancarrow T, Pereg D, Plusquellec P, Potyrala M, Receveur O, Shearer RG (2010) Environmental contaminants and human health in the Canadian Arctic. *Science of the Total Environment* 408:5165–5234
- Dudarev AA, Chupakhin V, Odland JO, Reiersen LO, Chashchin V (2010) A follow-up study of blood levels of persistent toxic substances (PTS) among indigenous peoples of the coastal Chukotka, Russia, 2001–2007. *J Circumpolar Health Supplements* 7
- Dudarev AA (2012) Dietary exposure to persistent organic pollutants and metals among Inuit and Chukchi in Russian Arctic Chukotka. *Int J Circumpolar Health* 71:18592
- Dudarev AA, Chupachin VS, Ivanova ZS, Lebedev GB (2012a) Specificity of exposure of the indigenous dwellers of coastal and inland Chukotka to polychlorinated biphenyls. *Gigiena i Sanitariia* 4:22–28 [In Russian]



- Dudarev AA, Mizernyuk VN, Nikanov AN (2012) Persistent organic pollutants and diabetes mellitus in the indigenous population and newcomers to Kola Lapland. *Toxicol Vesnik* 4:7–13
- Dufour R (1988) The otitis media among Inuit children. Proposal for a new approach. *Arctic Med Res* 47(Suppl 1):659–665
- Elzanaty S, Rignell-Hydbom A, Jonsson BA, Pedersen HS, Ludwicki JK, Shevets M, Zveyezday V, Toft G, Bonde JP, Rylander L, Hagmar L, Bonefeld-Jorgensen E, Spano M, Bizzaro D, Manicardi GC, Giwercman A (2006) Association between exposure to persistent organohalogen pollutants and epididymal and accessory sex gland function: multicentre study in Inuit and European populations. *Reprod Toxicol* 22(4):765–773
- Everett CJ, Matheson EM (2010) Biomarkers of pesticide exposure and diabetes in the 1999–2004 national health and nutrition examination survey. *Environ Int* 36(4):398–401
- Ferla R, Calo V, Cascio S, Rinaldi G, Badalamenti G, Carreca I, Surmacz E, Colucci G, Bazan V, Russo A (2007) Founder mutations in BRCA1 and BRCA2 genes. *Ann Oncol* 18(Suppl 6):vi93–98
- Fisk A, DeWit C, Wayland M, Kuzyk Z, Burgess N, Letcher R, Braune B, Blum S, Norstrom R, Lie E, Larson N, Skaare J, Muir D (2005) An assessment of the toxicological significance of anthropogenic contaminants in Canadian Arctic wildlife. *Sci Total Environ* 351–353:57–93
- Fredslund SO, Bonefeld-Jørgensen EC (2012) Breast cancer in the Arctic – changes over the past decades. *Int J Circumpolar Health* 71:19155
- Friberg JT, Melbye M (2008) Cancer patterns in Inuit populations. *Lancet Oncol* 9(9):892–900
- Friberg J, Koch A, Wohlfahrt J, Storm HH, Melbye M (2003) Cancer in Greenlandic Inuit 1973–1997: a cohort study. *Int J Cancer* 107(6):1017–1022
- Geier DA, Kern JK, King PG, Sykes LK, Geier MR (2012) Hair toxic metal concentrations and autism spectrum disorder severity in young children. *Int J Environ Res Public Health* 9(12):4486–4497
- Ghisari M, Long M, Bonefeld-Jørgensen EC (2013) Genetic polymorphisms in CYP1A1, CYP1B1 and COMT genes in Greenlandic Inuit and Europeans. *Int J Circumpolar Health* 72:21113
- Ghisari M, Eiberg H, Long M, Bonefeld-Jørgensen EC (2014) Polymorphisms in Phase I and Phase II genes and breast cancer risk and relations to persistent organic pollutant exposure: a case–control study in Inuit women. *Environ Health* 13(1):19
- Giwercman AH, Rignell-Hydbom A, Toft G, Rylander L, Hagmar L, Lindh C, Pedersen HS, Ludwicki JK, Lesovoy V, Shvets M, Spano M, Manicardi GC, Bizzaro D, Bonefeld-Jørgensen EC, Bonde JP (2006) Reproductive hormone levels in men exposed to persistent organohalogen pollutants: a study of Inuit and three European cohorts. *Environ Health Perspect* 114(9):1348–1353
- Grandjean P (2013) Only one chance. How environmental pollution impairs brain development—and how to protect the brains of the next generation. Oxford University Press, New York
- Grandjean P, Herz KT (2011) Methylmercury and brain development: imprecision and underestimation of developmental neurotoxicity in humans. *Mt Sinai J Med* 78(1):107–118
- Grandjean P, Weihe P, Jørgensen PJ, Clarkson T, Cernichiari W, Videro T (1992) Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. *Arch Environ Health* 47:185–195
- Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, Murata K, Sorensen N, Dahl R, Jørgensen PJ (1997) Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol Teratol* 19(6):417–442
- Grandjean P, Murata K, Budtz-Jørgensen E, Weihe P (2004) Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort. *J Pediatr* 144(2):169–17
- Grandjean P, Henriksen JE, Choi AL, Petersen MS, Dalgard C, Nielsen F, Weihe P (2011) Marine food pollutants as a risk factor for hypoinsulinemia and type 2 diabetes. *Epidemiology* 22(3):410–417
- Grandjean P, Weihe P, Nielsen F, Heinzow B, Debes F, Budtz-Jørgensen E (2012a) Neurobehavioral deficits at age 7 years associated with prenatal exposure to toxicants from maternal seafood diet. *Neurotoxicol Teratol* 34(4):466–472
- Grandjean P, Andersen EW, Budtz-Jørgensen E, Nielsen F, Mølbak K, Weihe P, Heilmann C (2012b) Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. *JAMA* 307:391–397
- Grandjean P, Gronlund C, Kjaer IM, Jensen TK, Sorensen N, Andersson AM, Juul A, Skakkebaek NE, Budtz-Jørgensen E, Weihe P (2012c) Reproductive hormone profile and

- pubertal development in 14-year-old boys prenatally exposed to polychlorinated biphenyls. *Reprod Toxicol* 34(4):498–503
- Halling J, Petersen MS, Jorgensen N, Jensen TK, Grandjean P, Weihe P (2013) Semen quality and reproductive hormones in Faroese men: a cross-sectional population-based study of 481 men. *BMJ Open* 3(3)
- Hansen S, Nieboer E, Odland JO, Wilsgaard T, Veyhe AS, Sandanger TM (2010) Levels of organochlorines and lipids across pregnancy, delivery and postpartum periods in women from Northern Norway. *J Environ Monit* 12(11):2128–2137
- Hansen S, Nieboer E, Sandanger TM, Wilsgaard T, Thomassen Y, Veyhe AS, Odland JO (2011) Changes in maternal blood concentrations of selected essential and toxic elements during and after pregnancy. *J Environ Monit* 13(8):2143–2152
- Haugen TB, Tefre T, Malm G, Jonsson BA, Rylander L, Hagmar L, Bjorsvik C, Henrichsen T, Saether T, Figenschau Y, Giwercman A (2011) Differences in serum levels of CB-153 and p,p'-DDE, and reproductive parameters between men living south and north in Norway. *Reprod Toxicol* 32(3):261–267
- Hauser R, Altshul L, Chen Z, Ryan L, Overstreet J, Schiff I, Christiani DC (2002) Environmental organochlorines and semen quality: results of a pilot study. *Environ Health Perspect* 110(3):229–233
- Heilmann C, Grandjean P, Weihe P, Nielsen F, Budtz-Jorgensen E (2006) Reduced antibody responses to vaccinations in children exposed to polychlorinated biphenyls. *PLoS Med* 3(8), e311
- Heilmann C, Budtz-Jorgensen E, Nielsen F, Heinzow B, Weihe P, Grandjean P (2010) Serum concentrations of antibodies against vaccine toxoids in children exposed perinatally to immunotoxicants. *Environ Health Perspect* 118(10):1434–1438
- Joensen UN, Jorgensen N, Rajpert-De Meyts E, Skakkebaek NE (2008) Testicular dysgenesis syndrome and Leydig cell function. *Basic Clin Pharmacol Toxicol* 102(2):155–161
- Jorgensen N, Carlsen E, Nermoen I, Punab M, Suominen J, Andersen AG, Andersson AM, Haugen TB, Horte A, Jensen TK, Magnus O, Petersen JH, Vierula M, Toppari J, Skakkebaek NE (2002) East–west gradient in semen quality in the Nordic-Baltic area: a study of men from the general population in Denmark, Norway, Estonia and Finland. *Hum Reprod* 17(8):2199–2208
- Julvez J, Smith GD, Golding J, Ring S, Pourcain BS, Gonzalez JR, Grandjean P (2013) Prenatal methylmercury exposure and genetic predisposition to cognitive deficit at age 8 years. *Epidemiology* 24(5):643–650
- Jusko TA, De Roos AJ, Schwartz SM, Lawrence BP, Palkovicova L, Nemessanyi T, Drobna B, Fabisikova A, Kocan A, Sonneborn D, Jahnova E, Kavanagh TJ, Trnovec T, Hertz-Picciotto I (2010) A cohort study of developmental polychlorinated biphenyl (PCB) exposure in relation to post-vaccination antibody response at 6-months of age. *Environ Res* 110(4):388–395
- Kallenborn R, Halsall C, Dellong M, Carlsson P (2012) The influence of global distribution on fate processes and distribution of anthropogenic persistent organic pollutants. *J Environ Monit* 14(11):2854–2869
- Kim Y, Kim BN, Hong YC, Shin MS, Yoo HJ, Kim JW, Bhang SY, Cho SC (2009) Co-exposure to environmental lead and manganese affects the intelligence of school-aged children. *Neurotoxicology* 30(4):564–571
- Kirkegaard J (2012) Incidence of pancreatic cancer in Greenland 2000–2010. *Int J Circumpolar Health* 71:18368
- Laird BD, Goncharov AB, Chan HM (2013) Body burden of metals and persistent organic pollutants among Inuit in the Canadian Arctic. *Environ Int* 59:33–40
- Lanier AP, Day GE, Kelly JJ, Provost E (2008) Disparities in cancer mortality among Alaska Native people, 1994–2003. *Alaska Med* 49(4):120–125
- Lee DH, Lee IK, Porta M, Steffes M, Jacobs DR Jr (2007) Relationship between serum concentrations of persistent organic pollutants and the prevalence of metabolic syndrome among non-diabetic adults: results from the National Health and Nutrition Examination Survey 1999–2002. *Diabetologia* 50(9):1841–1851
- Madigan MP, Ziegler RG, Benichou J, Byrne C, Hoover RN (1995) Proportion of breast cancer cases in the United States explained by well-established risk factors. *J Natl Cancer Inst* 87(22):1681–1685

- Moysich KB, Menezes RJ, Baker JA, Falkner KL (2002) Environmental exposure to polychlorinated biphenyls and breast cancer risk. *Rev Environ Health* 17(4):263–277
- Mozaffarian D, Shi P, Morris JS, Spiegelman D, Grandjean P, Siscovick DS, Willett WC, Rimm EB (2011) Mercury exposure and risk of cardiovascular disease in two U.S. cohorts. *N Engl J Med* 364(12):1116–1125
- Mozaffarian D, Shi P, Morris JS, Grandjean P, Siscovick DS, Spiegelman D, Willett WC, Rimm EB, Curhan GC, Forman JP (2012) Mercury exposure and risk of hypertension in US men and women in 2 prospective cohorts. *Hypertension* 60(3):645–652
- Muckle G, Dewailly E, Ayotte P (1998) Prenatal exposure of Canadian children to polychlorinated biphenyls and mercury. *Can J Public Health* 89(Suppl 1):22–27
- Murata K, Weihe P, Budtz-Jorgensen E, Jorgensen PJ, Grandjean P (2004) Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury. *J Pediatr* 144(2):177–183
- Navas-Acien A, Umans JG, Howard BV, Goessler W, Francesconi KA, Crainiceanu CM, Silbergeld EK, Guallar E (2009) Urine arsenic concentrations and species excretion patterns in American Indian communities over a 10-year period: the Strong Heart Study. *Environ Health Perspect* 117(9):1428–1433
- Nielsen AB, Davidsen M, Bjerregaard P (2012) The association between blood pressure and whole blood methylmercury in a cross-sectional study among Inuit in Greenland. *Environ Health* 11:44
- Nøst TH, Breivik K, Fuskevåg OM, Nieboer E, Odland JØ, Sandanger TM (2013) Persistent organic pollutants in Norwegian men from 1979 to 2007: intraindividual changes, age-period-cohort effects, and model predictions. *Environ Health Perspect* 121(11–12):1292–1298
- Nøst TH, Vestergren R, Berg V, Nieboer E, Odland JØ, Sandanger TM (2014) Repeated measurements of per- and polyfluoroalkyl substances (PFASs) from 1979 to 2007 in males from Northern Norway: assessing time trends, compound correlations and relations to age/birth cohort. *Environ Int* 67:43–53
- Olsen GW, Burris JM, Ehresman DJ, Froehlich JW, Seacat AM, Butenhoff JL, Zobel LR (2007) Half-life of serum elimination of perfluorooctanesulfonate, perfluorohexanesulfonate, and perfluorooctanoate in retired fluorochemical production workers. *Environ Health Perspect* 115(9):1298–1305
- Patel CJ, Bhattacharya J, Butte AJ (2010) An Environment-Wide Association Study (EWAS) on type 2 diabetes mellitus. *PLoS One* 5(5), e10746
- Petersen MS, Halling J, Bech S, Wermuth L, Weihe P, Nielsen F, Jorgensen PJ, Budtz-Jorgensen E, Grandjean P (2008) Impact of dietary exposure to food contaminants on the risk of Parkinson's disease. *Neurotoxicology* 29(4):584–590
- Punab M, Zilaitiene B, Jorgensen N, Horte A, Matulevicius V, Peetsalu A, Skakkebaek NE (2002) Regional differences in semen qualities in the Baltic region. *Int J Androl* 25(4):243–252
- Richthoff J, Rylander L, Hagmar L, Malm J, Giwercman A (2002) Higher sperm counts in Southern Sweden compared with Denmark. *Hum Reprod* 17(9):2468–2473
- Richthoff J, Rylander L, Jonsson BA, Akesson H, Hagmar L, Nilsson-Ehle P, Stridsberg M, Giwercman A (2003) Serum levels of 2,2',4,4',5,5'-hexachlorobiphenyl (CB-153) in relation to markers of reproductive function in young males from the general Swedish population. *Environ Health Perspect* 111(4):409–413
- Robins JC, Marsit CJ, Padbury JF, Sharma SS (2011) Endocrine disruptors, environmental oxygen, epigenetics and pregnancy. *Front Biosci (Elite Ed)* 3:690–700
- Roman HA, Walsh TL, Coull BA, Dewailly E, Guallar E, Hattis D, Marien K, Schwartz J, Stern AH, Virtanen JK, Rice G (2011) Evaluation of the cardiovascular effects of methylmercury exposures: current evidence supports development of a dose–response function for regulatory benefits analysis. *Environ Health Perspect* 119(5):607–614
- Rusiecki JA, Baccarelli A, Bollati V, Tarantini L, Moore LE, Bonfeld-Jorgensen EC (2008) Global DNA hypomethylation is associated with high serum-persistent organic pollutants in Greenlandic Inuit. *Environ Health Perspect* 116(11):1547–1552

- Schell LM, Gallo MV, Ravenscroft J, DeCaprio AP (2009) Persistent organic pollutants and anti-thyroid peroxidase levels in Akwesasne Mohawk young adults. *Environ Res* 109(1):86–92
- Schug TT, Janesick A, Blumberg B, Heindel JJ (2011) Endocrine disrupting chemicals and disease susceptibility. *J Steroid Biochem Mol Biol* 127(3–5):204–215
- Sibinski LJ (1987) Final report of a two year oral (diet) toxicity and carcinogenicity study of fluorochemical FC-143 (perfluorooctanane ammonium carboxylate) in rats. 3 M Company/RIKER Exp. No. 0281CR0012; 8EHQ-1087-0394, 1–4
- Snyder OB, Kelly JJ, Lanier AP (2006) Prostate cancer in Alaska Native men, 1969–2003. *Int J Circumpolar Health* 65(1):8–17
- Sorensen N, Murata K, Budtz-Jorgensen E, Weihe P, Grandjean P (1999) Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age. *Epidemiology* 10:370–375
- Spillman MA, Manning NG, Dye WW, Sartorius CA, Post MD, Harrell JC, Jacobsen BM, Horwitz KB (2010) Tissue-specific pathways for estrogen regulation of ovarian cancer growth and metastasis. *Cancer Res* 70(21):8927–8936. doi:10.1158/0008-5472.CAN-10-1238, Epub 19 Oct 2010
- Swan SH, Elkin EP, Fenster L (1997) Have sperm densities declined? A reanalysis of global trend data. *Environ Health Perspect* 105(11):1228–1232
- Swan SH, Elkin EP, Fenster L (2000) The question of declining sperm density revisited: an analysis of 101 studies published 1934–1996. *Environ Health Perspect* 108(10):961–966
- ten Tusscher GW, Steerenberg PA, van Loveren H, Vos JG, von dem Borne AE, Westra M, van der Slikke JW, Olie K, Pluim HJ, Koppe JG (2003) Persistent hematologic and immunologic disturbances in 8-yearold Dutch children associated with perinatal dioxin exposure. *Environ Health Perspect* 111:1519–1523
- Thurston SW, Bovet P, Myers G, Davidson PW, Georger LA, Shamlaye C, Clarkson TW (2007) Does prenatal methylmercury exposure from fish consumption affect blood pressure in childhood? *Neurotoxicology* 28:924–930
- Toft G, Pedersen HS, Bonde JP (2004) Semen quality in Greenland. *Int J Circumpolar Health* 63(Suppl 2):174–178
- Toft G, Rignell-Hydbom A, Tyrkiel E, Shvets M, Giwercman A, Lindh CH, Pedersen HS, Ludwicki JK, Lesovoy V, Hagmar L, Spano M, Manicardi GC, Bonefeld-Jorgensen EC, Thulstrup AM, Bonde JP (2006) Semen quality and exposure to persistent organochlorine pollutants. *Epidemiology* 17(4):450–458
- Valera B, Dewailly E, Poirier P (2008) Cardiac autonomic activity and blood pressure among Nunavik Inuit adults exposed to environmental mercury: a cross-sectional study. *Environ Health* 7:29
- Valera B, Dewailly E, Poirier P (2009) Environmental mercury exposure and blood pressure among Nunavik Inuit adults. *Hypertension* 54(5):981–986
- Valera B, Dewailly E, Poirier P (2011) Impact of mercury exposure on blood pressure and cardiac autonomic activity among Cree adults (James Bay, Quebec, Canada). *Environ Res* 111(8):1265–1270
- Valera B, Muckle G, Poirier P, Jacobson SW, Jacobson JL, Dewailly É (2012) Cardiac autonomic activity and blood pressure among Inuit children exposed to mercury. *Neurotoxicology* 33:1067–1074
- Valera B, Dewailly E, Poirier P (2013) Association between methylmercury and cardiovascular risk factors in a native population of Quebec (Canada): a retrospective evaluation. *Environ Res* 120:102–108
- Veyhe AS, Hansen S, Sandanger TM, Nieboer E, Odland JO (2012) The Northern Norway mother-and-child contaminant cohort study: implementation, population characteristics and summary of dietary findings. *Int J Circumpolar Health* 71:18644
- Vine MF, Stein L, Weigle K, Schroeder J, Degnan D, Tse CK, Backer L (2001) Plasma 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) levels and immune response. *Am J Epidemiol* 153(1):53–63
- Virtanen JK, Laukkanen JA, Mursu J, Voutilainen S, Tuomainen TP (2012) Serum long-chain n-3 polyunsaturated fatty acids, mercury, and risk of sudden cardiac death in men: a prospective population-based study. *PLoS One* 7(7), e41046

- Weber JP (2002) The AMAP ring test: a laboratory intercomparison program for POPs in human serum. Presented at the AMAP conference and workshop: impacts of POPs and Mercury on Arctic environments and humans, Tromsø
- Weisglas-Kuperus N, Sas TC, Koopman-Esseboom C, van der Zwan CW, De Ridder MA, Beishuizen A, Hooijkaas H, Sauer PJ (1995) Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. *Pediatr Res* 38(3):404–410
- Weisglas-Kuperus N, Patandin S, Berbers GA, Sas TC, Mulder PG, Sauer PJ, Hooijkaas H (2000) Immunologic effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. *Environ Health Perspect* 108(12):1203–1207
- Yang M, Park MS, Lee HS (2006) Endocrine disrupting chemicals: human exposure and health risks. *J Environ Sci Health C Environ Carcinog Ecotoxicol Rev* 24(2):183–224
- Yorifuji T, Debes F, Weihe P, Grandjean P (2011) Prenatal exposure to lead and cognitive deficit in 7- and 14-year-old children in the presence of concomitant exposure to similar molar concentration of methylmercury. *Neurotoxicol Teratol* 33(2):205–211
- Zhu AZ, Binnington MJ, Renner CC, Lanier AP, Hatsukami DK, Stepanov I, Watson CH, Sosnoff CS, Benowitz NL, Tyndale RF (2013) Alaska Native smokers and smokeless tobacco users with slower CYP2A6 activity have lower tobacco consumption, lower tobacco-specific nitrosamine exposure and lower tobacco-specific nitrosamine bioactivation. *Carcinogenesis* 34(1):93–101

## Chapter 4

# Effects of Pollutant Exposure on Human Health as Studied with Selected EU Projects

Arja Rautio

**Abstract** Investigations on the precise impact of environmental pollutants on human health are difficult to study, because many factors, like genetic background, age, sex, diseases, exposure history and environment, affect at the same time and to varying degrees. Environmental factors include mixtures of contaminants and other chemical compounds to which individuals are commonly exposed. Knowledge about the effects of mixtures is mainly missing and difficult to study at population level. The incidence of many diseases has increased and it has been assumed that pollutants may have a contributing effect especially in the exposed populations.

Exposure during the fetal period is in special attention in the epidemiological and mechanistic (in vitro) studies, because fetal stage is the most vulnerable during human life. During the last decade there have been several research projects under the framework of the EU, which have focused on potential toxicity at low exposure levels of environmental contaminants to child development (like ENRICO, OBELIX, CLEAR, INUENDO, ArcRisk, PHIME). One goal of these research programmes has been to collect all the existing evidence about the associations between environmental contaminants and measured health outcomes. In this chapter the main aims and results of these projects will be introduced and discussed. The evaluation of potential health risks and their magnitude is needed.

**Keywords** Environmental health • Contaminants • Toxic metals • Epidemiological studies • Risk assessment • Exposure

---

A. Rautio (✉)

Centre for Arctic Medicine, Thule Institute, University of Oulu, Oulu, Finland  
e-mail: [arja.rautio@oulu.fi](mailto:arja.rautio@oulu.fi)

## Abbreviations

AMAP	Arctic Monitoring and Assessment Program
ArcRisk	Arctic Health Risks: Impacts on health in the Arctic and Europe owing to climate-induced changes in contaminant cycling—research project
CHICOS	Developing a Child Cohort Research Strategy for Europe—research project
CLEAR	Comparison of health risks in populations in the Arctic and selected areas in Europe due to the spreading of contaminants resulting from climate change; Climate change, Environmental contaminants and Reproductive health—research project
COPHES	Consortium to perform human biomonitoring on a European scale—research project
DALY	Disability-adjusted life years
DDE	Metabolite of DDT
DDT	Dichlorodiphenyltrichloroethane
DEMOCOPHES	Demonstration of a study to coordinate and perform human biomonitoring on a European scale—research project
EBoDE	Environmental Burden of Disease in European countries—research project
EDC	Endocrine disrupting compounds
ENRIECO	Environmental Health Risks in European Birth Cohorts—research project
EU	European Union
HBM	Human Biomonitoring Value
INUENDO	Biopersistent organochlorines in diet and human fertility: Epidemiological studies of time-of-pregnancy and semen quality in Inuit and European populations—research project
OBELIX	OBesogenic Endocrine disrupting chemicals: LInking prenatal eXposure to the development of obesity later in life—research project
PCB153	Polychlorobiphenyl 153
PHIME	Public Health Impact of long-term, low-level Mixed element Exposure in susceptible population strata—research project
POPs	Persistent Organic/toxic Pollutants

### 4.1 Approaches to Describe Risks for Human Health

Environmental Burden of Disease in European countries (EBoDE) project evaluated nine environmental risk factors in six European countries (Belgium, Finland, France, Germany, Italy and the Netherlands), for making integrated

measures of environmental health effects to be used for setting priorities in environmental health policies and research (Hänninen et al. 2014). Disability-adjusted life years (DALYs) were estimated for benzene, dioxins, secondhand smoke, formaldehyde, lead, traffic noise, ozone, particulate matter (PM<sub>2.5</sub>). Airborne particulate matter was the leading risk factor for human health, followed by secondhand smoke and traffic noise. Radon, dioxins and formaldehyde were in the middle class of risk factors. As main conclusions, the researchers point out the importance to produce information for health policies and research needs, resource allocation, identification of susceptible groups, and targets for efficient exposure reduction, and international exposure monitoring standards would enhance data quality and improve comparability.

Studies on the impact of environmental pollutants on human health are difficult to undertake and interpret, since there are several confounding factors influencing health at the same time. The most important ones are genetic and other individual factors (like age, gender, diseases). From the environment where we live there are many chemicals affecting at the same time, and the effects of the mixtures of chemical compounds are difficult to study, and we have very little knowledge about those. Environmental health effects may vary considerably with regard to their severity and type of disease. The most sensitive groups are pregnant mothers, fetuses and children and usually those groups have been under research.

One of extreme interesting region is Arctic. It has been found that there are a lot of chemicals which are transported to the North by winds or with ocean currents depending their chemical structure and properties. In the Arctic the persistent organic/toxic pollutants (POPs) stay in the environment, accumulate in food chains through which humans are exposed. At the moment many of those POPs have already banned, but all the time there will be new chemical compounds which have the same kind of chemical characteristics which could have possibility to transfer and stay in the Arctic regions. During the last 20 years there have been in the Arctic a monitoring program for the blood levels of POPs and toxic metals in pregnant women (Arctic Monitoring and Assessment Program, AMAP) in all Arctic countries, and many child-mother cohorts have been established to research the health of newborns and child development. There have been published already several reports, and the newest one is from 2015 (AMAP 2015), where the time trends of many POPs and toxic metals have been found to decline. It also shows the importance of global actions, like restrictions and bans of POPs and toxic metals, to reduce emissions of contaminants. However, many of those still exist in the soil and ice in the Arctic and they may release to the food chains, especially due to warming climate.

## 4.2 Research Projects of European Community

During the last years there have been several attempts to approach the important question about the environmental contaminants and their risks for human health. Within the framework of the EU Sixth and Seventh Framework Programmes



**Table 4.1** Environmental contaminants and human health outcomes, EU-funded research projects

---

*ArcRisk*, Arctic Health Risks: Impacts on health in the Arctic and Europe owing to climate-induced changes in contaminant cycling, 2009–2014; [www.arcrisk.eu](http://www.arcrisk.eu)

---

*CHICOS*, Developing a Child Cohort Research Strategy for Europe, 2010–2013, <http://www.chicosproject.eu/>

---

*CLEAR*, Comparison of health risks in populations in the Arctic and selected areas in Europe due to the spreading of contaminants resulting from climate change; Climate change, Environmental contaminants and Reproductive health, 2009–2013; and *INUENDO*, Biopersistent organochlorines in diet and human fertility. Epidemiological studies of time-of-pregnancy and semen quality in Inuit and European populations, 2002–2005

---

*COPHES and DEMOCOPHES*, Consortium to perform human biomonitoring on a European scale, 2004–2010 and *DEMOCOPHES*, Demonstration of a study to coordinate and perform human biomonitoring on a European scale, 2010–2012, <http://www.eu-hbm.info/cophes/news/project-completed>

---

*ENRIECO*, Environmental Health Risks in European Birth Cohorts, 2009–2011, <http://enrieco.org>

---

*OBELIX*, OBesogenic Endocrine disrupting chemicals: LInking prenatal eXposure to the development of obesity later in life, 2009–2013, <http://www.theobelixproject.org/>

---

*PHIME*, Public Health Impact of long-term, low-level Mixed element Exposure in susceptible population strata, 2006–2011, [http://www.med.lu.se/labmedlund/amm/forskning/haelsorisker\\_av\\_metaller/phime](http://www.med.lu.se/labmedlund/amm/forskning/haelsorisker_av_metaller/phime)

---

(2007–2013) there were several projects focusing on the effects of environmental contaminants on human health. Among those could be mentioned nine projects which are more detailed discussed in this Chapter (Table 4.1).

### 4.2.1 *ArcRisk*

The *ArcRisk* project was looking at the linkages between environmental contaminants, climate change and human health and aimed at supporting European policy development in these areas. The key findings in the *ArcRisk* projects are (see more [www.arcrisk.eu](http://www.arcrisk.eu)):

Model simulations conducted under *ArcRisk* suggest that climate change will affect contaminant pathways, mobility and levels in the Arctic and elsewhere. Both legacy and more recently introduced organic chemicals can be found in Arctic marine foodstuffs which are major source for exposure, and much of this contamination is due to long-range transport. Health risks associated with contaminant exposure are greatest among the vulnerable groups, like developing fetus and children and there should be dietary guidance to indigenous populations in relation to chemical contaminants in traditional goods. However, it is difficult to link health outcomes to specific contaminants and harmonization of study protocols are needed for estimation of the links of health effects and exposure.

One important part of the *ArcRisk* project was the policy recommendations, which focus also on the future actions. It is necessary to keep in mind that the avoiding future adverse effects on human health from exposure to contaminants

requires an overall strategy that integrates policies and measures to reduce use and releases of contaminants, monitoring of environmental levels and human exposure, education and risk communication, and where necessary food consumption advice to critical groups.

### **4.2.2 CHICOS**

The main aim of the CHICOS was to develop an integrated strategy for birth cohort research in Europe for the next 15 years through coordination of the most important European birth cohorts. The CHICOS recommendations included support for establishing the infrastructure for a European-wide database platform, including new cohorts that cover groups of European population that are underrepresented in birth cohort research, continuing follow-up of existing European cohorts, combining data from birth cohorts, routine registries, and other data sources, and integrating knowledge translation and public and policy engagement (see more, <http://www.chicosproject.eu/>).

### **4.2.3 CLEAR and INUENDO**

The aim of the CLEAR project was to investigate the possible impact of global climate change on reproductive health in Arctic and European populations. There were three cohorts consisted of 1400 pregnant women and 600 spouses from Greenland, Poland and Ukraine. The cohorts were established during the INUENDO project and followed-up in the CLEAR project and also more samples were collected for analyses of new contaminants (like brominated and fluorinated contaminants, phthalates and bisphenol A) and follow-up was done. The main results of the CLEAR project (see more [http://cordis.europa.eu/project/rcn/92242\\_en.html](http://cordis.europa.eu/project/rcn/92242_en.html)) were:

The study demonstrated that climate change will not directly cause major changes in environmental POP exposure levels in the Arctic, and it did not indicate that adult exposure to current levels of environmental contaminants have marked influence on male and female fertility or child growth and development. The researchers emphasized that CLEAR study has examined male reproductive health in much more detail than female reproductive health and child development, and pronounced long-term effects of fetal exposure on adult reproductive health has not been addressed.

The researchers found some polymorphisms in androgen and estrogen receptors and enzymes which may have influence on the exposure levels of contaminants. There were several methodological challenges in testing the significance of associations between exposures and health outcomes. According to the toxicokinetic model developed in the CLEAR project, the duration of exclusive breastfeeding, birth weight and maternal blood levels were the most important predictors of child exposure (Verner et al. 2013), and modeling exposure-response relationships for multiple, correlated exposures (exposure mixtures) was introduced (Lenters et al. 2015).

The risk assessment was difficult, since there were statistical robustness of exposure-response associations, clinical relevance of outcomes, biological plausibility, causal inference and potential sources of bias, and weight of the evidence based on systematic literature reviews.

#### **4.2.4 *COPHES and DEMOCOPHES***

The projects of the EU framework for human biomonitoring surveys, like COPHES and DEMOCOPHES, have collected the experts from different European countries to work together in the questions of exposure characterization and risk assessment. In COPHES/DEMOCOPHES projects performed the first harmonized human biomonitoring survey in 13 out of 17 European countries (1800 mother-child pairs) by harmonized protocols and procedures e.g. in mercury levels correlated to national fish consumption and urinary cadmium levels were associated with environmental quality and food quality.

The results of these projects showed that combining human biomonitoring and environmental data provided added value for (the evaluation of) evidence-informed policy making, and more collaboration with applications of new technologies and transnational research are important on the way forward (see more e.g. Smolders et al. 2014; Casteleyn et al. 2015).

#### **4.2.5 *ENRIECO***

European birth cohorts collect a wealth of data on environmental exposures in the past and ongoing studies funded by EC and national programs. The aim of the ENRIECO project was to increase knowledge on specific environment and health causal relationships in pregnancy and birth cohorts, and improve the use of existing data and facilitate collaboration among these studies (altogether 37 European birth cohorts were involved, see <http://www.enrieco.org/>). Collaborative analyses from existing data of several European birth cohorts have done from outdoor air pollution, water contamination, allergens, biological contaminants, molds, POPs and tobacco smoke exposure (Gehring et al. 2013).

The main reasons for collaboration between population studies are replication, studying heterogeneity, and increasing statistical power to study small relative risks, rare events and complex interactions. Key success factors for collaborative analyses are common definitions of main exposure and health variables. As conclusions, there is a need for harmonization of study plans (aims, protocols, data, health outcomes, confounding factors) and methodologies used and also in environmental exposure assessment.

### **4.2.6 OBELIX**

The OBELIX project investigated if prenatal exposure to endocrine disrupting compounds (EDC), like dioxins, in food plays a role in the development of obesity and related disorders later in life. Multidisciplinary approach with epidemiology, neonatology, endocrinology, toxicology, analytical chemistry and risk assessment was used. EDCs may mimic the actions of hormones in pregnancy and childhood, and they are also suspected to promote obesity by disturbing metabolic and endocrine pathways in body. By animal and mechanistic (in vitro) studies EDCs affected body weight and adipose cell functions and DNA demethylation. The OBELIX project concluded that perinatal exposure to low levels of EDCs affects endocrine signalling pathways that may lead to changes in body weight (Iszatt et al. 2015).

### **4.2.7 PHIME**

The aim of the PHIME project was to study if the exposure levels of the general population in Europe and other countries cause toxic effects in susceptible individuals. The exposure to lead and cadmium seems to be rather similar in many European countries. The exposure to mercury differs, according to varying fish intake and amalgam fillings. The levels of cadmium seem to be stable, and there was association of low-level cadmium and decrease of bone mineral density.

The PHIME project has also increased information about the molecular mechanisms for the uptake of metals in plants. Individuals may be at different risk due to their genetic background, the toxicokinetics of mercury, arsenic, lead and cadmium was changed, and the toxicodynamics of arsenic, lead, cadmium and manganese according the genetic variations (Visnjevec et al. 2014). All this should be considered in risk assessment and prevention, and the risk may be varied between individuals and populations.

## **4.3 Networks for Biomonitoring and Joint Studies with Pooled Samples**

In many of the EU-funded projects the focus have been in forming networks for following, reaching and giving advice to the policy-makers about the situation of environmental contaminants and their risks for human health in Europe, Arctic and also in other countries. These projects suggest more collaboration, harmonizing of the human biomonitoring programs and study designs, forming joint databases from national cohorts and making pooled samples for having more evidence about the causal relationship between contaminant levels and human health.

The advantages of the joint database are clearly seen, e.g. the product of the ENRIECO and OBELIX and in collaboration with ArcRisk and CLEAR researchers

was a study about the effects of PCBs and DDE on birth weight (Govarts et al. 2012). This database covered maternal and cord blood and breast milk samples of 7990 women enrolled in 15 study populations from 1990 through 2008. Using identical variable definitions, the authors performed a linear regression of birth weight on estimates of cord serum concentration of PCB153 and *p,p'*-DDE, adjusted for gestational age and selected covariates for each cohort. The summary estimates by meta-analysis and performed analyses of interactions were made. The meta-analysis including all cohorts indicated a birth weight decline of 150 g per 1 µg/L increase in PCB153, and DDE was associated with a 7 g decrease in birth weight (Govarts et al. 2012).

#### 4.4 Epidemiological Studies: Weight of Evidence

There may be synergistic effects when more than one pollutant is present at the same time, and the composition of these pollutant mixtures varies from one geographical region to another. There have been published a lot of associations between the exposure of the environmental persistent contaminants and health outcomes (like cancers, different types of dysfunctions, adverse effects on child development).

In the environmental research meta-analysis is difficult to do, e.g. only 43 studies from 2752 were eligible for meta-analysis when studied the causality of exposure for persistent organic pollutants and Type 1 and Type 2 diabetes (Taylor et al. 2013). There was too much variation across studies to permit a detailed meta- or pooled analysis. In the Arctic populations the association between environmental contaminants and various health outcomes is even more difficult since there are small number of studies from small populations and regions and mixed results (Singh et al. 2014).

Reviews and meta-analyses of original scientific articles are needed to evaluate potential health effects and their magnitude. ArcRisk project was focused on correlations between exposure to environmental contaminants and detected health outcomes and a new method, the synthesis of regression coefficients was developed in combining findings across different published studies with different statistical content (see more Nieminen et al. 2013a, b, 2015). It is a big challenge to combine the information from different studies together, since every study has its own design and methodological approaches, different measurement scales and there is also lack of necessary information needed for summarizing and meta-analyzing the magnitude of effects.

In the ArcRisk studies there as found a weak correlation between birth weight and exposure to PCBs but no correlation between birth weight and exposure to DDTs and no correlation between the sex ratio of newborns and exposure to PCBs (Nieminen et al. 2013b). All the future work in the magnitude of effects needs more harmonizing the study plans, careful information about the basic data and selection of variables, careful description of statistics used and reporting practices. All this is needed for other researchers who can later utilize the results in their work. In many epidemiological studies the principal aim is identical, but different studies use different statistical methods.

## 4.5 Biological Guideline Values and Risk Assessment: Case of Mercury

Humans are exposed to toxic substances from different sources within the environment, usually by ingestion, inhalation and dermal absorption. The risk assessment process, which incorporates hazard identification, exposure assessment, dose–response assessment and risk characterizations is used to quantify the probability of harmful effects on human health. This scientific evidence-based methodology is currently used for evaluating non-cancer hazard and cancer risk in environmental and occupational settings. The concentration levels of the contaminants in blood are generally followed, and it provides the sum of exposure from different routes. How accurate it is depends on the elimination half-life of the contaminants in blood and accumulation in tissues.

One answer for estimating the harmful levels for human health is to use the biological guideline values, which are used to protect the populations exposed. Here is one example, the different mercury values published by different organizations to evaluate the exposure limit (see Table 4.2).

**Table 4.2** Biological guideline values for mercury (Hg)

Me Hg	Blood	Reference dose	5.8 µg/L	Rice et al. (2003)
Total Hg	Blood	Intervention level children, pregnant/ childbearing women	8 µg/L	Legrand et al. (2010)
Total Hg	Blood	Females (>50 years), males (>18 years) at increasing risk	>20 µg/L	Health Canada (1999)
Total Hg	Blood	Females (>50 years), Males (>18 years)	>100 g/µL	Health Canada (1999)
Reference values for Hg in blood or urine (HBM Commission 2003, 2005)				
Hg	Urine	Children (6–12 years) without amalgam fillings	0.7 µg/L	
		Adults (18–69 years) without amalgam filling	1.0 µg/L	
Hg	Blood	Children (6–12 years), fish consumption ≤3 times/month	1.5 µg/L	
		Adults (18–69 years), fish consumption ≤3 times/month	2.0 µg/L	
Human biomonitoring values for mercury in blood and urine (HBM Commission 1999)				
			HBM I	HBMII
Hg	Blood	Children and adults	5 µg/L	15 µg/L
Hg	Urine	Children and adults	5 µg/g crea, 7 µg/L	20 µg/g crea, 25 µg/L

*HBM I* represents the concentration of a substance in humans below which there is no risk or adverse health effects and no need for action, *HBM II* represents the concentration of a substance in humans above which there is an increased risk for adverse health effects and urgent need to reduce the exposure and to provide individual biomedical care (an intervention or action level)

The PHIME and ArcRisk project made collaboration especially in the mercury research. Mercury levels in human blood have been several-fold in the populations of the Arctic Canada and Greenland, but now declined to the levels found in those populations living in the Mediterranean region, Northern Norway and Arctic Russia. Risk communication is challenging in the case of high mercury levels, and it is really important for the populations, especially the pregnant women and children. Fish and seafood are healthy food, but at the same time the main source of mercury. Methylmercury is very toxic for cells and organs, and it affects neurodevelopment of fetus and children.

The potential toxicity at low exposure levels of environmental contaminants and toxic metals have been researched in many of the EU-funded projects, especially in PHIME. The toxicity of the mercury on central nervous system of fetuses and myocardium in adults has been markedly modified by nutrition, and the toxicokinetics of mercury has been found to be modified by genetic factors. In the ArcRisk project Nieminen et al. (2015) studied whether differences in the number of reported outcome variables exists between the papers with non-significant findings compared to papers with significant findings using papers on maternal exposure to mercury and child development as an example. Elevated number of outcome variables was found in papers with reported non-significant associations between maternal mercury and outcomes, and the literature review did not find uniform evidence that there is a relationship between the maternal mercury level and child development. To combat the bias, better reporting, registration of all studied parameters and their systematic inclusion in meta-analyses should be done.

## 4.6 Contaminants and Placental Transport

To reach the fetal circulation, contaminants must cross the placenta, the main interface between mother and fetus by passive diffusion or active placental transfer processes. Based on analyses of cord blood, it is known that the fetus is exposed to environmental contaminants present in the maternal circulation. In addition to their physiological substrates, transporter proteins may also transfer foreign compounds such as therapeutic agents, environmental pollutants and chemical carcinogens bearing structural resemblance to their physiological substrates.

Placental transport was studied in CLEAR, PHIME and ArcRisk projects. The distribution of contaminants between maternal blood, cord blood and placenta are usually related. If compounds are metabolized in the fetus or placenta, metabolites may accumulate and cause toxic effects, and fetal and maternal serum levels may differ (Vizcaino et al. 2014). A strong correlation has been observed between concentrations in the maternal and fetal compartments for perfluorinated compounds, PCBs and DDE. The function and genetic polymorphisms of transporter proteins may cause person-to-person variation in fetal exposure to environmental contaminants, which may affect individual risk for adverse events after exposure to harmful compounds. This has been found in the uptake of methylmercury in placenta, and

may lead to accumulation during early development (Llop et al. 2014) in the PHIME project. Also other metals, like cadmium may modulate fetal exposure to other harmful compounds by changing the activity of transporters in the human placenta (Kummu et al. 2012).

## 4.7 Toxicokinetic Modelling

It is possible to extrapolate body burden and exposure to the whole lifespan of the population under certain assumptions by using pharmaco/toxicokinetic modeling in exposure and risk assessment. This approach was used in the cases of PCB153, DDE and POPs (see Verner et al. 2013; Abass et al. 2013) in CLEAR, ArcRisk and OBELIX projects, and toxicokinetic models were found to be valid methods in future risk prediction. According studies of Abass et al. (2013) the birth during the 1960s and 1970s has led to high lifelong exposure and body burdens remain elevated. By combining results from epidemiologic studies with pharmaco/toxicokinetic analyses, it would be helpful in identifying underlying confounding factors. The answers to the important questions—what is the total contaminant burden people acquire over their lifespan and what are the long-term health effects—require more multidisciplinary research and the toxicokinetic modelling approaches presented above could be one means for estimating human health risk.

Bioaccumulation of the lipid-soluble POPs leads to high levels in breast milk, and a new toxicokinetic method for the total risk assessment of POPs for women (breastfeeding mothers) was developed by Čupr et al. (2011). The method depends on the backward calculation model for life-long POPs exposure of breastfeeding women. The total risk shows that the main risk-posing group continues to be the PCBs (Václavíková et al. 2014).

Also CoZMoMAN model has been used in the ArcRisk project in person-specific predictions of life course concentrations of PCBs in the individual Norwegians (Nøst et al. 2013). The rank correlation between measurements and predictions from both the CoZMoMAN model and regression analyses was strong. The time-variant mechanistic model CoZMoMAN has been useful in estimating prenatal, postnatal and childhood exposure to PCB153 under scenarios of hypothetical and realistic maternal fish consumption (Binnington et al. 2014).

## 4.8 The Incorporation of Mechanistic Studies in Human Health Risk Assessment

The aim of *in vitro* characterization is to produce relevant information on metabolism and interactions in order to anticipate and ultimately predict what could happen *in vivo* in humans. Numerous *in vitro* models are available and each model has advantages and disadvantages. The combination of mechanistic and human *in vivo*



studies was done in the CLEAR, ArcRisk and OBELIX projects. This multidisciplinary way produced a lot of results to be used, e.g. in the overall process of human risk assessment. Examples of the incorporation of *in vitro* biotransformation studies into human health risk assessment have been published by Abass et al. (2014a, b).

## 4.9 Conclusions

During the last 10 years several multidisciplinary EU-funded projects have done a great work in researching the associations between the exposure of environmental contaminants and human health. There have been approaches towards multidisciplinary research e.g. collaboration between researchers in climate change, environmental and human health and epidemiological and mechanistic studies; and also building new monitoring and research networks and forming joint databases for solving the problems of small sample size or differences in methodology and study design in estimating the risk of contaminant exposure for human health. New methodologies have been developed in searching the ways to show the causality and the magnitude of effects. All these research projects have already given important information to the policy-makers and it would be as important as these efforts were started also to continue that work.

Do we need to put attention to the environment, contaminants and human health in the future? Absolutely we need to do that. Although the environmental levels of legacy POPs are generally declining, concentrations of some (such as PCBs) have remained relatively stable. Trends in contaminant concentrations may vary in different geographical areas of the Arctic and Europe (new hot spots may develop) and in different populations (especially indigenous populations), and this must be taken into account. Extreme events associated with global climate change, such as tsunamis and floods, might also affect food and water security, possibly increasing the incidence of contaminated food items. The impact of climate change on future human exposure to POPs will largely depend on food, e.g. the amount and type of fish consumed (wild or farmed fish) and other seafood. At present, it is not possible to draw final conclusions about future trends in exposure or the related trends in human health.

Vulnerable subgroups exist owing to, among others, genetic background, exposure history, and age. Although there are currently few data with which to draw conclusions about the implications of such vulnerabilities, it is already clear that the fetal stage is the most sensitive during human life and so fetal exposure deserves special attention. In the lifestyle of a population, there are possibly more significant exposure scenarios than the oral route, and which are not considered in conventional risk assessment.

We need better collaboration between the monitoring programs and researchers for building databases, but also for using the already existing data for new type of analyses (e.g. biostatistical, modeling) in multidisciplinary way. Better toxicokinetic models would require larger sample sizes, better analytical accuracy, fewer assumptions in exposure assessment. Human biomonitoring studies of environmental contaminants can be used together with the expected exposure routes to build up such

models. The models can be further used to predict future levels of similar chemicals in blood or to predict changes in exposure, such as could result from changes in the levels of the chemicals in fish and game. All these efforts are needed when estimating the risks of environmental contaminants on human health.

## References

- Abass K, Huusko A, Nieminen P, Myllynen P, Pelkonen O, Vähäkangas K, Rautio A (2013) Estimation of health risk by using toxicokinetic modeling: a case study of polychlorinated biphenyl PCB153. *J Hazard Mater* 261:1–10
- Abass K, Reponen P, Mattila S, Rautio A, Pelkonen O (2014a) Comparative metabolism of benfuracarb in *in vitro* mammalian hepatic microsomes model and its implications for chemical risk assessment. *Toxicol Lett* 224:290–299
- Abass K, Reponen P, Mattila S, Rautio A, Pelkonen O (2014b) Human variation and CYP enzyme contribution in benfuracarb metabolism in human *in vitro* hepatic models. *Toxicol Lett* 224:300–309
- AMAP Assessment 2015: Human health in the Arctic. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway, p. 165
- Binnington MJ, Quinn CL, McLachlan MS, Wania F (2014) Evaluating the effectiveness of fish consumption advisories: modeling prenatal, postnatal and childhood exposures to persistent organic pollutants. *Environ Health Perspect* 122:178–186
- Casteleyn L et al (2015) A pilot study on the feasibility of European harmonized Human Biomonitoring: strategies towards a common approach, challenges and opportunities. *Environ Res*. doi:10.1016/j.envres.2014.10.028
- Čupr P, Mikeš O, Krsková A, Černá M (2011) Long-term trends of POPs in human milk in Czech Republic and application for human health risk assessment. *Environmental Health Perspectives*. 23th ISEE annual meeting, Barcelona, 13–16 Sept 2011, Abstract Book: P-0472
- Gehring U, Casas M, Brunekreef B, Bergström A et al (2013) Environmental exposure assessment in European birth cohorts: results from the ENRIECO project. *Environ Health* 2013;12:8
- Govarts E, Nieuwenhuijsen M, Schoeters G, Ballester F, Bloemen K, de Boer M, Chevrier C, Eggesbø M, Guxens M, Krämer U, Legler J, Martínez D, Palkovicova L, Patelarou E, Ranft U, Rautio A, Skaalum Petersen M, Slama R, Stigum H, Toft G, Trnovec T, Vandentorren S, Weihe P, Weisglas Kuperus N, Wilhelm M, Wittsiepe J, Bonde JP, OBELIX/ENRIECO (2012) Prenatal exposure to polychlorinated biphenyls (PCB) and dichlorodiphenyldichlorethylene (DDE) and birth weight: a meta-analysis within 12 European birth cohorts. *Environ Health Perspect* 120:162–170
- Hänninen O, Knol AB, Jantunen M, Lim TA, Conrad A, Rappolder M et al (2014) Environmental burden of disease in Europe: assessing nine risk factors in six countries. *Environ Health Perspect* 122:439–446. doi:10.1289/ehp.1206154
- HBM Commission (1999) Stoffmonographie Quecksilber – referenz – und human-biomonitoring-werte (HBM). German Human Biomonitoring Commission. *Bundesgesundheitsblatt - Gesundheitsforschung - Gesundheitsschutz* 42:522–532
- HBM Commission (2003) Aktualisierung der Referenzwerte für Blei, Cadmium, und Quecksilber im Blut und im Urin von Erwachsenen. German Human Biomonitoring Commission *Bundesgesundheitsblatt - Gesundheitsforschung - Gesundheitsschutz* 46:1112–1113
- HBM Commission (2005) Neue und aktualisierte Referenzwerte für Schadstoffgehalte in Blut und Urin von Kindern – Arsen, Blei, Cadmium und Quecksilber German Human Biomonitoring Commission. *Bundesgesundheitsblatt - Gesundheitsforschung - Gesundheitsschutz* 48:1308–1312
- Health Canada (1999) Methylmercury in Canada. III. Medical Services Branch, Ottawa

- Iszatt N, Stigum H, Verner M-A, White RA et al (2015) Prenatal and postnatal exposure to persistent organic pollutants and infant growth: a pooled analysis of seven European birth cohorts. *Environ Health Perspect* 123:730–736
- Kummu M, Wallin K, Sieppi E, Rautio A, Vähäkangas K, Myllynen P (2012) Cadmium inhibits ABCG2 transporter function in BeWo and MCF-7/MTX cells. *Placenta* 33:859–865. doi:10.1016/j.placenta.2012.10.021
- Legrand M, Feeley M, Tikhonov C, Schoen D, Li-Muller A (2010) Methylmercury blood guidance values for Canada. *Can J Public Health* 101:28–31
- Lenters V, Portengen L, Rignell-Hydbom A, Jönsson B, AG et al (2015) Prenatal phthalate, per-fluoroalkyl acid, and organochlorine exposures and term birth weight in three birth cohorts: multi-pollutant models based on elastic net regression. *Environ Health Perspect*. doi:10.1289/ehp.1408933
- Llop S, Engström K, Ballester F, Franforte E, Alhamdow A, Pisa F, Tratnik JS, Mazej D, Murcia M, Rebagliato M, Bustamante M, Sunyer J, Sofianou-Katsoulis A, Prasouli A, Antonopoulou E, Antoniadou I, Nakou S, Barbone F, Horvat M, Broberg K (2014) Polymorphisms in ABC transporter genes and concentrations of mercury in newborns – evidence from two Mediterranean birth cohorts. *PLoS One* 9, e97172
- Nieminen P, Lehtiniemi H, Huusko A, Vähäkangas K, Rautio A (2013a) Polychlorinated biphenyls (PCBs) in relation to secondary sex ratio—a systematic review of published studies. *Chemosphere* 91:131–138
- Nieminen P, Lehtiniemi H, Vähäkangas K, Huusko A, Rautio A (2013b) Summarizing the reported findings between quantitative exposure and outcome variables from epidemiologic studies in a consistent form: The effect of exposure to polychlorinated biphenyls (PCBs) on birth weight as an example. *Epidemiol Biostat Public Health* 10, e8854
- Nieminen P, Abass K, Vähäkangas K, Rautio A (2015) Statistically non-significant papers in environmental health studies included more outcome variables. *Biomed Environ Sci* 28(9):666–673. doi:10.3967/bes2015.093
- Nøst TH, Breivik K, Fuskevåg OM, Nieboer E, Odland JØ, Sandanger TM (2013) Persistent organic pollutants in Norwegian men from 1979 to 2007: intraindividual changes, age–period–cohort effects, and model predictions. *Environ Health Perspect* 121:1292–1298
- Rice DC, Schoeny R, Mahaffey K (2003) Methods and rationale for derivation of a reference dose for methylmercury by the U.S. EPA. *Risk Anal* 23:107–115
- Singh K, Bjerregaard P, Chan HM (2014) Association between environmental contaminants and health outcomes in Indigenous populations of the Circumpolar North. *Int J Circumpolar Health* 73:25808. doi:10.3402/ijch.v73.25808
- Smolders R et al (2014) Interpreting biomarker data from the COPHES/DEMOCOPHES twin projects: Using external exposure data to understand biomarker differences among countries. *Environ Res*. doi:10.1016/j.envres.2014.08.016
- Taylor KW, Novak RF, Anderson HA, Birnbaum LS, Blystone C, Devito M, Jacobs D, Köhrle J, Lee DH, Rylander L, Rignell-Hydbom A, Tornero-Velez R, Turyk ME, Boyles AL, Thayer KA, Lind L (2013) Evaluation of the association between persistent organic pollutants (POPs) and diabetes in epidemiological studies: a national toxicology program workshop review. *Environ Health Perspect* 121:774–783
- Václavíková J, Mikeš O, Černá M, Čupr P (2014) Toxicokinetic modeling of selected POPs in human breast milk: prediction of chronic daily intake and health risks for Czech women in 1994–2009. Arctic frontiers conference, Abstract. Part II: Health and environment in the Arctic, Tromsø
- Verner M-A, Sonneborn D, Lancz K, Muckle G, Ayotte P, Dewailly É et al (2013) Toxicokinetic modeling of persistent organic pollutant levels in blood from birth to 45 months of age in longitudinal birth cohort studies. *Environ Health Perspect* 121:131–137
- Visnjevec AM, Kocman D, Horvat M (2014) Human mercury exposure and effects in Europe. *Environ Toxicol Chem* 33:1259–1270
- Vizcaino E, Grimalt JO, Fernandez-Somoano A, Tardon A (2014) Transport of persistent organic pollutants across the human placenta. *Environ Int* 65:107–115

# Chapter 5

## Organic Metal Species as Risk Factor for Neurological Diseases

Sören Meyer, Till Weber, Robert Haferkorn-Starke, Tanja Schwerdtle, and Julia Bornhorst

**Abstract** This chapter will focus on organic metal species of environmental concern that can exercise some influence on neurological disorders. A variety of organic metal species were identified in the last couple of years and their concentrations in the environment are rising. Moreover cases of overnutrition are increasing and due to the fortification of various organic metallic compounds in our diet there are growing anxieties that food ingredients may inadvertently be contributing to neurological disorders. This chapter provides a summary of organic metal species that have been linked with neurological disorders including its exposure pathways (especially diet), and a possible risk in the context of consumers safety pointing out gaps in the actual research. The list includes agents which have no known biological role in humans as organic species of mercury, tin, lead and arsenic. Besides the classical organometals also aluminium is illuminated. Additionally those, such as iron and manganese which are essential for life but can be toxic when absorbed in excess amounts will be discussed.

**Keywords** Neurological diseases • Diet • Organic metal species

### 5.1 Methylmercury

The heavy metal mercury (Hg) is ubiquitously distributed in the environment. Through natural effects including volcanic emissions and anthropogenic effects including waste disposal and mining, elemental mercury ( $\text{Hg}^0$ ) and inorganic mercury (iHg:  $\text{Hg}_2^{2+}$ ,  $\text{Hg}^{2+}$ ) are distributed in the environment. The environmentally most common organic mercury species methylmercury (MeHg) is formed by biomethylation of inorganic mercury in phytoplankton, by both

---

S. Meyer • T. Weber • R. Haferkorn-Starke • T. Schwerdtle • J. Bornhorst (✉)  
Department of Food Chemistry, Institute of Nutritional Science, University of Potsdam,  
Arthur-Scheunert-Allee 114-116, 14558 Nuthetal, Germany  
e-mail: [julia.bornhorst@uni-potsdam.de](mailto:julia.bornhorst@uni-potsdam.de)

sulfate- and iron-reducing bacteria, and bioaccumulates in the aquatic food chain (FAO/WHO 2011).

Apart from occupational exposure, diet is the main contributor to Hg exposure. While total Hg in non-seafood predominantly consists of iHg, 80–100 % of total Hg in fish and other seafood is represented by MeHg (EFSA 2012).

After ingestion the iHg displays a low absorption rate in the gastro intestinal tract. iHg is mainly excreted via faeces and urine with the kidney being the critical target organ for iHg toxicity. MeHg is rapidly intestinally absorbed. Furthermore, it is also well known to transfer across other physiological barriers including the blood brain barrier, the blood liquor barrier and the blood placenta barrier, resulting in neurotoxic and neurodevelopmental effects (FAO/WHO 2011; EFSA 2012; Carocci et al. 2014; Bridges and Zalups 2010).

In 2012 the European Food Safety Authority (EFSA) Scientific Panel on Contaminants in the Food Chain established a tolerable weekly intake (TWI) for MeHg of 1.3 µg/kg body weight (b.w.), expressed as mercury. While biomonitoring data indicates that generally MeHg intake in Europe is below the TWI, individually higher levels were observed. This is especially of concern to pregnant women because biomarker evaluation showed that non-toxic maternal concentrations could be potentially toxic to the unborn child (EFSA 2012).

Poisoning after intake of highly contaminated fish in Minamata, Japan, clearly showed the capability of MeHg as neurotoxicant (Harada 1995). Up to date there are multiple possible mechanisms discussed in literature which might be the mechanism behind MeHg induced neurotoxicity. The high affinity of all Hg species to sulfhydryl groups leads to a variety of effects like depolymerization of tubulin in the cytoskeleton, inhibition of protein biosynthesis and disruption of redox status via glutathione (GSH) reduction; complex formation of MeHg with the sulfhydryl group of cysteine is followed by an active transport of MeHg-Cysteine and thus to a rise in bioavailability (EFSA 2012; Bridges and Zalups 2010; Aschner et al. 2010; Syversen and Kaur 2012). Induction of oxidative stress and disruption of neurotransmitter systems could also be observed (Aschner et al. 2010).

While little is known about direct effects of MeHg on neurological diseases, studies suggest possible links between Hg exposure, Alzheimer's disease (AD) and amyotrophic lateral sclerosis (ALS) because of the comparable symptoms of mercury toxicity (including tremor, extremity weakness and ataxia) (Carocci et al. 2014). Elevated Hg blood levels could be shown in AD patients and in vitro studies showed the ability of Hg to increase production and reduce degradation of beta-amyloid peptides which play a significant role in AD (Chin-Chan et al. 2015). It has to be noted, that partial demethylation of MeHg takes place in the brain and thus reported association between iHg or total Hg concentrations and neurological diseases might also partly account for MeHg (Syversen and Kaur 2012).

Interestingly, although some epidemiological studies provide evidence for an association between fetal MeHg exposure and neurological impairments of infants, a study investigating the effect of high dietary MeHg exposure of women early in pregnancy could not find direct associations with negative neurodevelopmental

outcomes for the infants, with positive nutritional factors possibly outweighing the toxic effects (EFSA 2012; Strain et al. 2015). Also Nakamura et al. could not find any correlation between biomarker mercury levels and neurological outcomes in residents of the Taiji region but could correlate the concentrations to Se levels in whole blood (Nakamura et al. 2014).

Triggered by these outcomes the EFSA aimed to carry out a risk benefit analysis on the benefits of long-chain polyunsaturated fatty acids (LCPUFA) in fish/seafood compared to the risk of MeHg in fish/seafood. They concluded that a general approach for Europe is actually not possible especially because of the variety of fish species and local differences in food consumption patterns (EFSA 2015).

In order to further understand the risk of MeHg exposure it is important that the underlying mechanisms of toxicity are elucidated and counteracting effects of potential positive nutritional factors of fish consumption like LCPUFA or essential trace elements like selenium have to be evaluated (Nakamura et al. 2014; EFSA 2015). To reach this goal, there is a need for further epidemiological studies evaluating consistent, comparable biomarkers of MeHg exposure. While there are many used biomarkers of mercury exposure, speciation of mercury species has to be facilitated to minimize the risk of over- or underestimation of MeHg exposure (Berglund et al. 2005).

Following the need for speciation, the toxicological risk assessment has to focus on the different modes of action of different Hg species considering the co-exposure through dietary intake and after dealkylation in the brain.

Focusing on the origin of neurological diseases like AD and ALS, the role of MeHg before and after demethylation may be of neglected importance.

Due to the persistence of Hg species in the environment new approaches to minimize exposure through contaminated food have to be identified and individual local food consumption patterns have to be evaluated especially for women in childbearing age.

## 5.2 Organotin

Tin (Sn) is a naturally occurring element with a variety of inorganic and organic tin species of industrial importance. Organotin compounds are covalently bound to one or more organic substituents, such as methyl, ethyl, butyl or phenyl groups. Major applications for organotins are polyvinyl chloride (PVC) heat stabilizer, biocides, catalysts, agrochemicals and glass coatings. However, the use is largely specific for the different organotins and do not overlap with tri-substituted compounds not suitable as stabilizers for PVC and mono- substituted and di-substituted compounds not suitable for use as biocides. In general, organotin compounds are released to the environment through their production with exposure occurring by inhalation, ingestion and dermal absorption. Thereby, the most significant route of human exposure is diet but data are very limited. Relevant endpoints of exposure include developmental toxicity, immunotoxicity, endocrine disruption and neurotoxicity but the degree differs across the group as a whole (ATSDR 2005). Methyltins appear to

have a great potential to cause neurotoxicity with triethyltin (TET) and trimethyltin (TMT) by far being the most potent neurotoxins. Since neurotoxicity of dialkyltin or trialkyltin compounds has only been shown in animal studies and only a few descriptions of their human toxicity have been reported, this chapter will focus on exposure, possible modes of action and risks for TMT being the most studied organotin neurotoxicant (Mitra et al. 2013).

Since TMT is used in industry and agriculture as a constituent in fungicides and plastic production (Boyer 1989), human exposure may occur primarily through accidental and occupational exposure (Saary and House RA 2002; Jiang et al. 2000; Yoo et al. 2007). A risk assessment for TMT is rare due to the limited data available (ATSDR 2005). The highest acute NOAEL was 0.7 mg/kg/day for neurological effects in rats, while 1 mg/kg/day was a serious neurological LOAEL in rats (self-mutilating and aggressive behaviour) with doses  $\geq 2$  mg/kg/day being lethal (Snoeij et al. 1985; Bouldin et al. 1981). In the few sub-chronic studies available, the lowest LOAEL was 0.05 mg/kg/day for impaired performance of rat pups in a learning task, but there was no dose-response relationship (Noland et al. 1982).

TMT has been shown in humans and in animals to cause neuronal necrosis, particularly in the hippocampus and other structures in the limbic system. The morphological changes resulted in behavioural alterations such as aggression, memory loss and unresponsiveness (ATSDR 2005; Geloso et al. 2011). While the mechanism by which TMT induces neurodegeneration are still not conclusively clarified, many hypothesis are discussed in literature. Experimental evidence emphasize that TMT-induced neurodegeneration is a complex event including excitotoxicity, neuroinflammation, mitochondrial dysfunction and oxidative stress (Geloso et al. 2011; Billingsley et al. 2006; Corvino et al. 2014; Kaur and Nehru 2013). Additionally, it is proposed that behavioural deficits observed in a study with rats could account by the impairment of endogenous glutathione homeostasis which results in a death of neurons in the hippocampal region (Kaur and Nehru 2013). At the cellular level mitochondrial dysfunction might be a factor of cell death through the involvement of a mitochondrial membrane bound protein, stannin, selectively expressed by TMT-sensitive cells (Billingsley et al. 2006). Some of the therapeutic approaches proposed succeeded only in attenuating TMT neurotoxicity without complete remission (Corvino et al. 2013) indicating that the development of new therapeutic strategies awaits further evaluation.

Deciphering the underlying modes of action of TMT-induced neurotoxicity is of central importance especially since TMT-induced hippocampal injury is not accompanied by a blood brain barrier disruption (Harry and Lefebvre d'Hellencourt 2003; Lattanzi et al. 2013). Furthermore, concerns are rising that TMT-induced cell death is caused by different pathogenic pathways, probably acting different in *in vivo* and *in vitro* models (Lattanzi et al. 2013). This may offer a clue for future studies. Anyway, also the underlying mechanisms of TET need to be further investigated since TET is acting differently as TMT, affecting structure and function of the nervous system resulting in brain and spinal cord swelling (Kyriakides et al. 1990).

Further studies especially chronic exposure studies, which have not been carried out up to our knowledge, will provide data which are urgently needed to carry out an adequate risk assessment. Actually, only very few data are available on the effects of TET and TMT in humans deriving from unintentional occupational exposures. Data to estimate organotin exposure, particularly dietary exposure are needed whereby also the underlying species should be taken into account since they all have different end-points of toxicity. Up to now insufficient information is available to assess risk to the terrestrial environment. Additionally, epidemiological studies need to be carried out.

### 5.3 Lead

Naturally, lead (Pb) occurs in general in its +2 oxidation state in various ores. Historically, the main sources of inorganic lead were lead-based paintings and plumbing as well as solders used in food packaging. Today due to regulations and rethinking especially houses built before 1950 with old painting and plumbing are most concerning for lead uptake for the general population (Goyer 1996; Mason et al. 2014; Mielke et al. 1999). Alkylated organic lead compounds are mostly man-made and can be characterised by a carbon atom of one or more organic molecules bound to the lead atom. Tetraalkyllead was used for example in the past as gasoline additive and was a huge contributor to lead pollution in the environment. In most western countries the usage of lead as antiknock additive is nowadays forbidden by law, but still accounts for a large proportion of air emission in countries where leaded gasoline is still used (Lovei 1999). However, because of governmental prevention strategies, a reduction in blood lead levels has been observable over the last decades and children are no longer in the range of concern for lead toxicity (Nichani et al. 2006; Pirkle et al. 1998). Blood lead levels reflect an acute exposure, whereas bone lead levels are better reflective of cumulative exposure over time (ATSDR 2007).

In the European Union legislative limits exist for food and drinking water. They reach from 0.1 mg/kg for example for meat up to 0.3 mg/kg for leaf vegetables. However, up to 1.5 mg/kg are allowed in mussels (European 2006). The legislative limit for drinking water is set to 0.01 mg/L (European 1998). Contaminated drinking water as well as animal meat, fruits and vegetables are just one possible way of human exposure. Other routes are dermal uptake and vastly more important inhalation of lead-containing particles out of anthropogenic sources (Olympio et al. 2009). These two exposure routes are probably the major pathways for organolead uptake for the general population (Abadin and Pohl 2010). In Europe, the average consumer is exposed to 0.36–1.24 µg lead/kg b.w. by food. In case of specific diets with high consumption of game meat for example, the uptake can increase to 1.98–2.44 µg/kg b.w. due to lead-based ammunition. However, lead uptake by outdoor air is with 0.002 µg/kg b.w. low (EFSA 2010).

In general, the presence of lead in the body causes damage to the central nervous system and several underlying mechanisms are discussed. Thereby, especially bivalent



lead can act as a pharmaceutical agent substituting bivalent calcium and, to a lesser extent bivalent zinc. Interferences with the calcium release from mitochondria for example are resulting in oxidative stress. This causes mitochondrial self-destruction ending in activation of apoptosis and cell death. Additionally, lead interferes with neurotransmitter release disrupting the function of the GABAergic and dopaminergic systems (Mason et al. 2014; Goldstein 1993; Guilarte et al. 1993).

Furthermore, lead interacts with other body systems supporting the function of the central nervous system. These include hypertension, impaired renal functions and vitamin D deficiency as well as preterm birth (ATSDR 2007). Lead encephalopathy is one severe neurological effect of lead exposure caused by very high doses. Symptoms include headache, mental dullness up to memory loss, coma and death in case of long-time lead exposure (Kumar et al. 1987). Children are affected at much lower concentrations and additionally, the central nervous system is more vulnerable to toxic agents compared to the mature one. Accordingly, neural proliferation, differentiation and plasticity are strongly impaired by lead causing neurobehavioral deficits (Olympio et al. 2009). The lead exposure-related neurodevelopmental deficits are associated with the loss of IQ points, impairment with school performance resulting in substantial economic losses to society (Gould 2009; Grandjean and Herz 2015).

Over the last decades the lead levels considered as tolerable for children have been repeatedly lowered. Beginning in the 1960s with blood lead levels of 60  $\mu\text{g/L}$ , they have been lowered to 30  $\mu\text{g/L}$  in 1975, to 25  $\mu\text{g/L}$  in 1985 and finally to 10  $\mu\text{g/L}$  in 1991. Today a safe level of lead is not derivable any more. Therefore, factors as age of exposure, duration of blood lead elevation and characteristics of the child's rearing environment have to be considered and are too variable. Consequently, there is no lead blood concentration that can be considered as safe in general (Olympio et al. 2009).

More reliable biomarker would help to evaluate the risk after lead exposure and prevent damage at an early stage. But except blood and hard to determine bone lead levels, no respectable biomarker has been discovered so far. However, there are new modern methods and imaging techniques to investigate and document for example the reduction of gray matter (cortex) volume. This can be used especially for the prefrontal cortex in adults with increased childhood lead exposure (Cecil et al. 2008). Early clinical detection particularly in children helps to protect their developing central nervous system and intervention at this stage may aid in reversal of certain posttoxicity complications.

Whereas effects of inorganic lead are well described in literature and lots of studies exist, the available data about organolead compounds like alkylated lead is scarce and most studies were already carried out in the 1990s. Since tetraalkyllead was used worldwide as a gasoline additive it is still persistent in the environment.

There should be more studies investigating degradation of organolead compounds and their contamination of food by uptake from contaminated soil. Additionally, more work has to be carried out on toxicity and toxicokinetics of these compounds. For both inorganic and organic lead epidemiological research on international populations is currently very limited (Mason et al. 2014).

Even though forbidden, humans are exposed to lead by paintings and plumping of old houses, but the restrictions already resulted in lower blood lead levels.

Nevertheless, there still might be other “silent” sources of lead and the World Health Organization recommended constant research in this particularly area (WHO 1998). This is associated with strict controls of products entering the European Union suspected to contain lead for example children’s toys and cosmetics.

## 5.4 Arsenic

Arsenic (As) is ubiquitously present in various forms in nature. The variety of arsenic species can be separated into inorganic (e.g. arsenite, arsenate) and organic arsenic compounds (e.g. arsenobetaine, arsenosugars, arsenolipids). Organic arsenicals are classified by an arsenic carbon bond and are generally assumed to be less toxic than inorganic species. Interestingly, inorganic compounds are predominant in drinking water and terrestrial food especially in rice, whereas organic arsenicals are particularly present in marine fish and other seafood. In these food levels are reaching from 1 to 100 mg As/kg wet weight and are up to 100-fold higher than in terrestrial food. Thereby, water-soluble organic arsenicals are mostly present in algae, whereas fat-soluble compounds are predominant in fatty fish. Diet is the main source of arsenic for humans but exposure depends on the type of food, the conditions of growth like the type of soil, water quality and geochemical activities as well as further food processing conditions (EFSA 2009).

Whereas arsenicals in food and drinking water are contaminants since arsenic is not considered as an essential element, some arsenicals are used as pesticide, for animal fattening or as pharmaceuticals. Even though highly toxic, Trisenox is for example authorized in the treatment of leukaemia and due to lack of alternatives patients with sleeping sickness are cured with Melarsoprol (Gibaud and Jaouen 2010).

In contrast to drinking water, which is regulated in many countries by a legislative limit of 0.01 mg/L repatriated to a recommendation of the World Health Organization (WHO) (European 1998; WHO 2001), the only regulatory limits for food products are currently applied in China. For rice, the legislative limit is for example set to 0.2 mg inorganic As/kg. This might be due to the circumstances that cereal and cereal products especially rice and rice-based products contain high amounts of inorganic As (0.1–0.4 mg As/kg dry weight) caused by accumulation of arsenic after root uptake from soil (USDA 2014). Nevertheless, the European Commission introduced maximum levels for inorganic arsenic in rice and rice products in 2016.

A chronic ingestion as well as inhalative exposure towards inorganic arsenic has been associated with incidences for tumours of the lung, skin and bladder. The International Agency for Research on Cancer (IARC) classified inorganic arsenic as a human carcinogen (group 1) (IARC 2012). Further, chronic ingestion is associated with skin lesions, cardiovascular diseases, abnormal glucose metabolism, diabetes, disturbance of foetal and infant development as well as neurotoxicity (EFSA 2009; IARC 2012).

Especially exposure to lower concentrations are increasing the susceptibility to cognitive dysfunctions (Naujokas et al. 2013). Accumulation could be shown for

inorganic as well as organic arsenic species like methylated arsenicals in many parts of the brain. Thereby, arsenic alters cognitive functions particularly learning and memory capability during childhood, but adults are also affected as critical processes are often damaged. Effects seem to be depending on concentration, timing, and duration of exposure. However, adverse effects can even appear weeks or month after initial acute poisoning (Tyler and Allan 2014). Destruction of axonal cylinders and compositional changes caused by the disruption of the neurofilament and the microtubule network may play a major key in arsenite neurotoxicity, since it does not affect gene expression (Vahidnia et al. 2007).

The toxicological properties of organic arsenicals are depending on their molecular nature and the majority of these compounds occur in the pentavalent oxidation state (Feldmann and Krupp 2011). A wide range of toxicity is observable in this group of compounds reaching from the as non-toxic categorised arsenobetaine to the as highly toxic considered thio-analogue of dimethylarsinic acid namely thio-dimethylarsinic acid. The toxicity of this compound is comparable to that of trivalent arsenic species, which are generally assumed to be highly toxic. The toxicity of inorganic arsenic is probably caused by its metabolism to dimethylarsinic acid, where trivalent arsenicals are intermediates (Naranmandura et al. 2007; Raml et al. 2007; Wang et al. 2015). However, less is known about arsenosugars and arsenolipids, which both are common arsenicals in contaminated marine food. Because of their metabolism to dimethylarsinic acid as well, which is considered as possible carcinogenic to humans (IARC 2012), the evidence concerning a risk to human health especially after long term exposure cannot be excluded. *In vitro* studies demonstrated that arsenosugars are significantly less toxic than arsenite and did not exert genotoxicity (Andrewes et al. 2004; Leffers et al. 2013a; Leffers et al. 2013b). In contrast, it was demonstrated that arsenic-containing hydrocarbons, one class of arsenolipids, display toxicity in the same concentration range as arsenite, but are different in their toxic mode of action (Meyer et al. 2014a; Meyer et al. 2014b). Because of the high lipophilic character of arsenolipids they are possibly transported by passive diffusion across physiological barriers like the intestine or the blood brain barrier. Consequently, they might be able to enter the brain and cause neurotoxicological effects. First evidences for this assumption have already been reported but especially in this area further investigations are urgently needed to characterise the risk of organic arsenicals in particular of arsenolipids to human health.

Even though many data are available in literature about arsenic toxicity further studies are needed to elucidate the underlying mechanism. In case of inorganic arsenicals the reasons for carcinogenesis are still not completely understood. The mode of action seems to be indirectly for example by inhibition of DNA repair, induction of reactive oxygen and nitrogen species attacking DNA or epigenetic disorders, since direct effects were rarely observable (Hughes et al. 2011).

Data about organic arsenical especially concerning arsenolipids are scarce and besides further *in vitro* studies, investigating the neurotoxicological potential, studies with experimental animals have to be performed. These are necessary to evaluate the risk of these food-relevant arsenicals for human health and finally, to carry out a

risk assessment. Therefore, exposure assessment is also needed, since the actual burden for the consumer cannot be estimated. This requires an improvement and simplification of arsenic speciation analysis techniques. The high variety in arsenic toxicity due to the different species demonstrate that differentiation is a very important tool.

Since classical rodent animal models are not useful in case of arsenic carcinogenesis among others due to strong differences in the toxicokinetics of inorganic arsenic between rodents and humans further epidemiological studies have to be carried out. They will display if a high consumption of seafood containing especially organic arsenicals is associated with increased incidence of health-related effects like neurological dysfunctions or cancer.

## 5.5 Aluminium

Aluminium (Al), omnipresent since Al is the most abundant metal in the earth's crust, appears to have no physiological requirement (Verstraeten et al. 2008). However, human intervention (soil acidification, food additives, Al-containers etc.) increases the human body burden of aluminium (Exley 2003; Exley 2009).

Al occurs in nature in only one oxidation state (+3) which normally seeks out complexing agents with oxygen-atom donor sites such as carboxylate, phosphate and sulphate groups among others. Therefore, Al is frequently found as aluminosilicates, hydroxides, phosphates, sulphates and cryolite (Krewski et al. 2007). Human exposure to Al is ensured mainly via air, food and water with diet being the main source for individuals who are not exposed occupationally. Thereby a variety of Al compounds are produced and used for different purposes, such as in water treatment, papermaking, fire retardants, food additives, preservatives, colours, cosmetics and pharmaceuticals. Al alloys with other metals find also application in consumer appliances, food packaging and cookware (EFSA 2008). Infant formulae are especially enriched with the highest potential exposure being from soy-based formula (Committee on Toxicity (COT) 2013).

Oral  $\text{Al}^{3+}$  is readily bioavailable with generally 0.5 % of intake or less. Oral  $\text{Al}^{3+}$  bioavailability is increased in the presence of citrate or lactate in food while it may be decreased by silicon-containing compounds. Additionally bioavailability is also inversely related to iron status. While bone can contain approximately 60 % of total aluminium in the body, lung, muscle, liver and brain contain approximately 25 %, 10 %, 3 % and 1 % respectively (FAO/WHO 2012).

The EFSA established a tolerable weekly intake (TWI) of 1 mg/kg b.w./week and confirmed this conclusion in 2011 (EFSA 2008). Mean dietary exposure from water and food in non-occupational exposed adults assessed in several European countries, varied from 0.2 to 1.5 mg/kg b.w./week; this corresponds in a 60 kg adult in a dietary uptake of 1.6–13 mg aluminium per day. Children represent the group with the highest potential exposure to aluminium per kg body weight due to the higher food intake than adults when expressed to body weight. The potential esti-

mated uptake ranged from 0.7 to 2.3 mg/kg b.w./week. The EFSA also considered that the TWI is likely to be exceeded in a significant part of the European population (EFSA 2008). In 2011, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) of 2 mg/kg b.w.. In the EU certain aluminium compounds are permitted with restriction and since 1<sup>st</sup> of February 2014 calcium aluminosilicate, betonit and kaolin are not permitted (European 2012). A technical report of the EFSA further evaluated that the mean and 95<sup>th</sup> percentile dietary exposure estimates to five aluminium-containing food additives [E 523, E 541 (i, ii), E 554, E 556 and E 559] for five population groups (toddlers, children, adolescents, adults and the elderly) largely exceed the TWI established by the EFSA and the PTWI established by the JECFA (EFSA 2013a).

The main toxic effects of Al are on the brain, the nervous system and the kidney. However, this chapter will focus on the link between Al exposure and neurological disorders especially since the brain is considered to be the most vulnerable organ to the toxic manifestations of Al. Thereby, Al has been reported by several studies being a risk factor for the development of the most common neurodegenerative disorder Alzheimer's disease (Tomljenovic 2011). Although the exact mechanisms are still unknown, Al is known to affect multiple systems in the brain (Exley 2014a). For example oxidative stress appears to play a significant role through the potentiation of damaging redox activity or the disruption of calcium signaling (Kumar and Gill 2014). Al is also competing effectively with essential metals, in particular magnesium and iron, in essential processes. For example, an altered metabolism is resulting of the higher binding affinity of Al over magnesium for ATP consequently interfering with magnesium-catalyzed enzyme activities (Exley 2009). Additionally, recent research identified that the homeostasis of copper is altered by Al (Page et al. 2012).

In order to further understand the risks arising from Al exposure it is important to identify the biological effects of Al and we will need to further our understanding of the body burden of Al. The most significant factor about its potential danger as a neurotoxin is its omnipresence in life. It is highly probable that our use of Al will increase in the future. It is suggested that tomorrow's generation will have higher body burden of Al (Exley 2013). The body burden is spread among the tissues, but small amounts of Al over lifetime favors brain tissues as the site of bioaccumulation. Concerns about Al exposure in the human diet persist since several years. The EFSA Panel does not consider exposure to Al via food to constitute a risk for developing Alzheimer's disease based on the available scientific data despite that it could be implicated in the etiology of Alzheimer's disease. Further research is necessary to clarify if Al can be and probably is a contributor to neurodegenerative diseases. Additionally, we will need to implement measures to reduce body burden to the lowest practical limit and relating them to any subsequent changes in health-related indices (Exley 2014b). Although there are only limited studies for therapy or treatment, Davenward et al. (2013) demonstrated that lowering the body burden of aluminium in individuals with moderate-to-severe AD concomitantly, leads to clinically significant improvements in cognitive performance in some individuals (Davenward et al. 2013). These experiments offer therapeutic strategies but further research in this

field is of central importance. Further areas of concern include the limited knowledge of Al bioavailability and address whether the aluminium species significantly affects aluminium pharmacokinetics, such as oral bioavailability, and aluminium-induced effects, such as the association between aluminium consumption and cognitive loss. It is worrying that up to now no data are available on the absorption of Al in infants specifically (Committee on Toxicity 2013). Especially in the background that kidney function is not fully developed at birth resulting in a lower elimination of Al we should critically look at the high Al content in some infant formulas (Committee on Toxicity 2013). In general, the biological responses may be highly specific to the form of Al but data regarding Al speciation are limited up to now.

There is a need for further epidemiological studies that look at different Al species of ingestion and long-term studies on chronic Al exposure need to be carried out. Additionally the public should be better informed on potential health risks especially in the background that a high percentage of the world's population uses alternative self-medication and herbal treatments for prophylactic purposes.

## 5.6 Iron

Iron (Fe) is the fourth frequent element and the most abundant transition metal in the earth's surface. Furthermore, it is an essential trace element for all organisms (Ganz 2013). Iron naturally occurs in its +2 (reducing agent) and +3 (oxidising agent) oxidation state in human and animal cells and plays an important role in lots of oxygen- and electron-transferring enzymes. Likewise, the most important ferrous compounds are part of Haem proteins, like haemoglobin and myoglobin in blood (EFSA 2006).

For humans the main source of iron is animal meat, but it is also present in some fruit and vegetables as well as in legumes and cereals. In food production, iron compounds are used for technological purposes only, for example iron oxide as a colouring agent, or ferrous gluconate for oxidative colouring of olives (European 2008).

In literature, the total body iron content of a healthy person is ranging from 45 to 60 mg/kg b.w., which can be divided into functional iron, transport iron and depot iron (BfR 2004). Epidemiological studies demonstrate that the daily intake of iron for men is between 14.6 and 15.1 mg and for women ranging from 11.6 to 12.3 mg in the middle age group (Schulze et al. 2001). Based on this knowledge, in the European Union legislative limits exist for drinking water and food. The limit in drinking water is set to 0.2 mg/L (European 1998) and the maximum residue in food reaches from 20 mg/kg for sodium ferrous cyanide up to 150 mg/kg for ferrous gluconate as colouring agent for olives (European 2008). Since 2004, the European Union authorised iron as food supplement and since 2006 for enrichment in food (BfR 2004).

However, the German Federal Institute for Risk Assessment (BfR) concluded that an uncontrolled enrichment of food with iron has to be seen critically because there might be a link between high iron depots and an increased risk for cardiovascular diseases (BfR 2004). A potentially hazardous reactivity is given due to the

oxidative potential of iron. This is linked with increasing oxidative stress and earlier aging of cells (BfR 2008). To prevent this, an effective control mechanism exists regulating the iron balance in organisms. In organism iron is generally linked to functional proteins like haemoglobin, to transport molecules like transferrin as well as to depot proteins like ferritin. Consequently, iron is kept away from oxidative reactions and cannot cause cellular damage (Hershko et al. 1988). Massive iron overload, however, leads to an uncontrolled rising of the iron pool accompanied with increasing binding capacity of ferritin. The excess iron can bind to proteins with low molecular weight and non-transferrin-bound iron (NTBI) occurs. NTBI usually occurs in the plasma in case of hereditary haemochromatosis and thalassaemia. In vitro studies show that NTBI results in free hydroxyl radicals and peroxidation of the lipids membrane (Hershko et al. 1988; Schumann 2001). In further studies a link between thalassaemia or haemochromatosis patients and liver cell damages along with transferrin saturation > 70 % was observable (Le Lan et al. 2005; Jensen et al. 2003; Piga et al. 2009). Furthermore, a connection between iron homeostasis and cellular death, severe dysfunctions along with Alzheimer's, Parkinson's and Huntington's diseases exists. For these diseases high iron concentration in the brain are often ascertainable (Salvador et al. 2010; Ganz and Nemeth 2011) which is also related with the development of oxidative stress.

In consideration of missing studies, the supply situation of the population and the not controlled enrichment of iron in food, the Federal Institute for Risk Assessment recommended not to use iron in food supplements and other food. Hence, iron supplementation should only be carried out under medical observation (BfR 2004).

Even though knowledge about the mechanism of oxidative stress improved in the last couple of years and more information about the neuropathology of Alzheimer's disease is available, the role of iron in organisms is still not fully understood. However, further findings in those fields could be fundamental for the understanding of the modes of action of iron and its role as a contributor in neurodegenerative diseases. In addition, interactions with other metals have to be evaluated since iron can promote their oxidative capacities resulting in higher oxidative stress levels.

## 5.7 Manganese

Manganese (Mn) is the 12<sup>th</sup> most abundant element in the earth's crust and the fifth most abundant metal, usually occurring with iron. Typically Mn exists as oxides, carbonates and silicates and earth erosions, resulting in the omnipresence of Mn in air, soil and waterways. Although Mn can exist in 11 oxidation states the most biologically important Mn compounds are those that contain Mn<sup>2+</sup> and Mn<sup>3+</sup>. The ubiquitous trace element is essential for normal growth development and cellular homeostasis. It is crucial for brain development and function of a variety of enzyme families including glutamine synthetase, arginase, pyruvate carboxylase and Mn

superoxide dismutase. Thereby, it is regulating development, energy metabolism, digestion, immune function, reproduction and antioxidant defences (Santamaria 2008; Costa and Aschner 2015).

Mn exposure of the general population arises from both natural and anthropogenic sources with dietary intake as primary route. Rich Mn sources are nuts, chocolate, cereal-based products, crustaceans and molluscs, pulses, and fruits and fruit products. The main contributors in the diet of adults are cereal based products, vegetables, fruits and fruit products, beverages and drinking water (Barceloux 1999). Mn is also used in several industrial settings including the production of steel, the production of dry-cell batteries, glass and fireworks and it is used in chemical manufacturing, in the leather and textile industries (Flynn and Susi 2009). Moreover, Mn can be found in fungicides, as a component of an antiknock gasoline additive [(Methylcyclopentadienyl)mangantricarbonyl (MMT)] or as a contrast reagent for medical magnetic resonance imaging (Mangafodipir) (ATSDR 2008). Due to the insufficient available data to derive an average requirement or a population reference intake, an Adequate Intake (AI) is proposed. Mean intake of Mn in adults in the EU are around 3 mg/day while the intake for infants aged from 7 to 11 months is proposed to be 0.02–0.5 mg/day (EFSA 2013b). The US Institute of Medicine considered an AI of 2.3 mg/day for men and 1.8 mg/day for women and set an “Upper Limit” of 11 mg/day (IOM 2002).

Due to its ubiquitous presence in most foodstuffs, in industrial countries Mn deficiency is practically non-existing and has only been observed in animal studies (Friedman et al. 1987). However, exceeding the homeostatic range Mn can result in an irreversible condition known as manganism that shares similar neuropathology with Parkinson’s disease (PD), with dopaminergic (DAergic) cell loss associated with motor and cognitive deficits (Racette 2014). Manganism patients exhibit symptoms including tiredness, behavioural changes, and delayed neurological disturbances characterized by dystonia or kinesia (Olanow 2004). Up to now clarity about the underlying mechanisms of Mn-induced neurotoxicity is lacking. Elevated brain Mn levels have been associated with an impaired iron homeostasis, oxidative stress, mitochondrial dysfunction, induction of protein aggregation, excitotoxicity and DNA repair. Importantly, the highest Mn levels are found in the caudate-putamen, globus pallidus, substantia nigra, and subthalamic nucleus (Flynn and Susi 2009; Tuschl et al. 2013; Bowman et al. 2011).

In order to understand the risks arising from Mn exposure it is of central importance to decipher the underlying mechanism and its transport. Thereby, information about the specific Mn-species should further be used to answer in more detail the questions about the interrelation of the species to the molecular mechanisms (Michalke and Fernsebner 2014). Especially since Mn also represents a risk factor for Parkinson’s disease and alterations in neuronal handling of Mn have also been observed in the context of Huntington’s disease. Moreover, identifying the mechanisms of Mn is essential for the development of new therapeutic strategies against manganism since pharmaceuticals against Parkinson’s disease are not effective (Olanow 2004; Guilarte 2010). Furthermore, additional studies



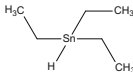
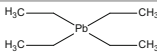
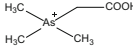
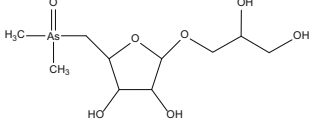
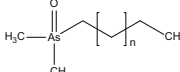


are necessary to consider a threshold of toxicological concerns, which would be important for highly susceptible populations including infants or patients receiving parenteral nutrition (Aschner and Aschner 2005; Alves et al. 1997). Thereby, it should be mentioned that in infant formula 100-fold higher Mn levels than in breast milk have been observed (Ljung et al. 2011). Recent studies also predict an impact of Mn on epigenetic changes and some populations are highly susceptible towards Mn due to genetic polymorphisms of metabolic enzymes or Mn transporters (Kim et al. 2015). In general less is known about genetic interactions in Mn-induced neurotoxicity (Chakraborty et al. 2015; Bornhorst et al. 2014). Consequently, Mn exposure levels in populations with different genetic backgrounds need to be studied and individuals carrying genotypes resulting in a higher susceptibility to Mn exposure need to be identified. Assessment of Mn intake and status is actually difficult due to the lack of an adequate biomarker. Therefore, the identification of a sensitive and selective biomarker of exposure is required.

## 5.8 Conclusion

In everyday life, human beings are exposed to levels of biologically available organic metal species and it is highly probable that our burden will increase in the future. Thereby, for most of the organic metal species, the most important route of exposure is diet. This chapter is briefly summarising the available data regarding occurrence in food, possible modes of action and risks of the organic species of mercury, tin, lead and arsenic. Besides the classical organometals also aluminium, iron and manganese are described. The illuminated metals had been implicated to be a risk factor for neurological disorders pointing out the central importance in deciphering underlying modes of neurotoxic action and the importance of an adequate risk assessment. However, to be able to evaluate this, there are various future needs in research. A new exposure paradigm is needed that is correlating species specific oral bioavailability and metal-induced effects, especially metal-induced neurological effects. Actually there are only limited data available regarding species specific effects at all. Additionally, a lot has been implicated for interactions of the metals with other metal homeostasis which should be taken in consideration especially those which are essential. In general, less is known about individual susceptibility towards metal-induced neurotoxicity. The effect of genetic polymorphisms of metabolic enzymes and transporters of the respective metal needs to be established as well as alterations of the metal-responsive proteins and epigenetic changes due to metal exposure. In order to minimize the effects of exposure, further biological monitoring should be done with more sensitive and selective biomarkers for the individual metals and metal species. Altogether, it should be of our central interest to lower body burden of organic metal species having no biological role. Additionally, new therapeutic strategies against metal-induced neurotoxic effects need to be evaluated (Table 5.1).

**Table 5.1** Most common organic species and structures of Hg, Sn, Pb and As and its neurotoxic potential

Element	Most common organic species and structures	Neurotoxic potential
Hg	Methylmercury 	X
Sn	Trimethyltin 	X
	Triethyltin 	X
Pb	Tetraethyllead 	X
As	Arsenobetaine 	Unidentified
	Arsenosugar e.g. Dimethylarsinoyl-sugar-glycerol 	Not investigated
	Arsenolipid e.g. As-containing hydrocarbons 	Not investigated

X identified

## References

- Abadin HG, Pohl HR (2010) Alkyllead compounds and their environmental toxicology. *Met Ions Life Sci* 7:153–164. doi:[10.1039/BK9781847551771-00153](https://doi.org/10.1039/BK9781847551771-00153)
- Alves G, Thiebot J, Tracqui A, Delangre T, Guedon C, Lerebours E (1997) Neurologic disorders due to brain manganese deposition in a jaundiced patient receiving long-term parenteral nutrition. *JPEN J Parenter Enteral Nutr* 21(1):41–45
- Andrewes P, Demarini DM, Funasaka K, Wallace K, Lai VW, Sun H, Cullen WR, Kitchin KT (2004) Do arsenosugars pose a risk to human health? The comparative toxicities of a trivalent and pentavalent arsenosugar. *Environ Sci Technol* 38(15):4140–4148
- Aschner JL, Aschner M (2005) Nutritional aspects of manganese homeostasis. *Mol Aspects Med* 26(4-5):353–362. doi:[10.1016/j.mam.2005.07.003](https://doi.org/10.1016/j.mam.2005.07.003)
- Aschner M, Onishchenko N, Ceccatelli S (2010) Toxicology of alkylmercury compounds. *Met Ions Life Sci* 7:403–434. doi:[10.1039/bk9781847551771-00403](https://doi.org/10.1039/bk9781847551771-00403)
- ATSDR (2005) Toxicological profile for Tin and tin compounds. US Department of Health and Human Service, Washington, DC
- ATSDR (2007) Toxicology profile for lead. US Department of Health and Human Service, Washington, DC
- ATSDR (2008) Draft toxicological profile for manganese. US Department of Health and Human Service, Atlanta, GA
- Barceloux DG (1999) Manganese. *J Toxicol Clin Toxicol* 37(2):293–307

- Berglund M, Lind B, Bjornberg KA, Palm B, Einarsson O, Vahter M (2005) Inter-individual variations of human mercury exposure biomarkers: a cross-sectional assessment. *Environ Health* 4:20. doi:[10.1186/1476-069X-4-20](https://doi.org/10.1186/1476-069X-4-20)
- BfR (2004) Use of minerals in foods toxicological and nutritional-physiological aspects Part II
- BfR (2008) Frequently asked questions and answers about iron in food
- Billingsley ML, Yun J, Reese BE, Davidson CE, Buck-Koehntop BA, Veglia G (2006) Functional and structural properties of stannin: roles in cellular growth, selective toxicity, and mitochondrial responses to injury. *J Cell Biochem* 98(2):243–250. doi:[10.1002/jcb.20809](https://doi.org/10.1002/jcb.20809)
- Bornhorst J, Chakraborty S, Meyer S, Lohren H, Brinkhaus SG, Knight AL, Caldwell KA, Caldwell GA, Karst U, Schwerdtle T, Bowman A, Aschner M (2014) The effects of pdr1, djr1.1 and pink1 loss in manganese-induced toxicity and the role of alpha-synuclein in *C. elegans*. *Metallomics* 6(3):476–490. doi:[10.1039/c3mt00325f](https://doi.org/10.1039/c3mt00325f)
- Bouldin TW, Goines ND, Bagnell RC, Krigman MR (1981) Pathogenesis of trimethyltin neuronal toxicity. Ultrastructural and cytochemical observations. *Am J Pathol* 104(3):237–249
- Bowman AB, Kwakye GF, Hernandez EH, Aschner M (2011) Role of manganese in neurodegenerative diseases. *J Trace Elem Med Biol* 25(4):191–203. doi:[10.1016/j.jtemb.2011.08.144](https://doi.org/10.1016/j.jtemb.2011.08.144)
- Boyer IJ (1989) Toxicity of dibutyltin, tributyltin and other organotin compounds to humans and to experimental animals. *Toxicology* 55(3):253–298
- Bridges CC, Zalups RK (2010) Transport of inorganic mercury and methylmercury in target tissues and organs. *J Toxicol Environ Health B Crit Rev* 13(5):385–410. doi:[10.1080/10937401003673750](https://doi.org/10.1080/10937401003673750)
- Carocci A, Rovito N, Sinicropi MS, Genchi G (2014) Mercury toxicity and neurodegenerative effects. *Rev Environ Contam Toxicol* 229:1–18. doi:[10.1007/978-3-319-03777-6\\_1](https://doi.org/10.1007/978-3-319-03777-6_1)
- Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, Altaye M, Egelhoff JC, Wessel S, Elangovan I, Hornung R, Jarvis K, Lanphear BP (2008) Decreased brain volume in adults with childhood lead exposure. *PLoS Med* 5(5), e112. doi:[10.1371/journal.pmed.0050112](https://doi.org/10.1371/journal.pmed.0050112)
- Chakraborty S, Chen P, Bornhorst J, Schwerdtle T, Schumacher F, Kleuser B, Bowman AB, Aschner M (2015) Loss of pdr-1/parkin influences Mn homeostasis through altered ferroportin expression in *C. elegans*. *Metallomics* 7(5):847–856. doi:[10.1039/c5mt00052a](https://doi.org/10.1039/c5mt00052a)
- Chin-Chan M, Navarro-Yepes J, Quintanilla-Vega B (2015) Environmental pollutants as risk factors for neurodegenerative disorders: Alzheimer and Parkinson diseases. *Front Cell Neurosci* 9:124. doi:[10.3389/fncel.2015.00124](https://doi.org/10.3389/fncel.2015.00124)
- Committee on Toxicity (COT) (2013) Statement on the potential risks from aluminium in the infant diet. pp 1–24
- Corvino V, Marchese E, Michetti F, Geloso MC (2013) Neuroprotective strategies in hippocampal neurodegeneration induced by the neurotoxicant trimethyltin. *Neurochem Res* 38(2):240–253. doi:[10.1007/s11064-012-0932-9](https://doi.org/10.1007/s11064-012-0932-9)
- Corvino V, Marchese E, Podda MV, Lattanzi W, Giannetti S, Di Maria V, Cocco S, Grassi C, Michetti F, Geloso MC (2014) The neurogenic effects of exogenous neuropeptide Y: early molecular events and long-lasting effects in the hippocampus of trimethyltin-treated rats. *PLoS One* 9(2), e88294. doi:[10.1371/journal.pone.0088294](https://doi.org/10.1371/journal.pone.0088294)
- Costa L, Aschner M (2015) Manganese in health and disease. Royal Society of Chemistry, Cambridge, UK
- Davenward S, Bentham P, Wright J, Crome P, Job D, Polwart A, Exley C (2013) Silicon-rich mineral water as a non-invasive test of the ‘aluminum hypothesis’ in Alzheimer’s disease. *J Alzheimers Dis* 33(2):423–430. doi:[10.3233/JAD-2012-12123192280Q5860P8543M](https://doi.org/10.3233/JAD-2012-12123192280Q5860P8543M)
- EFSA (2006) Tolerable upper intake levels for vitamins and minerals
- EFSA (2008) Scientific Opinion of the Panel on Food Additives, Flavourings, Processing Aids and Food Contact Materials (AFC). Safety of aluminium from dietary intake. *EFSA J* 6(7):1–34
- EFSA (2009) EFSA Panel on Contaminants in the Food Chain (CONTAM); Scientific Opinion on arsenic in food. *EFSA J* 7(10):1351–1355
- EFSA (2010) Scientific opinion on lead in food. *EFSA J* 8(4):1–151
- EFSA (2012) Scientific Opinion on the risk for public health related to the presence of mercury and methylmercury in food. *EFSA J* 10:1–241

- EFSA (2013) Dietary exposure to aluminium-containing food additives Supporting Publications EN-411
- EFSA (2013b) Scientific Opinion on dietary reference values for manganese. *EFSA J* 11(11):1–44
- EFSA (2015) Statement on the benefits of fish/seafood consumption compared to the risks of methylmercury in fish/seafood. *EFSA J* 13(1):1–36
- European Union (1998) Corrigendum to Council Directive 98/83/EC of 3 November 1998 on the quality of water intended for human consumption (OJ L 330 of 5.12.1998)
- European Union (2006) Regulation (EC) No 1881/2006 of 19 December 2006 setting maximum levels for certain contaminants in foodstuffs (Text with EEA relevance) (OJ L 364, 20.12.2006, p 5)
- European Union (2008) Regulation (EC) No 1333/2008 of the European Parliament and of the Council of 16 December 2008 on food additives (Text with EEA relevance)
- European Union (2012) Commission Regulation (EU) No 380/2012 of 3 May 2012 amending Annex II to Regulation (EC) No 1333/2008 of the European Parliament and of the Council as regards the conditions of use and the use levels for aluminium-containing food additives
- Exley C (2003) A biogeochemical cycle for aluminium? *J Inorg Biochem* 97(1):1–7, doi:[S0162013403002745](https://doi.org/10.1016/S0162013403002745) [pii]
- Exley C (2009) Darwin, natural selection and the biological essentiality of aluminium and silicon. *Trends Biochem Sci* 34(12):589–593. doi:[10.1016/j.tibs.2009.07.006](https://doi.org/10.1016/j.tibs.2009.07.006)S0968-0004(09)00167-4
- Exley C (2013) Human exposure to aluminium. *Environ Sci Process Impacts* 15(10):1807–1816. doi:[10.1039/c3em00374d](https://doi.org/10.1039/c3em00374d)
- Exley C (2014a) Why industry propaganda and political interference cannot disguise the inevitable role played by human exposure to aluminum in neurodegenerative diseases, including Alzheimer’s disease. *Front Neurol* 5:212. doi:[10.3389/fneur.2014.00212](https://doi.org/10.3389/fneur.2014.00212)
- Exley C (2014b) What is the risk of aluminium as a neurotoxin? *Expert Rev Neurother* 14(6):589–591. doi:[10.1586/14737175.2014.915745](https://doi.org/10.1586/14737175.2014.915745)
- FAO/WHO (2011) Safety evaluation of certain food additives and contaminants. WHO food additives series, vol 63. Mercury, pp 605–685
- FAO/WHO (2012) Safety evaluation of certain food additives and contaminants: prepared by the seventy-fourth meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Food Additives Series, vol 65. pp 3–86
- Feldmann J, Krupp EM (2011) Critical review or scientific opinion paper: arsenosugars—a class of benign arsenic species or justification for developing partly speciated arsenic fractionation in foodstuffs? *Anal Bioanal Chem* 399(5):1735–1741. doi:[10.1007/s00216-010-4303-6](https://doi.org/10.1007/s00216-010-4303-6)
- Flynn MR, Susi P (2009) Neurological risks associated with manganese exposure from welding operations—a literature review. *Int J Hyg Environ Health* 212(5):459–469
- Friedman BJ, Freeland-Graves JH, Bales CW, Behmardi F, Shorey-Kutschke RL, Willis RA, Crosby JB, Trickett PC, Houston SD (1987) Manganese balance and clinical observations in young men fed a manganese-deficient diet. *J Nutr* 117(1):133–143
- Ganz T (2013) Systemic iron homeostasis. *Physiol Rev* 93(4):1721–1741. doi:[10.1152/physrev.00008.2013](https://doi.org/10.1152/physrev.00008.2013)
- Ganz T, Nemeth E (2011) Hepcidin and disorders of iron metabolism. *Annu Rev Med* 62:347–360. doi:[10.1146/annurev-med-050109-142444](https://doi.org/10.1146/annurev-med-050109-142444)
- Geloso MC, Corvino V, Michetti F (2011) Trimethyltin-induced hippocampal degeneration as a tool to investigate neurodegenerative processes. *Neurochem Int* 58(7):729–738. doi:[10.1016/j.neuint.2011.03.009](https://doi.org/10.1016/j.neuint.2011.03.009)
- Gibaud S, Jaouen G (2010) Arsenic-based drugs: from fowler’s solution to modern anticancer chemotherapy. In: Jaouen G, Metzler-Nolte N (eds) Medicinal organometallic chemistry, vol 32, Topics in organometallic chemistry. Springer, Berlin Heidelberg, pp 1–20. doi:[10.1007/978-3-642-13185-1\\_1](https://doi.org/10.1007/978-3-642-13185-1_1)
- Goldstein GW (1993) Evidence that lead acts as a calcium substitute in 2nd messenger metabolism. *Neurotoxicology* 14(2–3):97–102
- Gould E (2009) Childhood lead poisoning: conservative estimates of the social and economic benefits of lead hazard control. *Environ Health Perspect* 117(7):1162–1167. doi:[10.1289/ehp.0800408](https://doi.org/10.1289/ehp.0800408)
- Goyer RA (1996) Results of lead research: prenatal exposure and neurological consequences. *Environ Health Perspect* 104(10):1050–1054

- Grandjean P, Herz KT (2015) Trace elements as paradigms of developmental neurotoxicants: lead, methylmercury and arsenic. *J Trace Elem Med Biol* 31:130–134. doi:[10.1016/j.jtemb.2014.07.023](https://doi.org/10.1016/j.jtemb.2014.07.023)
- Guilarte TR (2010) Manganese and Parkinson's disease: a critical review and new findings. *Environ Health Perspect* 118(8):1071–1080. doi:[10.1289/ehp.0901748](https://doi.org/10.1289/ehp.0901748)
- Guilarte TR, Miceli RC, Altmann L, Weinsberg F, Winneke G, Wiegand H (1993) Chronic prenatal and postnatal Pb<sup>2+</sup> exposure increases [3H]MK801 binding sites in adult rat forebrain. *Eur J Pharmacol* 248(3):273–275
- Harada M (1995) Minamata disease: methylmercury poisoning in Japan caused by environmental pollution. *Crit Rev Toxicol* 25(1):1–24. doi:[10.3109/10408449509089885](https://doi.org/10.3109/10408449509089885)
- Harry GJ, Lefebvre d'Helencourt C (2003) Dentate gyrus: alterations that occur with hippocampal injury. *Neurotoxicology* 24(3):343–356. doi:[10.1016/S0161-813X\(03\)00039-1](https://doi.org/10.1016/S0161-813X(03)00039-1)
- Hershko C, Peto TE, Weatherall DJ (1988) Iron and infection. *Br Med J (Clin Res Ed)* 296(6623):660–664
- Hughes MF, Beck BD, Chen Y, Lewis AS, Thomas DJ (2011) Arsenic exposure and toxicology: a historical perspective. *Toxicol Sci* 123(2):305–332. doi:[10.1093/toxsci/kfr184](https://doi.org/10.1093/toxsci/kfr184)
- IARC (2012) A review of human carcinogens. Part C: Arsenic, metals, fibres, and dusts. IARC Monographs, pp 196–211
- IOM (2002) Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium and zinc. pp 1–20
- Jensen PD, Jensen FT, Christensen T, Eiskjaer H, Baandrup U, Nielsen JL (2003) Evaluation of myocardial iron by magnetic resonance imaging during iron chelation therapy with deferoxamine: indication of close relation between myocardial iron content and chelatable iron pool. *Blood* 101(11):4632–4639. doi:[10.1182/blood-2002-09-2754](https://doi.org/10.1182/blood-2002-09-2754)
- Jiang GB, Zhou QF, He B (2000) Tin compounds and major trace metal elements in organotin poisoned patient's urine and blood measured by gas chromatography flame photometric detector and inductively coupled plasma mass spectrometry. *Bull Environ Contam Toxicol* 65:277–284
- Kaur S, Nehru B (2013) Alteration in glutathione homeostasis and oxidative stress during the sequelae of trimethyltin syndrome in rat brain. *Biol Trace Elem Res* 153(1–3):299–308. doi:[10.1007/s12011-013-9676-x](https://doi.org/10.1007/s12011-013-9676-x)
- Kim G, Lee HS, Seok Bang J, Kim B, Ko D, Yang M (2015) A current review for biological monitoring of manganese with exposure, susceptibility, and response biomarkers. *J Environ Sci Health C Environ Carcinog Ecotoxicol Rev* 33(2):229–254. doi:[10.1080/10590501.2015.1030530](https://doi.org/10.1080/10590501.2015.1030530)
- Krewski D, Yokel RA, Nieboer E, Borchelt D, Cohen J, Harry J, Kacew S, Lindsay J, Mahfouz AM, Rondeau V (2007) Human health risk assessment for aluminium, aluminium oxide, and aluminium hydroxide. *J Toxicol Environ Health B Crit Rev* 10(Suppl 1):1–269. doi:[10.1080/10937400701597766](https://doi.org/10.1080/10937400701597766)
- Kumar V, Gill KD (2014) Oxidative stress and mitochondrial dysfunction in aluminium neurotoxicity and its amelioration: a review. *Neurotoxicology* 41:154–166. doi:[10.1016/j.neuro.2014.02.004](https://doi.org/10.1016/j.neuro.2014.02.004) S0161-813X(14)00027-8
- Kumar S, Jain S, Aggarwal CS, Ahuja GK (1987) Encephalopathy due to inorganic lead exposure in an adult. *Jpn J Med* 26(2):253–254
- Kyriakides MA, Sawyer RT, Allen SL, Simpson MG (1990) Mechanism of action of triethyltin on identified leech neurons. *Toxicol Lett* 53(3):285–295
- Lattanzi W, Corvino V, Di Maria V, Michetti F, Geloso MC (2013) Gene expression profiling as a tool to investigate the molecular machinery activated during hippocampal neurodegeneration induced by trimethyltin (TMT) administration. *Int J Mol Sci* 14(8):16817–16835. doi:[10.3390/ijms140816817](https://doi.org/10.3390/ijms140816817)
- Le Lan C, Loreal O, Cohen T, Ropert M, Glickstein H, Laine F, Pouchard M, Deugnier Y, Le Treut A, Breuer W, Cabantchik ZI, Brissot P (2005) Redox active plasma iron in C282Y/C282Y hemochromatosis. *Blood* 105(11):4527–4531. doi:[10.1182/blood-2004-09-3468](https://doi.org/10.1182/blood-2004-09-3468)
- Leffers L, Ebert F, Taleshi MS, Francesconi KA, Schwerdtle T (2013a) *In vitro* toxicological characterization of two arsenosugars and their metabolites. *Mol Nutr Food Res* 57(7):1270–1282. doi:[10.1002/mnfr.201200821](https://doi.org/10.1002/mnfr.201200821)
- Leffers L, Wehe CA, Huwel S, Bartel M, Ebert F, Taleshi MS, Galla HJ, Karst U, Francesconi KA, Schwerdtle T (2013b) *In vitro* intestinal bioavailability of arsenosugar metabolites and presys-

- temic metabolism of thio-dimethylarsinic acid in Caco-2 cells. *Metallomics* 5(8):1031–1042. doi:[10.1039/c3mt00039g](https://doi.org/10.1039/c3mt00039g)
- Ljung K, Palm B, Grandér M, Vahter M (2011) High concentrations of essential and toxic elements in infant formula and infant foods—a matter of concern. *Food Chem* 127(3):943–951
- Lovei M (1999) Eliminating a silent threat. World Bank support for the global. Phaseout of lead from gasoline. The George Foundation, India
- Mason LH, Harp JP, Han DY (2014) Pb neurotoxicity: neuropsychological effects of lead toxicity. *Biomed Res Int* 2014:840547. doi:[10.1155/2014/840547](https://doi.org/10.1155/2014/840547)
- Meyer S, Matissek M, Muller SM, Taleshi MS, Ebert F, Francesconi KA, Schwerdtle T (2014a) In vitro toxicological characterisation of three arsenic-containing hydrocarbons. *Metallomics* 6(5):1023–1033. doi:[10.1039/c4mt00061g](https://doi.org/10.1039/c4mt00061g)
- Meyer S, Schulz J, Jeibmann A, Taleshi MS, Ebert F, Francesconi KA, Schwerdtle T (2014b) Arsenic-containing hydrocarbons are toxic in the in vivo model *Drosophila melanogaster*. *Metallomics* 6(11):2010–2014. doi:[10.1039/c4mt00249k](https://doi.org/10.1039/c4mt00249k)
- Michalke B, Fernsebner K (2014) New insights into manganese toxicity and speciation. *J Trace Elem Med Biol* 28(2):106–116. doi:[10.1016/j.jtemb.2013.08.005](https://doi.org/10.1016/j.jtemb.2013.08.005)
- Mielke HW, Gonzales CR, Smith MK, Mielke PW (1999) The urban environment and children's health: soils as an integrator of lead, zinc, and cadmium in New Orleans, Louisiana, U.S.A. *Environ Res* 81(2):117–129. doi:[10.1006/enrs.1999.3966](https://doi.org/10.1006/enrs.1999.3966)
- Mitra S, Gera R, Siddiqui WA, Khandelwal S (2013) Tributyltin induces oxidative damage, inflammation and apoptosis via disturbance in blood-brain barrier and metal homeostasis in cerebral cortex of rat brain: an in vivo and in vitro study. *Toxicology* 310:39–52. doi:[10.1016/j.tox.2013.05.011](https://doi.org/10.1016/j.tox.2013.05.011)
- Nakamura M, Hachiya N, Murata KY, Nakanishi I, Kondo T, Yasutake A, Miyamoto K, Ser PH, Omi S, Furusawa H, Watanabe C, Usuki F, Sakamoto M (2014) Methylmercury exposure and neurological outcomes in Taiji residents accustomed to consuming whale meat. *Environ Int* 68:25–32. doi:[10.1016/j.envint.2014.03.005](https://doi.org/10.1016/j.envint.2014.03.005)
- Naranmandura H, Iyata K, Suzuki KT (2007) Toxicity of dimethylmonothioarsinic acid toward human epidermoid carcinoma A431 cells. *Chem Res Toxicol* 20(8):1120–1125. doi:[10.1021/tx700103y](https://doi.org/10.1021/tx700103y)
- Naujokas MF, Anderson B, Ahsan H, Aposhian HV, Graziano JH, Thompson C, Suk WA (2013) The broad scope of health effects from chronic arsenic exposure: update on a worldwide public health problem. *Environ Health Perspect* 121(3):295–302. doi:[10.1289/ehp.1205875](https://doi.org/10.1289/ehp.1205875)
- Nichani V, Li WI, Smith MA, Noonan G, Kulkarni M, Kodavor M, Naheer LP (2006) Blood lead levels in children after phase-out of leaded gasoline in Bombay, India. *Sci Total Environ* 363(1–3):95–106. doi:[10.1016/j.scitotenv.2005.06.033](https://doi.org/10.1016/j.scitotenv.2005.06.033)
- Noland EA, Taylor DH, Bull RJ (1982) Monomethyl- and trimethyltin compounds induce learning deficiencies in young rats. *Neurobehav Toxicol Teratol* 4(5):539–544
- Olanow CW (2004) Manganese-induced Parkinsonism and Parkinson's disease. *Ann N Y Acad Sci* 1012:209–223
- Olympio KP, Goncalves C, Gunther WM, Bechara EJ (2009) Neurotoxicity and aggressiveness triggered by low-level lead in children: a review. *Rev Panam Salud Publica* 26(3):266–275
- Page KE, White KN, McCrohan CR, Killilea DW, Lithgow GJ (2012) Aluminium exposure disrupts elemental homeostasis in *Caenorhabditis elegans*. *Metallomics* 4(5):512–522. doi:[10.1039/c2mt00146b](https://doi.org/10.1039/c2mt00146b)
- Piga A, Longo F, Duca L, Roggero S, Vinciguerra T, Calabrese R, Hershko C, Cappellini MD (2009) High nontransferrin bound iron levels and heart disease in thalassemia major. *Am J Hematol* 84(1):29–33. doi:[10.1002/ajh.21317](https://doi.org/10.1002/ajh.21317)
- Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC (1998) Exposure of the U.S. population to lead, 1991–1994. *Environ Health Perspect* 106(11):745–750
- Racette BA (2014) Manganism in the 21st century: the Hanninen lecture. *Neurotoxicology* 45:201–207. doi:[10.1016/j.neuro.2013.09.007](https://doi.org/10.1016/j.neuro.2013.09.007)
- Raml R, Rumpfer A, Goessler W, Vahter M, Li L, Ochi T, Francesconi KA (2007) Thio-dimethylarsinate is a common metabolite in urine samples from arsenic-exposed women in Bangladesh. *Toxicol Appl Pharmacol* 222(3):374–380. doi:[10.1016/j.taap.2006.12.014](https://doi.org/10.1016/j.taap.2006.12.014), S0041-008X(06)00488-1 [pii]

- Saary MJ, House RA (2002) Preventable exposure to trimethyl tin chloride: a case report. *Occup Med* 52(4):227–230
- Salvador GA, Uranga RM, Giusto NM (2010) Iron and mechanisms of neurotoxicity. *Int J Alzheimers Dis* 2011:720658. doi:[10.4061/2011/720658](https://doi.org/10.4061/2011/720658)
- Santamaria AB (2008) Manganese exposure, essentiality and toxicity. *Indian J Med Res* 128(4):484–500
- Schulze MB, Linseisen J, Kroke A, Boeing H (2001) Macronutrient, vitamin, and mineral intakes in the EPIC–Germany cohorts. *Ann Nutr Metab* 45(5):181–189. doi:[10.1159/000046727](https://doi.org/10.1159/000046727)
- Schumann K (2001) Safety aspects of iron in food. *Ann Nutr Metab* 45(3):91–101. doi:[10.1159/000046713](https://doi.org/10.1159/000046713)
- Snoeiij NJ, van Iersel AA, Penninks AH, Seinen W (1985) Toxicity of triorganotin compounds: comparative in vivo studies with a series of trialkyltin compounds and triphenyltin chloride in male rats. *Toxicol Appl Pharmacol* 81(2):274–286
- Strain JJ, Yeates AJ, van Wijngaarden E, Thurston SW, Mulhern MS, McSorley EM, Watson GE, Love TM, Smith TH, Yost K, Harrington D, Shamlaye CF, Henderson J, Myers GJ, Davidson PW (2015) Prenatal exposure to methyl mercury from fish consumption and polyunsaturated fatty acids: associations with child development at 20 mo of age in an observational study in the Republic of Seychelles. *Am J Clin Nutr* 101(3):530–537. doi:[10.3945/ajcn.114.100503](https://doi.org/10.3945/ajcn.114.100503)
- Syversen T, Kaur P (2012) The toxicology of mercury and its compounds. *J Trace Elem Med Biol* 26(4):215–226. doi:[10.1016/j.jtemb.2012.02.004](https://doi.org/10.1016/j.jtemb.2012.02.004)
- Tomljenovic L (2011) Aluminum and Alzheimer’s disease: after a century of controversy, is there a plausible link? *J Alzheimers Dis* 23(4):567–598. doi:[10.3233/JAD-2010-101494VQ1P78553222661M](https://doi.org/10.3233/JAD-2010-101494VQ1P78553222661M)
- Tuschl K, Mills PB, Clayton PT (2013) Manganese and the brain. *Int Rev Neurobiol* 110:277–312. doi:[10.1016/B978-0-12-410502-7.00013-2](https://doi.org/10.1016/B978-0-12-410502-7.00013-2)
- Tyler CR, Allan AM (2014) The effects of arsenic exposure on neurological and cognitive dysfunction in human and rodent studies: a review. *Curr Environ Health Rep* 1:132–147. doi:[10.1007/s40572-014-0012-1](https://doi.org/10.1007/s40572-014-0012-1)
- USDA (2014) China’s maximum levels for contaminants in food. Foreign Agriculture Service Global Agriculture Information Network Report CH14058
- Vahidnia A, van der Voet GB, de Wolff FA (2007) Arsenic neurotoxicity—a review. *Hum Exp Toxicol* 26(10):823–832. doi:[10.1177/0960327107084539](https://doi.org/10.1177/0960327107084539)
- Verstraeten SV, Aimo L, Oteiza PI (2008) Aluminium and lead: molecular mechanisms of brain toxicity. *Arch Toxicol* 82(11):789–802. doi:[10.1007/s00204-008-0345-3](https://doi.org/10.1007/s00204-008-0345-3)
- Wang QQ, Thomas DJ, Naranmandura H (2015) Importance of being thiomethylated: formation, fate, and effects of methylated thioarsenicals. *Chem Res Toxicol* 28(3):281–289. doi:[10.1021/tx500464t](https://doi.org/10.1021/tx500464t)
- WHO (1998) Human exposure to lead. Report on the Human Exposure Assessment Location (HEAL) Programme. Meeting held in Bangkok, Thailand
- WHO (2001) Arsenic and arsenic compounds. Environmental health criteria, vol 224. Geneva
- Yoo CI, Kim Y, Jeong KS, Sim CS, Choy N, Kim J, Eum JB, Nakajima Y, Endo Y, Kim YJ (2007) A case of acute organotin poisoning. *J Occup Health* 49(4):305–310

# Chapter 6

## Integrated Assessment of Policies for Reducing Health Impacts Caused by Air Pollution

Rainer Friedrich

**Abstract** Air pollution in the EU is still causing considerable health impacts, thus further reduction of air pollution is necessary. However, air pollution control policies are often expensive and have side effects, especially by increasing or decreasing greenhouse gas emissions and thus influencing climate change. An integrated assessment of air pollution control policies thus has to take the effects on climate change into account and furthermore has to ensure, that the most efficient policy, i.e. the bundle of measures with the highest net benefit is chosen. Here, a methodology for carrying out an integrated assessment of air pollution control policies, the impact pathway approach, is described. The methodology includes the estimation of utility losses and is able to assess as well technical measures (that change emission factors, e.g. by using improved filters) as non-technical measures (e.g. changes in behaviour/activities, e.g. using public transport after an increase in fuel taxes). The method is then applied for identifying the most effective and efficient measures for reducing air pollution from transport activities in Europe. The results show, that the most efficient transport policies for improving air quality and protecting climate are more use of bicycles and e-bikes, better traffic management, rail replacing air transport, shore based electricity and tighter emission limits for ships, tighter EURO limits (EURO 7), low emission zones in cities ( $\geq$ EURO V), improved tyres, brake pads and road cover and promotion of CNG and electric drive.

**Keywords** Integrated assessment • Air pollution control • Greenhouse gas reduction • Transport policies

---

R. Friedrich (✉)  
University of Stuttgart, Hessbruehlstr, 49 A, 70565 Stuttgart, Germany  
e-mail: [Rainer.Friedrich@ier.uni-stuttgart.de](mailto:Rainer.Friedrich@ier.uni-stuttgart.de)



## 6.1 Introduction

Air pollution as well in developed as in developing countries is—despite considerable efforts to reduce emissions of air pollutants at least in Europe and North America—still causing high health impacts. By far the highest health risks are caused by fine particles (especially PM<sub>2.5</sub>). Recent estimates suggest that the life time per person is on the average shortened by between 3 and 9 months (e.g. CAFE 2005) in Western Europe caused by a lifelong exposure to PM<sub>2.5</sub>. Please note that PM<sub>2.5</sub> is a mixture of different species, including secondary aerosols that are formed in the air from gaseous precursors, especially NO<sub>2</sub>, SO<sub>2</sub>, NH<sub>3</sub> and NMVOC. Further to primary and secondary fine particles NO<sub>2</sub> is causing considerable health impacts followed by ozone.

The European Commission has reacted to this challenge by launching a number of directives as well for controlling emissions as for controlling ambient concentrations of pollutants. The Air Quality Directive, controlling the ambient concentration of a number of pollutants, has recently (2008) been revised. A major improvement was adding new air quality objectives for PM<sub>2.5</sub>, including limit values and exposure related objectives.

However the limit values are still frequently exceeded, furthermore even if all limit values would have been met, considerable health impacts caused by fine particles and NO<sub>2</sub> would occur, as the concentration response relationships are assumed to be linear and for fine particles without threshold. A further reduction of air pollution is thus necessary. And indeed plans for reducing air pollution further are made from the EU to the city level.

However implementing policies and measures to reduce emissions of air pollutants has not only advantages, which are first of all the reduced health and environmental impacts caused by the reduction of the emission of air pollutants, but also more or less severe disadvantages, especially negative economic and social impacts. These include in most cases costs, which finally lead to a reduction of income in the population. Other disadvantages might be utility losses, i.e. losses of comfort or time. In addition, there are side effects, that may be positive or negative, e.g. changes in emissions of greenhouse gases and thus climate change impacts.

When making decisions about air pollution control policies, all these benefits, damages and costs should be taken into account. In other words, an integrated assessment should be made for helping decision makers to make decision by providing all relevant information. The basic idea is, that we live in a highly complex cross-linked world, where any decision has a lot of different impacts. And of course at least all important impacts should be taken into account when making the decision. Thus we define Integrated Assessment (IA) as a multidisciplinary process of synthesizing knowledge across scientific disciplines with the purpose of providing all relevant information to decision makers to help to make decisions. Of course what is relevant and thus should be integrated depends on the question to be answered respectively the decision to be taken. In the following, the different levels where integration takes place, are demonstrated on the example of PM<sub>10</sub>/PM<sub>2.5</sub> health impacts.

So, if the primary aim is to reduce health impacts caused by fine particles in an efficient way, firstly all sources of PM emissions should be taken into account, as the most efficient measures should be selected, however it is not known in advance, for which emission sources the most efficient reduction measures are available. Thus as well anthropogenic sources like transport, industry, energy conversion, households, agriculture as biogenic and natural sources like sea salt, particles from erosion, a.s.o. should be analysed. Of course, natural emission can in most cases not be reduced, but they cause a base concentration, that cannot be changed and thus limits the effect of measures that reduce anthropogenic emissions. Furthermore PM consists to a large part of secondary particles that are formed by chemical transformation in the air, especially ammonium nitrate, ammonium sulphate and organic secondary aerosols. Thus, not only primary PM emissions, but also emissions of the precursors of secondary aerosols like  $\text{SO}_2$ ,  $\text{NO}_x$ ,  $\text{NH}_3$ , NMVOC have to be considered.

Secondly, having identified emission reduction measures or policies, we find, that nearly all measures have an effect not only on PM or PM precursor emissions, but also on emissions of other substances like greenhouse gases. For instance if we reduce electricity demand, this will reduce PM emissions from electricity production, e.g. in a coal fired power plant, but it will also reduce emissions of many other pollutants including  $\text{CO}_2$  emissions. And in a full assessment, these 'secondary' benefits should also be taken into account when assessing the efficiency of the measure. Other measures, like the reduction of wood stove use or the burning of VOC in flue gases have a positive effect on particle or precursor emissions, but on the other hand lead to a substantial additional emission of  $\text{CO}_2$ , thus partly or fully compensating the positive effect of the measure with regard to air pollution control by additional climate change. So, these secondary benefits or burden also have to be taken into account in an integrated assessment.

Furthermore, we assume, that any activity that is causing emissions increases the utility or welfare of the person that is carrying out the activity. There would be no reason to conduct the activity, if this would not be true. Thus if by an air pollution control measure the activity is changed, there might be a loss in the utility for the emission source operators. For instance, if someone changes from using his private car to using a bicycle, not only emissions are reduced, but the trips with the bicycle are also much less expensive than with the private car. But as many people still prefer using private cars, obviously for them the utility gain of using the private car is higher than the additional costs compared to bicycle rides.

Furthermore, to make the assessment transparent and reproducible, it should be a quantitative analysis, i.e. by quantifying the relevant impacts and costs. Furthermore, to make the impacts, costs a.s.o. comparable, they should be transformed into a common unit, preferable a monetary unit (e.g. €). Monetary units have the advantage, that the value of the unit is defined independently from the assessment, thus monetary values per unit of damage can be transferred from a contingent valuation study, where they are measured, to the assessment situation with so called benefit transfer methods. Furthermore units are conceivable, everybody has a clear understanding of the value of 1000€, but how much would be 1000 utility points?

Once damages and benefits have been converted into damage or benefit costs, they can be directly compared with the costs and thus a cost-benefit analysis can be made. In sum, for policies to reduce air pollution at least the following features have to be calculated:

- Monetized avoided health and environmental impacts caused by the reduction of emissions of air pollutants (per year)
- Monetized impacts or avoided impacts of climate change caused by changes in emissions of greenhouse gases (per year)
- Annuity of costs
- Annual utility gains or losses, if applicable.

In the following the methodology for estimating these impacts is explained:

## 6.2 Assessing Impacts of Avoided Emissions of Air Pollutants: The Impact Pathway Approach

The principal methodology used to create unit assessment factors is the impact pathway approach (see Fig. 6.1). It is a bottom-up-approach, which can be used to estimate environmental costs by following the pathway from source of emissions via quality changes of air, soil and water to physical impacts. The approach was developed within a series of EU research projects known under the name ExternE. The aim of these projects was to estimate and monetize socio-environmental

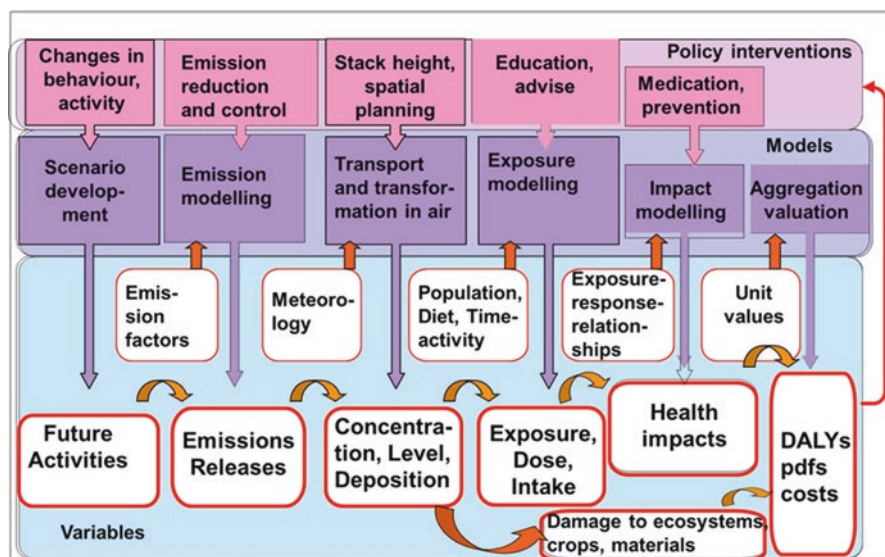


Fig. 6.1 The impact pathway or full chain approach

damages to support the internalization of external costs as a strategy to balance the purely economic dimension with social and environmental aspects.

The advantage of this approach is that the events linking a 'burden' to an 'impact' and the monetary valuation are considered sequentially, therefore providing a logical and transparent way of quantifying externalities (Bickel and Friedrich 2005).

The impact pathway approach consists of four main steps:

1. generating scenarios of activities, that cause emissions, and modelling of emissions of pollutants;
2. simulation of transport and chemical transformation in the atmosphere, the latter leading to changes in concentrations of secondary pollutants, e.g. secondary anorganic and organic aerosols, ozone, NO<sub>2</sub>;
3. assessment of impacts (to human health, ecosystems, materials) and
4. monetary valuation of the impacts.

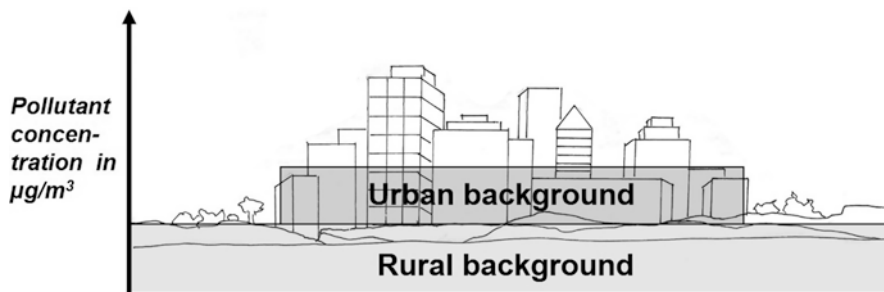
An illustration of the impact pathway is shown in Fig. 6.1.

A project, whose environmental performance and sustainability should be assessed, e.g. the construction of a bypass road or a new power plant leads to changes in activities, this means to a change of km driven in the road network or to a change in electricity generation in the power plants. For examples, due to a bypass a lot of cars will use the bypass, whereas the traffic density in the city might decrease. In general two scenarios describing the values of activities are built, one without and one with the project.

In a further step, the emissions are modelled for each scenario using emission factors per unit of activity, e.g. the NO<sub>x</sub> emissions per km driven with a EURO 5 gasoline car in an urban area. As we have to take into account the chemical processes occurring in the atmosphere, we can not only focus on the emissions of the project, but have to take into account the concentration of the substances, that react with the emitted pollutants. Thus we need to generate a full emission data set covering all emission sources in the relevant region. Furthermore, as projects are planned for future periods, we need scenarios of the future development of activities and emissions.

Following this, the concentrations of pollutants in environmental media, esp. air and soil, and in food have to be modelled. For this step, the release heights, the location, meteorological parameters and the concentration of other pollutants, that react with the emitted substance and thus forms secondary pollutants have to be taken into account. It is important to note that not only local damage has to be considered—air pollutants are transported and chemically transformed in the atmosphere and will cause considerable damage hundreds of kilometres away from the source. So local, European wide and hemispheric modelling is required. With our tool ECOSENSE<sup>®</sup> we use a parametric version of the 'Unified EMEP model' (2003) for the regional atmospheric modelling.

There is a distinguishable difference between the background concentration in a city and the background concentration in the surrounding area, as depicted in Fig. 6.2. Therefore, the concentrations calculated with the chemical transport and transformation models for larger grid cells have to be adjusted to take into account, that the



**Fig. 6.2** Rural background concentration and additional urban background concentrations (Urban Increment), (Ortiz and Friedrich 2013)

concentration in the part of the grid cell, where urban areas are located and where people live and are exposed is higher than in the rural parts.

To accomplish this, the urban increment (i.e., the difference between regional and urban background pollutant concentrations) has to be included in the analysis. The urban increment model, developed by Torras Ortiz and Friedrich (2013), can be used for estimating this PM<sub>10</sub> and PM<sub>2.5</sub> increment for large European cities.

The increment for PM is calculated using a regression function, that uses parameter values such as wind speed, size of the urban area and the primary emissions released from low-height sources in the urban area (Ortiz and Friedrich 2013).

Most health impacts from air pollutants are caused by inhalation of the pollutants, thus health impacts are related to the concentration of pollutants in the inhaled air, which is different from the background concentration in the air in cities. Epidemiological studies usually correlate health impacts with concentrations measured or modelled at a fixed place (e.g. the urban background concentration), and thus such concentrations are used to estimate health effects. However it would be better to use the exposure (i.e. the concentrations at the places and times where and when people sojourn) to estimate health impacts for two reasons.

First, the contribution of emissions to the background concentration is different from the contribution to exposure. For example, urban traffic emissions are emitted in street canyons and so nearer to the windows of houses than the emissions of central heating devices. This increases the effectiveness of measures in the transport sector.

Second, some changes in behaviour only change the exposure, but not the background concentration. If for instance a person stays inside instead of carrying out an outside activity, this will change his exposure but not the background concentration.

Exposure models are currently developed, e.g. in the EC project HEALS ([www.heals-eu.eu](http://www.heals-eu.eu)), but not yet ready for use. So, deviating from the scheme in Fig. 6.1, for the exemplary results shown later concentrations are used to estimate health impacts. To estimate the health effects caused by background concentration in cities, concentration-response functions are used, that link the exposure changes to changes

**Table 6.1** Risk functions for mortality risks from exposure to PM<sub>2.5</sub> and NO<sub>2</sub> (WHO 2013)

Pollutant	Relative risk (95 % C.I.) All cause natural mortality >30 years
PM <sub>2.5</sub> (per 10 µg/m <sup>3</sup> )	1.062 (1.04–1.083)
NO <sub>2</sub> (per 10 µg/m <sup>3</sup> above 20 µg/m <sup>3</sup> )	1.055 (1.03–1.080), up to 33 % overlap

in health effects. The health effects of simple pathway pollutants are calculated using impact functions. To construct an impact function, three components are needed:

- the relative risk for a certain health endpoint to occur per exposed person per unit of increase of pollutant concentration,
- the background rate of disease within the population and
- the proportion of the population exposed.

Recently, WHO published a report on ‘Health risks of air pollution in Europe—HRAPIE project Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide’ (WHO 2013) which contains recommendations for relative risks to be used for cost-benefit analysis. These risk function will be used for the analysis. The most important concentration–response functions are shown in Table 6.1 for mortality risk from exposure to PM<sub>2.5</sub> and NO<sub>2</sub>. Please note that the mortality risks from PM<sub>2.5</sub> are considerably higher than those of NO<sub>2</sub>, as the function for NO<sub>2</sub>, but not the one for PM<sub>2.5</sub>, has a threshold (20 µg/m<sup>3</sup>). Furthermore, WHO estimates, that if both functions are applied together, results cannot be simply added, as around one third of the additional cases are counted in both results.

There are a larger number of further risk functions in the WHO (2013) study. A transformation of the relative risks given there into impact functions can be made using the background rates for the illness analysed. For Europe, these can be found e.g. in Rabl et al. (2014).

After having calculated the health impacts and the number of cases for the different endpoints, there are two different possibilities to aggregate the results. A possible unit to compare health impacts are DALYs, disability adjusted life years. However, for comparing health impacts with environmental impacts and costs, it is preferable to use monetary values; i.e. the transformation of health impacts into €, using contingent valuation. This approach is especially suitable for cost-benefit analyses.

The results are monetary values attributed to certain health effects, including market and non-market costs. These values express the willingness to pay to avoid these effects and—for morbidity only—the cost of illness (e.g. cost for the treatment of the illness in the hospital, medicine) as well as the production loss due to illness-related absence from work. The valuation of an asthma attack, for example, should not only include the cost of medical treatment, but also the willingness-to-pay (WTP) to avoid residual suffering. In general, the WTP costs of avoiding a risk of getting ill are much larger than the costs for the treatment and the production losses.

**Table 6.2** Selected monetary values relating to specific health end-points in €2010 (INTARESE 2011)

Health end-point	Central value in	
	€ <sub>2010</sub> per case	
Life expectancy reduction—value of life year lost	60,000	Euro
Respiratory hospital admissions	2990	Euro
Cardiac hospital admissions	2990	Euro
Work loss days (WLD)	441	Euro
Restricted activity days (RADs)	194	Euro
Minor restricted activity days (MRAD)	57	Euro
Lower respiratory symptoms	57	Euro
Cough days	57	Euro

Meanwhile, a large number of studies for monetizing health risks is available. These studies determine the willingness to pay to avoid a small risk of getting ill with different diseases, from a restricted activity day to non-fatal cancer, or a small risk of dying earlier, i.e. reducing life expectancy. A range of methodologies is used to determine the WTP. Stated preference methods use surveys; the most important methods are contingent valuation and attribute based choice modelling. Revealed preference methods observe the behaviour of people, often used methods are hedonic pricing and the averting behaviour method. Stated preference methods can be used universally for any assessment, while revealed preference methods have a more limited application.

For the health endpoints, for which concentration-response-relationships are known, sufficiently reliable WTP studies are available. To use these values, they have to be transferred from the context (location, year,...) of the WTP study to the context of the integrated assessment study. This is done with benefit transfer methods. One of the simplest benefit transfer methods is to adjust the monetary unit value per health endpoint with the ratio of the purchase power parity adjusted income per capita for the context of the WTP study and the context of the integrated assessment study.

Some exemplary monetary values used here for the valuation of the different endpoints can be found in Table 6.2.

One has to be aware, that only small risks can be monetized. Losing a life year with certainty or high probability is not accepted in a society; to prevent the death of a concrete person that suffers for instance from maritime distress or is trapped in a coal mine, everything possible would be done to rescue him regardless of the costs. Thus a value of 60,000€ means that a person would pay  $10^{-5} * 60,000$  € to avoid a probability of  $10^{-5}$  to have an accident.

The values given in the previous table are monetary values applicable for impacts of emissions released in the year 2010. For the calculation of monetary values for future years, an uplift factor has to be taken into account to reflect the higher willingness-to-pay (WTP) for avoiding health impacts.

As a result of the EU project NEEDS, evidence was found, that monetary values for health risks for future years increase with an inter-temporal elasticity to GDP per capita growth of 0.7–1.0 according to the following formula:

$$\text{WTP for year}(2010 + n) = \text{WTP for year}2010 * (1 + g)^{0.85*n}$$

With  $g$  = average growth rate of per capita income, WTP = willingness to pay.

In addition to health impacts, another damage category is ecosystem damage expressed as biodiversity loss. The impacts on biodiversity are expressed at species level, eventually leading to a Potentially Affected Fraction (PAF) or Potentially Disappeared Fraction (PDF) of species. For the terrestrial biodiversity losses due to eutrophication and acidification in Europe an approach developed by Ott et al. (2006) is used. Biodiversity losses are estimated in PDF, i.e. the potentially disappeared fraction of species per  $\text{m}^2$  and year compared to the number of species that would be present without the human intervention summed up over the area (in  $\text{m}^2$ ) and the years, where biodiversity losses occur. The monetary valuation is based on restoration costs of  $0.52\text{€}_{2010}/\text{PDF}$  (Ott et al. 2006). This approach assumes the costs to be investment expenditures required to offset damages done to the ecosystems by anthropogenic activity. This can be done by restoring the ecosystem or reinstating an environmental condition that is a perfect substitute for the lost ecosystem service.

### 6.3 Application of the Pathway Approach

The process of assessing emission changes with the impact pathway approach obviously is quite complex and laborious. To facilitate the practical use of the method, the methodology has been used to generate ‘unit values’, i.e. unit impacts or risks per kg of pollutant emitted. These unit values are provided with a software tool named ECOSENSE (available at [www.externe.info](http://www.externe.info)). As input emissions of PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, SO<sub>2</sub> and NH<sub>3</sub> have to be provided, distinguishing between release in the different European countries, emission from high stacks, low stacks or in a street canyon, release in rural areas, cities or large agglomerations. Impacts and monetized impacts are then provided.

### 6.4 Uncertainties

Individual sources of uncertainty have to be identified and quantified. It is appropriate to group them into different categories, even though there may be some overlap:

- i. data uncertainty, e.g. slope of a dose–response function, cost of a day of restricted activity and deposition velocity of a pollutant;
- ii. model uncertainty, e.g. assumptions about causal links between a pollutant and a health impact, assumptions about form of a dose–response function (e.g. with



- or without threshold), and choice of models for atmospheric dispersion and chemistry;
- iii. uncertainty about policy and ethical choices, e.g. discount rate for intergenerational costs, and value of statistical life;
  - iv. uncertainty about the future e.g. the potential for reducing crop losses by the development of more resistant species;
  - v. idiosyncrasies of the analyst e.g. interpretation of ambiguous or incomplete information.

The first two categories (data and model uncertainties) are of a scientific nature and can be analysed by using statistical methods. Results show a geometric standard deviation of ca. 2–3, which means that the true value could be 2–3 times smaller or larger than the median estimate. The largest uncertainties lie in the exposure-response function for health impacts and the value of a life year lost—current research is directed towards reducing these uncertainties, which reflect our limited knowledge. These results are discussed in more detail in Spadaro and Rabl (2008).

Furthermore, certain basic assumptions have to be made such as the discount rate, the valuation of damage in different parts of the world, the treatment of risks with large impacts or the treatment of gaps in data or scientific knowledge. Here, a sensitivity analysis should be carried out demonstrating the impact of different choices on the results. Decisions then would sometimes necessitate a choice of the decision-maker about the assumption to be used for the decision. This would still lead to a decision process that is transparent and, if the same assumptions were used throughout different decisions, consistent.

## 6.5 Assessment of Greenhouse Gas Emissions

Policies that reduce air pollution in most cases also influence greenhouse gas emissions and policies that are designed to reduce greenhouse gas emissions in most cases also decrease or increase emissions of air pollutants. The results of a case study of assessing climate change policies carried out in the EU research project HEIMTSA indicates, that the impacts of climate policies on air pollution and human health are—expressed as external costs—about as important as the impacts on climate change. While most climate policies reduce environmental human health impacts, some like biomass burning in small stoves or insulation of buildings with new tight windows increase health risks substantially.

Thus an integrated assessment of policies for air pollution control has to include an assessment of the changes of greenhouse gas emissions. While the estimation of changes of greenhouse gas emissions is already included in newer integrated assessments, the question remains, how greenhouse gas emissions should be compared with health risks within a cost-benefit analysis. As estimates of marginal damage costs of climate change show a very wide range of results, we propose to use estimated marginal avoidance costs for reaching the EU's short and long term aims for

**Table 6.3** Marginal avoidance costs (MAC) in the EU to reach the ‘2° aim’, discount rate 5%/a, based on results of Kuik et al. (2009)

	2010	2020	2030	2040	2050
€ <sub>2010</sub> /t CO <sub>2</sub>	36 (20–63)	58 (33–103)	95 (54–167)	155 (88–272)	252 (143–443)

greenhouse gas reduction, e.g. those estimated in a meta study by Kuik et al. (2009). If we use the declared aim of the European Commission to limit the temperature increase of earth’s surface to 2° within a world-wide strategy, marginal avoidance costs shown in Table 6.3 are estimated. Thus, as the exemplary results shown in Chap. 7 are for 2020, 58€ per t of CO<sub>2,eq</sub> are used.

## 6.6 Assessment of Utility Losses

The overall aim of the activities of humans is to maximize their well-being or welfare. For example, if someone decides to use his own car to travel, although using public transport or the bicycle would be much cheaper, there must be some welfare or utility gain of using the car that compensates for the additional costs. A part of this utility gain might be time gains (if the car is faster than public transport or bicycle use). But in addition there are further advantages, e.g. more comfort including a guaranteed seat, feeling safer, no waiting times resp. more flexibility and so on. As these are legitimate arguments, they should be taken into account within an integrated assessment.

The methodology, that can be used to quantify such utility gains or losses, is the ‘rule of one-half’. If a user shifts from a more expensive technique with costs  $x$  to a technique with costs  $y$  ( $x > y$ ) after the costs  $x$  have been increased by  $\Delta x$ , the average utility loss would be  $x + \Delta x/2 - y$ , the income gain would be  $x - y$ , thus the average net utility loss  $\Delta x/2$ . Utility gains and losses would be estimated for all non-technical measures, where a price change (e.g. by implementing a tax) leads to an enhanced use of a cheaper activity or the omission of the activity without replacement.

## 6.7 Assessment of Policies and Measures for Reducing Air Pollution in the Transport Sector

In the following an example for applying the methodology is shown, namely the results of an integrated assessment of policies and measures for reducing air pollution in the transport sector. The results have been generated within the EU project TRANSPHORM ([www.transphorm.eu](http://www.transphorm.eu)).

The first step was to identify measures that reduce the emissions of air pollutants (and greenhouse gases) in the transport sector, covering all transport modes. In a

screening process the following potentially effective measures have been chosen for the assessment:

Measures in urban areas:

- Enhanced use of bicycles (15–30 % of all trips in cities)
- Enhanced use of public transport (30–35 % of all trips in cities)
- More electric cars (2.5 % of passenger car fleet)
- Car pooling
- Better traffic management
- In low emission zones only EURO5 cars are allowed
- Ban of through traffic of heavy duty vehicles
- City toll for city centers reducing private traffic by 15 %
- Parking management
- 10 % of buses are hybrid buses
- Freight consolidation center

Measures on non-urban roads:

- Speed limit 110 km/h on motorways
- Speed limit 80 km/h on rural roads

Measures for all road transport:

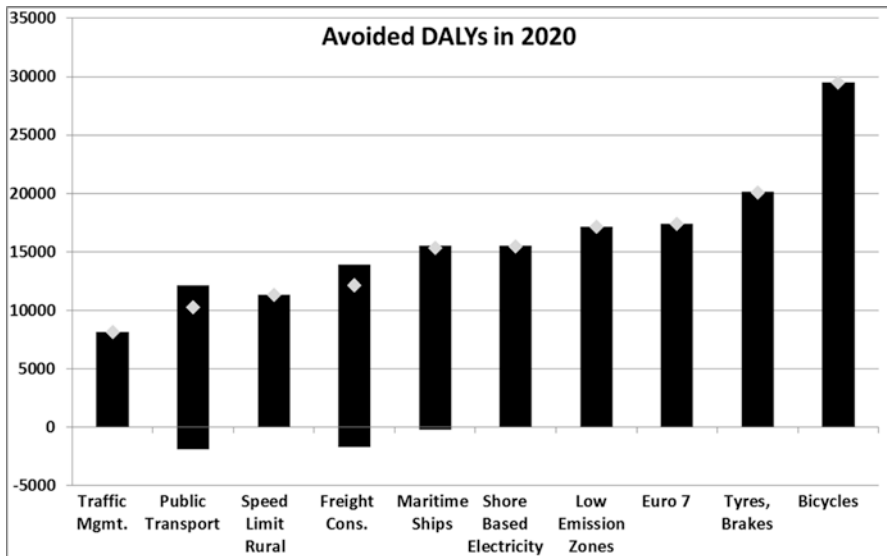
- Road pricing 1.5 cent per km
- Fuel tax is increased by 20 %
- EURO 7 is implemented in early 2019, especially reducing NO<sub>x</sub> emission limits further
- Improved tyres and brakes: 30 % reduction in PM10 emissions from abrasion
- 10 % share of biofuels
- 10 % cars running with CNG (compressed natural gas).

Other modes than road:

- Tighter NO<sub>x</sub> emission limits for inland ships
- Tighter NO<sub>x</sub> and SO<sub>2</sub> emission limits for sea going vessels
- Electricity for ships in the harbour provided by connection to grid
- Tax for kerosene
- Air transport for distances <500 km shifted to rail

For all these measures, the effects of implementing the measure in the EU, i.e. the reduction of health risks, when implementing the measure, is calculated using the methodology described in Chap. 2. The result of this analysis, the reduction of health impacts caused by the EU wide implementation of the measures, concretely the 10 most effective measures are shown in Fig. 6.3.

So for 2020, the enhanced use of bicycles turns out to be most effective of the measures followed by the enhanced use of long lasting tyres and brake pads. The 30,000 DALYs gained per year can be translated into a gain of life expectancy of roughly 20 min per person in Europe and per 1 year operation of the enhanced bicycle use. The small negative benefits (i.e. damage) for public transport and



**Fig. 6.3** Avoided health impacts in Europe in 1000 DALYs (disability adjusted life years) per year for the year 2020, caused by implementation of emission reduction measures in the EU

freight consolidation are due to the additional electricity production for public transport resp. electrical small trucks.

For 2030, the ranking changes. The introduction of EURO 7 standards are now the most effective measures, as in 2030 80% of the cars are EURO 7 cars, whereas in 2020 it have been only 20%. As in 2030 mainly EURO 6 cars and trucks are used, the effect of all measures reducing emissions of road transport is reduced.

All measures are also reducing the greenhouse gas (GHG) emissions. Ranking the measures according to their GHG reduction gives the result shown in Fig. 6.4.

The most important measures are now measures for air transport, especially the shift from air to rail for small distances of less than 500 km.

To be able to combine health and climate effects, we have to convert the damages into a common unit, here €. For the health damage, this is done using marginal damage costs as described in Chap. 2, for the greenhouse gas emissions an avoidance cost approach is used as described in Chap. 5. The result is shown in Fig. 6.5.

In the next step, the costs for implanting the measures are taken into account, the net benefits (benefits=avoided damages minus costs) are estimated. Shifting from private cars to the use of bicycles or public transport is saving money, if we assume, that the bicycle or public transport users disestablish their cars and if needed use car sharing. Bicycles are much cheaper than public transport, so the enhanced bicycle use ranks highest in the list of the most efficient measures (Fig. 6.6).

In the last step, we add utility losses as described in Chap. 5. Especially abandoning the own car and shifting to the use of bicycles or public transport will result in quite large utility losses, whereas technical measures (long-lasting tyres and brakes,

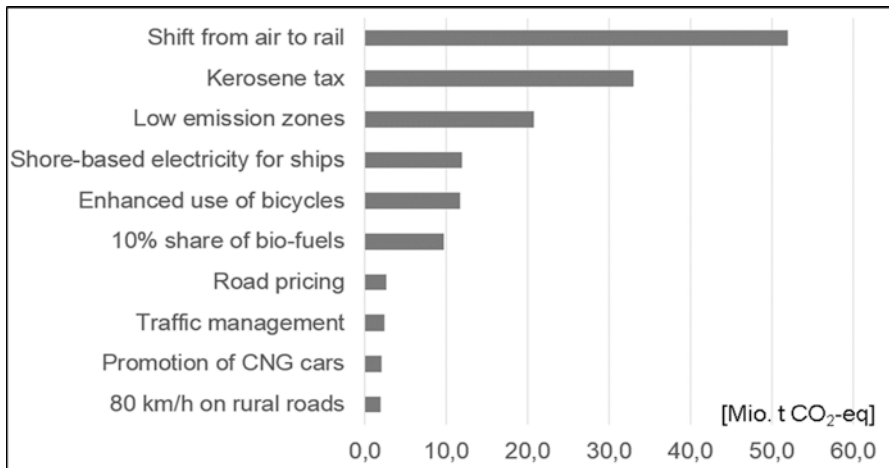


Fig. 6.4 Ranking of measures according to their GHG emission reduction in Europe 2020

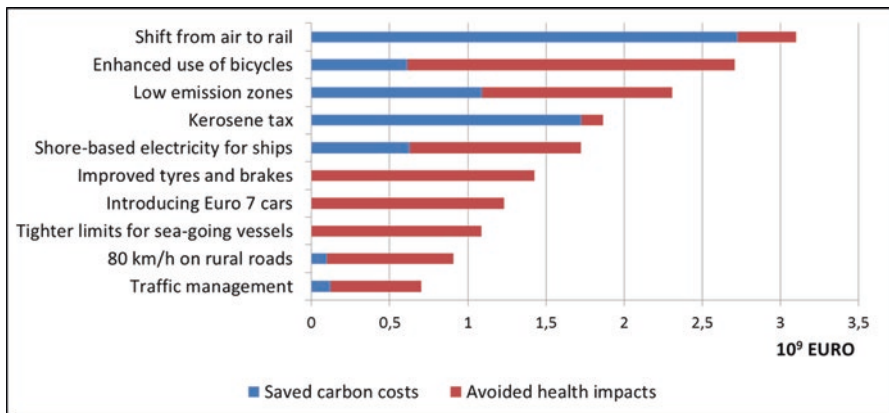


Fig. 6.5 Effectiveness of measures with regard to reduction of health and climate impacts 2020

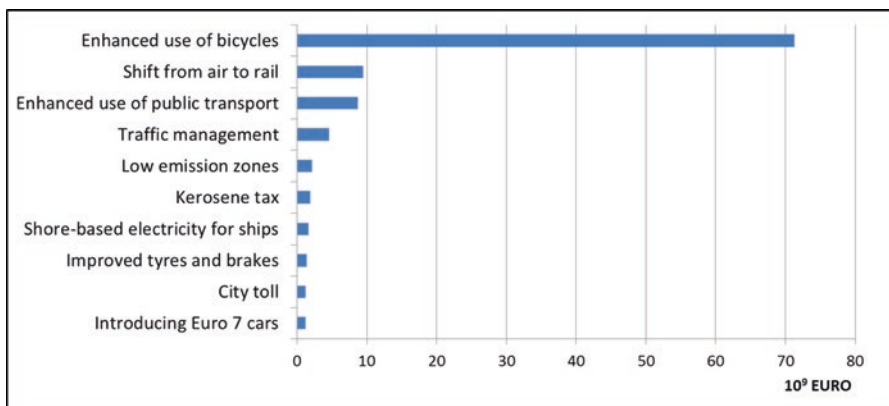
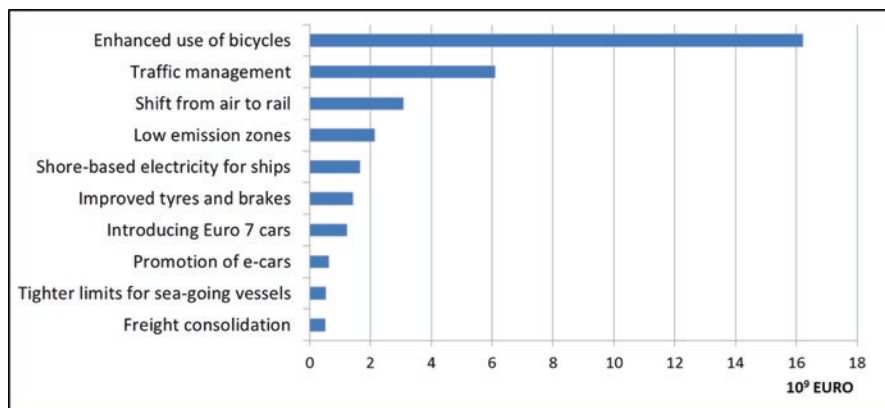


Fig. 6.6 The ten most efficient measures with the highest net benefit for Europe 2020



**Fig. 6.7** Most efficient measures taking account of utility losses; the net benefit=avoided health and climate damages minus costs minus utility losses (where relevant) are shown

EURO 7) do not have significant utility losses. Thus when subtracting utility losses, the net benefit of enhanced bicycle use is largely reduced, although this measure stays to be the most efficient one, whereas the more costly use of public transport vanishes from the top ten list (Fig. 6.7).

## 6.8 Conclusions

A methodology and tools for carrying out an integrated assessment have been developed and are available. The assessment of health impacts caused by air pollution as part of an integrated assessment is carried out using the ‘impact pathway’ or ‘full chain’ approach. A detailed web guidebook on how to carry out this approach can be found on ‘[www.integrated-assessment.eu](http://www.integrated-assessment.eu)’. The tool ECOSENSE for assessing health and environmental impacts of emissions of air pollution with a simplified approach can be found at ‘[www.externe.info](http://www.externe.info)’.

The methodology is constantly improved and extended. Recent improvements are especially the modelling of the urban increment and the inclusion of new concentration-response functions for NO<sub>2</sub>.

The use of the methodology has been demonstrated by assessing air pollution control measures in the transport sector. Further new elements are the inclusion of ‘non-technical’ measures (that change activities instead of emission factors) and the quantification of utility losses.

The results show, that the most efficient transport policies for improving air quality and protecting climate are:

- more use of bicycles and e-bikes,
- traffic management,
- rail replacing air transport,

low emission zones in cities,  
 shore based electricity and tighter emission limits for ships,  
 improved tyres and brakes with longer lifetime,  
 tighter EURO limits (EURO 7) and  
 promotion of CNG and electric drive.

However, further analyses for other sectors than transport (e.g. Theloke et al. 2013) suggest, that more effective and efficient measures can be found in other sectors than transport, especially in the agricultural sector. These measures include a reduction in meat consumption, the mounting of dust filters in hog houses and an optimized fertilizer application.

## References

- Air Quality Directive (2008) Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe. OJ L 152, pp 1–44, 11 June
- Bickel P, Friedrich R (eds) (2005) ExternE—externalities of energy methodology 2005 update. [http://www.externe.info/externe\\_d7/?q=node/30](http://www.externe.info/externe_d7/?q=node/30)
- CAFE (2005) Baseline scenario for the Clean Air for Europe Programme. [http://ec.europa.eu/environment/archives/cafes/activities/pdf/cafes\\_scenario\\_report\\_1.pdf](http://ec.europa.eu/environment/archives/cafes/activities/pdf/cafes_scenario_report_1.pdf)
- Kuik O, Brander L, Tol R (2009) Marginal abatement costs of greenhouse gas emissions: a meta-analysis. *Energy Policy* 37:S1395–S1403. doi:10.1016/j.enpol.2008.11.040
- Ortiz ST, Friedrich R (2013) A modelling approach for estimating background pollutant concentrations in urban areas. *Atmos Pollut Res* 4(2):147–156
- Ott W et al (2006) Assessment of biodiversity losses. [http://www.needs-project.org/RS1b/RS1b\\_D4.2.pdf](http://www.needs-project.org/RS1b/RS1b_D4.2.pdf)
- Rabl A, Spadaro JV, Holland M (2014) How much is clean air worth? Calculating the benefits of pollution control. Cambridge University Press, Cambridge
- Spadaro JV, Rabl A (2008) Estimating the uncertainty of damage costs of pollution: a simple transparent method and typical results. *Environ Impact Assess Rev* 28(2–3):166–183. doi:10.1016/j.eiar.2007.04.001
- Theloke J, Kuhn A, Kugler U, Friedrich R, Stern R, Jörß W, Bultjes P (2013) Berechnung von Kosteneffektivitäten im Projekt PAREST, UBA Texte, 50/2013. PAREST. <http://www.umweltbundesamt.de/publikationen/berechnung-von-kosteneffektivitaeten-im-projekt>
- Unified EMEP Model (2003) Unified EMEP Model description. [http://www.emep.int/index\\_model.html](http://www.emep.int/index_model.html)
- WHO (2013) Health risks of air pollution in Europe—HRAPIE project recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide. World Health Organization. [http://www.euro.who.int/\\_\\_data/assets/pdf\\_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf?ua=1](http://www.euro.who.int/__data/assets/pdf_file/0006/238956/Health-risks-of-air-pollution-in-Europe-HRAPIE-project,-Recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.pdf?ua=1)

# Chapter 7

## Monetary and Non-Monetary Measures of Health Benefits from Exposure Reduction

Milan Ščasný and Vojtěch Máca

**Abstract** Multiple pathways in which environmental hazards may affect human health translate to diversity of adverse impacts of impaired health on human welfare. Impaired health may in turn directly affect production or consumption opportunities at individual, household as well as societal level. While at a methodological level the channels of economic effects of impaired health are extensively identified and discussed it is considerably more challenging to establish respective empirical estimates. This chapter outlines approaches to economic valuation of health benefits from exposure reduction, discusses relevance of cost categories for various physical impacts and points to challenges in linking epidemiological findings to welfare implications. A succinct overview of available monetary values for a range of health outcomes is provided.

**Keywords** Health benefits • Health risk valuation • Non-market valuation • Value of a statistical life • Cost-of-illness • Willingness to pay • QALY • DALY • Damage function • Impact pathway analysis • ExternE

### 7.1 Introduction

Economics as a discipline studies how people make choices among competing alternatives, restricted by price and subject to resource constraints. To account for health, standard economic models of consumer behaviour were adapted to reflect the notion that many goods, services and behaviours that influence health are valued just for their influence on health. In other words people spend money and their time to “produce health”. This should be understood rather broadly, referring not only to goods and services produced and sold in market, but also other non-market factors, such as time, cultural and psychological aspects, institutional settings etc.

---

M. Ščasný (✉) • V. Máca  
Environment Center, Charles University,  
Jose Martiho 2/407, 162 00 Prague, Czech Republic  
e-mail: [Milan.Scasny@czp.cuni.cz](mailto:Milan.Scasny@czp.cuni.cz)



Looking from the other side, health contributes to individual utility (or social welfare)<sup>1</sup> in multiple ways. First, health directly affects utility because people prefer to be healthy rather than sick, or to be alive rather than to be dead. Second, the enjoyment of consumption of other goods and services is (at least in part) influenced by the health status so that marginal utility derived from consumption is partly conditional on health status. Third, good health is instrumental to an individual's (or community's) capability to undertake desired activities and to generate income that allows them to consume goods or leisure time spent with family and friends.

Unlike consumption of most goods and services that yields welfare directly, consumption of health goods and services does not provide utility per se. People incur these expenses simply because they believe the health good or the health service promote their health. As an analogue, consumers do not demand energy per se either, but they rather demand energy services such as a warm home, lighting, or washed dishes. In this case, the demanded energy service is a result of combining the energy use with household domestic durable goods (such as boiler, bulb, or dishwasher). Accordingly, the key direct determinants of economic welfare are the consumption of non-health goods and services, leisure, and health status. Health status of an individual is then a function of activities to avert illness, such as taking vitamins, exogenous factors such as exposure to pollution, and individual characteristics of given person.

Consequently, the impact of impaired health on an individual (or her household) can be measured in exactly the same terms—consumption of market and non-market non-health goods and services, leisure, and health status. Taking example of out-pocket expenditures, impaired health will drive up household consumption of health-related goods and services at expense of other non-health goods and services. In the same vein, increase in time spent seeking care or in work incapacity will reduce production of goods or provision of services, and indirectly (via reduced ability to produce income) also consumption.

There are other viewpoints though. From a perspective of a firm, absenteeism and presenteeism can reduce productivity and/or produce quality and consequently affect profits, diminishing consumption (or investment) opportunities of firm owners. At a societal level impaired health is related to (generally rising) expenditures on health care services, social security payments (i.e. disability or unemployment benefits), although impaired health may also lead to reduced room for governments to provide public goods and redistribute income. An important distinction between

---

<sup>1</sup> In welfare economics, social welfare is represented by social welfare function that is a mapping of individual preferences or judgments of everyone in the society as to collective choices. The functional form of social welfare depends on normative assumption on equity. For instance, in most contexts, a social welfare function provides a societal preference based on only individual utility functions. In the utilitarian or Benthamian social welfare fiction, the social welfare is measured as the total or sum of individual utilities (or incomes). An alternative can be based on the product rather than on the sum. In contrast, the Rawlsian approach (so called, max-min) measures the social welfare of society on the basis of the welfare of the least well-off individual member of society, i.e. the societal welfare is maximized if and only if the income of the poorest person in society is maximized without regard for the income of other individuals.

the concepts of individual (household) utility versus social welfare is that societies may also care about distributional issues, although a pragmatic approach commonly taken in societal benefit-cost analyses often uses a simplification in that societal welfare is a sum of welfare of individuals.

## 7.2 Economic Valuation of Health Impacts

### 7.2.1 *Damage Function Approach*

To assess the economic value of health impacts, the general approach currently used is based on the damage function approach (EC 2000; Alberini et al. 2015; Hunt et al. 2016). The damage function approach (in Europe also called impact pathway analysis, IPA) measures economic benefits of reduced (increased) exposures to pollution.

In brief, the IPA is an analytical procedure examining the sequence of processes through which polluting emissions result into external damages. The method allows to estimate the marginal physical impact and the marginal cost of pollution from certain emission or area source, as a function of technology and of the location of the plant. The IPA comprises of four basic steps: (1) selection of the reference emission source, determination of the technology used and of the harmful emissions released, (2) calculation of changes in pollutant concentrations for all affected regions using atmospheric dispersion models, (3) estimation of physical impacts from exposure using concentration-response functions (CFRs), and (4) translation of the physical impacts into monetary impacts by using appropriate valuation methods (see, Máca et al. 2012; Ščasný et al. 2015). These four general steps of the IPA can be described by following schematic chain.

#### Schematic IPA



In order to establish a causal relationship between pollution and human morbidity and mortality, the IPA uses the concentration-response functions calibrated using epidemiological and toxicological studies. Hunt et al. (2016) provides the latest review of these functions for ambient air pollution, while Alberini et al. (2015) review the unit values for the airborne pollution related health points.

(continued)

(continued)

Monetized impacts on human health are the most important category among all impacts expressed in monetary terms that are covered by the ExternE's IPA, and the impacts on mortality several times exceed the impacts on morbidity. The evidence points to underestimation of morbidity impacts of airborne pollution in health impact assessment. As a respond, Hunt et al. (2016) suggest marking up mortality costs by at least 10–15 % in order to broadly correct the health benefit estimates in the case of neglected morbidity impacts.

The IPA procedure has been incorporated into EcoSense tool—the integrated atmospheric dispersion and exposure assessment model<sup>2</sup>—that determines a range of impacts on human health, buildings and materials, biodiversity, and crop yields.

The ExternE's IPA method is very similar to an integrated assessment model used in the American studies to connect emissions to changes in concentrations, human exposures, physical effects and monetary damages by the Air Pollution Emission Experiments and Policy model (APEEP, see Muller and Mendelsohn 2007; Muller et al. 2011; Groosman et al. 2011). OECD's air pollution and human health review makes a comparison among several models including MIT's APEEP, IIASA's GAINS, and ExternE's IPA approach (Alberini et al. 2015). The limitation of damage function approach is its static setup—the number of adverse effects is calculated making no allowance for behavioural changes due to pollution.

### 7.2.2 *Willingness to Pay*

In order to measure a change in welfare, one needs a yardstick. There are in fact two mirroring measures, willingness to pay (WTP) for an improvement in one's health and willingness to accept (WTA) a compensation for its deterioration (this may also be posited as WTP for a decrease and WTA a compensation for an increase in health risks). Albeit these are two sides of the same coin, the relation between them is far from trivial. Standard economic theory suggests that differences regularly observed in WTP vs. WTA empirical studies are due to the income and substitution effects, since the two welfare measures evaluate the changes at the initial level, or new level of utility, respectively. Moreover, WTP is bounded by individual's (or her household) income, unlike WTA that can be in principle unrestricted. Apart from this

---

<sup>2</sup>EcoSense uses air transport models to control changes in the atmospheric concentration of pollutants at the local, regional and global level. An internet accessible version of EcoSense (EcoSenseWeb1.3) was developed within the NEEDS project (Preiss and Klotz 2008), see the details at <http://ecosenseweb.ier.uni-stuttgart.de/>.

income effect, numerous studies has pointed to other factors as well, including to what extent what is being valued is an “ordinary” market good (Horowitz and McConnell 2002), aversion to potential losses relative to potential gains, often called endowment effect (Kahneman et al. 1990), or subjects’ misconceptions in value elicitation process (Plott et al. 2005).

### 7.2.3 Cost Components of Health Impacts

The goal of valuation of health impacts is to capture all the components that may induce a change in welfare. The microeconomic approach builds upon trade-offs people are making in decisions about alternative uses of their resources that indicate the relative value they place to competing uses. Since many of these goods (and services) are normally traded on market, measuring these using monetary terms is a convenient reference of their willingness to pay.

From a societal perspective the economic values of health impacts associated with exposure to environmental hazards comprise the following five components:

1. medical (mitigating) expenses associated with treatment of illnesses,
2. defensive or aversive expenditures associated with attempts to prevent illnesses and other effects on welfare,
3. lost of productivity,
4. disutility (dis-welfare) associated with the symptoms (pain, suffer, and other inconveniencies) and lost opportunities for leisure activities,
5. welfare loss due to change in life expectancy or risk of premature death.

The mitigating treatment costs (1) and the avertive expenditures (2) used to be placed under same category named as *resource costs*. The first four components are related to morbidity, whereas the last one is associated with mortality impacts. A sum over the five cost components gives the total costs of health impacts. Hunt et al. (2016) point on possible overlap and double-counting when the morbidity cost components are summed cross them, however. For instance, elicited WTP to avoid the disutility associated with certain illness may also include financial and non-financial concerns in her assessment of her loss of welfare. The averting expenditures involved by moving house need not be separable from the disutility and productivity cost component. If medical treatment is effective, it will likely also reduce both the disutility and productivity costs (ibid.). It is worth to mention different practice in the performance of valuation studies in Europe and the Northern America. In many valuation studies being conducted in the Northern America, respondents are asked to state a value for both cost-of-illness and willingness-to-pay for disutility, such as Chestnut et al. (2006), as Americans bear more of the costs of illness, particularly they bear directly the mitigation costs. In contrast to Europe, most of the costs associated with illness used to be more covered by various public health and social insurance programs.

*Treatment cost* or ‘direct costs’ are the expenses incurred because of the illness including medical care, home care, travel costs etc. Any direct medical costs and non-medical costs used to mitigate consequences of the illness. These costs may be derived in top-down or bottom-up manner from healthcare statistics or in single studies (e.g. pharmacoeconomic or cost studies). There are wide differences in cost estimates for same health outcomes with respect to perspective taken (viewpoint of an individual, healthcare provider, or public authority), differences in standards of treatment (average length of hospital stay, drugs prescriptions etc.), as well as a lack of data (e.g. share between inpatient/outpatient treatment for particular illnesses). Among numerous efforts to provide standardized guidance, a widely acknowledged U.S. EPA’s Cost-of-Illness Handbook stands out, detailing precisely a bottom-up approach for estimating direct medical costs (Abt Associates Inc. 2007). On the top of direct medical costs this cost category should also include the opportunity cost of time spent in obtaining treatment.

*Avertive expenditures* may include the costs involved by relocation to area of lower air pollution, staying inside, and similar other.

*Loss of productivity* is an indirect cost category that expresses the value that has been lost due to an absence from work because of illness. The two dominant approaches are human capital and friction costs methods, both are however criticized on number of issues such as ignoring unpaid labour or deficiencies in theoretical founding (see e.g. Krol et al. 2013). Even the convention for the most obvious—valuation of time lost from work (or valuation of unpaid labour if pursued)—remains unsettled, but some of the divergences origin from different perspectives taken in the calculation. Measures commonly used include employees’ added value to the firm, employees’ gross income, average national gross income or GDP per employee (or per capita), or insurance payments; there is no consensus on superiority of one of the measures. Valuation of unpaid labour can also be measured by different approaches; the two dominant methods are replacement cost and opportunity cost. In public policy evaluations it is common to assert societal perspective focusing on productivity loss when the ability of productive, working-age individuals to work is affected. This should ideally account not only for absenteeism, but also for reduced productivity (called “presenteeism”), compensation mechanisms (i.e. compensations for lost work), and multiplier effects (affected co-workers’ productivity in team-dependant production). Hunt et al. (2016) also includes opportunity costs related to leisure time due to the health impact to the same category as the loss of productivity. Welfare due to impact on leisure time is however usually reflected in elicited willingness to pay to avoid all other inconveniences associated with illness and as a consequence leisure time used to be part of the fourth component.

*Disutility* part should complement individual loss of welfare from pain and suffering. Resource costs and loss of productivity does not capture the total welfare loss because these largely miss inconveniences, suffering and pain associated with illness (apart from some minor overlaps such as pain killers). As such they can only provide a lower bound of the overall change in utility measured by willingness-to-pay or willingness-to-accept to avoid the illness or reduce risk of contracting/

developing illness. Alberini and Krupnick (2000) have estimated that the total magnitude of willingness to pay is 1.6–2.3 times larger than the sum of treatment costs and loss of productivity.

*Welfare loss due to premature death* is standard measure to derive the economic costs of mortality related impacts. As a matter of fact, any men will die sooner or later, and no life can be saved. However, regulation may affect survival rate over certain time and across certain population. The economic approach to valuing premature mortality risk reduction does not trace whose life will be saved but rather values small change in mortality risk across a wider population.

The effect on probability of dying can be translated into two virtual statistical measures called life-years-loss or statistical lives lost. Basically, taking into consideration life expectancies of each person or each subgroup of a population, all cases of premature mortality across all age groups can be recalculated into total years “lost” or total lives “saved” in a given population. For example, if a policy reduces the risk of dying by 1 in 10,000 in a population of 10,000 people, this policy will generate one statistical life in that population.

Economists then make an effort to derive a monetary value for small changes in mortality risk. If the willingness to pay for certain risk reduction is divided by the magnitude of the risk valued, a value of a statistical life (VSL) is derived. The VSL—also denoted as Value of Prevented Fatality, VPF—is defined as individual’s marginal rate of substitution between this risk and wealth. For small changes in the probability of survival WTP is approximately a product of change in probability and individual’s marginal rate of substitution between wealth and the probability of survival. As explained by Ščasný and Alberini (2012), the VSL can equivalently be described as the total willingness to pay by a group of  $N$  people experiencing a uniform reduction of  $1/N$  in their risk of dying.<sup>3</sup>

The concept of VSL is generally deemed as the appropriate and theoretically sound construct for *ex ante* policy analyses, when the identities of the people whose mortality rates are affected by the policy are not known yet. For valuation of change in mortality risks there is also another approach based on life expectancy change<sup>4</sup> using Value Of Life Year (VOLY). While the VSL approach usually applies single value per each fatality, the VOLY approach makes distinction in the differing length of remaining life expectancy. The (mean) life expectancy change (or rather mean number of life years lost) may differ substantially by different health risks and across different age sub-populations. It is argued that for some health outcomes the mortality is only brought forward by several months (e.g. deaths among elderly with chronically impaired health during heat waves) and attributing

---

<sup>3</sup>To illustrate, consider a group of 10,000 individuals, and assume that each of them is willing to pay €200 to reduce his, or her, own risk of dying by 1 in 10,000. The VSL implied by this WTP is €200/0.0001, or €2 million.

<sup>4</sup>Life expectancy is defined as the average number of years remaining for an individual or a group of people at a given age, assuming age-specific mortality rates. Statistically speaking, life expectancy is an integral of survival function between the actual age and infinite. Naturally, the survival rate equals to 1 minus the probability of dying.

VSL in this case may be perceived as incomparable impact to say traffic fatality involving young adult.<sup>5</sup> Using VOLY in health impact assessment can hence offer recommendations in conflict with those obtained by using VSL. As pointed out by Alberini et al. (2015), as long as the VOLY and VSL are constant with respect to age, the policy that affect the chance of surviving of younger adults would be concluded to offer greater benefits than the policy with completely effect on older adults if the VOLY is used. By contrast, the two policies would provide the same benefits if VSL is used. Regardless these contrasting outcomes, the monetary equivalent of both VSL and VOLY should be based on the willingness to pay for a small reduction in the risk of dying (Hammit 2007).

### 7.2.4 Valuation Methods

The first three cost components of the health impacts may be (at least in part) monetized using market prices or available proxy values (especially relevant in public healthcare) and are commonly summed to what is being denoted as cost-of-illness (COI). This allows—though with some limitations—for attributing market prices to goods and services consumed in course of treatment (1), prevention expenditures (2), or loss of earnings (or productivity) during illness (3).

Monetary valuation of the dis-welfare due to pain, suffer, and other inconveniences associated with illness (4) and risk of premature death (5) can, in the absence of market, be obtained using the following two options:

- conducting a new, original study using non-market valuation techniques, or
- transferring estimates from existing valuation studies (using benefit transfer techniques).

Non-market valuation can infer preferences either revealed in real market transactions (revealed preferences) or stated by survey respondents to a hypothetical question mimicking decision situation in market (stated preferences). The two elementary approaches of revealed preference are hedonic price and household production approach. Hedonic price approach aims at infer how a price of a market good changes when attributes of closely related non-market good changes, for example how a real estate price changes in response to land contamination (or the opposite, land clean-up). If the change in health risks from such contamination is known, this allows us to estimate (implicit) discount for such a risk increase. Hedonic price approach has been traditionally used in the U.S. for valuation of occupational mortality risks because (in theory) the wages offered by an employer and accepted by her employee in labour market should *inter alia* reflect the health risks of a particular occupation. In choosing better remunerated but slightly riskier job one makes an implicit trade-

---

<sup>5</sup>Note that both metrics are used frequently in Europe, while in the USA, Scientific Advisory Board of U.S. EPA argues against using different values for mortality risk reductions for differently-aged adults and recommends to continue using assumption of an age-independent willingness to pay for mortality risk reductions (cf. EPA 2010).

off between money (wage) and health (safety). By observing many such choices at the market we are able to estimate the marginal value of such a risk in working population. Such calculation is particularly useful when dealing with valuation of premature mortality as we will discuss in the next subsection.

The household production approach follows the logic of economic model of household production briefly outlined in introduction section and assumes that individual (or her household) derives utility not from goods per se but from their influence on health. One of the revealed preference methods—avertive behaviour—thus measures expenditures on changing risk to health. Adhering to the example of land contamination used before, here this may represent the costs of water purification.

There are various limitations related to use of particular non-market valuation technique. The revealed preference approach—which is often preferred as it is based on observation of existing markets—is not always able to address a good that matches a health outcome defined in exposure-response functions (i.e. it may be difficult to isolate value of health from other co-benefits of averting activity).<sup>6</sup>

Contingent valuation (CVM) or discrete choice experiments (DCE) belong to stated preference methods frequently used to derive willingness-to-pay (or willingness-to-accept a compensation). The key difference between CVM and DCE is in how WTP/WTA is elicited. In contingent valuation willingness to pay (or willingness to accept) is inferred more or less directly, without choosing among several alternatives, by asking e.g. “*Would you consider paying X euros to decrease your risk of lung cancer from 2 in 1,000 to 1 in 1,000?*”. In contrast, the discrete choice experiments usually present several alternatives—when one may be current state—that are each described by several characteristics, varying their levels across the alternatives (Carson and Louviere 2011).

These methods are however sensitive to various biases originating from the hypothetical format such as strategic behaviour of respondents (incl. incentive to overstate values when real payment is not expected), credibility of the valuation scenario, free-rider problem, sensitivity to scope of good values, or general distrust in payment vehicle used in the valuation scenario, such as distrust of public authorities’ use of public funds in case when contingent product is a public good provided by public authority.

The benefit transfer method uses the values estimated earlier for actual policy need. This may entail change in context (e.g. using valuation of health effects from air pollution exposure to similar health effects from exposure to chemicals), transfer from other country or transfer of values in time. The logic behind is based on strong assumption that original context and actual policy need are perfectly substitutable.

---

<sup>6</sup>The revealed preference approach (but stated preference approach alike) tends to assume *homo economicus* conditions—that individuals make fully informed decisions in pursuing their market transactions. The reality tends to be more vivid and valuations based on revealed preferences may be confounded by various market imperfections/asymmetries and/or restricted to segments of population, not to mention difficulties in inferring individual perceptions of cost and effectiveness of averting activity or time horizon one may assume in her spending (or job) decision.



One option to account for possible differences is to transfer not only unit values (typically mean WTP) but entire benefit function (i.e. a function with coefficients of influential factors that can be supplemented with relevant numbers from actual policy background).

A careful attention should be paid to implications of benefit transfer over time and countries. This particularly concerns quality of original study, use of discount rate(s) to express present value of the future costs, equity issues when transferring values across countries and time (e.g. decision on use of market exchange rates or conversion by purchasing parity power), distributional impacts (e.g. income weighting), and how to account for intra-generational equity.

### 7.3 Review of Empirical Evidence on Monetary Valuation of Health Impacts

#### 7.3.1 Mortality

The VSL estimates differ in number of characteristics, namely (1) whether the risk reduction is from public or private effort, (2) whether the risk relates to acute or chronic mortality, (3) whether a latency of risk is presented, and (4) whether a specific cause of death is presented (i.e. death from cancer vs. in an accident). We will discuss this later on in conjunction with cancer risk valuation.

There is a wealth of empirical evidence on VSL, recently summarized in OECD meta-analysis of mortality valuation studies in environmental, health and transport policies providing more than 900 VSL estimates (OECD 2012). The OECD meta-analysis recommends average adult VSL for OECD countries in a range of \$1.5–\$4.5 million, with a base value \$3 million; for EU-27, the corresponding range is \$1.8 to \$5.4 million, with a base value \$3.6 million (in 2005 USD). General recommendations for adjusting VSL base values are:

- no adjustment for income within country, for transfers between countries adjustment with GDP per capita and income elasticity of VSL of 0.8 (based on OECD 2012; and further applied, for instance, in OECD 2014; or WHO Regional Office for Europe and OECD 2015),
- no adjustment of WTP for adults vs. own child due to limited evidence and unresolved issues, although earlier recommendation suggested a factor of 1.5–2 to be used for regulation targeted on reducing children’s risk;
- no adjustment for health status and background risk due to limited evidence;
- no adjustment for cancer or dread as the OECD meta-analysis shows no clear evidence in favour of a cancer premium<sup>7</sup>;

---

<sup>7</sup>In fact, a cancer premium was found in meta-analysis of full unscreened dataset, but not in quality-screened models, cf. OECD (2012, p. 132).

- no adjustment of VSL for public vs. private good (and altruistic) context due to limited evidence and unresolved issues;
- morbidity preceding death—addictiveness (i.e. separate treatment) of morbidity costs is suggested.

In Europe, the central estimate of VSL used in the Clean Air for Europe cost-benefit analysis (Holland 2014) is €1.09 million (median) and €2.22 million (mean, 2008 euros). The central values of VSL used by U.S. Environmental Protection Agency and by U.S. Department of Transportation are \$9.4 million and \$9.2 million, respectively (recalculated in 2013 dollars by Robinson and Hammitt 2015). Both of these values are based on qualitative reviews, although the former one is based on a literature review published in the 1990s, the later one is based on a review conducted during 2012 and 2013.

Much less studies have focused on eliciting Value Of Life Year (VOLY). Originally for use in ExternE methodology, Rabl (2003) developed a procedure to convert VSL to VOLY, but later VOLY was elicited directly in a CVM survey conducted in nine European countries (Desaigues et al. 2011), providing the values of VOLY at €57,700 (mean) and €138,700 (median).<sup>8</sup> The VOLY values based on Desaigues et al. study have been widely used in cost-benefit analyses of many EU-wide policies, such as CBA on Clean Air for Europe, The VOLY value has been also used to derive the economic costs of the quality-of-life based measures, such as QALY's or DALY's.

Valuation of cancer risk attracts considerable attention judging by more than 50 studies identified in recent review by authors (Ščasný et al. 2014b; Alberini and Ščasný 2013; Alberini and Ščasný 2014). Majority of these studies used stated preference approaches to address issues affecting WTP such as dread related to cancer, effect of latency, altruism, type of cancer or how the reduction in cancer risk is delivered.

The cancer premium (or sometimes cancer differential) is posited as capturing elements of dread and fear of cancer, as well as pain and suffering from the period of illness preceding death. In CBA praxis such adjustment for cancers to default mortality risk reduction value (VSL) is sometimes applied, but studies are not consistent in these findings and recommendations from OECD meta-analysis argue against adjustment of VSL base values and also U.S. EPA's Scientific Advisory Board warned against a unique cancer premium value.

There is rather mixed evidence with respect to WTP for risk reduction for child and adult, and for self and other member of their household. For example an US study found that parents are willing to pay 2.5 times more to reduce skin cancer risks to their children than to themselves (Dickie and Gerking 2002), and another study found that parents were willing to pay about 30 % more to reduce risk of can-

---

<sup>8</sup>Alberini et al. (2015) review of VOLY studies identifies a couple of earlier studies that examined willingness to pay for an extension in expected remaining lifetime usually of about couple of months that would be experienced at older ages, see, for instance, Johannesson and Johannsson (1996), Morris and Hammitt (2001), or Chilton et al. (2004). In most cases, criticism has been raised on the basis of their elicitation method and/or small sample sizes.

cer to their child in the Czech Republic (Alberini and Ščasný 2011), but the latter study also found no statistical difference between WTP for adult and WTP for her child in Italy. More recently, Alberini and Ščasný (2016) conducted similar survey in Canada and got similar results for Canadians as they found earlier for the Czech parents—VSL for adults is estimated at \$6.7 million, while VSL for children is \$8.8 million (in Canadian 2015 dollars). In terms of cause of death, the child premium is the largest for respiratory diseases, 56%, and the smallest for cancer (24%).

A prolonged time between initial exposure and increased cancer incidence has intrigued research into possible decline of WTP with latency, i.e. actual WTP discounts future health impairment. Two recent studies find near-zero discount rate over latency period (Hammitt and Haninger 2010; Alberini and Ščasný 2011), but several other studies suggest the opposite and the implicit discount rate ranges in most cases between 5 and 15% (Van Houtven et al. 2008; Cameron and DeShazo 2013; Tsuge et al. 2005; Alberini and Ščasný 2016).

### 7.3.2 *Morbidity*

Recently Hunt (2011) in his overview summarized epidemiological evidence linking heavy metals to health impacts and health impacts to monetary values. Most of reviewed metals (arsenic, cadmium, hexavalent chromium, lead, mercury and nickel) affect health in both ingestion and inhalation pathways leading to wide range of health outcomes. Hunt's review addresses chronic and severe endpoints like cancers, still birth and adverse pregnancy outcomes, IQ loss, anaemia and renal dysfunction. Human exposures though vary from mere incidental one-time to life-long and similarly induced health effects are not restricted to severe impairments but milder and/or sub-clinical health effects (e.g. irritation or sensitization) are likely to be much more frequent in the population. Multitude of pathways, metal-specific persistence in the environment, exposure environs, latency of development of effects from exposure (or the opposite time-lag between decrease in emissions and decrement in health risk, called cessation-lag), all pose significant challenges and add to uncertainty in damage cost calculations (Bachmann 2006). Earlier study by Serup-Hansen et al. (2004) for Danish Ministry of Environment elaborated total cost estimates for health impacts related to chemicals including contact allergy and lung cancer. A recent study for European Chemicals Agency (Ščasný et al. 2014a, b) estimated willingness to pay to avoid health outcomes related to chemicals, some of them highly relevant to heavy metal exposures (skin sensitization, kidney injury, neurodevelopmental disorders, very low birth weight or 'generic' cancer case). Based on comprehensive surveys in three EU countries, EU-wide values were derived for future use in socio-economic analyses and/or health impact assessments.

Beyond those studies estimating WTP for avoiding particular health outcomes, there are a few studies that employ entire (or part of) impact pathway approach to estimate damage costs of exposures to heavy metals. Spadaro and Rabl (2004)

developed a simplified model for quantifying health (i.e. physical) impacts of toxic emissions of arsenic, cadmium, chromium, mercury, nickel and lead. Several of ExternE projects (NewExt, GREENSENSE, OMNITOX, ESPREME, DROPS) elaborated impact pathway methodology further and provided estimates of impacts in DALYs as well as monetary values (Bachmann 2006; Friedrich 2007; Rabl et al. 2014). Spadaro and Rabl (2008) estimated global average neurotoxic impacts and costs due to mercury emissions and found that average of the marginal damage costs is about \$1,500/kg (with dose threshold) or \$3,400/kg (without threshold). On a country level neurotoxic impacts of airborne lead emissions from waste-to-energy plant were estimated by Pizzol et al. (2010) in a range of 41–83 €/kg based on impact-pathway modelling of IQ-loss in children. IQ losses from lead and mercury airborne emissions including monetary valuation are incorporated in a Danish integrated modelling system EVA (Brandt et al. 2013). External costs of emissions of cadmium contained in phosphorus fertilizers to soil were explored by Pizzol et al. (2014). For a cumulative long-term cadmium exposure via dietary intake the external costs were estimated at 334 €/kg (using 3% discount rate).

In the very recent study commissioned by OECD, a core set of pollutant-health (morbidity) combinations was recommended for outdoor air pollution (Hunt et al. 2016). This set meets the criteria of being unbiased, credible and implementable, and can be applied in health impact assessment in OECD countries, China and India. This review identified five pollutant-health pairs that specifically include (a) respiratory and cardiovascular hospital admissions linked to PM and to ozone, (b) restricted activity days and associated work loss days in relation to PM and/or ozone, (c) chronic bronchitis in adults in relation to PM, (d) acute bronchitis in children aged 6–18 years resulting from PM<sub>10</sub>, and (e) acute lower respiratory illness in children aged <5 years resulting from PM<sub>10</sub>.

## 7.4 Non-Monetary Measures of Health Benefits

### 7.4.1 Health-Related Quality of Life Indices

A vivid stream of research and practice has evolved on non-monetary measures generalizing impact of health impairment on one's life. These are based on subjective assessment of actual health status of a person affected by certain illness/disability as compared to person in a perfect state. Such measures sum duration of health state adjusted by quality or disability (QALY, DALY).<sup>9</sup> Unlike for WTP, for QALY (or

---

<sup>9</sup>QALY stands for quality-adjusted life years, while DALY stands for disability adjusted life years; there are also other indices such as healthy-years equivalents (HYE), but not as frequently used as QALYs and DALYs. The key difference between QALY and DALY is that their indices of life quality (for QALY) and disability (for DALY) are reversed to one another but in both cases normalized to a scale between 0 and 1. Disregarding other minor differences, a QALY of 0.7 is roughly equal to DALY of 0.3.

DALY) it is sufficient to estimate change in quality of life from health impairment and duration of a health state under consideration, essentially without much concern about great deal of determinants that may affect the WTP (cf. Hammitt 2002; Hofstetter and Hammitt 2002). There are several ways to derive utility weights for QALY measure, either directly using visual analogue scales, time trade-offs or standard gambles, or indirectly by means of standardized questionnaires (such as EuroQoL or SF-36). Disability weights, in contrast, were originally defined by experts (cf. Gold et al. 2002) but recently abandoned for a set of weights elicited from paired comparisons by a large sample of general public (Salomon et al. 2012).

The relative ease of estimation of these measures has stimulated numerous attempts to find a simple transformation of QALY (or DALY) to WTP, what would allow us to evaluate a broad range of health outcomes affected by public policies, simply because a wealth of QALY and DALY weights exists for much broader scope<sup>10</sup> of health outcomes and these could be transferred easily without much care for context and other determinants. Unfortunately, these simplifications (such as disregarding non-health determinants of WTP) also imposes substantial restrictions on individual preferences and many of these restrictions are not particularly realistic (cf. Hammitt and Haninger 2010) meaning that a simple transformation of QALYs (or DALYs) to WTP is generally not feasible (Robinson et al. 2013; Gyrd-Hansen and Kjær 2012).

#### ***7.4.2 Non-Monetary Benefits of Metals' Exposure Reduction***

The appeal of quantification of impacts from exposure to heavy metals in QALYs or DALYs lies in simple unification of morbidity and mortality and simplified methods linking DALYs/QALYs to adverse effect indicators, e.g. ED<sub>10</sub> (exposure dose associated with 10% risk). Crettaz et al. (2002) and Pennington et al. (2002) elaborated such a valuation of human health impacts in terms of DALYs for cancer and non-cancer effects in Life Cycle Impact Assessment (LCIA) methodology, respectively. These approaches were elaborated further in ExternE impact-pathway methodology (Bachmann 2006; Friedrich 2007; Rabl et al. 2014), but in the latter served primarily as a means to arrive at monetary values assuming equality between VOLY and WTP per DALY. Even without attempting to convert QALYs or DALYs to monetary terms, LCIA approach is far from settled. Pizzol et al. (2011) compared different LCIA methodologies to find a poor agreement between methods both in terms of relative contribution of each metal and of the metals in total to the total impact on human health. According to the authors these differences stem from different techniques used to calculate the characterization factors and also due to number of metals included in each method.

---

<sup>10</sup>Cost-effectiveness Analysis Registry at <https://research.tufts-nemc.org/cear4/> provides a large collection of (QALY) utility weights. Salomon et al. (2012) provide 220 disability weights, originally devised for WHO's Global Burden of Disease Study 2010.

## 7.5 Conclusions

The health impacts are usually measured in terms of premature mortality or new cases of certain illness. In health economics, these impacts are translated in the health-related quality of life indices, such as QALY's and DALY's, that both allow aggregation across all the adverse health effects in order to express them through one health impact indicator. This approach allows performing the cost-effectiveness analysis of various policy designs or regulatory programs. On the other hand, using the health-related quality of life indices does not allow performing the cost-benefit analysis, since involved economic costs and health impacts are expressed in quite different units, i.e. money and life years. Despite some attempts to attain monetary value of VOLY on the health-related quality of life indices, this economic assessment is not generally justified, as it can be applied only under very restrictive (not realistic) assumptions.

In contrast, deriving the willingness to pay for avoiding disutility associated with premature mortality or illness, the resource costs, and the opportunity costs for each health outcome is theoretically sound and appropriate approach to perform the cost-benefit analysis. This approach is time and resource intensive, as each cost component has to be derived for each health outcome under consideration. In fact, empirical literature on health benefit valuation is not huge, but on the other hand this literature has been growing fast for last decades and it can serve a bulk of economic values for continuously growing number of health outcomes.

Quantifying the economic benefits of health impacts due to policies require to examine the full chain of impact pathways. Damage function approach embedded in ExetnE's IPA is just such method that can serve performing both the cost-effectiveness analysis relying on the health-related quality of life indices as well as the cost-benefit analysis using the willingness to pay approach.

## References

- Abt Associates Inc. (2007) Cost of illness handbook. U.S. Environmental Protection Agency, Washington, DC, <http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=901A0E00.TXT>
- Alberini A, Krupnick A (2000) Cost-of-illness and willingness-to-pay estimates of the benefits of improved air quality: evidence from Taiwan. *Land Econ* 76:37–53. doi:10.2307/3147256
- Alberini A, Ščasný M (2011) Context and the VSL: evidence from a stated preference study in Italy and the Czech Republic. *Environ Resour Econ* 49(4):511–538
- Alberini A, Ščasný M (2013) Does the cause of death matter? The effect of dread, controllability, exposure and latency on the VSL. *Ecol Econ* 94:143–155
- Alberini A, Ščasný M (2014) Stated-preference study to examine the economic value of benefits of avoiding selected adverse human health outcomes due to exposure to chemicals in the European Union (ECHA/2011/123)—Part III: Carcinogens. Study prepared for the European Chemicals Agency by the Charles University Prague (Environment Center) and VU University Amsterdam (Faculty of Earth and Life Sciences), Sept 2014
- Alberini A, Ščasný M (2016) One or Many VSLs? Evidence from a Survey of the Public in Canada. Report on the VERHI-in-Canada Project submitted to Health Canada

- Alberini A, Bigano A, Post J (2015) Air pollution and human health: approaches and issues when valuing the cost of inaction. ENV/EPOC(2015)10. OECD, Paris
- Bachmann TM (2006) Hazardous substances and human health: exposure, impact and external cost assessment at the European scale. Elsevier, Amsterdam
- Brandt J et al (2013) Contribution from the ten major emission sectors in Europe and Denmark to the health-cost externalities of air pollution using the EVA model system—an integrated modelling approach. *Atmos Chem Phys* 13(15):7725–7746
- Cameron TA, DeShazo JR (2013) Demand for health risk reductions. *J Environ Econ Manag* 65(1):87–109. doi:10.1016/j.jeem.2012.05.008
- Carson RT, Louviere JJ (2011) A common nomenclature for stated preference elicitation approaches. *Environ Resour Econ* 49(4):539–559
- Chestnut LG et al (2006) The economic value of preventing respiratory and cardiovascular hospitalizations. *Contemp Econ Policy* 24:127–143
- Chilton S, Covey J, Jones-Lee M, Loomes G, Metcalf H (2004) Valuation of health benefits associated with reductions in air pollution: final report. DEFRA, London
- Crettaz P et al (2002) Assessing human health response in life cycle assessment using ED10s and DALYs: part 1—Cancer effects. *Risk Anal* 22(5):931–946
- Desaigues B et al (2011) Economic valuation of air pollution mortality: a 9-country contingent valuation survey of value of a life year (VOLY). *Ecol Indic* 11(3):902–910
- Dickie M, Gerking S (2002) Parents' valuation of latent health risks to their children. Working Paper 06-2002, Department of Economics, University of Central Florida. <https://itd.bus.ucf.edu/cdn/economics/workingpapers/2002-06.pdf>
- EC (2000) Externalities of energy: 2000 update. European Commission, DG Research
- EPA (2010) Valuing mortality risk reductions for environmental policy: a white paper. U.S. Environmental Protection Agency, Washington, DC, [http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0563-1.pdf/\\$file/EE-0563-1.pdf](http://yosemite.epa.gov/ee/epa/eerm.nsf/vwAN/EE-0563-1.pdf/$file/EE-0563-1.pdf)
- Friedrich R (2007) ESPREME—estimation of willingness-to-pay to reduce risks of exposure to heavy metals and cost-benefits analysis for reducing heavy metals occurrence in Europe. Publishable Final Activity Report, IER University of Stuttgart. [http://espreme.ier.uni-stuttgart.de/download/Final\\_Activity\\_Report.pdf](http://espreme.ier.uni-stuttgart.de/download/Final_Activity_Report.pdf)
- Gold MR, Stevenson D, Fryback DG (2002) HALYS and QALYS and DALYS, Oh My: similarities and differences in summary measures of population Health. *Annu Rev Public Health* 23:115–134
- Groosman B, Muller NZ, O'Neill-Toy E (2011) The ancillary benefits from climate policy in the United States. *Environ Resour Econ* 50(4):585–603
- Gyrd-Hansen D, Kjær T (2012) Disentangling WTP per QALY data: different analytical approaches, different answers. *Health Econ* 21:222–237
- Hammit JK (2002) QALYs versus WTP. *Risk Anal* 22(5):985–1001
- Hammit JK (2007) Valuing changes in mortality risk: lives saved versus life years saved. *Rev Environ Econ Policy* 1(2):228–240
- Hammit JK, Haninger K (2010) Valuing fatal risks to children and adults: effects of disease, latency, and risk aversion. *J Risk Uncertain* 40(1):57–83
- Hofstetter P, Hammit JK (2002) Selecting human health metrics for environmental decision-support tools. *Risk Anal* 22(5):965–983
- Holland M (2014) Cost-benefit analysis of final policy scenarios for the EU Clean Air Package. Version 2 Corresponding to IIASA TSAP Report #11, Version 1. EMRC, Laxenburg
- Horowitz JK, McConnell KE (2002) A review of WTA/WTP studies. *J Environ Econ Manag* 44(3):426–447
- Hunt A (2011) Policy interventions to address health impacts associated with air pollution, unsafe water supply and sanitation, and hazardous chemicals. OECD Environment Working Papers No. 35, OECD, Paris
- Hunt A, Ferguson J, Hurley F, Searl A (2016) Social costs of morbidity impacts of air pollution. OECD Environment Working Papers, No. 99, OECD, Paris

- Johannesson M, Johannsson P (1996) To be, or not to be, that is the question: an empirical study of the WTP for an increased life expectancy at an advanced age. *J Risk Uncertain* 13:163–174
- Kahneman D, Knetsch JL, Thaler R (1990) Experimental tests of the endowment effect and the Coase theorem. *J Polit Econ* 98(6):1325–1348
- Krol M, Brouwer W, Rutten F (2013) Productivity costs in economic evaluations: past, present, future. *Pharmacoeconomics* 31(7):537–549. doi:10.1007/s40273-013-0056-3
- Máca V, Melichar J, Ščasný M (2012) Internalization of external costs of energy generation in central and eastern European countries. *J Environ Dev* 21(2):181–197. doi:10.1177/1070496512442504
- Morris J, Hammitt JK (2001) Using life expectancy to communicate benefits of health care programs in contingent valuation studies. *Med Decis Mak* 21(6):468–478
- Muller NZ, Mendelsohn RO (2007) Measuring the damages of air pollution in the United States. *J Environ Econ Manag* 54(1):1–14
- Muller NZ, Mendelsohn RO, Nordhaus WD (2011) Environmental accounting for pollution in the United States economy. *Am Econ Rev* 101(5):1649–1675
- OECD (2012) Mortality risk valuation in environment, health and transport policies. OECD, Paris
- OECD (2014) The cost of air pollution: health impacts of road transport. OECD, Paris. doi:10.1787/9789264210448-5-en
- Pennington D et al (2002) Assessing human health response in life cycle assessment using ED10s and DALYs: part 2—Noncancer effects. *Risk Anal* 22(5):947–963
- Pizzol M et al (2010) External costs of atmospheric Pb emissions: valuation of neurotoxic impacts due to inhalation. *Environ Health* 9:9
- Pizzol M et al (2011) Impacts of “metals” on human health: a comparison between nine different methodologies for Life Cycle Impact Assessment (LCIA). *J Clean Prod* 19(6–7):646–656
- Pizzol M, Smart JCR, Thomsen M (2014) External costs of cadmium emissions to soil: a drawback of phosphorus fertilizers. *J Clean Prod* 84:475–483
- Plott CR et al (2005) The willingness to pay-willingness to accept gap, the “endowment effect”, subject misconceptions, and experimental procedures for eliciting valuations. *Am Econ Rev* 95(3):530–545
- Preiss P, Klotz VE (2008) V1. 3: user’s manual and “description of updated and extended draft tools for the detailed site-dependent assessment of external costs.” Stuttgart: Institute of Energy Economics and the Rational Use of Energy, University of Stuttgart, p 63
- Rabl A (2003) Interpretation of air pollution mortality: number of deaths or years of life lost? *J Air Waste Manag Assoc* 53(1):41–50
- Rabl A, Spadaro JV, Holland M (2014) How Much is Clean Air Worth?: Calculating the Benefits of Pollution Control. Cambridge University Press, Cambridge, pp 1–671
- Robinson LA, Hammitt JK (2015) Research synthesis and the value per statistical life. *Risk Anal* 35(6):1086–1100
- Robinson A et al (2013) Estimating a WTP-based value of a QALY: the “chained” approach. *Soc Sci Med* 92:92–104
- Salomon JA et al (2012) Common values in assessing health outcomes from disease and injury: disability weights measurement study for the Global Burden of Disease Study 2010. *Lancet* 380(9859):2129–2143
- Ščasný M, Alberini A (2012) Valuation of mortality risks attributable to climate change: investigating the effect of survey administration modes on a VSL. *Int J Environ Res Public Health* 9(12):4760–4781
- Ščasný M et al (2014a) Stated-preference study to examine the economic value of benefits of avoiding selected adverse human health outcomes due to exposure to chemicals in the EU. Service contract to ECHA, Charles University Environment Center, Prague. <http://echa.europa.eu/support/socio-economic-analysis-in-reach/willingness-to-pay-to-avoid-certain-health-impacts>
- Ščasný M, Máca V, Zvěřinová I (2014b) Valuation of REACH-relevant health outcomes: a literature review. Service contract for the European Chemicals Agency No. ECHA/2011/123, Dec 2012



- Ščasný M, Massetti E, Melichar J, Carrara S (2015) Quantifying the ancillary benefits of the Representative Concentration Pathways on air quality in Europe. *Environ Resour Econ* 62(2):383–415
- Serup-Hansen N, Gudum A, Munk Sorensen M (2004) Valuation of chemical related health impacts. Miljøministeriet, Copenhagen, 929(929). <http://www2.mst.dk/udgiv/publications/2004/87-7614-295-7/pdf/87-7614-296-5.pdf>
- Spadaro JV, Rabl A (2004) Pathway analysis for population-total health impacts of toxic metal emissions. *Risk Anal* 24(5):1121–1141
- Spadaro JV, Rabl A (2008) Global health impacts and costs due to mercury emissions. *Risk Anal* 28(3):603–613
- Tsuge T, Kishimoto A, Takeuchi K (2005) A choice experiment approach to the valuation of mortality. *J Risk Uncertain* 31(1):73–95
- Van Houtven G, Sullivan MB, Dockins C (2008) Cancer premiums and latency effects: a risk tradeoff approach for valuing reductions in fatal cancer risks. *J Risk Uncertain* 36(2):179–199. doi:10.1007/s11166-008-9032-2
- WHO Regional Office for Europe, OECD (2015) Economic cost of the health impact of air pollution in Europe: clean air, health and wealth. WHO Regional Office for Europe, Copenhagen

# Chapter 8

## Capturing the Health Effects of Environmental and Industrial Policy in a Macroeconomic Model

Hector Pollitt and Ben Gardiner

**Abstract** Assessments of industrial or environmental policy are often based on macroeconomic models which typically neglect the effects of the policies on human health. Cost Benefit Analyses can include these effects but often in a rather simplistic manner, for example neglecting lagged effects and distributional impacts. However, it has been shown that the impacts on human health of such measures can be large and can even outweigh the standard economic impacts, as they cumulate over time.

In this chapter we discuss how health effects could be integrated fully into a macroeconomic modelling framework that can also be used to predict the levels of certain pollutants through scenario analysis. Rather than converting all the health impacts of the pollution to monetary ‘externalities’, as is standard in neoclassical economics, we outline an approach that would consider the wider implications of the direct impacts (e.g. on labour productivity or healthcare costs) to be assessed within the same modelling framework. The outputs of the model thus represent an integrated analysis of physical and economic impacts in each scenario.

The chapter discusses some of the key assumptions that are required to conduct such an analysis and demonstrates how the choice of economic model can be important in determining overall outcomes.

**Keywords** Macroeconomic modelling • E3ME • Integrated assessment • Healthcare • Labour productivity

---

H. Pollitt (✉) • B. Gardiner  
Cambridge Econometrics, Covent Garden, Cambridge, CB1 2HT, UK  
e-mail: [hp@camecon.com](mailto:hp@camecon.com)

## 8.1 Introduction

### 8.1.1 Overview

This chapter explores how links can be established between environmental and/or industrial policy and their potential health and wider economic impacts, as represented in modelling tools. We discuss the shortcomings of existing approaches and how they could be improved upon, providing an example based on the E3ME macro-econometric model (<http://www.e3me.com>) that is commonly used for environmental policy analysis in Europe.

### 8.1.2 *The Role of Macroeconomic Modelling in Policy Analysis*

Governmental organisations use two standard approaches for assessing possible future policy, both of which may rely on (macroeconomic) modelling. The European Commission applies a multi-criteria analysis that is described in its *Better Regulation Guidelines* (European Commission 2015). Macroeconomic modelling plays an important role in the quantitative part of this assessment but, while the approach can produce both estimates of macroeconomic and health outcomes, these are generally from separate analyses. The alternative approach, as applied by the UK government (HM Treasury 2013), is Cost Benefit Analysis (CBA). CBA includes both economic and health impacts (amongst other factors) and expresses each one in monetary terms. Macroeconomic modelling may be used to obtain the economic impacts, usually expressed in GDP terms. The health effects are estimated as unit ‘damage costs’ which are then added to or subtracted from the economic GDP result to give a broader indicator of welfare.

In this chapter we argue that both approaches may miss important interactions between human health and wider economic welfare. For example, workers that are not fit to work may withdraw from the labour market. This will also affect the rest of the labour market; firms may be stuck with less efficient workers, or bottlenecks may allow remaining workers to charge more for their labour. While all of this will have an impact on GDP, the extent of this impact will be highly dependent on other factors. There will also be important distributional outcomes that are missing from a standard CBA (and also often from multi-criteria analysis).

In summary the treatment in existing quantitative approaches does not attempt to address the complexities and possible interaction effects between environmental policy, human health and the wider economy.

## **8.2 The Types of Health Effects That Might Be Included in a Macroeconomic Model**

### ***8.2.1 Introduction***

Before describing how health effects could be included in a macroeconomic model, we must be clear about the particular health effects that we are covering and how they are defined. Some of these are summarised below—the list is not intended to be comprehensive and could depend on the types of environmental and industrial policies that are being assessed. These are the ‘exogenous’ factors that are not modelled but which could be entered into a macroeconomic model; we do not here discuss secondary, indirect effects, which the macroeconomic model itself would be designed to determine.

### ***8.2.2 Health Care Costs***

Perhaps the most obvious inputs to a macroeconomic model are the direct treatment costs for accident or illness that are incurred, or are avoided, by additional regulation. The macroeconomic impact of these costs is likely to vary by country, for example depending on whether the health care is funded by the state through taxation, by private insurance schemes, or by the individual. Particularly for developing countries we must consider whether the individual would be able to meet the costs; if not then we would expect to see more severe impacts in the other categories.

### ***8.2.3 Mortality Rates***

Population is usually present in macroeconomic models. The exact treatment depends on the type of model (see next section) but each person in the economy can be expected to contribute to production (if of suitable working age and active in the labour force) and also to contribute to the consumption of goods and services. Entry into and exit from the working age population is largely taken from official population projections which themselves rely on fertility and mortality rates. Policies which have the potential to affect these demographic rates would need to be captured in the model through additional assumptions, as they would not be part of the baseline (population) projection.

### **8.2.4 *Labour Market Participation and Public Benefits***

If an individual suffers from a serious illness they may be unable to work and will therefore withdraw from the labour force. In countries with a social security system they will receive disability benefits. Leaving aside migration, reducing the size of the active labour force will constrain the level of output that an economy can produce so environmental policy that results in a higher labour force may result in higher overall production levels.

One point that is often neglected in this type of analysis is the role of carers. If an individual that is no longer able to work requires constant caring, this could also lead to another individual effectively withdrawing from the formal labour force.

### **8.2.5 *Labour Productivity***

Even if an individual is still able to work, their labour productivity is likely to be reduced if they are ill. This means that they will be able to produce less for a given amount of time working. They may also work fewer hours, for example to attend medical appointments. Overall, the ratio of output per worker would be expected to fall if there was a higher incidence of illness. If environmental policy reduced rates of illness it could therefore be possible to increase total economy-wide productivity rates.

## **8.3 Designing a Counterfactual Scenario**

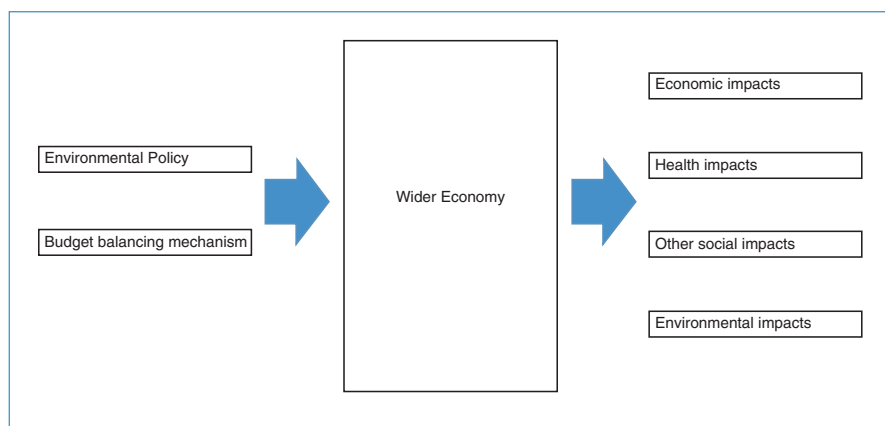
### **8.3.1 *Introduction***

Macroeconomic models typically operate through a series of scenarios. First of all a baseline case is determined, usually on the basis of business-as-usual assumptions. The aim of the baseline is not necessarily to provide a highly accurate forecast of future outcomes, but instead to be used as a comparator for the policy scenarios.

The policy scenarios are then defined as the baseline plus the addition of one or more policy inputs. The differences between the outcomes of the policy scenarios and the baseline represent the policy impacts, based on *ceteris paribus* principles.

### **8.3.2 *Designing Scenarios of Environmental Policy***

Figure 8.1 summarises the inputs to and outputs of a scenario of environmental policy. The two main inputs to such a scenario are likely to be the direct compliance cost to business and a representation of the health impacts of the policies.



**Fig. 8.1** Scenario inputs and outputs. *Source:* Authors' representation

In addition, an assumption is usually made about government fiscal neutrality. This means that overall the government balance is unchanged between the baseline case and the policy scenarios, i.e. that there is no fiscal stimulus or contraction in the policy scenario, which could bias the results of the analysis. It is sometimes necessary to adjust another tax rate to ensure fiscal neutrality is observed; note that the choice of tax to change can be important (see Barker et al. 2009, for a discussion).

### 8.3.3 *The Main Dimensions to Consider*

There are several important dimensions that must be taken into account when designing the scenarios. The main ones are:

- Geographical coverage—Many of the health impacts of a poor environment are highly localised, for example the effects of poor air or water quality will be most felt by those who are close to the source of pollution.
- Economic sectors—Environmental regulations often focus on heavy industry and the power sector, with some additional constraints on transport. It is these sectors that are most likely to bear the burden of meeting regulatory requirements.
- Socio-economic and demographic group—The age and gender of the individuals affected are important. For example, if the impacts fall on those who are retired then there will be less impact on the labour market. Socio-economic status is also important, especially with regards to ability to pay for medical treatment.
- Time period—CBA largely ignores timing effects, or applies a simple multiplicative rate to discount future costs. However, many of the effects can take many years to be felt. For example, the economic impacts of pollution that slows learning development in children will not manifest itself until those children enter the labour market, 10–20 years later.

### **8.3.4 *Ensuring the Changes Are ‘Net’***

The scenario-based approach described earlier in this section is designed to assess the ‘net’ effects of environmental policy. By this we mean that it should take into account impacts, both positive and negative, across the whole economy, comparing the policy scenario to what otherwise would happen. When we design the scenario inputs we must ensure that they are also on a net basis.

When assessing healthcare costs this issue is especially important, as treatment costs are usually highest at the end of a person’s lifespan. So while reduced incidence of illness may reduce short-term healthcare costs, the same individual may live longer and require a similar level of treatment at a later date.

When designing the scenario inputs for a macroeconomic model, careful consideration must thus be given to how the effects are measured. If only the ‘gross’ effects are considered (i.e. the short-term reduction in costs, without taking into account longer-term impacts) then this must be made clear in the analysis as an underlying assumption.

## **8.4 Different Types of Macroeconomic Models**

### **8.4.1 *Introduction***

Once the scenarios have been designed, there is a question about the type of economic model to use. Several different types of model exist, with each one offering a different interpretation both of how the economy works (see Keen 2011 for a discussion), and also a different interpretation of how health impacts would affect macroeconomic relationships. This section outlines the main types of model that may be applied. In the next section, we expand in detail as to how a scenario of environmental policy with health effects could be modelled in one particular modelling framework.

### **8.4.2 *Input-Output and Multiplier Analysis***

#### **8.4.2.1 Introduction**

Input-output analysis is a demand-driven approach that focuses on supply chains. The input-output table details the interactions between different sectors of the economy. The other modelling approaches below also incorporate input-output analysis but fit it within a broader macroeconomic framework. An economic multiplier is estimated by inverting the matrix of the input-output table; this gives an estimate of how much total output increases in response to a unit increase in demand.

The working assumption to the approach is that all industries operate below capacity and that they are able to increase output if the demand for their product increases. There are no prices in input-output analysis, or any representation of supply constraints.

#### **8.4.2.2 Coverage of the Key Dimensions**

Of the four dimensions listed in Sect. 8.3.3, input-output analysis is only able to cover economic sectors in a high level of detail. Input-output tables are typically produced at national level (although they could be produced for local areas in theory, the scale of the data collection exercise makes this unusual) and for a single base year. They have no representation of different demographic or socio-economic groups.

#### **8.4.2.3 Potential Coverage of Health Effects**

It is relatively straightforward to measure the economy-wide effects of a change in healthcare expenditure by entering a demand shock to that sector in the input-output table. This shock could be balanced by a corresponding change in other spending (either by households or government) to ensure that changes are net overall.

However, it is much more difficult to estimate the other health impacts of environmental analysis using input-output analysis alone. The approach is able to estimate changes in consumption patterns (e.g. to different mortality rates) but, due to its lack of representation of the production side, supply shocks are much more difficult to incorporate.

Most difficult of all is attempting to assess a change in labour productivity. Standard input-output analysis does not include labour at all and even a version that is extended to include labour has severe limitations. For example, as labour is entirely determined by the current level of production, which is largely fixed, an increase in the level of production per worker would result in a lower required number of workers.

#### **8.4.2.4 Summary**

In summary, input-output analysis is in most cases not a suitable tool for carrying out an economic assessment of the health effects of environmental policy. We therefore turn attention to more sophisticated modelling approaches.



### 8.4.3 *CGE Models*

#### 8.4.3.1 Introduction

Computable General Equilibrium (CGE) models have become the standard tool for long-run macroeconomic analysis since the 1970s. They are derived from neoclassical microeconomic theory and ensure consistency ('closure') at the macro level between different economic and financial balances. Commonly used CGE models include GTAP (Hertel 1999), GEM-E3 (Capros et al. 2013) and the Monash model (Dixon and Rimmer 2002).

The basic assumption of CGE models is one of optimisation. It is assumed that individuals and firms act 'rationally' in their own self-interest to optimise their own interests, and that markets optimise by allowing prices to adjust to balance supply and demand. The supply-driven nature of the model follows from this assumption—prices adjust so that all available resources are used productively and the economy as a whole optimises its level of economic output. The only way to increase output levels is to increase the resources available with which to produce, as adding any constraint (i.e. regulation) on the optimisation process can only lead to lower output overall.

The parameters in CGE models are usually either drawn from econometric studies or 'calibrated' using a single base year of data. The GTAP database (<https://www.gtap.agecon.purdue.edu/databases/v8/>) provides a standard data set for global CGE models.

#### 8.4.3.2 Coverage of the Key Dimensions

CGE models usually operate at national or international level. Regional models exist (e.g. state level in the US) but there are very few models for local areas. As CGE models incorporate input-output tables, they have the same coverage of sectors, but face the same restrictions for being used at local level.

Some CGE models are disaggregated to provide information split by socio-economic group. The constraint on this aspect of the analysis is the data rather than the theoretical modelling framework.

CGE models are also used to assess future scenarios, so can at least in part address the time dimension of health impacts. However, the dynamic properties of CGE models are limited so the accumulation of health effects over time does not fit naturally into the modelling framework.

#### 8.4.3.3 Potential Coverage of Health Effects

The supply-oriented structure of CGE models makes them quite suitable for some aspects of incorporating health effects. For example, in the basic 'Cobb-Douglas' labour-capital production function with constant returns to scale, an increase in labour productivity (assumed to take place through an improvement to the average

health of the labour force) can be adjusted relatively easily by increasing L in the following equation, implying that there are more labour resources available:

$$\text{Output} = A * L^\beta * K^{(1-\beta)}$$

where A is Total Factor Productivity, L is the amount of labour used, K is the amount of capital used and the betas represent the marginal productivity of additional capital and labour.

This does not lead to a one-to-one increase in output, as the value of beta is less than one, but it is fairly trivial to calculate the change in output for any given level of beta. Within the model structure prices will adjust so that demand and supply are fully consistent and all available resources are used. So while although there is an easy representation of labour productivity in the model structure, the wider assumption that all resources are used and higher productivity automatically leads to higher demand and production levels is more questionable.

It is also possible to estimate the effects of shifts in expenditure in and out of the healthcare sector by adjusting the utility levels of the different sectors, inducing the shifts in expenditure. The input output linkages in the model will allow for the analysis of supply chain impacts in the same way as input-output analysis described above.

#### 8.4.3.4 Summary

In summary, CGE models could be used to provide a macroeconomic representation of health effects from environmental policy but there are some limitations in the modelling approach that must be recognised. There are also assumptions that must be taken into account when interpreting model results. It is important to be aware of the type of question that CGE models are meant to answer:

*Given these constraints, what is the maximum level of economic production?*

This is not the same as the type of question that scenario analysis often is used for:

*If this policy is implemented, what will be the impact on economic production levels?*

With these findings in mind, we now turn attention to a more simulation-based approach.

### 8.4.4 Macro-Econometric Models

#### 8.4.4.1 Introduction

Macro-econometric models fall roughly between static input-output analysis and CGE modelling. They have an input-output structure at their core and extend this to cover other economic indicators (e.g. household consumption, international trade) and prices. Macro-econometric models are in general simulation, rather than optimisation, tools that estimate the outcomes of shocks to the economic system.

Prices in macro-econometric models do not adjust fully. While it is assumed that supply will adjust to meet demand, as in input-output analysis, supply constraints and changes in prices are important features of the model. However, the working assumption is that there is usually spare capacity in the economy which, if drawn upon, could lead to higher rates of economic production and employment.

The parameters in macro-econometric models are usually derived through econometric equations, as the model name suggests (although they could theoretically be derived in other ways). This provides a strong empirical basis for the analysis but does require a much larger data set and means that the models can be subject to the Lucas Critique (Lucas 1976) of using past performance to predict behaviour under different circumstances. However, it should be noted that the parameters in CGE models are subject to the same criticism.

The E3ME model, which we describe in the next section, is an example of a macro-econometric model. Another example is the GINFORS model (Lutz et al. 2010).

#### **8.4.4.2 Coverage of the Key Dimensions**

Like CGE models, macro-econometric models usually operate at national level. There are some examples of regional models (e.g. the UK MDM-E3 model developed from Barker and Peterson 1988) but the data requirements mean that local area modelling is difficult within a full model structure. The same input-output constraint described above also applies to macro-econometric models and they are able to offer similar sectoral coverage.

Macro-econometric models are typically more open to the idea of different socio-economic groups with different consumption patterns but in practice are limited by the available data in this respect so there is little difference in coverage. However, one important advantage over CGE models is in modelling changes over time, as the models encompass both stocks and flows (rather than just flows in a standard CGE model) and are able to estimate transition paths as well as long-run outcomes.

#### **8.4.4.3 Potential Coverage of Health Effects**

The macro-econometric models are more heterogeneous in their detailed approach than the CGE models, especially in their treatment of supply-side constraints. In the next section we will show how such constraints are modelled using the E3ME model. In general, however, they are able to offer an adequate representation of health effects.

Household and government spending can be increased or decreased to the health-care sector as required, depending on the level of demand. Additional spending may be drawn from elsewhere or could come in the form of additional borrowing. Unlike in CGE models, the banking sector in macroeconomic models is able to provide additional financing (i.e. increasing the money supply) if there is sufficient demand for new loans.

It is also possible to model supply shocks in macro-econometric models. In the labour market, where potential supply is given explicitly by the available labour force, shocks can be entered by changing the number of available workers and the model will solve. Modelling changes in labour productivity is potentially trickier as the model may not know whether higher productivity leads to more output or fewer workers—however, as we shall see in the next section, it is possible to factor in productivity changes in some models.

#### **8.4.4.4 Summary**

In summary, macro-econometric models have their underlying assumptions which the model user and the policy maker who interprets the results must be aware of. Overall, however, the basic principles of a tool that simulates changes in policy rather than finds optimal outcomes for society is much more appropriate for an assessment of the health effects of environmental policy. The models are better suited for incorporating the build-up of health effects with impacts that might last long into the future. The empirical basis on which behavioural responses are estimated, while far from perfect, gives the best guide possible for estimating the impacts of future policy outcomes.

### ***8.4.5 Summary of How Health Effects May Be Represented***

In this section we have discussed three different modelling approaches, how they cover the key dimensions of the analysis and how they may be able to incorporate health effects that were identified in Sect. 8.2. Table 8.1 summarises the information.

## **8.5 Case Study: Simulating Health Impacts in the E3ME Modelling Framework**

### ***8.5.1 Introduction to E3ME***

In this section we describe in more detail how the health impacts of environmental policy could be assessed using the E3ME macroeconomic model. The model is described briefly below before we discuss how each individual impact can be entered and the data required to do so. Further information about E3ME, including recent applications and the full model manual (Cambridge Econometrics 2014), can be found at the website [www.e3me.com](http://www.e3me.com).

**Table 8.1** Overview of model capabilities

	Input-output	CGE	Macro-econometric
<i>Key dimensions</i>			
Geographical	Usually national, sometimes regional but not local	Usually national, sometimes regional but not local	Usually national, sometimes regional but not local
Sectoral	Reasonable	Reasonable	Reasonable
Socio-economic groups	None	Limited	Limited
Over time	Not available	Typically long-term only (can be made dynamic)	Long-term and short-term
<i>Health impacts</i>			
Healthcare costs	Possible	Possible	Possible
Mortality rates	Changes in consumption only	Focused on changes in production	Accounts for changes in production and consumption
Labour market participation	Not covered	Covered	Covered
Labour productivity	Not covered	Covered—assumed to lead to higher production	Covered—could lead to higher production or lower employment

*Source:* Authors' analysis

E3ME is a macro-econometric model of the world's economic and energy systems and the environment. It was originally developed through the European Commission's research framework programmes and is now widely used in Europe and beyond for policy assessment, for forecasting and for research purposes. The most recent version of the model has global coverage and splits the world into 59 regions. The model includes 69 economic sectors in Europe and 43 economic sectors in the rest of the world. Its historical database covers the period 1970–2014 and it projects forward annually up to 2050. Recent applications of the model include inputs for the official analysis of the EU's 2030 climate and energy targets (Pollitt et al. 2014). E3ME has also been used to look at the economic damages related to local air pollution in Ščasný et al. (2009) and Barker and Rosendahl (2000).

Figure 8.2 summarises how the different components of the model fit together. In this section we describe how the feedbacks from environment to economy (top left arrow) could be expanded to include health effects. With this feedback in place the model is able to offer a comprehensive assessment of the impacts of environmental policy, including health effects.

E3ME has previously been used in the past to estimate the health-economic impacts of exposure to heavy metal pollutants, such as lead and cadmium, as part of the EU DROPS research project (<http://drops.nilu.no/>). Many of the linkages described below are developed from the research that was carried out in that project and generalised for wider environmental policy.

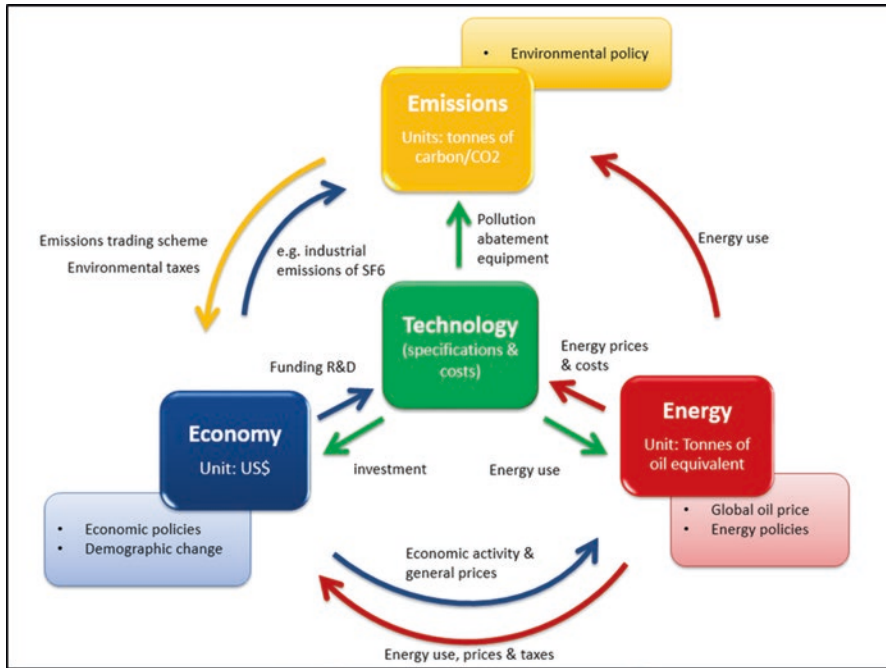


Fig. 8.2 E3ME modules. Source: Cambridge Econometrics (2014). Used with permission

### 8.5.2 Forming Model Inputs

At present there is no health module in E3ME but it would be possible to build this in, adding a box between environment and economy to Fig. 8.2. However, a simpler approach would be to carry out these calculations off-model and use an approach based on a parameterisation of these calculations. Whichever approach is adopted, the first stage in the calculation is to estimate the incidence of illness or disease linked to emissions of a specific pollutant. This estimate can then in turn be used to produce estimates of changes in healthcare costs, labour productivity, etc.

The advantage of building the calculations into the model is that it is possible to take into account the non-linearities that are an important feature of dose-response functions. The advantage of the parameterised approach is that it is technically easier to implement. In the sections below we outline the linkages back to the economy based on estimates that could be derived using either approach.

### 8.5.3 Healthcare Costs

Healthcare expenditure features in two parts of the model: final household expenditure and final government consumption. In both cases the revenues accrue to the health sector which may adjust employment levels to match demand.

Usually a budget balancing mechanism will be put in place, i.e. if healthcare spending increases then it is at the expense of other expenditure. For households the standard assumption is that other expenditures are reduced proportionately (although this could be modified, e.g. to focus on luxuries rather than necessities). Governments can either reduce expenditure in other areas or raise taxes to pay for the increased healthcare requirements.

### **8.5.4 Mortality Rates**

Population is an exogenous assumption to E3ME and is therefore quite easy to change. There are two main effects in the model from changing population:

- Aggregate rates of consumption, which are estimated on a per capita basis multiplied by population, will be affected
- The potential available labour force is adjusted

E3ME breaks the population down to 5-year age bands and splits by gender, which are important for determining the size of the potential labour force (ideally they would also determine consumption patterns but the data do not support this). Information about mortality rates should therefore also be broken down by age group and gender.

### **8.5.5 Labour Market Participation**

The labour force, or labour supply, is defined as those in employment plus those who are unemployed. In E3ME the labour force is calculated by multiplying working age population (see above) by a set of participation rates, again split by gender and age band. Participation rates are estimated endogenously in the model but the equations do not take into account health effects—hence it is necessary to step in and make additional changes.

In making these changes we need to gather information about the number of people who are unable to work due to illness, and also make an assumption about the share of these people who otherwise would have been either working or seeking work. Ideally an allowance should be made for individuals who drop out of the labour force to take on caring responsibilities.

A change in labour supply will affect primarily the unemployment rate (labour supply minus employment), which will in turn feed into the determination of wage rates and industrial unit costs. A moderate sized change in labour supply could therefore have important impacts throughout the economy.

### 8.5.6 *Labour Productivity*

As a macro-econometric model, E3ME does not make assumptions about the full use of capacity. Under normal economic conditions it can usually be assumed that there is spare capacity available in the economy, represented by economists as the 'output gap'. The difference between actual and potential production levels are not observable, however, so the output gap is estimated, in part based on activity rates in labour markets (where capacity, i.e. number of people, is known).

E3ME also does not have an explicit representation of potential output but has an estimate based on a set of econometric 'normal' output equations. These equations are an extrapolation of previous trends in the economy that give a measure of expected future output, on which capacity is based. If actual production increases in relation to expected output then there may be constraints on output, represented by:

- Increases in product prices
- Increased investment in new capacity
- Import substitution
- Increases in wage rates and working hours

A reduction in labour productivity, for example due to illness, is assumed to reduce available capacity, leading to all of these outcomes. The functional form used is similar to the Cobb-Douglas function, described in Sect. 8.4.3.3, except that E3ME does not assume constant returns to scale and so the  $(1 - \beta)$  term can be replaced with a separate value between zero and one. It is still necessary, however, to make an assumption about the marginal productivity of labour (how much capacity increases in response to higher labour productivity, represented by  $\beta$  in the equation).

Reductions in labour productivity due to illness could be expected to accumulate over time, until the workers affected either recover or leave employment. As above, it is therefore useful to have an estimate of the share of workers affected in each age/gender group.

### 8.5.7 *Expected Macroeconomic Outcomes*

Figure 8.3 shows the main linkages within the model framework. The four inputs from health impacts are shown in bold-italic type in the four corners. The most important outcomes from the modelling (GDP, employment and unemployment) appear in the centre of the figure. Solid lines represent relationships in the model that are formed either through accounting balances or assumptions, while the dashed lines represent the econometric equations.

It is immediately clear that the feedbacks are highly complex and there are many interdependencies in the modelling system. Furthermore, the figure only shows the most important linkages. There are further linkages as well (e.g. labour participation rates are also determined by GDP and unemployment rates) and the economic



mechanisms that determine the direct effects of environmental policy are not included. Furthermore, the model itself of course represents a simplification of the actual real-world processes.

Although not shown on the diagram there are also a mix of linear (e.g. the calculation of unemployment) and log-linear (e.g. the econometric equations) relationships. This means that the context in which the analysis is carried out is important. For example a policy that is designed to create employment will be ineffective if there are no people available to fill the new positions. This suggests that a simple approach of subtracting a value from GDP to estimate damage costs could be inaccurate under different economic conditions.

Overall, however, we can trace through some of the broad impacts of reducing the incidence of illness:

- A reduction in healthcare costs leads to a shift in spending away from healthcare and towards other products. The impact of this on GDP is ambiguous, it is likely to depend on the relative import content of the purchased goods and the displaced health services. A higher share of domestic content in the products consumed will lead to higher GDP.
- A reduction in mortality rates will mean higher levels of consumption by households. This will lead to higher tax receipts and, potentially, government expenditure (not shown in the figure). Aggregate GDP will be higher, although GDP per capita may be affected less. A reduction in mortality rates may also mean a larger available labour force. This will in the long run lead to lower wage rates (through adjustments in employment and unemployment levels) and lower business costs. Improved competitiveness will lead to lower inflation rates, higher (real) household spending and an improvement in the trade balance. Ultimately, GDP will increase.
- An increase in labour participation rates will also lead to an increase in labour supply, with the same effects as described above.
- Finally an improvement in labour productivity will increase the capacity of domestic firms, allowing them to reduce prices and increase production levels. Lower prices lead to competitiveness benefits, higher domestic spending and an improvement in international trade. Firms may also benefit from lower wage rates and a direct beneficial trade substitution effect (through more capacity for domestic production). Although there is one potential negative effect, lower investment (as there is less need for new capacity), the positive effects from lower prices are likely to outweigh this and GDP increases overall.

The picture is thus broadly positive in economic terms and even more so in welfare terms because households would much prefer to spend their income on things other than healthcare. The question for policy makers is how these benefits would compare against the potential costs of implementing the environmental regulation. E3ME provides a framework that can address this question.

### 8.6 Conclusions

Macroeconomic models are used extensively for the quantitative assessment of environmental policy. By combining the energy system with the wider economy, these models are able to estimate the impacts of energy or environmental policy on indicators such as GDP and employment. Current models are not, however, well suited to understanding the health impacts of such policies.

The approach that has been applied most widely has been to estimate health costs in monetary terms and to subtract this from the GDP results in the model. However, as is clear in Fig. 8.3, this approach ignores the complexity of the underlying relationships and may not be appropriate to use in different macroeconomic contexts. Furthermore, it cannot be used easily to estimate impacts on other macroeconomic indicators, such as employment. There is thus merit in assessing how the macroeconomic modelling frameworks available can be adapted to better account for health impacts.

We considered three different modelling frameworks that could be applied potentially. Each one has a different focus and a different set of comparative advantages and disadvantages. We found that input-output analysis is not an appropriate tool to carry out the analysis, CGE modelling can partially address the issues, while a macro-econometric model better meets the requirements, although still with some shortcomings.

The final part of the chapter outlined how the framework offered by the E3ME macroeconomic model could be adapted to take into account health impacts. We found that the relationships are complex and there are many potential interactions with the wider economy. However, the mechanisms within the model in general

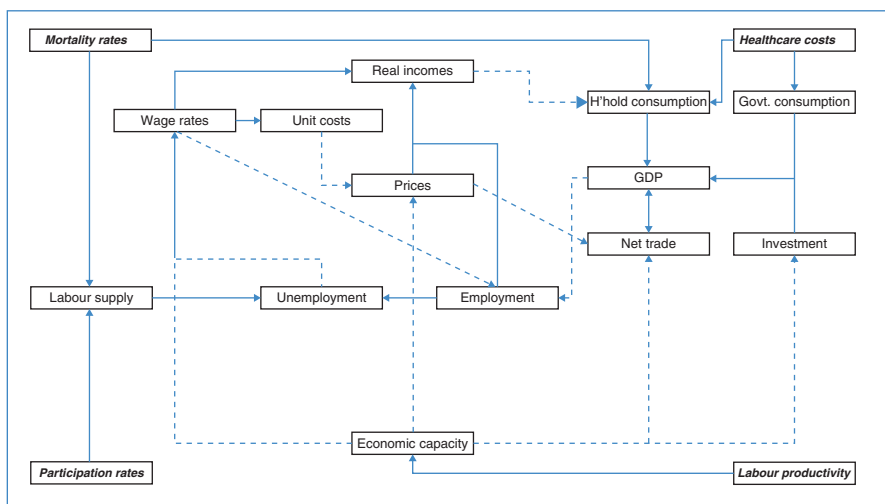


Fig. 8.3 Main model linkages. Source: Authors' analysis

point to a positive outcome from reducing incidences of illness. The value of such a tool to policy makers is that it can allow a comparison of the benefits from improving public health with the initial costs of implementing the environmental policy, all within a single assessment framework.

## References

- Barker TS, Peterson W (1988) *The Cambridge multisectoral dynamic model*. Cambridge University Press, Cambridge
- Barker TS, Rosendahl KE (2000) 'Ancillary benefits of GHG mitigation in Europe: SO<sub>2</sub>, NO<sub>x</sub> and PM<sub>10</sub> reductions from policies to meet Kyoto targets using the E3ME model and EXTERNE valuations', *Ancillary benefits and costs of greenhouse gas mitigation*. Proceedings of an IPCC co-sponsored workshop, OECD, Paris, Mar 2000
- Barker TS, De-Ramon SD, Pollitt H (2009) Revenue recycling and labour markets: effects on costs of policies for sustainability. In: Bosetti V, Gerlagh R, Schleicher SP (eds) *Modelling sustainable development transitions to a sustainable future*. Edward Elgar, Cheltenham and Northampton, MA
- Cambridge Econometrics (2014) E3ME manual, version 6.0. [www.e3me.com](http://www.e3me.com)
- Capros P, Van Regemorter D, Paroussos L, Karkatsoulis P (2013) GEM-E3 model documentation. JRC Technical Report, Publications Office of the European Union, Luxembourg. <http://ftp.jrc.es/EURdoc/JRC83177.pdf>
- Dixon PB, Rimmer MT (2002) *Dynamic, general equilibrium modelling for forecasting and policy: a practical guide and documentation of MONASH*. North-Holland, Amsterdam
- European Commission (2015) *Better Regulation guidelines*. European Commission document SWD, 115. [http://ec.europa.eu/smartregulation/impact/commission\\_guidelines/docs/iag\\_2009\\_en.pdf](http://ec.europa.eu/smartregulation/impact/commission_guidelines/docs/iag_2009_en.pdf)
- Hertel T (1999) *Global trade analysis: modeling and applications*. Cambridge University Press, Cambridge
- HM Treasury (2013) *The green book: appraisal and evaluation in central government*. TSO, London, [https://www.gov.uk/government/uploads/system/uploads/attachment\\_data/file/220541/green\\_book\\_complete.pdf](https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/220541/green_book_complete.pdf)
- Keen S (2011) *Debunking economics—revised and expanded edition: The naked emperor dethroned?* Zed, London and New York
- Lucas R (1976) *Econometric policy evaluation: a critique*. In: Brunner K, Meltzer A (eds) *The Phillips curve and labor markets*. North-Holland, Amsterdam
- Lutz C, Meyer B, Wolter MI (2010) *The global multisector/multicountry 3E-model GINFORS. A description of the model and a baseline forecast for global energy demand and CO<sub>2</sub>-emissions*. *Int J Global Environ Issues* 10(1–2):25–45
- Pollitt H, Alexandri E, Chewpreecha U, Klaassen G (2014) *Macroeconomic analysis of the employment impacts of future EU climate policies*. *Clim Pol*. doi:[10.1080/14693062.2014.953907](https://doi.org/10.1080/14693062.2014.953907)
- Ščasný M, Pollitt H, Chewpreecha U, Píša V (2009) *Analysing macroeconomic effects of energy taxation by the econometric E3ME model*. Prepared for the special issue on Environmental Public Finance of *Czech Journal of Economics and Finance*

# Chapter 9

## Transition to a Low Carbon Economy; Impacts to Health and the Environment

Rebecca J. Thorne

**Abstract** As carbon dioxide (CO<sub>2</sub>) emissions and other greenhouse gases (GHGs) represent both a great environmental challenge in terms of climate change and a problem for human health and social issues, their emission must be mitigated. Technological pathways to a low carbon economy include improving resource application/energy intensity, or by cutting carbon intensity through promoting low carbon technologies such as renewable energy or carbon capture and storage (CCS). For these technologies to be a truly effective option to mitigate climate change, they must be sustainable and be protective of the environment and human health over the long-term. Whilst analysis of most initiatives focuses on direct GHG quantification, other benefits and potential impacts are often not considered, and indirect emissions are often overlooked. In this chapter, the benefits of adapting to a low carbon economy are assessed, and technological adaptation strategies discussed along with associated impacts to human health and the environment. In particular, CCS is focused upon and life cycle assessment (LCA) shown as a particularly useful tool to give a holistic view of the impacts relating to mitigation options. These are important decision criteria for policymakers, and should be dealt with as such.

**Keywords** Climate change • Impacts • Human health • Environment • CCS

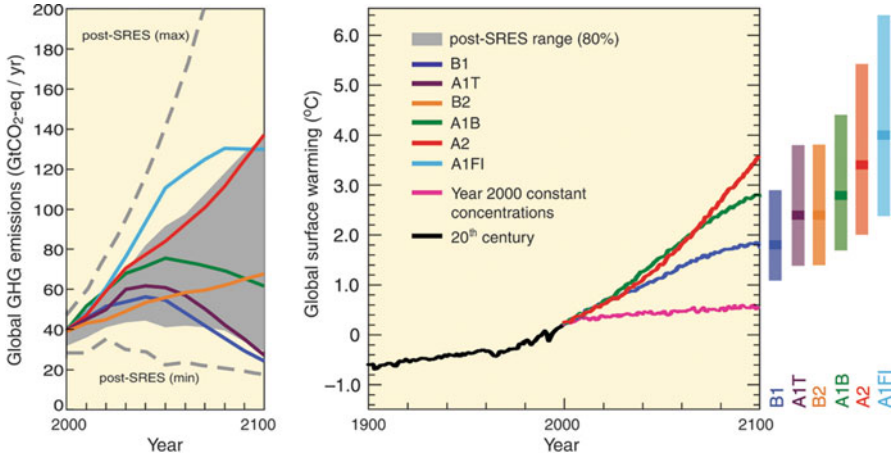
### 9.1 Introduction to Climate Change and Associated Impacts

According to figures from the Intergovernmental Panel on Climate Change (IPCC), the interpretation of the temperature record indicates a slight increase in global average annual temperatures in the last 150 years of 0.76 °C (IPCC 2007a). It is generally accepted by the scientific community that the main cause of this ‘global warming’ is the increase in atmospheric concentrations of greenhouse gases (GHGs) due to human activity, including carbon dioxide (CO<sub>2</sub>), methane (CH<sub>4</sub>) and nitrous

---

R.J. Thorne (✉)

Department of Environmental Impacts and Economics (IMPEC),  
NILU - Norwegian Institute for Air Research, PO Box 100, 2027 Kjeller, Norway  
e-mail: [rjt@nilu.no](mailto:rjt@nilu.no)



**Fig. 9.1** Global GHG emissions and emission scenarios. Taken from IPCC (2007b). *Note: Left Panel:* Global GHG emissions (in GtCO<sub>2</sub>-eq) in the absence of climate policies: six illustrative SRES marker scenarios (coloured lines) and the 80th percentile range of recent scenarios published since SRES (post-SRES) (grey shaded area). Dashed lines show the full range of post-SRES scenarios. The emissions include CO<sub>2</sub>, CH<sub>4</sub>, N<sub>2</sub>O and F-gases. *Right Panel:* Solid lines are multi-model global averages of surface warming for scenarios A2, A1B and B1, shown as continuations of the twentieth-century simulations. These projections also take into account emissions of short-lived GHGs and aerosols. The pink line is not a scenario, but is for Atmosphere-Ocean General Circulation Model (AOGCM) simulations where atmospheric concentrations are held constant at year 2000 values. The bars at the right of the figure indicate the best estimate (solid line within each bar) and the likely range assessed for the six SRES marker scenarios at 2090–2099. All temperatures are relative to the period 1980–1999

oxide (N<sub>2</sub>O) (IPCC 2007a). CO<sub>2</sub>, whose atmospheric concentration has risen from pre-industrial levels of 280–380 ppm in 2005, is considered the main GHG and responsible for two thirds of the enhanced greenhouse effect (IPCC 2007a).

There is general acceptance that the world must take both preventive and mitigating action against global warming, and reduce CO<sub>2</sub> emissions to the atmosphere. Predictions have been made that if continued in a ‘business-as-usual’ (BAU) scenario, significant climate change will occur by the end of the century increasing the global surface temperature by 1–6 °C depending on emission scenario [several scenarios have been developed, but for an example see Fig. 9.1, where further reading on each scenario described can be found in the Special Report on Emissions Scenarios (SRES) (IPCC 2000)]. It is now accepted by many countries that ‘dangerous’ climate change will occur if the global-mean temperature increases over a 2 °C limit compared to a pre-industrial baseline (Gosling et al. 2011).

In response to the global warming threat, the United Nations Framework Convention on Climate Change (UNFCCC) established the Kyoto protocol in 1997, deeming it necessary to mitigate GHG emissions. For the temperature increase to be limited to 1.8 °C (below the 2 °C threshold), CO<sub>2</sub> emissions must stabilise in the long term around 400 ppm (Enkvist et al. 2010). This corresponds with global

CO<sub>2</sub>-eq emissions peaking at around 55 GtCO<sub>2</sub>-eq per year in the year 2040, before declining to under 30 GtCO<sub>2</sub>-eq per year by the end of the century (Fig. 9.1). A target reduction for Annex I (developed) countries of 5.2% compared to the 1990 baseline was set for the first Kyoto protocol commitment period of 2008–2012. Although this was an important first step towards mitigation action, these targets were not fully met. For example, the EU, as one of the major contributors towards Kyoto Protocol foundation, had a 8% GHG reduction target in 2008–2012, but the 15 states who were EU members in 1997 when the Kyoto Protocol was adopted (EU-15) only made a 4.3% reduction in GHG emissions (Lau et al. 2012). During the second Kyoto commitment period of 2013–2020, Annex I parties have committed to reduce GHG emissions by at least 18% below 1990 levels.

Other emission targets are also in place. In 2009, the EU pledged a unilateral GHG reduction target of 20% below 1990 levels by 2020, with at least 20% of EU energy from renewables and a 20% increase in energy efficiency also by 2020 (the 2020 climate and energy package). In addition, in 2014 the EU pledged a unilateral GHG reduction target of 40% below 1990 levels by 2030, coupled with at least 27% of EU energy from renewables and a 27% increase in energy efficiency (the 2030 climate and energy framework). Most recently at the Paris Climate Conference (COP21) held in December 2015, 195 countries adopted the first ever universal and legally binding global climate deal to limit global warming to well below 2 °C above a pre-industrial baseline, with an aim of 1.5 °C (EC 2015a). The agreement states a need for global GHG emissions to peak as soon as possible, and undertake rapid reductions thereafter. In advance of the conference, countries accounting for approximately 90% of global CO<sub>2</sub> emissions submitted their climate plans in terms of Intended Nationally Determined Contributions (INDCs). This showed a gap of 15–17 Gt between what countries have promised to do, and the emissions reductions needed to peak emissions by 2020 and stay on the 2 °C trajectory (IRENA 2015a). According to analysis, if all current INDCs are fully implemented, the temperature will still rise roughly 2.7 °C by the end of the century (IRENA 2015a).

If insufficient measures are taken to mitigate global warming, significant impacts are likely to occur relating to rising sea levels, ocean acidification, and issues to ecosystems and biodiversity, water resources and food security at a global level (see Gosling et al. 2011, for a review). As a result of these climatologic factors, human health is threatened both by direct effects of temperature and climatologic instability, but also by secondary effects such as those resulting from impacts to agriculture, access to clean water, shifting distribution of disease, migration, competition for scarce resources and the potential for armed conflict (Louis and Hess 2008). Figure 9.2 summarises the relationships between the driving forces for local climate change, pressure on the environment, human hazard exposure and health impacts, using the Driving Force-Pressure-State-Exposure-Effect-Action (DPSEEA) framework developed by the World Health Organisation (WHO) (Kjellstrom and McMichael 2013).

The Fourth Assessment Report (AR4) from the IPCC estimated that the global-mean sea level rise could be up to 0.38 m for a world with 2 °C increased temperatures (IPCC 2007c), although more recent reports suggest that this is an underestimate

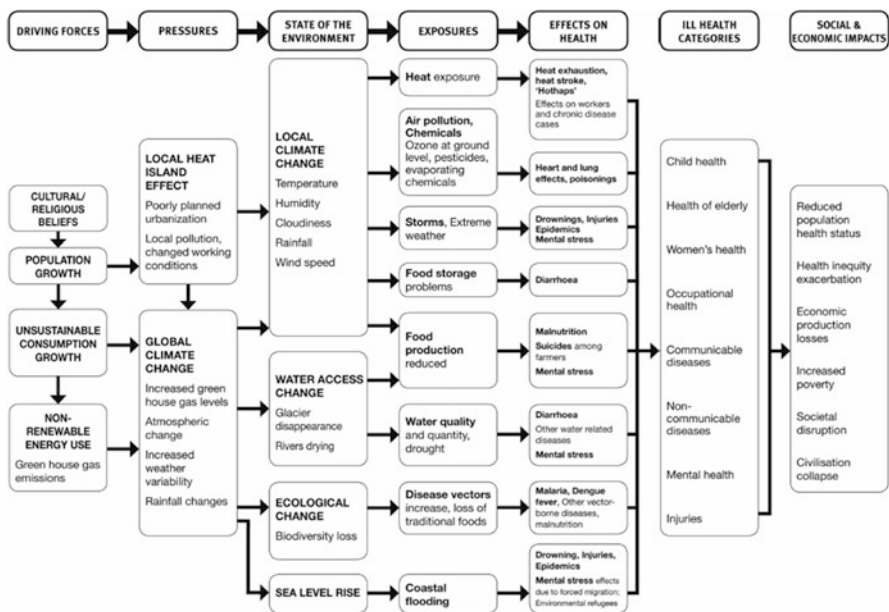


Fig. 9.2 DPSEEA framework for climate change and global public health. Taken from Kjellstrom and McMichael (2013)

(Rahmstorf 2010). Impacts upon society resulting from this include flooding, with 63–102 million people estimated flooded and 5–20 % of coastal wetlands estimated lost, and southeast Asia representing the worst affected region (IPCC 2007c). Exposure to flooding and other natural hazards is increased by the destruction of mangroves that protect coasts from tidal surges (Jäger and Kok 2007). Globally, the cost of this has been estimated at around \$2–400,000 million per year (Dasgupta et al. 2009; van Vuuren et al. 2011). Due to ocean absorption of CO<sub>2</sub>, surface pH is projected to decrease by 0.3–0.4 units by 2100 with a 3–4 °C temperature increase, leading to under-saturation of aragonite, changes to oxygen levels, redistribution of fish, and adverse effects on calcifying organisms (IPCC 2007c). Due to these effects (and others on other ecosystems), the potential for abrupt negative changes in ecosystems and ecosystem services is great, with extinctions possible for 20–30 % of plant and animal species assessed (IPCC 2007c). Impacts may be felt by humans, with resulting large-scale crises and migration.

Climate change impacts to water resources and agriculture could have a significant impact on human welfare. Globally, it is expected that there will be an overall net negative impact on water resources, and drought-affected areas will increase (IPCC 2007c). For example, Gosling and Arnell (2011) estimated using a hydrological model that for a 2 °C increase in temperature, up to 2.202 billion people may experience increased water scarcity. Similarly, with the same temperature increase, it has been estimated that around 59 % of the global population would have a blue

water shortage (fresh surface and groundwater), and 39% would experience a shortage of both blue and green water (precipitation on land stored in soil) (Rockstroem et al. 2009). According to Rockstroem et al. (2009), green water dominates in food production; thus countries which are severely water short may not be able to produce enough food for their population unless green water is managed well. On the other hand, enriched CO<sub>2</sub> concentrations may have a positive effect on crop productivity (IPCC 2007c). Nevertheless, according to Tubiello et al. (2007), this may be offset by a negative factors for crop growth such as pests, weeds and diseases and extreme events, and evidence suggests that crops grown under elevated CO<sub>2</sub> conditions may have a reduced protein and mineral content (Ainsworth and McGrath 2010). It will only be through understanding and simulating local and region level climate-crop processes that the impacts can be understood.

Direct effects to human health (i.e. mortality) may also result from climate change and climate variability. These include risks from extreme weather [over the past 20 years, natural disasters have claimed more than 1.5 billion lives, and affected more than 200 million people annually (Jäger and Kok 2007)], and increased risk of disease with an estimated extra 2.5 billion people at risk of dengue (IPCC 2007c) and 100,000 extra deaths from malaria across Africa in 2050 (van Vuuren et al. 2011). For temperature-related mortality, evidence is conflicting due to the balance of heat and cold related mortality (Gosling et al. 2011); Hayashi et al. (2010) estimate that one million heat-related global deaths could be avoided by 2100 if CO<sub>2</sub> levels are stabilised at 450 ppm instead of 650 ppm. However, overall the authors suggest that the increased temperatures caused by climate change could actually reduce overall mortality due to the larger decrease of cold-related mortality, although Gosling et al. (2011) note that these conclusions are debateable. Climate variation may also reduce local and regional air quality, with resulting respiratory and cardiac disease (Ebi and McGregor 2008), as well as affecting the environmental fate of other pollutants such as mercury (Hg) (Sundseth et al. 2015). For the latter, methylation and re-emission from soil and water may increase with higher temperatures, with resulting impacts to human health (Sundseth et al. 2015; Pacyna et al. 2010). Nonetheless, local conditions must again be studied in order to accurately predict effects.

A complication to the situation is that human impacts relating to the aforementioned factors are not distributed equally amongst people. According to the United Nations (Jäger and Kok 2007), vulnerability itself depends on sensitivity to impacts, exposure and adaption capacity, with the most vulnerable groups including the poor, indigenous populations, women and children. In recent years, countries that have an equitable income distribution and medical treatment availability have had larger life expectancy gains than others. Thus, poverty represents a major factor in vulnerability; these people lack essential services, making them especially susceptible to environmental and socioeconomic change (Jäger and Kok 2007), and developing countries may thus be the most affected by climate change (Beg et al. 2002). More than 90% of the people exposed to natural disasters live in the developing world, and due to factors such as reduced state social protection schemes and poor infrastructure, chronic disease and conflict, capacity to adapt is reduced (Jäger and Kok 2007).



In fact, according to St Louis and Hess (2008), the distribution of most severe health burdens would be almost inverse to the global distribution of GHG emissions, although the experience of the 2003 heatwave in Europe shows that high-income countries may also be adversely affected (Haines et al. 2006). Importantly, the leading health risks in poorest countries including malnutrition, unsafe drinking water, poor-quality nutrients and use of low-quality fuels, are all climate sensitive. Effects of climate change since the end of the 1990s are already being observed (Louis and Hess 2008); the World Health Organisation (WHO) has predicted that deaths per year attributable to climate change have already reached 154,000, with the greatest burden in sub-Saharan Africa and South Asia (WHO 2002). Although international trade has helped remove many from poverty, outsourcing natural resource extraction, production and manufacturing to developing countries leads to challenges with environmental impacts and unsustainable patterns of consumption (Jäger and Kok 2007). Worsening the situation, as noted by the Brundtland Commission (WCED 1987), is that environmental degradation contributes to the 'downward spiral of poverty'. Poorest people suffer the greatest water deficit due to location, poor infrastructure and lack of finances, and poor access to material assets at the household level is part of a cycle of impoverishment, vulnerability and environmental change (Jäger and Kok 2007).

To increase robustness towards environmental and socioeconomic change, sustainable development must be achieved and poverty must be addressed in all countries across all sectors. In particular, local and global governance should be integrated for policy making (Jäger and Kok 2007). This was highlighted at the COP21 conference, with governments agreeing to provide continued and enhanced international support for adaptation to developing countries (EC 2015a). Some studies (e.g. Arnell et al. 2011) show that climate change impacts may not be totally eliminated by climate policy alone; science and technology has the potential to reduce vulnerability, but has also added to the climate change risks faced by people and the environment (Jäger and Kok 2007). Overall, there are strong synergies between improving human well-being and reducing vulnerability from environment, development and human rights perspectives (Jäger and Kok 2007). However, many uncertainties exist between understanding the association of climate and human systems.

## **9.2 Co-Benefits to Air Quality Resulting from a Low Carbon Economy, and Associated Impacts**

A low carbon society would not only mitigate the aforementioned effects of climate change (conveying direct benefits), but in many cases, methods of reducing CO<sub>2</sub> emissions have additional impacts on other types of emissions, such as air pollutants including particulate matter (PM), ozone (O<sub>3</sub>), carbon monoxide (CO), sulphur dioxide (SO<sub>2</sub>) and nitrogen oxides (NO<sub>x</sub>). This is as a result of the fact that these air pollutants are emitted from many of the same processes as GHGs.

**Table 9.1** Sources and health effects of air pollution. Adapted from the U.S. EPA (2012)

Pollutant	Main source(s)	Health effects
Ozone (O <sub>3</sub> )	Secondary pollutant typically formed in sunlight by chemical reaction of NO <sub>x</sub> and volatile organic compounds (VOCs)	Decreases lung function and causes respiratory symptoms, aggravates asthma and other lung diseases which can lead to premature mortality
Particulate Matter (PM)	Emitted through chemical reactions including fuel combustion, industrial processes, agriculture and unpaved roads	Short-term exposure can aggravate heart/lung diseases. Long term exposure can lead to heart and lung disease and premature mortality
Lead (Pb)	Smelters and other metal industries, combustion of leaded gasoline, waste incinerators and battery manufacturing	Damages the developing nervous system lowering IQ and impacting learning, memory and behaviour. In adults, cardiovascular, renal and early anaemia effects may occur
Nitrogen oxides (NO <sub>x</sub> )	Fuel combustion and wood burning	Aggravates lung disease leading to respiratory symptoms and increased susceptibility to respiratory infection
Carbon monoxide (CO)	Fuel combustion (mainly vehicles)	Reduces the amount of oxygen reaching organs and tissues, aggravates heart disease
Sulphur dioxide (SO <sub>2</sub> )	Fuel combustion, electric utilities and industrial processes and natural sources	Aggravates asthma and increased respiratory symptoms. Contributes to particulate formation

Air pollutants derive from almost all economic and societal activities but mainly the combustion of fossil fuels (Guerreiro et al. 2014a, b). In brief, PM originates both from primary particles that are directly emitted, as well as secondary particles that are produced from chemical reactions involving PM precursors. O<sub>3</sub> is a secondary pollutant formed in the troposphere following emissions of precursors, and CO, SO<sub>2</sub>, NO<sub>x</sub> and toxic metals are mainly emitted during fuel combustion and/or industrial activity (Guerreiro et al. 2014a, b). Organic air pollutants include benzo(a) pyrene (BaP), a polycyclic aromatic hydrocarbon formed mainly from organic material combustion, and benzene, mainly released during incomplete combustion of vehicle fuels (Guerreiro et al. 2014a, b).

Indirect co-benefits on air pollution resulting from climate change mitigation policies and the reduction in levels of pollutants other than GHGs have been described (Haines 2012; Patz et al. 2014; Woodcock et al. 2009; Kjellstrom and McMichael 2013). Air pollutants can affect health in many ways, but in general can be linked to respiratory and cardiovascular disease, decreased lung function, increased frequency and severity of respiratory symptoms and susceptibility to respiratory infection, effects to the nervous system (such as IQ loss), cancer and premature death. Table 9.1 gives a break-down of the sources of some key air pollutants, and their effect on human health. The United States Environmental Protection Agency (U.S. EPA) note that those with pre-existing health conditions, older adults, children and diabetics appear at a greater risk (U.S. EPA 2012).

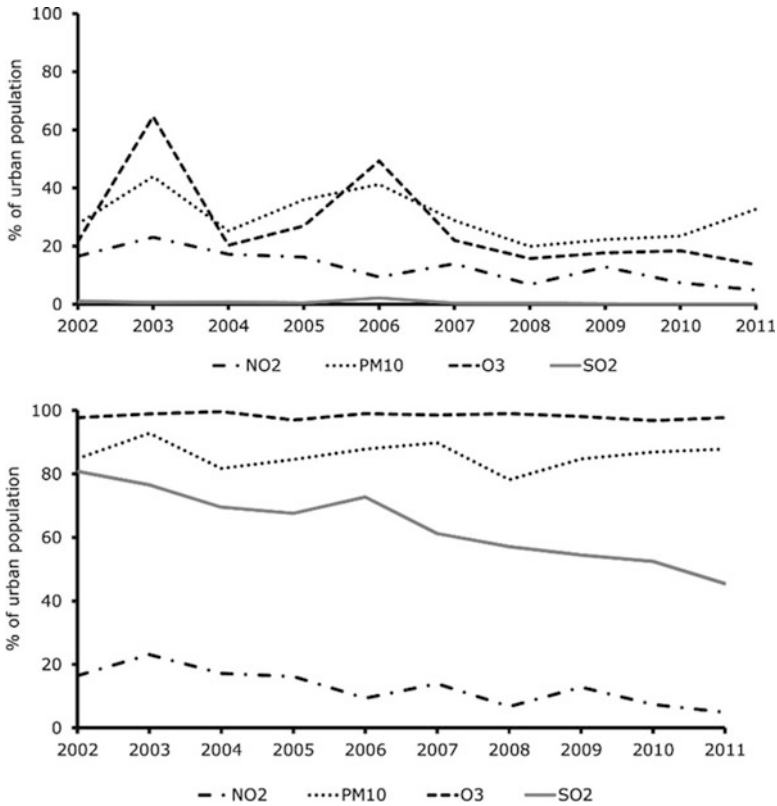
According to Guerreiro et al. (2014b), PM poses the greatest risk in terms of the potential to harm human health, penetrating into the respiratory system leading to health problems and premature mortality. Toxic metals (e.g. Hg) can also reside in or be attached to PM and impact human health when inhaled. Co-benefits in terms of reductions of levels of these pollutants may thus have a substantial, immediate impact on population health (Haines et al. 2009, 2006), and are important to provide additional rationale for GHG mitigation, as well as to address international health priorities such as the UN Millennium Development Goals (MDGs) (Haines et al. 2009). As such, quantifying the impact of air pollution on public and environmental health has become an increasingly critical component in recent years for policy discussion.

Many studies have demonstrated the link between air pollutants in urban areas and adverse health risks due to air pollution (Ebi and McGregor 2008; Reiss et al. 2007; Lippmann 2014; Henschel and Chan 2013; WHO 2013). Health risks due to air pollution in urban areas are particularly an issue, since recent data indicates that more than 70 % of global population live in cities (Costa et al. 2014). Current pollution levels, especially of PM, O<sub>3</sub> and BaP thus impact on large parts of the population (Fig. 9.3); for example, it has been estimated that 20–44 % of the EU-27 urban population were exposed to concentrations of PM<sub>10</sub> in excess of the EU air quality daily limit (Guerreiro et al. 2014b). Additionally, in low-income countries, indoor air pollution is also a large issue for health (Haines 2012).

Air pollution can also directly and indirectly adversely impact the environment. For example, O<sub>3</sub> can damage vegetation, negatively impacting its growth and reducing its ability to uptake CO<sub>2</sub> (U.S. EPA 2012). SO<sub>x</sub> can cause acidification, and NH<sub>3</sub> emissions (93 % of which derive from agriculture) exert pressure on ecosystems due to eutrophication (Guerreiro et al. 2014b).

In many countries levels of air pollutants are declining. In the United States of America (U.S.), nationwide air quality has improved significantly in recent years (U.S. EPA 2012). Nevertheless, in 2010, 124 million people lived in U.S. counties that exceeded one or more national ambient air quality standard (NAAQS) (U.S. EPA 2012). Levels of six common pollutants have declined since 1990 when the Clean Air Act was amended, including ground level O<sub>3</sub> (8-h O<sub>3</sub> declined 17 %), PM pollution (24-h PM<sub>10</sub> declined by 38 %), Pb (3-month average Pb declined 83 %), NO<sub>2</sub> (annual NO<sub>2</sub> declined 45 %), CO (8-h CO declined 73 %) and SO<sub>2</sub> (annual SO<sub>2</sub> declined 75 %) (U.S. EPA 2012). This is thought to result from the development of cleaner cars, industries and consumer products (U.S. EPA 2012). In Europe also, air pollution shows some improvement. In the EU-27 block between 2002 and 2011, emissions of primary PM<sub>10</sub> and PM<sub>2.5</sub> decreased by 14 % and 16 % respectively, SO<sub>x</sub> emissions decreased by 50 %, NO<sub>x</sub> decreased by 27 %, NH<sub>3</sub> emissions decreased by 7 % and non-methane volatile organic compounds (NMVOCs) decreased by 23 % (Guerreiro et al. 2014b).

Despite improving trends, there is a close connection between the climate and air quality, and climate change itself is likely to have an impact on general air pollution levels. Climate change may directly affect local and regional air quality through changes in chemical reaction rates or changes in boundary layer heights, which



**Fig. 9.3** The percentage of the EU-27 urban population potentially exposed to air pollution exceeding allowable EU air quality standards (*top*), and WHO air quality guidelines (*bottom*). Taken from Guerreiro et al. (2014b)

affect the mixing of pollutants and synoptic airflow that governs pollutant transport (Ebi and McGregor 2008). In particular, O<sub>3</sub> and PM pollutants are strongly influenced by shifts in weather conditions (U.S. EPA 2012; Ebi and McGregor 2008). Thus, e.g., the U.S. Environmental Protection Agency (U.S. EPA) has concluded that climate change may produce a 2–8 ppm increase in summertime average ground-level O<sub>3</sub> concentrations, further exacerbate O<sub>3</sub> concentrations on days when weather is already favouring high O<sub>3</sub> concentrations, lengthen the O<sub>3</sub> season, and produce both increases and decreases in PM pollution over different U.S. regions (U.S. EPA 2012).

As these air pollutants also contribute to climate change (tropospheric O<sub>3</sub> is a GHG and particulates absorb and scatter light and change cloud reflectivity, lifetime and precipitation), a positive feedback loop may occur. As before, the overall impact would not be distributed equally, but rather the more vulnerable portions of society would suffer most.

### 9.3 Technological Pathways to a Low Carbon Economy

Technological pathways to a lower carbon economy include improving resource application and/or achieving structural change, developing higher levels of industrial efficiency, or by cutting carbon intensity through the promotion of low carbon technologies such as renewable energy or carbon capture and storage (CCS). Economic/political solutions also play an important role. The Kyoto Protocol proposes three mechanisms to assist developed ratified nations to achieve their emission reductions; the clean development mechanism that promotes environmentally friendly foreign investments to earn saleable emission reduction credits, the joint implementation mechanism allowing a country to earn emission reduction units from emission reduction elsewhere, and emission trading (Lau et al. 2012). The COP21 agreement also emphasises a combination of technological and economic factors for reaching GHG emission targets (EC 2015a).

For a transition to a low carbon economy to be successful it must be sustainable and protective of the environment and human health over the long term, and not conflict with the economic growth of a country. In practice, a combination of transitional pathways must be adopted; this is shown by the 2020 and 2030 targets for the EU, which aim to increase both the production of energy from renewable sources and increase energy efficiency. For example, the 20-20-20 targets introduced by the European Union aim to increase both the production of renewable energies by 20% and energy efficiency by 20% by 2020, in order to achieve a 20% (or even 30%) reduction in GHG emissions (EC 2009). The energy and industrial sectors must play a central role in achieving the GHG emission mitigation required for climate stabilisation. This was demonstrated in a study by Riahi et al. (2007), which found that in each of their stabilisation scenarios, 85% of total mitigation was in the energy and industrial sectors. Agriculture and forestry played a less important role in emission reductions in absolute terms, but are nonetheless important for net GHG emissions.

For full mitigation to be achieved, significant implementation barriers must be overcome. For example, studies have shown that less than 30% of potential GHG mitigation may be achieved by 2030 due to both cost and non-cost related barriers (Smith et al. 2007). As a result, policies must be implemented that convey benefits (incentives) for climate, economic, social and environmental sustainability (Smith et al. 2007). Subsidies also play a part to make certain technologies competitive; a study showed that in 2012, EU government interventions to support renewable energies were \$<sub>2012</sub> 41 billion, and support for energy efficiency was \$<sub>2012</sub> 9 billion (ECOFYS 2014a). Determining GHG mitigation measures hinges on comprehensive understanding of local conditions and the historical relationship between economic development, energy consumption and CO<sub>2</sub> emissions—especially for developing countries (Liao and Cao 2013). Some synergies already exist between climate change policies and sustainable development, such as energy efficiency, renewable energy, transport and sustainable land-use policies (Beg et al. 2002); if fully understood, these may alleviate mitigation barriers. For example, Yedla et al.

(2005) found that local pollutant mitigation strategies in transportation planning would mitigate GHGs, as well as attract international funding for transport infrastructure development in developing countries (Yedla et al. 2005).

Nevertheless, there may be large trade-offs where developing countries are dependent on indigenous coal and are required to switch to cleaner and more expensive fuels to limit emissions (Beg et al. 2002). Thus, policies must recognise the diverse situations of developing countries with respect to their level of economic development, their vulnerability to climate change and their ability to adapt or mitigate (Beg et al. 2002). This is reflected in the COP21 agreement, which emphasises large differences in the roles of developed and developing countries (EC 2015a). In addition, targeted (local) policy making may be required for countries where industrial structural change is occurring rapidly in an unequal manner across regions, such as China (Tian et al. 2014). In short, according to Beg et al. (2002), “Recognition of how climate change is likely to influence other development priorities may be a first step toward building cost-effective strategies and integrated, institutional capacity in developing countries to respond to climate change.”

### ***9.3.1 Improving Resource Application and Energy Intensity***

Delivering the same service or product but using less energy is one of the most cost-effective options for reducing GHG emissions. This is underlined by the EU target that all member states must achieve a 20% increase in energy efficiency by 2020; in practice this means that EU energy consumption should not exceed 1,483 Mtoe of primary energy (EC 2012). Energy consumption in the EU has already been reduced. EU primary energy consumption peaked in 2006 (around 1,721 Mtoe) and decreased to reach 1,584 Mtoe in 2012 (EC 2014), although much of this reduction can be assigned to a reduced economic productivity. To ensure 2020 targets are met, the Energy Efficiency Directive (EED) was created, that, for example, details measures for improving the energy efficiency of public buildings. Energy efficiency is also considered important in the U.S.; research published by McKinsey & Company (Granade et al. 2009) shows that the U.S. economy has the potential to reduce annual non-transportation energy consumption by 23% by 2020, eliminating more than \$1.2 trillion in waste and abating 1.1 Gt of GHG emissions annually.

Measures to increase efficiency vary depending on the sub-sector and activity in question. For combustion activities, fuel switching and use of alternate fuels can offer important CO<sub>2</sub> reductions, and in some cases can also lower operating costs (IEA 2012). Table 9.2 gives the energy content and unabated emission factors for CO<sub>2</sub> as specified by the 2006 IPCC Guidelines for National Greenhouse Gas Inventories (IPCC 2006) for some commonly combusted fuels. The differences in energy content values and CO<sub>2</sub> emission factors goes some way towards explaining the global shift away from coal combustion to natural gas (Riahi et al. 2007). However, additional investment is often required to utilise alternative fuels, and

**Table 9.2** Default CO<sub>2</sub> emission factors, representing unabated emissions from stationary combustion installations, and the energy density for the three major forms of fossil fuels. Data is sourced from the IPCC (2006)

Fuel	CO <sub>2</sub> emission factor (kg per TJ fuel)	Energy density (TJ per Gg fuel)
Coal (sub-bituminous)	96,100 (92,800–100,000)	18.9 (11.5–26.0)
Crude oil	73,300 (71,100–75,500)	42.3 (40.1–44.8)
Natural gas	56,100 (54,300–58,300)	48.0 (46.5–50.4)

regulatory or institutional barriers can inhibit their use (IEA 2012). Some developed regions with vastly improved input efficiency in resource-related heavy manufacturing demonstrate the potential for reducing CO<sub>2</sub> emissions in regions lagging in input efficiency (Tian et al. 2014). For cement production, fuel switching may lead to large efficiency gains, whilst for aluminium production, new technologies are required for gains in efficiency (IEA 2012). For transport, lower carbon-emission motor vehicles with more efficient engines and alternative fuels are being implemented, and an increased focus put on ‘active’ transport (e.g. walking or cycling) (Woodcock et al. 2009). In summary, specific efficiency improvement measures can be identified in virtually every sector, from buildings (Perez-Lombard et al. 2008) to agriculture (Pimentel et al. 1983), and should be identified on a case-by-case basis.

As with other GHG mitigation strategies, energy efficiency and public health are closely linked. Haines et al. (2009) and Haines (2012) reviewed and modelled energy initiatives to curb GHG emissions in energy, residential construction, urban transport and agricultural systems, and found many global health co-benefits (including amongst the poor). For each sector, the potential links between reduction in GHGs and health appeared strong, and e.g. switching to low carbon fuels, lowering consumption of animal products and using clean-burning cook stoves could reduce disease on national to regional scales. For example, for household energy, it was found that over 10 years, replacing 15 million inefficient cook-stoves with improved efficiency stoves could avert an estimated two million deaths during the period, as well as saving energy (Haines 2012). For transport, Woodcock et al. (2009) found that a reduction in CO<sub>2</sub> emissions from an increase in active travel and reduced use of motor vehicles had larger health benefits than from the use of lower emission motor vehicles alone. Policies to increase energy efficiency should account for these co-benefits.

### 9.3.2 *Low Carbon Technologies*

Evidence suggests that introducing low carbon technologies into the energy sector is key to mitigating GHG emissions and climate change (Valentine 2011; Lau et al. 2012; Song et al. 2015; Hastik et al. 2015; Panwar et al. 2011; Chang 2013). In addition to the mitigation of environmental impacts, this may bring co-benefits

**Table 9.3** Main renewable energy sources and their usage. Adapted from Demirbas (2006)

Energy source	Energy conversion and usage options
Hydropower	Power generation
Modern biomass	Heat/power generation, pyrolysis, gasification, digestion
Geothermal	Urban heating, power generation, hydrothermal, hot dry rock
Solar	Solar home system/dryers/cookers
Direct solar	Photovoltaic, thermal power generation, water heaters
Wave	Numerous designs
Tidal	Barrage, tidal stream

promoting the local economy through creating opportunities for industrial development and contributing towards energy security (Valentine 2011). Of the possible technologies investigated in order to achieve mitigation targets, renewables, nuclear and CCS are regarded as important options.

Renewable energy—energy that comes from resources replenished on a human timescale—is considered a clean source of energy that will minimise environmental impacts, minimise secondary waste and is sustainable for economic and social needs; both now and in the future (Panwar et al. 2011). Table 9.3 gives an overview of renewable energy sources, with more detailed reviews available elsewhere (Demirbas 2006; Raghuvanshi et al. 2008; Panwar et al. 2011). Recent estimates of the current share of renewable energy sources in global energy vary, e.g., from 19.1% (REN21 2015) to >22% (IRENA 2015a). For Europe, the share of renewables in gross final energy consumption was considered to be 15.3% in 2014 (ECOFYS 2014b). The most rapid growth, and the largest increase in capacity, is thought to have been led by wind, solar photovoltaics and hydropower. Projecting to the future, renewables are predicted to play an important role (REN21 2015). Within Europe, the EU considers that renewable energy is essential for an energy transformation to occur, as it “...contributes to all of the Energy Union objectives: the delivery of security of supply, a transition to a sustainable energy system with reduced greenhouse gas emissions, industrial development leading to growth and jobs and lower energy costs for the EU economy” (EC 2015b). The EU has set a target of 20% energy from renewable sources by 2020 [and 10% use of biofuels in transport (EC 2015b)], growing to at least 27% by 2030. The role of renewables was also specifically emphasised in the COP21 agreement (EC 2015a).

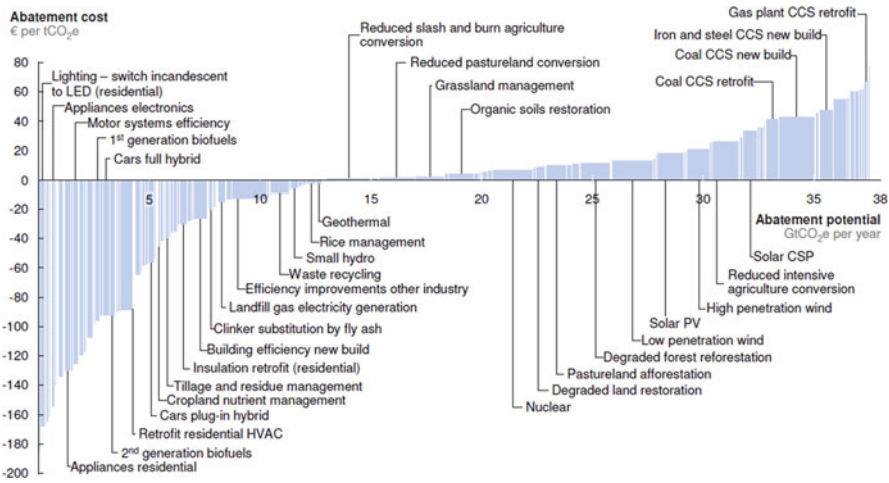
Nuclear energy is considered clean energy but its inclusion in the renewable energy list is a subject of major debate. In recent years nuclear energy has also been seen as a GHG mitigation pathway that conveys energy security, [providing 2.6% of global final energy consumption in 2013 (REN21 2015)], but it is less attractive in terms of accessibility, affordability and resilience (Valentine 2011). Additional safety considerations since the Fukushima disaster of March 2011 have also impacted its appeal (Valentine 2011). For a review of nuclear energy, see e.g. Pearce (2012).



A major challenge for the uptake of renewables in the large scale is breaking through the technological hold that fossil fuels have on energy provision (Valentine 2011). Due to the relatively fragmented structure of the renewable energy sector, a large issue is cost, coupled with lack of public understanding and political support (Valentine 2011). Although current engineering consensus is that up to 20% energy from intermittent sources can be incorporated into most existing electricity networks without additional storage capacity or backup generators (Valentine 2011), a persisting belief exists that renewables would lead to unstable electricity networks. Additionally, depending on the renewable energy type, conflicts may occur for land with pre-existing ecosystem services (Hastik et al. 2015)—benefits provided by ecosystems to humans that depend on biodiversity as the total sum of life. National renewable energy associations exist in most countries, but as of yet there is an insufficient collaborative effort of transnational renewable energy associations to lobby against (and unseat) fossil fuels (Valentine 2011).

Considering the likelihood for continued dominance of fossil fuels in the years to come, an additional concept to renewable energy is to continue to burn fossil fuels, but abate resulting CO<sub>2</sub> emissions in the same way that technological fixes were devised in the past to abate SO<sub>2</sub>, NO<sub>x</sub> and PM emissions. This is termed carbon capture and storage (CCS—which may relate to post combustion, pre combustion or oxy-fuel technology), and is predicted to play an important role in GHG mitigation in addition to other transitional pathways. Use of CCS is not a sustainable GHG mitigation option in the long run; fossil fuels are a finite resource whose prices are now for the first time being predominantly driven by demand-side pressures not by supply-side manipulations (Valentine 2011). Nonetheless, by reducing CO<sub>2</sub> emissions from large (and current) industrial point sources, CCS is an enabling technology that will allow the continued use of fossil fuels for power generation and combustion in industrial processes, during the transition to cleaner renewable technologies. The International Energy Agency (IEA) CCS Technology Roadmap (IEA 2013) even goes so far as to note that CCS is the ‘only’ technology available to mitigate GHG emissions from large-scale fossil fuel usage in fuel transformation, industry and power generation. To achieve the 2 °C at least cost, the IEA considers that 4000 million tonnes per annum (Mtpa) CO<sub>2</sub> needs to be captured and stored in 2040, growing to around 6000 Mtpa in 2050 (IEA 2015). Similarly, CCS may be the only technology able to achieve an actual reduction in GHG emissions, and hold the global temperature rise to an aim of 1.5 °C, according to the COP21 agreement (EC 2015a).

Abatement costs of mitigation options were compared by McKinsey & Company (Enkvist et al. 2010), presenting the technical potential of emissions abatement per specific mitigation option and the associated cost per tCO<sub>2</sub>-eq saved, taking into account required up-front investment and associated operating costs. According to the cost curve (Fig. 9.4), increasing energy price expectations lead to abatement technologies being net profit positive. IPCC cost estimates for adaptation to climate change under a BAU scenario are between 1 and 5% of average global GDP. According to Enkvist et al. (2010), the abatement cost estimate across all sectors is



**Fig. 9.4** Global GHG abatement cost curve beyond BAU-2030. Taken from Enkvist et al. (2010). *Note:* The curve presents an estimate of the maximum potential of all technical GHG abatement measures below \$80 per tCO<sub>2</sub>-eq if each lever was pursued aggressively. It is not a forecast of what role different abatement measures and technologies will play

\$6 per tCO<sub>2</sub>-eq (excluding transportation), meaning mitigation would come at a net profit to society, representing a lower societal cost than estimated for adaptation. When specifically comparing the GHG mitigation costs of different low carbon technologies, CCS was found to be a part of the lowest cost mitigation options. Despite its high capital costs, the IEA (2013) finds that without it, overall costs to halve GHG emissions by 2050 rise by 70%. Once relatively low-cost technologies (such as various renewable options) are fully exploited—because of limits in their availability, or in countries where these technologies are not an option- CCS becomes a competitive option.

To directly compare the overall competitiveness of various electricity generating technologies, the levelised cost of electricity (LCOE) is often used to normalise figures. This represents the per kW cost in real \$ of building and operating a generating plant over an assumed financial life and duty cycle, divided to equal annual payments, and reflects all costs, building time as well as expected lifetime. Table 9.4 gives the LCOE for non-renewable and fossil fuel options in new power plants, based on U.S. Energy Information Administration statistics (U.S. EIA 2015). The values do not include any government or state incentives, meaning the actual cost to society is reflected. At present, natural gas, geothermal and coal are the most economic electricity generating technologies, although the cost of other sources are falling as technology improves (IRENA 2015b). Solar PV module costs have fallen by as much as 80% since 2009 (IRENA 2015b), and wind turbine prices have fallen by almost a third since 2009 (IRENA 2015b). Whilst LCOE is a convenient summary of the overall competitiveness, actual plant investment decisions are affected

**Table 9.4** Cost comparison of electricity generating technologies. Adapted from the U.S. EIA, Annual Energy Outlook (2015)

Energy source	Plant type		Range for total system LCOE (\$ <sub>2013</sub> per MWh)		
			Minimum	Average	Maximum
Fossil fuels	Coal	Conventional	87.1	95.1	119.0
		Advanced	106.1	115.7	136.1
		Advanced with CCS	132.9	144.4	160.4
	Natural gas	Conventional combined cycle	70.4	75.2	85.5
		Advanced combined cycle	68.6	72.6	81.7
		Advanced with CCS	93.3	100.2	110.8
	Nuclear	Advanced	91.8	95.2	101.0
Renewable	Geothermal		43.8	47.8	52.1
	Biomass		90.0	100.5	117.4
	Wind	Onshore	65.6	73.6	81.6
		Offshore	169.5	196.9	269.8
	Solar	PV	97.8	125.3	193.3
		Thermal	174.4	239.7	382.5
	Hydroelectric		69.3	83.5	107.2

*Note:* The values for each source are given for a different capacity factor

by the specific technological and regional characteristics of a project, which are influenced by other technological and political factors (U.S. EIA 2015).

In reality, it is likely that attaining a low carbon economy must occur through a combination of transitional pathways. In one study focusing upon Japan, China and India by Okagawa et al. (2012), the authors found appropriate energy strategies differ between regions due to the uneven pre-existing nuclear energy, CCS potential and renewable energy potential, resource endowments and levels of economic development. They found that mitigation targets could be achieved if nuclear energy and CCS are not available, but that significant enhancement of renewable energy is needed in such a case. Riahi et al. (2007), found the three top ranked mitigation options to be deployment of biomass, nuclear and efficiency improvements, whilst large-scale CCS should be used on a large scale for unfavourable scenario baselines (e.g. the coal-intensive scenario A2, see Fig. 9.1) or in combination with stringent stabilisation targets. Nevertheless, according to the IEA nine of ten technologies that hold potential for energy and CO<sub>2</sub> emissions savings are failing to meet deployment objectives needed to transition to a low-carbon future (IEA 2012). Irrespective of mitigation pathway taken, the model by McKinsey & Company suggests that delaying abatement action by 10 years would half abatement potential to 19 GtCO<sub>2</sub> (Enkvist et al. 2010). The emissions trajectory would then exceed a 550 ppm stabilisation pathway as laid out by the IPCC, making it challenging to limit global warming to within 3 °C.

## 9.4 Case Study of CCS as a Climate Change Mitigation Technology

Despite the potential of CCS to mitigate climate change, globally there are only 15 large scale CCS projects in operation (see Table 9.5), with a further seven under construction (Global CCS Institute 2015). This represents a doubling since the start of the decade, with the total CO<sub>2</sub> capture of these 22 projects around 40 Mtpa (Global CCS Institute 2015). However, according to modelling by the IEA (2015), to limit the temperature rise to 2 °C at least cost, 4,000 Mtpa needs to be captured and stored in 2040, growing to around 6,000 Mtpa in 2050. Thus, the number of projects presently in operation is required to grow to thousands by the middle of the century.

Although commercialising CCS is not a technical challenge, policy and regulatory enhancements are key to incentivising in CCS. A major challenge is the high operational cost involved, despite the IEA indicating that CO<sub>2</sub> reduction costs are significantly increased without it (IEA 2012). There are also concerns that public opinion (dominantly affected by leakage fears) will affect the wide-spread implementation of CCS, and the technology appears imposed to some communities (Bachu 2008; Minh and Loisel 2011). Development of CCS as a low carbon technology requires a 'next generation' policy framework, that recognises the multitudes of risks and political challenges, along with the policy mechanisms needed to address them. Current CCS policies are limited; in Europe, the main piece of regulation for CCS is the EU CCS Directive on Geological Storage of Carbon Dioxide (Directive 2009/31/EC). As an enabling Directive, this means that CCS is not required to be developed, but establishes a legal framework for the environmentally safe geological storage of CO<sub>2</sub> for member states to follow. In the U.S., policy is somewhat mixed with regard to CCS; policy and regulations for onshore CCS in brine formations are set by the EPA, but are less certain for CCS with EOR. Overall, these barriers are reflected in the fact that since 2007, total CCS investment has been less than \$20 billion, compared to around 100 times that amount for renewable energies over the same timeframe.

Resolution of challenges (improving the opinion of the public and policy makers) can be achieved in part through full understanding of the risks and impacts resulting from the CCS process. Despite the fact that CCS is a relatively new technology and there is little experience on which to base failure rates and uncertainty levels (Wilday et al. 2011a), analysing the various risks and achieving a holistic view of the total impacts is crucial for further technology development. Aside from the global climate change effect of CO<sub>2</sub> returning to the atmosphere, local risks to health and safety and the environment resulting from use of CCS need to be properly assessed over a range of time scales. In addition to short term potential effects (from injection and storage), long term risks from storage must also be considered (Wilday et al. 2011a). The benefits of the CCS process (reduction of CO<sub>2</sub> emissions) may then be weighed up against the negative impacts in a holistic view of total impact, which is understandable for all.

**Table 9.5** Operational large scale CCS projects, as of 2015. Adapted from the Global CCS Institute (2015)

Project name	Location	Operation date	Industry	Capture type	Capture capacity (Mtpa)	Transport type	Storage type
Air Products Steam Methane Reformer EOR Project	U.S.	2013	Hydrogen production	Industrial separation	1.0	Pipeline	EOR
Boundary Dam Carbon Capture And Storage Project	Canada	2014	Power generation	Post-combustion capture	1.0	Pipeline	EOR
Century plant	U.S.	2010	Natural gas processing	Pre-combustion capture	8.4	Pipeline	EOR
Coffeyville Gasification Plant	U.S.	2013	Fertiliser production	Industrial separation	1.0	Pipeline	EOR
Enid Fertilizer CO <sub>2</sub> -EOR Project	U.S.	1982	Fertiliser production	Industrial separation	0.7	Pipeline	EOR
Great Plains Synfuel Plant and Weyburn-Midale Project	Canada	2000	Synthetic natural gas	Pre-combustion capture	3.0	Pipeline	EOR
In Salah CO <sub>2</sub> Storage	Algeria	2004	Natural gas processing	Pre-combustion capture	0.0 (injection suspended)	Pipeline	Dedicated geological storage
Lost Cabin Gas Plant	U.S.	2013	Natural gas processing	Pre-combustion capture	0.9	Pipeline	EOR

Petrobras Lula Oil Field CCS Project	Brazil	2013	Natural gas processing	Pre-combustion capture	0.7	Direct injection	EOB
Quest	Canada	2015	Hydrogen production	Industrial separation	1.0	Pipeline	Dedicated geological storage
Shute Creek Gas Processing Facility	U.S.	1986	Natural gas processing	Pre-combustion capture	7.0	Pipeline	EOB
Sleipner CO <sub>2</sub> Storage Project	Norway	1996	Natural gas processing	Pre-combustion capture	0.9	Direct injection	EOB
Snohvit CO <sub>2</sub> Storage Project	Norway	2008	Natural gas processing	Pre-combustion capture	0.7	Pipeline	Dedicated geological storage
Uthmaniyah CO <sub>2</sub> EOR Demonstration Project	Saudi Arabia	2015	Natural gas processing	Pre-combustion capture	0.8	Pipeline	EOB
Val Verde Natural Gas Plants	U.S.	1972	Natural gas processing	Pre-combustion capture	1.3	Pipeline	EOB

Note: EOB enhanced oil recovery

### 9.4.1 *Loss of Containment of CO<sub>2</sub> and Impacts to Human and Environmental Health*

Although CCS risks can be found all along the CCS chain, the main risk is the potential release of large quantities of CO<sub>2</sub> from surface installations and injection (Wilday et al. 2011a; Minh and Loisel 2011). CO<sub>2</sub> release may occur due to leakages (particularly in pipelines and storage facilities), or due to venting, required at various points of the CCS chain for pipeline depressurisation or operational reasons (Wilday et al. 2011b). Models for the release (and dispersion) of CO<sub>2</sub> have been developed in many studies, and can typically be divided into Gaussian/dense-gas or computational fluid dynamics (CFD) model types (Koornneef et al. 2012).

Captured CO<sub>2</sub> can be transported through a pipeline or in a ship. Based on an optimisation tool minimising transport cost, Morbee et al. (2010) estimated that 17,859 km pipeline will be required in Europe in 2050, along with 2,515 km of shipping routes, whilst Neele et al. (2011) found between 21,800 and 32,000 km of backbone CO<sub>2</sub> pipelines may be required, depending upon the scenario. Minh and Loisel (2011) predicted that 10% of the total CO<sub>2</sub> captured in 2050 would be transported by ships, reaching 450 MtCO<sub>2</sub> in 2050. Irrespective of transport method, leakage of CO<sub>2</sub> can occur due to corrosion of carbon steel pipelines or equipment, as a result of CO<sub>2</sub> content forming an acidic solution (Wilday et al. 2011b, a), or metal becoming brittle and fracturing (Mahgerefteh and Atti 2006; Wilday et al. 2011b). For shipping, when a leak is detected a jettisoning system must be activated (Han and Chang 2014), causing large CO<sub>2</sub> emissions around the ship.

Leakages are also expected from injection into storage sites. The IPCC currently estimates that flows from 1 to 2.2 MtCO<sub>2</sub> per year per well can be sustained, but in future Minh and Loisel (2011) estimate that the average storage scale will increase from 1 MtCO<sub>2</sub> per year to 8.8 MtCO<sub>2</sub> per year per site by 2050. The major leakage risk with injection is thought to be well failure due to the high pressures (Minh and Loisel 2011; Bachu 2008), with the estimated frequency of blowout per well per year estimated as  $1 \times 10^{-4}$ – $3 \times 10^{-4}$ . After injection, the security of CO<sub>2</sub> storage increases due to lower pressures and alternative CO<sub>2</sub> trapping mechanisms (Bachu 2008), but other risks for CO<sub>2</sub> leakage relate to the storage period itself. CO<sub>2</sub> buoyancy means that CO<sub>2</sub> will flow up and out if pathways are available, or if the well is poorly plugged (Wilday et al. 2011a; Bachu 2008). There is also a lack of knowledge of the nature and concentration of impurities that will be injected into the reservoir with the CO<sub>2</sub>, which can have important effects on hazards (Wilday et al. 2011a). Small quantities of H<sub>2</sub>S and SO<sub>2</sub>, as well as small quantities of non-condensable components (e.g. N, O, H) may be present in the CO<sub>2</sub>.

Although CCS is already performed (predominantly for EOR), due to the large proposed quantities and flow rates of CO<sub>2</sub> for CCS in the future, risks to human health are exacerbated (Minh and Loisel 2011). Consequences may be global or local, short or long term, and relate to health and safety and/or the environment. Short-term consequences of CO<sub>2</sub> leakage are those that pose an acute danger to life

(Bachu 2008). At atmospheric concentrations (0.038 %), CO<sub>2</sub> is not classed as toxic and is fundamental to photosynthetic and respiratory processes (Bachu 2008); but in high concentrations it is an asphyxiant, displacing oxygen in the air to below the 16% level required to sustain human life (hypoxia) (Wilday et al. 2011b, a; Bachu 2008). In addition, when inhaled at elevated concentrations CO<sub>2</sub> can increase the acidity of blood, triggering adverse effects on the respiratory, cardiovascular and central nervous systems (Wilday et al. 2011b, a; Bachu 2008). This has caused governing bodies to set both major hazards toxic dose criteria and offshore impairment criteria (Wilday et al. 2011a). For example, the UK occupational exposure limit is 0.5% for an 8 h time-weighted average, with a short-term exposure limit (STEL) of 1.5% for 15 min (Wilday et al. 2011b). As CO<sub>2</sub> is heavier than air, power stations and other buildings constructed with tunnels running beneath them will be vulnerable to CO<sub>2</sub> accumulation (Wilday et al. 2011b; de Lary et al. 2012). CO<sub>2</sub> can also have an indirect short-term effect on human health, because of its physical properties; for example, from explosive decompression (Reid 1979), cold burns and risk of ignition.

Long-term consequences of CO<sub>2</sub> leakage are generally those that effect ecosystems (Bachu 2008). CO<sub>2</sub> may dissolve in brine, react with rock, and remobilise trace metals or other problematic compounds (Wilday et al. 2011a). Dissolved CO<sub>2</sub> forms a weak carbonic acid, altering water pH (Bachu 2008; Carroll et al. 2014). Changes to pH of surrounding soil is likely to adversely affect the nutrient chemistry, redox-sensitive elements and trace metals, and plant growth (Bachu 2008). Water pH changes also induce geochemical reactions in shallow aquifers, potentially mobilising U.S. EPA regulated metal contaminants such as Pb and Hg (Atchley et al. 2013; Bachu 2008; Wang and Jaffe 2004) with associated human health and environmental risks (Siirila et al. 2012; Little and Jackson 2010). CO<sub>2</sub> leaking at the sea floor may affect the sediment biota by dissolving in seawater, locally affecting water pH and consequently marine life (Bachu 2008). Halsband and Kurihara (2013) describe how little studied meso- and bathy-pelagic species of the deep sea may be especially vulnerable to pH changes, as well as vertically migrating zooplankton, which require significant residence times at great depth as part of their life cycle. These represent a crucial link between primary producers and higher trophic levels such as fish, seabirds and marine mammals, and are important contributors to the biological pump (Halsband and Kurihara 2013). As such, consequences higher up the food chain need to be addressed.

Leakages from pipelines and storage failures may lead to relatively large flows of CO<sub>2</sub> but these are usually short lived, and based on statistics of underground gas storage, the frequency is likely to be low (Bachu 2008). Nonetheless, according to Bachu (2008), whilst the risk associated with any individual storage operation is likely to be low, the large number of such operations that will be needed by 2050 to make the strategy viable for climate change mitigation will increase the overall risk. Thus, using the CO<sub>2</sub> as a product, rather than a waste that must be stored, should be considered as a viable alternative to storage to reduce the risks involved.



### 9.4.2 *Other CCS Emissions and Impacts to Human and Environmental Health*

Although CCS leads to an overall reduction on CO<sub>2</sub> emissions, other undesirable emissions are produced (both directly and indirectly). Additional direct emissions from CCS vary depending on the specific CCS technology employed (Wilday et al. 2011b); i.e. post combustion, pre combustion or oxy-fuel. A good overview of emissions relating to the various technologies is given by Koornneef et al. (2012). For post-combustion, key extra emissions relate to the solvents themselves (amines or equivalent). Whilst emission of NH<sub>3</sub> from pulverised coal (PC) plants are negligible (Tzanidakis et al. 2013), amine-based solvents such as mono ethanolamine (MEA) can be emitted by the stack along with their degradation products including volatile organic compounds (VOCs) and NH<sub>3</sub> (Koornneef et al. 2012). Secondary amines, such as diethyl amine (DEYA) are also released during CO<sub>2</sub> capture due to volatilisation from the scrubbing solvent or degradation pathways (Karl et al. 2011). These compounds can also be emitted to water, which according to Koornneef et al. (2012) is an under-researched subject. The amine emissions vary depending on operating conditions, the amine that is used, and whether a water wash is installed (Thitakamol et al. 2007). Without water wash, use of MEA as a scrubbing solvent can result in emissions of 0.1–0.8 kg MEA per tonne CO<sub>2</sub> captured, which are minimised to between 0.01–0.03 kg per tonne CO<sub>2</sub> captured with water wash (Thitakamol et al. 2007; Koornneef et al. 2008). To reflect operating conditions with typical and with elevated emissions, Karl et al. (2011) described a best case scenario with emissions of 80 t per year MEA and 5 t per year DEYA, and a worst case scenario with emissions of 80 t per year MEA and 15 t per year DEYA. Other flue impurities may be influenced by CCS; for example, flue gas NO<sub>x</sub> may be enriched for pre-combustion technology due to the combustion characteristics of hydrogen rich fuel requiring a DeNO<sub>x</sub> installation, which in turn results in NH<sub>3</sub> emissions (Koornneef et al. 2012). In contrast, flue gas recycling (FGR) in oxy-fuel technology leads to a reduction of NO<sub>x</sub> formation during combustion (Koornneef et al. 2012).

In addition, due to the fact that CCS is an energy intensive technology, a reduction in the overall process efficiency occurs compared to a non-CCS unit (Table 9.6). Thus, if installed in a power station, CCS implies more fuel must be mined and combusted to produce the same output; e.g. Minh and Loisel (2011) found that 4.5 GtCO<sub>2</sub> captured translates to about 2.1 Gt extra coal. Thus, although direct CO<sub>2</sub> emissions from the plant itself are drastically decreased when CCS is fitted, other combustion products and upstream (indirect) emissions from fuel and material consumption are increased, as well as downstream emissions from waste processing (Tzanidakis et al. 2013). For the latter, as well as the extra waste relating to extra fuel combustion for the energy deficit, the composition of waste and by-products are also affected by CCS technology. In post-combustion technology, impurities in the flue gas react with amine-based solvents to form salts which are later separated from the solvent in a reclaiming and disposed of as sludge (Koornneef et al. 2012), influencing the distribution of trace elements (such as Hg) in the waste stream

**Table 9.6** Simplified overview of energy conversion and CO<sub>2</sub> capture efficiencies of power plants equipped with various CO<sub>2</sub> capture technologies. Adapted from Koornneef et al. (2012)

Capture process	Conversion technology	Generating efficiency (%)	Energy penalty of CO <sub>2</sub> capture (%)	Capture efficiency (%)
Post combustion	PC	30–40	8–13	85–90
	NGCC	43–55	5–12	85–90
Oxyfuel	PC	33–36	9–12	90–100
	GC and NGCC	39–62	2–19	50–100
Pre combustion	IGCC	32–44	5–9	85–90
	GC	43–53	5–13	85–100

*Note:* PC pulverised coal, NGCC natural gas combined cycle, GC gas cycle, IGCC integrated gasification combined cycle. Efficiencies are reported based on Lower Heating Values (LHV), assuming CO<sub>2</sub> product pressure of 11 MPa

(Thitakamol et al. 2007). Distribution of Hg is also influenced in oxyfuel combustion due to speciation variations; Davidson et al. (2003) suggest that the oxidised Hg produced here is more easily captured in flue gas control technologies, and would end up in waste streams rather than in the emitted flue gas. The energy demand and demands by CO<sub>2</sub> capture system also means extra water must be consumed (Koornneef et al. 2012).

Many studies have attempted to quantify overall additional power plant emissions, and detailed reviews are available. For example, Tzimas et al. (2007) provided insight regarding the potential of trade-offs between increased emissions of acid pollutants and decreased CO<sub>2</sub> emissions when CCS is implemented, suggesting that NO<sub>x</sub> emissions may be amplified across the power generation sector irrespective of technology type. In addition, for post combustion capture, deposition due to NH<sub>3</sub> emissions from the capture process increases considerably, and Tzanidakis et al. (2013) found that a potential post capture deployment of CCS will only be beneficial in relation to deposition due to SO<sub>2</sub> emissions. This was also demonstrated by Koornneef et al. (2010, 2012).

In terms of studies investigating the impact of these additional emissions to human health and the environment, amines have received the most focus in recent years, possibly because post-combustion CCS technology is the most advanced. Although the toxicity of MEA is well documented, and exposure guidelines are set, research towards chronic exposure effects and other toxicity end-points is lacking (Koornneef et al. 2012). Additionally MEA forms other compounds in the atmosphere whose effects are lesser quantified; A potential concern is the formation of carcinogenic nitrosamines, nitramines and amides that are products of the sunlight-induced reaction of ethanolamines and atmospheric oxidants such as NO<sub>x</sub> (Koornneef et al. 2012). In 2007, the Norwegian Institute for Air Research (NILU) initiated a project to study the effects of amine emissions to the environment, including MEA, AMP, MDEA and piperazine (in Koornneef et al. 2012). In addition to the effects to human health, damage to ecosystems can occur due to eutrophication and acidification from NH<sub>3</sub> release (Tzanidakis et al. 2013). In a study by Karl et al. (2011), the

**Table 9.7** Overview of compounds, exposure paths and associated toxicity. Adapted from Karl et al. (2011)

Compound	Exposure route	Toxicity
MEA	Inhalation	Human health, subchronic
	Aquatic environment	Algae/bacteria, chronic
DEYA	Inhalation	Human health, acute
	Aquatic environment	Algae/bacteria, chronic
Nitrosamines	Inhalation	Human health, carcinogenic
	Drinking water	Human health, carcinogenic
	Aquatic environment	Algae/bacteria, chronic
Nitramines	Drinking water	Human health, carcinogenic
	Aquatic environment	Fish, chronic
Formamide	Aquatic environment	Invertebrate, chronic
Acetamide	Inhalation	Human health, carcinogenic

authors found that maximum expected MEA deposition fluxes would exceed toxicity limits for aquatic organisms by about a factor of 3–7, depending on the scenario. Due to the formation of nitrosamines and nitramines, the estimated emissions of DEYA would be expected to be close to or exceed safety limits for drinking water and aquatic emissions (Karl et al. 2011). An overview of these compounds and their effects to human health and the environment is given in Table 9.7, adapted from Karl et al. (2011), and more details of the effects of nitrosamines and human health can be found in other studies (Ravnum et al. 2014; Zhang et al. 2014).

Other risks to human health include the exposure of the population to fine particulates (Hou et al. 2015; Lippmann 2014); primary PM<sub>10</sub>, PM<sub>2.5</sub> and secondary inorganic aerosols (SIA) (Schlesinger and Cassee 2003; Reiss et al. 2007). There is particularly a concern over human health regarding increased urban NO<sub>x</sub> concentrations (Tzanidakis et al. 2013; Bostrom et al. 1994). Additionally, risks posed by other gases produced by IGCC and oxy-fuel processes—oxygen and nitrogen—need to be considered with respect to their effects on human health (Wilday et al. 2011b). Nitrogen is produced in large quantities by air separation units, and is mixed with hydrogen for combustion in IGCC processes, representing an asphyxiation hazard. In addition, oxygen enhances combustion and IGCC processes will also produce CO<sub>2</sub> contaminated with H<sub>2</sub>S, which can react to produce iron sulphide—a pyrophoric substance (Wilday et al. 2011b).

### 9.4.3 Introduction to Methods for Assessing Impacts

Impacts to human health and the environment from any technology can be quantified using risk assessment or life cycle assessment (LCA). These techniques are complementary, with risk assessment generally providing an in-depth quantitative assessment of the human/environmental response to one particular toxicant, and

LCA providing a more general holistic assessment of the health/environmental impact relating to direct plus indirect emissions over the whole life cycle. A thorough assessment of impacts may also need to consider different time scales; for the case of CCS, some risks concern only the exploitation phase, whilst others may concern longer periods (e.g. storage) (Wilday et al. 2011a; Farret et al. 2010).

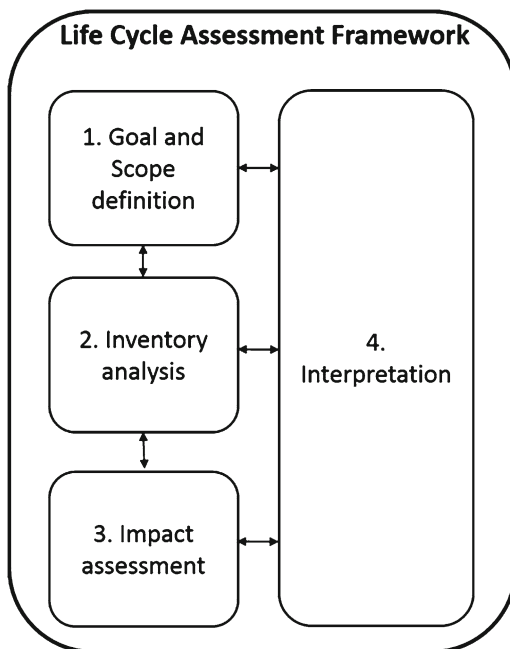
A quantitative risk assessment can theoretically be performed for any particular activity based on the product of probability and consequence. For example, the increased human health risk associated with groundwater contamination from potential CO<sub>2</sub> leakage into a potable aquifer was predicted by conducting a joint uncertainty and variability (JUV) risk assessment by Atchley et al. (2013). Although the estimated risk to human health from CO<sub>2</sub> leakages to potable groundwater was low, this study only considered one metal (Pb). Using a similar approach, Siirila et al. (2012) corroborated this and also found little cancer risk relating to Pb metal contamination. However, when the authors studied arsenic (As) contamination, the risk for cancer was orders of magnitude higher than for Pb. Carroll et al. (2014) also performed a risk assessment to study the potential impacts to groundwater quality due to CO<sub>2</sub> and brine leakage, and found that pH and total dissolved solid (TDS) levels would be likely to go beyond the ‘no impact thresholds’, although the risk to human health may not be significant for the simulations in the study. However, it is difficult to develop abatement strategies based on traditional risk assessments, as in general, only direct emissions from one stage of the CCS process are considered, and indirect emissions that are only evident from a life cycle perspective are often neglected or added as an afterthought.

The LCA method allows the full life-cycle cost of CCS ‘from cradle to grave’ to be considered in the context of the overall social, environmental and economic benefits, and inherent trade-offs relating to a range of factors determined (Hardisty et al. 2011; Tzanidakis et al. 2013). Variations in the scope of the assessment are varied, and can be on a wide scale or from a technical perspective on individual cases. In addition, a LCA may be consequential (i.e. comparing different technology options) or attributional (i.e. comparing specific options within a technology).

As part of the four stages of the LCA (which should adhere to ISO 14040:2006 International Standards), a goal and scope are set, life cycle emissions are inventoried (using an input/output approach), impacts are studied in a life cycle impact assessment (LCIA) and the LCA is interpreted. See Fig. 9.5. The range of impact categories can be ‘mid’ or ‘end’ point and are predominantly environmental e.g. climate change potential, biodiversity, energy usage, although human health can be considered in terms of ‘years of life lost’ (YOLL) or ‘daily adjusted life years’ (DALY). Weighting of factors and the method chosen can have a large effect on the LCIA ‘outcome’ in terms of environmental performance (Wilday et al. 2011a; Breedveld et al. 2010). Although less quantitative than a risk assessment in terms of specific effects to human and environmental health, the benefit of a LCA is that an overall, holistic view is gained.

Many studies have used LCA to compare the risks/benefits of using CCS to the base line case where no CCS is operational, and to compare different CCS technologies to each other. This type of analysis suggests that not all types of CCS are

**Fig. 9.5** Life cycle assessment framework



created equal in terms of their environmental impact (Hardisty et al. 2011); overall, GHG emissions leading to climate change are largely reduced by CCS, but other impact categories are less clear (Wilday et al. 2011a). Some studies find that additional life cycle emissions by up- and downstream process may result in a deterioration of the overall environmental performance compared to a power plant without CCS (Koorneef et al. 2012). A study by Singh et al. (2012) aimed to evaluate the impacts of the IEA scenarios from a global perspective, with results identifying generally positive co-benefits in relation to acidification and human health, with trade-offs relating to eutrophication due to  $\text{NH}_3$  emissions. Tzanidakis et al. (2013) commented that in relation to ecosystem impacts, an increase in  $\text{NH}_3$  emissions has the potential to impact significantly upon other policy targets such as National Emission Ceilings and the UNECE Gothenburg protocol, which aims to abate acidification, eutrophication and ground level  $\text{O}_3$ . Currently, 42 % of European terrestrial ecosystems are projected to remain at risk of nitrogen eutrophication in 2020, with consequent biodiversity loss (Posch et al. 2011). A general increase in water consumption has also been estimated with use of CCS, with an increase in water consumption of between 32 and 93 % for a 1 GWe power plant with CCS (Koorneef et al. 2012).

In terms of human health, when population exposure to total  $\text{PM}_{2.5}$  was used by Tzanidakis et al. (2013) as a proxy to calculate YOLL and DALY parameters, the impacts on human health appeared to be higher for the ‘no action’ scenario in which

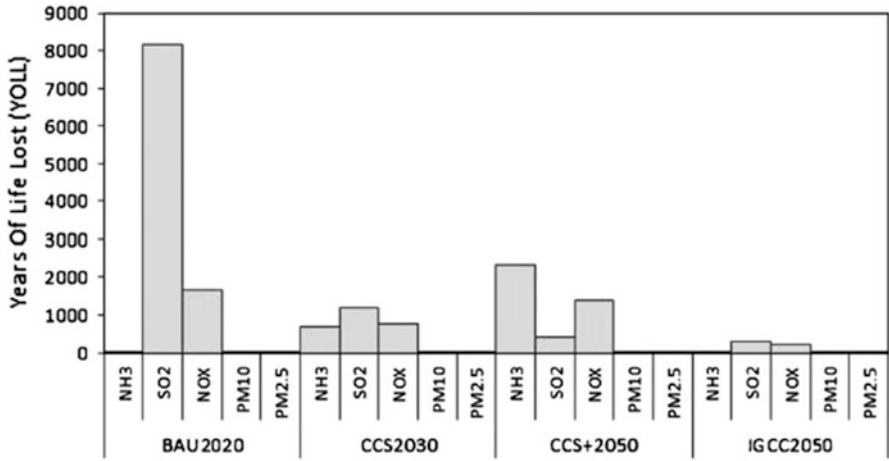


Fig. 9.6 Years-Of-Life-Lost (YOLL) attributable to BAU2020, CCS2030, CCS+2050 and IGCC2050 scenarios. Taken from Tzanidakis et al. (2013)

all plants are operational and without CCS (BAU2020) than for the ‘realistic’ potential development scenario (CCS2030) (Fig. 9.6). Total YOLLs are dominated by the power plant, with YOLLs attributed to other sectors being an order of magnitude less than the power plant. This finding was also corroborated by other authors (Singh et al. 2012). Exact emission flows and composition are uncertain, and trade-offs depend on the applied CCS technology in question.

When individuals cannot estimate the uncertainty of consequences from a new technology option they tend to build the worst potential scenario, denying or over-estimating potential risk (Slovic 1986). Although science based risk assessment should not be used as a direct guide for action, it is thus a beneficial instrument to avoid influence on policy making. For CCS, as with other technologies, a fair comparison requires evaluation of the consequences across the whole ‘life cycle’, from raw material extraction to waste disposal over all time spans, as focussing on one stage is often misleading (Matthews et al. 2002).

### 9.5 Summary

Although there is always a degree of uncertainty with understanding the relationship between physical processes, impact and climate models, evidence shows that significant changes are arising for many aspects of human and natural systems, and that GHG emissions from human activity are causing climate change. If no mitigation action is taken, it is predicted that global average surface temperatures will rise by between 1 and 6 °C by the end of the century. The IPCC has predicted that

if temperatures rise beyond 2 °C from pre-industrial levels, significant impacts will occur to human health and the environment, from which we may not recover. This is reflected by the COP21 Agreement which aims to keep the global rise in temperature (from pre-industrial levels) below 1.5 °C.

Effects to human health resulting from global warming may be direct (e.g. mortality from heatwaves and other extreme weather) or secondary (e.g. effects to agriculture and water resources). The virtual certainty is that climate change will have its most severe effects on the same poor, mostly tropical countries that are currently focused upon for global health efforts, and actions must be taken now to mitigate this outcome. Mitigating GHG emissions will not only avoid the health impacts resulting from climate change (both direct and secondary effects), but also convey co-benefits resulting from increased air quality. Pollutants such as PM, SO<sub>2</sub> and NO<sub>x</sub> are produced from many of the same processes as GHGs (mainly the combustion of fossil fuels), and are linked to a variety of respiratory and cardiovascular diseases. Climate change mitigation strategies may therefore also improve public health, especially in urban areas, and policies are thus highly connected.

Mitigation strategies may be in the form of a technological option (increasing the efficiency of current technology, or by utilising low carbon technology) or a social (change in behaviour) or economic solution. It is almost certain that a combination of these strategies must be adopted to reach mitigation targets. In addition, irrespective of GHG mitigation pathway, policy will prove central for success. Policies must be made based both on evidence and economic, technological and social feasibility, as well as potential health co-benefits. The share of low carbon energy is increasing in the energy market, but challenges still remain to break the hold of fossil fuels on the industry.

CCS represents an option that could mitigate GHG emissions during the transition to a lower carbon economy, allowing the continued use of fossil fuels. But despite these benefits, insufficient policy and doubts in public opinion represent significant barriers to its development. Understanding the risks and total impact of the technology, in terms of human and environmental health, may help allay public and policy-maker fears, thereby increasing CCS desirability. With further implementation of policies, economics may be improved (lower financial risk) of CCS, and technology deployment accelerated (Bachu 2008). LCA provides an overall framework for identifying and evaluating life cycle implications of any technology or mitigation option. However, it requires the support of risk assessment to go beyond simplistic assumptions about the implications of a discharge inventory, and both disciplines should be closely linked (see Matthews et al. 2002 for a discussion).

Overall, although GHG mitigation is progressing, progress is too slow. A model published by McKinsey & Company (Enkvist et al. 2010) suggests that delaying abatement action by 10 years would half abatement potential to 19 GtCO<sub>2</sub>, making it challenging to mitigate global warming to target levels. Thus, it is evident that more action must be taken now to prevent the major effects of climate change to human health and the environment in the coming years.

## References

- Ainsworth EA, McGrath JM (2010) Direct effects of rising atmospheric carbon dioxide and ozone on crop yields. In: Lobell D, Burke M (eds) *Climate change and food security: adapting agriculture to a warmer world*. Springer, New York
- Arnell NW, Van Vuuren DP, Isaac M (2011) The implications of climate policy for the impacts of climate change on global water resources. *Glob Environ Chang* 21:592–603
- Atchley AL, Maxwell RM, Navarre-Sitchler AK (2013) Human health risk assessment of CO<sub>2</sub> leakage into overlying aquifers using a stochastic, geochemical reactive transport approach. *Environ Sci Technol* 47:5954–5962
- Bachu S (2008) CO<sub>2</sub> storage in geological media: role, means, status and barriers to deployment. *Prog Energy Combust Sci* 34:254–273
- Beg N, Morlot JC, Davidson O, Afrane-Okesse Y, Tyani L, Denton F, Sokona Y, Thomas JP, La Rovere EL, Parikh JK, Parikh K, Rahman AA (2002) Linkages between climate change and sustainable development. *Clim Pol* 2:129–144
- Bostrom CE, Almen J, Steen B, Westerholm R (1994) Human exposure to urban air-pollution. *Environ Health Perspect* 102:39–47
- Breedveld L, Paltrinieri N, Wilday J, Farret R (2010) Application of LCA to carbon capture and storage, iNTeg-risk deliverable. D1.2.1.4.2B, Italy
- Carroll SA, Keating E, Mansoor K, Dai Z, Sun Y, Trainor-Guitton W, Brown C, Bacon D (2014) Key factors for determining groundwater impacts due to leakage from geologic carbon sequestration reservoirs. *Int J Greenhouse Gas Control* 29:153–168
- Chang SJ (2013) Solving the problem of carbon dioxide emissions. *Forest Policy Econ* 35:92–97
- Costa S, Ferreira J, Silveira C, Costa C, Lopes D, Relvas H, Borrego C, Roebeling P, Miranda AI, Teixeira JP (2014) Integrating health on air quality assessment-review report on health risks of two major European outdoor air pollutants: PM and NO<sub>2</sub>. *J Toxicol Environ Health B Crit Rev* 17:307–340
- Dasgupta S, Laplante B, Meisner C, Wheeler D, Yan J (2009) The impact of sea level rise on developing countries: a comparative analysis. *Clim Change* 93:379–388
- Davidson RM, Reeve DA, Sloss LL, Smith IM (2003) Trace elements—occurrence, emissions and control. IEA Clean Coal Centre, London
- de Lary L, Loschetter A, Bouc O, Rohmer J, Oldenburg CM (2012) Assessing health impacts of CO<sub>2</sub> leakage from a geological storage site into buildings: role of attenuation in the unsaturated zone and building foundation. *Int J Greenhouse Gas Control* 9:322–333
- Demirbas A (2006) Global renewable energy resources. *Energy Sources, Part A* 28:779–792
- Ebi KL, McGregor G (2008) Climate change, tropospheric ozone and particulate matter, and health impacts. *Environ Health Perspect* 116:1449–1455
- EC (European Commission) (2009) Directive 2009/28/EC of the European Parliament and of the Council of 23 April 2009 on the promotion of the use of energy from renewable sources and amending and subsequently repealing Directives 2001/77/EC and 2003/30. *Off J Eur Union* 5:5
- EC (European Commission) (2012) Directive 2012/27/EU of the European Parliament and of the Council of 25 October 2012 on energy efficiency, amending Directives 2009/125/EC and 2010/30/EU and repealing Directives 2004/8/EC and 2006/32/EC. European Commission, Brussels
- EC (European Commission) (2014) Europe 2020 indicators—climate change and energy. European Commission, Brussels. [http://ec.europa.eu/eurostat/statistics-explained/index.php/Europe\\_2020\\_indicators\\_-\\_climate\\_change\\_and\\_energy](http://ec.europa.eu/eurostat/statistics-explained/index.php/Europe_2020_indicators_-_climate_change_and_energy). Accessed 25 Aug 2015
- EC (European Commission) (2015a) Paris agreement. European Commission, Brussels. [http://ec.europa.eu/clima/policies/international/negotiations/future/index\\_en.html](http://ec.europa.eu/clima/policies/international/negotiations/future/index_en.html). Accessed 10 Jan 2016
- EC (European Commission) (2015b) Report from the commission to the European Parliament, the council, the European economic and social committee and the committee of the regions: renewable energy progress report. European Commission, Brussels



- ECOFYS (2014a) Subsidies and costs of EU energy. European Commission, Brussels
- ECOFYS (2014b) Renewable energy progress and biofuels sustainability. European Commission, Brussels
- Enkvist P-A, Dinkel J, Lin C (2010) Impact of the financial crisis on carbon economics: version 2.1 of the global greenhouse gas abatement cost curve. McKinsey
- Farret R, Hebrard J, Gombert P, Lahaie F (2010) Risk assessment CCS technology, iNTeg-risk deliverable D1.2.1.3. INERIS, Verneuil-en-Halatte
- Global CCS Institute (2015) Large scale CCS projects. Global CCS Institute, Melbourne, VIC, <https://www.globalccsinstitute.com/projects/large-scale-ccs-projects>. Accessed 23 Dec 2015
- Gosling SN, Arnell NW (2011) Simulating current global river runoff with a global hydrological model: model revisions, validation, and sensitivity analysis. *Hydrol Process* 25:1129–1145
- Gosling SN, Warren R, Arnell NW, Good P, Caesar J, Bernie D, Lowe JA, van der Linden P, O'Hanley JR, Smith SM (2011) A review of recent developments in climate change science. Part II: The global-scale impacts of climate change. *Prog Phys Geogr* 35:443–464
- Granade HC, Creyts J, Derkach A, Farese P, Nyquist S, Ostrowski K (2009) Unlocking energy efficiency in the U.S. economy: executive summary. McKinsey
- Guerreiro C, de Leeuw F, Foltescu V, Horalek J (2014a) Air quality in Europe—2014 report. European Environment Agency, Denmark
- Guerreiro CBB, Foltescu V, de Leeuw F (2014b) Air quality status and trends in Europe. *Atmos Environ* 98:376–384
- Haines A (2012) Health benefits of a low carbon economy. *Public Health* 126:S33–S39
- Haines A, Kovats RS, Campbell-Lendrum D, Corvalan C (2006) Climate change and human health: impacts, vulnerability, and mitigation. *Lancet* 367:2101–9
- Haines A, McMichael AJ, Smith KR, Roberts I, Woodcock J, Markandya A, Armstrong BG, Campbell-Lendrum D, Dangour AD, Davies M, Bruce N, Tonne C, Barrett M, Wilkinson P (2009) Health and Climate Change 6. Public health benefits of strategies to reduce greenhouse-gas emissions: overview and implications for policy makers. *Lancet* 374:2104–2114
- Halsband C, Kurihara H (2013) Potential acidification impacts on zooplankton in CCS leakage scenarios. *Mar Pollut Bull* 73:495–503
- Han SH, Chang D (2014) Dispersion analysis of a massive CO<sub>2</sub> release from a CO<sub>2</sub> carrier. *Int J Greenhouse Gas Control* 21:72–81
- Hardisty PE, Sivapalan M, Brooks P (2011) The environmental and economic sustainability of carbon capture and storage. *Int J Environ Res Public Health* 8:1460–1477
- Hastik R, Basso S, Geitner C, Haida C, Poljanec A, Portaccio A, Vrscaj B, Walzer C (2015) Renewable energies and ecosystem service impacts. *Renew Sustain Energy Rev* 48:608–623
- Hayashi A, Akimoto K, Sano F, Mori S, Tomoda T (2010) Evaluation of global warming impacts for different levels of stabilization as a step toward determination of the long-term stabilization target. *Clim Change* 98:87–112
- Henschel S, Chan G (2013) Health risks of air pollution in Europe—HRAPIE project: new emerging risks to health from air pollution—results from the survey of experts. World Health Organization (WHO), Geneva
- Hou X, Strickland MJ, Liao K-J (2015) Contributions of regional air pollutant emissions to ozone and fine particulate matter-related mortalities in eastern US urban areas. *Environ Res* 137:475–484
- IEA (International Energy Agency) (2012) Energy technology perspectives 2012: pathways to a clean energy system. Executive summary. IEA, Paris
- IEA (International Energy Agency) (2013) Technology roadmap carbon capture and storage, 2013th edn. IEA, Paris
- IEA (International Energy Agency) (2015) Energy technology perspectives 2015: pathways to a clean energy system. Executive summary. IEA, Paris
- IPCC (Intergovernmental Panel on Climate Change) (2000) Special report on Emissions scenarios: summary for policymakers. Cambridge University Press, Cambridge
- IPCC (Intergovernmental Panel on Climate Change) (2006) 2006 IPCC Guidelines for National Greenhouse Gas Inventories, Hayama, Japan. Institute for Global Environmental Strategies (IGES)

- IPCC (Intergovernmental Panel on Climate Change) (2007a) *Climate Change 2007: the physical science basis. Contributions of working group I to the fourth assessment report of the intergovernmental panel on climate change*. Cambridge University Press, Cambridge
- IPCC (Intergovernmental Panel on Climate Change) (2007b) *Climate Change 2007: synthesis report: contribution of working groups I, II and III to the fourth assessment report of the Intergovernmental Panel on Climate Change*. In: Pachauri RK, Reisinger A (eds) *IPCC fourth assessment report (AR4)*. IPCC, Geneva
- IPCC (Intergovernmental Panel on Climate Change) (2007c) *Summary for policymakers*. In: Parry ML, Canziani OF, Palutikof JP, Vanderlinden PJ, Hanson CE (eds) *Climate Change 2007: impacts, adaptation and vulnerability. Contribution of working group II to the fourth assessment report of the Intergovernmental Panel on Climate Change*. Cambridge University Press, Cambridge
- IRENA (International Renewable Energy Agency) (2015a) *Rethinking energy: renewable energy and climate change*. IRENA
- IRENA (International Renewable Energy Agency) (2015b) *Renewable power generation costs in 2014*. IRENA
- Jäger J, Kok MTJ (2007) *Vulnerability of people and the environment: challenges and opportunities*. In: Thywissen K (ed) *Global environment outlook 4 (GEO-4): environment for development*. United Nations Environment Programme, Nairobi
- Karl M, Wright RF, Berglen TF, Denby B (2011) *Worst case scenario study to assess the environmental impact of amine emissions from a CO<sub>2</sub> capture plant*. *Int J Greenhouse Gas Control* 5:439–447
- Kjellstrom T, McMichael AJ (2013) *Climate change threats to population health and well-being: the imperative of protective solutions that will last*. *Glob Health Action* 6:1–9
- Koornneef J, van Keulen T, Faaij A, Turkenburg W (2008) *Life cycle assessment of a pulverized coal power plant with post-combustion capture, transport and storage of CO<sub>2</sub>*. *Int J Greenhouse Gas Control* 2:448–467
- Koornneef J, Ramirez A, van Harmelen T, van Horssen A, Turkenburg W, Faaij A (2010) *The impact of CO<sub>2</sub> capture in the power and heat sector on the emission of SO<sub>2</sub>, NO<sub>x</sub>, particulate matter, volatile organic compounds and NH<sub>3</sub> in the European Union*. *Atmos Environ* 44:1369–1385
- Koornneef J, Ramirez A, Turkenburg W, Faaij A (2012) *The environmental impact and risk assessment of CO<sub>2</sub> capture, transport and storage—an evaluation of the knowledge base*. *Prog Energy Combust Sci* 38:62–86
- Lau LC, Lee KT, Mohamed AR (2012) *Global warming mitigation and renewable energy policy development from the Kyoto Protocol to the Copenhagen Accord—A comment*. *Renew Sustain Energy Rev* 16:5280–5284
- Liao H, Cao H-S (2013) *How does carbon dioxide emission change with the economic development? Statistical experiences from 132 countries*. *Glob Environ Chang* 23:1073–1082
- Lippmann M (2014) *Toxicological and epidemiological studies of cardiovascular effects of ambient air fine particulate matter (PM<sub>2.5</sub>) and its chemical components: Coherence and public health implications*. *Crit Rev Toxicol* 44:299–347
- Little MG, Jackson RB (2010) *Potential impacts of leakage from deep CO<sub>2</sub> geosequestration on overlying freshwater aquifers*. *Environ Sci Technol* 44:9225–9232
- Louis MES, Hess JJ (2008) *Climate change impacts on and implications for global health*. *Am J Prev Med* 35:527–538
- Mahgerefteh H, Atti O (2006) *Modeling low-temperature-induced failure of pressurized pipelines*. *AIChE J* 52:1248–1256
- Matthews HS, Lave L, Maclean H (2002) *Life cycle impact assessment: a challenge for risk analysts*. *Risk Anal* 22:853–860
- Minh H-D, Loisel R (2011) *Actuarial risk assessment of expected fatalities attributable to carbon capture and storage in 2050*. *Int J Greenhouse Gas Control* 5:1346–1358
- Morbee J, Serpa J, Tzimas E (2010) *The evolution of the extent and the investment requirements of a trans-European CO<sub>2</sub> transport network*. Publications Office of the European Union: European Commission, JRC—Institute for Energy, Luxembourg

- Neele F, Koenen M, van Deurzen J, Seebregts A, Groenenberg H, Thielemann T (2011) Large-scale CCS transport and storage networks in North-West and Central Europe. *Energy Procedia* 4:2740–2747
- Okagawa A, Masui T, Akashi O, Hijioka Y, Matsumoto K, Kainuma M (2012) Assessment of GHG emission reduction pathways in a society without carbon capture and nuclear technologies. *Energy Econ* 34:S391–S398
- Pacyna JM, Sundseth K, Pacyna EG, Jozewicz W, Munthe J, Belhaj M, Astrom S (2010) An assessment of costs and benefits associated with mercury emission reductions from major anthropogenic sources. *J Air Waste Manage Assoc* 60:302–315
- Panwar NL, Kaushik SC, Kothari S (2011) Role of renewable energy sources in environmental protection: a review. *Renew Sustain Energy Rev* 15:1513–1524
- Patz JA, Frumkin H, Holloway T, Vimont DJ, Haines A (2014) Climate change challenges and opportunities for global health. *JAMA* 312:1565–1580
- Pearce, JM (2012) Limitations of Nuclear Power as a Sustainable Energy Source. *Sustainability*, 4, 1173–1187
- Perez-Lombard L, Ortiz J, Pout C (2008) A review on buildings energy consumption information. *Energy Build* 40:394–398
- Pimentel D, Berardi G, Fast S (1983) Energy efficiency of farming systems: organic and conventional agriculture. *Agr Ecosyst Environ* 9:359–372
- Posch M, Aherne J, Hettelingh J-P (2011) Nitrogen critical loads using biodiversity-related critical limits. *Environ Pollut* 159:2223–2227
- Raghuvanshi SP, Raghav AK, Chandra A (2008) Renewable energy resources for climate change mitigation. *Appl Ecol Environ Res* 6:15–27
- Rahmstorf S (2010) A new view on sea level rise. *Nat Rep Clim Change* 4:44–45
- Ravnum S, Runden-Pran E, Fjellsbo LM, Dusinska M (2014) Human health risk assessment of nitrosamines and nitramines for potential application in CO<sub>2</sub> capture. *Regul Toxicol Pharmacol* 69:250–255
- Reid RC (1979) Possible mechanism for pressurized-liquid tank explosions or BLEVE's. *Science* (New York, N.Y.) 203:1263–1265
- Reiss R, Anderson EL, Cross CE, Hidy G, Hoel D, McClellan R, Moolgavkar S (2007) Evidence of health impacts of sulfate- and nitrate-containing particles in ambient air. *Inhal Toxicol* 19:419–449
- REN21 (Renewable Energy Policy Network for the 21st Century) (2015) Renewables 2015: global status report. Key findings 2015. Paris
- Riahi K, Grubler A, Nakicenovic N (2007) Scenarios of long-term socio-economic and environmental development under climate stabilization. *Technol Forecast Soc Chang* 74:887–935
- Rockstroem J, Falkenmark M, Karlberg L, Hoff H, Rost S, Gerten D (2009) Future water availability for global food production: the potential of green water for increasing resilience to global change. *Water Resour Res* 45
- Schlesinger RB, Cassee F (2003) Atmospheric secondary inorganic particulate matter: the toxicological perspective as a basis for health effects risk assessment. *Inhal Toxicol* 15:197–235
- Singh B, Stromman AH, Hertwich EG (2012) Environmental damage assessment of carbon capture and storage application of end-point indicators. *J Ind Ecol* 16:407–419
- Siirila ER, Navarre-Sitchler AK, Maxwell RM, McCray JE (2012) A quantitative methodology to assess the risks to human health from CO<sub>2</sub> leakage into groundwater. *Adv Water Resour* 36:146–164
- Slovic P (1986) Informing and educating the public about risk. *Risk Anal* 6:403–415
- Smith P, Martino D, Cai Z, Gwary D, Janzen H, Kumar P, McCarl B, Ogle S, O'Mara F, Rice C, Scholes B, Sirotenko O, Howden M, McAllister T, Pan G, Romanenkov V, Schneider U, Towprayoon S (2007) Policy and technological constraints to implementation of greenhouse gas mitigation options in agriculture. *Agr Ecosyst Environ* 118:6–28
- Song JN, Yang W, Higano Y, Wang XE (2015) Introducing renewable energy and industrial restructuring to reduce GHG emission: application of a dynamic simulation model. *Energy Convers Manag* 96:625–636

- Sundseth K, Pacyna JM, Banel A, Pacyna EG, Rautio A (2015) Climate change impacts on environmental and human exposure to mercury in the Arctic. *Int J Environ Res Public Health* 12:3579–3599
- Thitakamol B, Veawab A, Aroonwilas A (2007) Environmental impacts of absorption-based CO<sub>2</sub> capture unit for post-combustion treatment of flue gas from coal-fired power plant. *Int J Greenhouse Gas Control* 1:318–342
- Tian X, Chang M, Shi F, Tanikawa H (2014) How does industrial structure change impact carbon dioxide emissions? A comparative analysis focusing on nine provincial regions in China. *Environ Sci Pol* 37:243–254
- Tubiello FN, Soussana JF, Howden SM (2007) Crop and pasture response to climate change. *Proc Natl Acad Sci U S A* 104:19686–19690
- Tzanidakis K, Oxley T, Cockerill T, Apsimon H (2013) Illustrative national scale scenarios of environmental and human health impacts of Carbon Capture and Storage. *Environ Int* 56:48–64
- Tzimas E, Mercier A, Cormos C-C, Peteves SD (2007) Trade-off in emissions of acid gas pollutants and of carbon dioxide in fossil fuel power plants with carbon capture. *Energy Policy* 35:3991–3998
- U.S. EIA (United States Energy Information Administration) (2015) Annual energy outlook 2015: with projections to 2040. U.S. Energy Information Administration, Office of Communications, Washington, DC
- U.S. EPA (United States Environmental Protection Agency) (2012) Our nation's air: status and trends through 2010. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, North Carolina
- Valentine SV (2011) Emerging symbiosis: renewable energy and energy security. *Renew Sustain Energy Rev* 15:4572–4578
- van Vuuren DP, Isaac M, Kundzewicz ZW, Arnell N, Barker T, Criqui P, Berkhout F, Hilderink H, Hinkel J, Hof A, Kitous A, Kram T, Mechler R, Scricciu S (2011) The use of scenarios as the basis for combined assessment of climate change mitigation and adaptation. *Glob Environ Chang* 21:575–591
- Wang S, Jaffe PR (2004) Dissolution of a mineral phase in potable aquifers due to CO<sub>2</sub> releases from deep formations; effect of dissolution kinetics. *Energy Convers Manag* 45:2833–2848
- WCED (World Commission on Environment and Development) (1987) Our common future. Oxford University Press, Oxford and New York
- WHO (World Health Organization) (2002) Reducing risks: promoting healthy life. World Health Organisation (WHO), Geneva
- WHO (World Health Organization) (2013) Review of evidence on health aspects of air pollution—REVIHAAP project: technical report. World Health Organization (WHO), Copenhagen
- Wilday J, Paltrinieri N, Farret R, Hebrard J, Breedveld L (2011a) Addressing emerging risks using carbon capture and storage as an example. *Process Saf Environ Pro* 89:463–471
- Wilday J, Wardman M, Johnson M, Haines M (2011b) Hazards from carbon dioxide capture, transport and storage. *Process Saf Environ Prot* 89:482–491
- Woodcock J, Edwards P, Tonne C, Armstrong BG, Ashiru O, Banister D, Beevers S, Chalabi Z, Chowdhury Z, Cohen A, Franco OH, Haines A, Hickman R, Lindsay G, Mittal I, Mohan D, Tiwari G, Woodward A, Roberts I (2009) Health and Climate Change 2. Public health benefits of strategies to reduce greenhouse-gas emissions: urban land transport. *Lancet* 374:1930–1943
- Yedla S, Shrestha RM, Anandarajah G (2005) Environmentally sustainable urban transportation—comparative analysis of local emission mitigation strategies vis-a-vis GHG mitigation strategies. *Transp Policy* 12:245–254
- Zhang Y, Xu J, Zhang Y, Zhang J, Li Q, Liu H, Shang M (2014) Health risk analysis of nitrosamine emissions from CO<sub>2</sub> capture with monoethanolamine in coal-fired power plants. *Int J Greenhouse Gas Control* 20:37–42

# Chapter 10

## Unmasking Environmental Health Zorros: The Need for Involvement of Real Risk Communication Experts for Two-Way and Problem-Solving Communication Approaches

Hans Keune, Peter Van Den Hazel, and Frederic Bouder

**Abstract** In the literature about risk communication an evolution can be traced from traditional, one-way and problem focussed communication, restricted to the dissemination of information from experts to the public, to more modern, two-way and more problem solving oriented risk communication, with a focus on participation and cooperation between scientists, policy-makers and the public. Despite advances in theory and numerous initiatives in practice, traditional, one-way communication continues to dominate many attitudes towards the public communication of science as well as practices. Science should no longer hide behind expertism, elitist attitudes and non-transparent black box approaches. Despite good intentions of environmental health experts to help society tackle risks, unmasking these scientific Zorros is crucial to take practice and its practitioners and stakeholders serious. It is time for real professional risk communication expertise to be applied and involved in two-way directional and problem solving collaborations.

---

H. Keune (✉)

Research Institute for Nature and Forest, Belgian Biodiversity Platform, INBO, Kliniekstraat 25, Kliniekstraat 25, Brussels 1070, Belgium

Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium  
e-mail: [Hans.KEUNE@INBO.be](mailto:Hans.KEUNE@INBO.be)

P. Van Den Hazel

Public Health Services Gelderland-Midden, Arnhem, The Netherlands

International Network for Children's Health, Environment and Safety (INCHES), Dieren, The Netherlands

Health and Environment Alliance (HEAL), Brussels, Belgium

F. Bouder

Department of Technology and Society Studies, Maastricht University, Maastricht, The Netherlands

**Keywords** Risk communication • Environmental health • Experts • Two-way communication • Problem solving collaboration

## 10.1 Introduction

In the literature about risk communication an evolution can be traced from traditional, one-way communication, restricted to the dissemination of information from experts to the public, to more modern, two-way risk communication, with a focus on participation and cooperation between scientists, policy-makers and the public (Fischhoff 1995; Leiss 1996; McComas 2006). Traditional approaches to risk communication have often been based on what Wynne (1996) refers to as the deficit model: i.e. the assumption that clear, one-way communication of objective and sound scientific information from experts to the ignorant public is sufficient to make them aware of problems and respond accordingly. In most cases, the science is not simple and consensual, but involves ambiguities and uncertainties. Nor is the public mere recipient of information, but actor in the decision process of the strategies to improve and/or preserve situations and in management of the risks. Reflecting this, obligations for public disclosure of environmental information have for long been promulgated: for example under the Aarhus Convention (UNECE 1998), and through numerous ‘right to know-initiatives’ at local and national level.

Another important challenge in risk communication is on content: how to exchange meaningfully information regarding uncertain, complex and ambiguous knowledge (Renn 2008). How can science formulate confident, robust and clear messages when due to complexity, science struggles with uncertainties, unknowns, and ambiguities? How does the traditional scientific evidence base approach live up to expectations of clear communication and of solving problems without pleading for endless ever more detailed research and without too complicated messages due to lack of clear cut scientific understanding? There is a range of concerns in risk communication. Ragas et al. (2006) challenge the argument that communication should be restricted because of uncertainties, and argue that if the information is used by regulators, public managers and risk assessors, then the public equally ought to know. The belief that the public is unable to deal with complex issues can also be disputed (e.g. Marris et al. 2001). Withholding data regarding uncertainty is shown to often reduce trust (Frewer 2004; Van Kleef et al. 2007). As Slovic (2001) has stated, “The challenge is to communicate the risk estimates so that they are understandable and that the risks and associated uncertainty can be put into a personal perspective”.

Despite advances in theory and numerous initiatives in practice, the deficit model continues to dominate many attitudes towards the public communication of science (Davies 2008) as well as practices. Two-way communication is seen as inherently difficult and dangerous. The alternative view—that two-way communication helps

to make scientists and policy makers accountable and to empower the public—remains in the minority in many fields of science and policy. Much still has to be done to devise and promote more open, yet workable solution oriented approaches to the communication of science, risk and policy, in the context of complexity. In this chapter we will give some theoretical background to modern two-way risk communication combined with illustrations from practice, building on experiences in EU and national environmental health projects, highlighting challenges that after decades are still prominent.

## 10.2 Decades of Professional Two-Way Risk Communication Advice: How to...

### 10.2.1 A History of Two-Way Risk Communication Advice

Often cited and therefore important landmarks in the history of two-way risk communication advice are two seminal publications from the end of the last century. First Baruch Fischhoff's '*Risk Perception and Communication Unplugged: Twenty Years of Process*' (1995) elegantly summarizes both the message and its history (Table 10.1—after Fischhoff 1995):

Well, to be more precise, history at that moment in time. Or from a present day perspective we perhaps better call it its potential future as it could or should be, as even at present it still remains a challenge to change the dominance of one-way risk communication practice. Second, Stern and Fineberg in *Understanding Risk: Information Decisions in a Democratic Society* (1996) highlight the need for societal dialogue coupled with research practice. Both publications highlight the importance of a turn to more collaborative approaches combining scientific analysis with stakeholder involvement. Even before that, seminal more fundamental reflections about changing the mode of traditional science and science communication practice provided the breeding ground, so fertile, that one wonders why so unheard, or perhaps better, so neglected in real practice. Two examples are Rosenhead's *Rational*

**Table 10.1** Developmental stages in risk management

• All we have to do is get the numbers right
• All we have to do is tell them the numbers
• All we have to do is explain what we mean by the numbers
• All we have to do is show them they have accepted similar risks in the past
• All we have to do is show them that's a good deal for them
• All we have to do is treat them nice
• All we have to do is make them partners
• All of the above

*Analysis for a Problematic World. Problem Structuring Methods for Complexity, Uncertainty and Conflict* (1989) and *Uncertainty and Quality in Science for Policy* from Funtowicz and Ravetz (1990). Both publications contain pleas for new modes of balancing scientific uncertainty and societal challenges addressed by science, by opening up to more practice-relevant approaches and to practitioners and stakeholders. More recent examples in the field of environmental health echoing similar reflections and advice are Philippe Grandjean's *Non-Precautionary Aspects of Toxicology* (2005) and David Briggs' *A Framework for Integrated Environmental Health Impact Assessment of Systemic Risks* (2008).

These reflections share concerns and ambitions for better responses to the challenge of dealing with limited and ambiguous knowledge about societal important issues; in other words, of dealing with complexity. Whereas more traditional approaches such as the Santa Fé school (Kauffmann 1995; Holland 1998) merely believe that new scientific strategies in the face of complexity in the end will bring us closer to the modern scientific aim of ever more perfect knowledge and control, the critical complexity school (Cilliers 1998; Morin 2008; Kunneman 2010; Keune 2012) points out that limits of knowledge are inherent to complexity. The critical complexity thus points at the need for reduction of complexity, as we cannot fully embrace complexity. And it underlines the need for critical reflection on the normative basis for any simplification: why do we choose to take some elements of complexity into account, and other not? Methodological choices related to complexity cannot be objectified: they are open for discussion and for value laden preferences. This also has consequences for policy interpretation and policy action. Framing complexity is crucial: the complexity to be taken into account and the approach for dealing with that complexity is part of context specific negotiation amongst actors involved in the process of investigation and interpretation, and as such becomes negotiated complexity (Keune et al. 2013). This also poses the question who has the right to be involved in such negotiation? In principle all who have stake, such as the general public when it comes to important risk issues, can be considered for this. The core question of how to deal with limited knowledge on important societal issues in relation to environmental health is critical for risk communication.

We will next present some key aspects of two-way risk communication.

### 10.2.2 Who Communicate?

Risk communication is the act of conveying or transmitting information between parties about a range of areas including: levels of health or environmental risks; the significance or meaning of health or environmental risks; decisions, actions or policies aimed at managing or controlling health or environmental risks (Fewtrell and Bartram 2001). According to Bennett and Calman '*ongoing reciprocal communication among all interested parties is an integral part of the risk management process. Risk communication is more than the dissemination of information, and a major function is the process by which information and opinion essential to effective risk*



*management is incorporated into the decision*' (Bennett and Calman 1999). Thus, risk communication is an integral part of risk management.

Going back to earlier theories of communication (Laswell 1948), risk communication practice is often conceptualised as involving senders and receivers. There are a number of roles in risk communication that one might seek to incorporate according to Renn and Levine (1991). Some of these roles fit the senders of risk communication messages, other roles are applicable both for senders and receivers:

- Enlightenment role (aiming to improve risk understanding among target groups).
- Right-to-know (designed to disclose information about hazards to those who may be exposed).
- Attitude modification role (to legitimise risk-related decisions, to improve the acceptance of a specific risk source, or to challenge such decisions and reject specific risk sources).
- Legitimate function (to explain and justify risk management routines with a view to enhancing the trust in the competence and fairness of the management process).
- Risk reduction role (to enhance public protection through information about individual risk reduction measures).
- Risk reduction role (to enhance public protection through information about individual risk reduction measures).
- Behavioural change role (to encourage protective behaviour or supportive actions towards the communicating agency).
- Emergency readiness role (to provide guidelines or behavioural advice for emergency situations).
- Public involvement role (aiming at educating decision-makers about public concerns and perceptions).
- Participation role (to assist in reconciling conflicts about risk related controversies).

The general public is considered in most cases of risk communication the main target 'receiver' group. The involvement of the general public as stakeholder is needed to establish effective risk communication. However, beyond public commitments to increase public participation there is often little knowledge on how to engage with various "publics" (Löfstedt et al. 2011; Arvai and Rivers 2014). Communication should be perceived as a two directions process of providing information using different tools, understanding the reception of the information, reading the feedback and adjusting the information accordingly. Effective risk communication should be less about persuasion than about achieving fewer but better disagreements (Fischhoff 2013). In the eyes of the sender, the understanding of the reception of information has often to do with risk perception and acceptance. It is assumed

that acceptance of risks is greater when Fischhoff's (1995) eight developmental stages in risk management are met. As a precondition for engaging with stakeholders in a partnership, evidence needs to be communicated in a correct and intelligible manner, the ultimate health effect needs to be seen as not too big and the risk needs to be perceived as recognizable/manageable for all stakeholders, and finally the benefits need to be perceived as bigger than the risks.

The message sent has to be understood and the reciprocal exchange of information is fundamental to establish clear communication. The idea of the 'general public' as a target audience is generally misleading as there are many different groups of stakeholders, many 'publics'. Most stakeholders must be identified according to their position to the topic at hand.

Stakeholders are any individuals, groups of people, institutions (government or non-government) organisations or companies that may have a relationship with the project/program or other intervention at stake. They may—directly or indirectly, positively or negatively—affect or be affected by the process and/or the outcomes. Usually, different sub-groups have to be considered because within a certain group interests may be different (adapted from EU Project Cycle Management Manual 2004). In some projects or risk communication frameworks the following types of stakeholders are considered:

1. Those who are *important* to engage during a project or problem because they are important and/or influential in relation to the identified activities, e.g. environmental health managers and authorities, research institutes ("**developers and scientists**");
2. Those who are *influential* during and after a project, incident or programme, e.g. regional or local authorities (whether only advisory or with decision taking power) and other institutes influencing the public environment or health management or environment or health protection at local and/or downstream level ("**decision makers**");
3. Those who should apply or could be *instrumental in spreading the outcomes* or results (problematic, complex findings of research or planning scenarios, location specific management solutions, generic guidelines), e.g. public health authorities, national authorities dealing with public (environmental) health, existing local platforms/forums, NGOs, traditional authorities, patient organisations, etc. ("**end users**").

This approach, however, is increasingly challenging especially when it comes to the new roles of NGOs, the public and other interest groups. Since the 1990s regulators, especially in Europe, have experienced public distrust following large-scale incidents—from the BSE scandal in the UK to the dioxin in chickens

scandal in Belgium. As a result government has been under increased pressure to open up decision-making, which in practice means that influential stakeholders are increasingly able to influence risk communication (Löfstedt et al. 2011). On a global level, Sand (2003) has identified three main legal obligations to disclose information—and as a result, incentives to communicate:

- Disclosure to governments by environmental impact assessment statements;
- Disclosure to citizens under the ‘right-to know’ schemes;
- Disclosure to consumers through a variety of labelling schemes;

Therefore, increasingly, risk communication is seen as an integral part of the wider attempt to develop new forms of collective decision-making between regulators, industry, non-governmental organisations and the general public (Bouder and Löfstedt 2013). According to Renn, ‘effective communications must address, in as much detail as possible, the particular concerns of affected or interested parties in the specific case at hand (Renn 2008). In a majority of projects, incidents or cases there will be multiple stakeholder groups involved. In the case of power lines there are the inhabitants of houses under a high voltage line, but also the owners of the buildings in case of rental houses, the employees in case of companies, the local authorities and health services for the protection of public health, the power companies, maintenance staff and multiple other stakeholders. The risk communication to all of these stakeholders could have a different approach. These stakeholders will have different positions on the topic. The differences in interest will influence the risk perception and the way the communication will be conducted.

The senders of information in the process of risk communication are usually professionals linked to the authorities. Those authorities that are responsible for the health of the general public are usually the main stakeholders who convey messages related to incidents which might cause effects or alarm with the general public. There is a common misunderstanding that authorities have to possess all the available knowledge before communicating evidence or known facts. There is no societal obligation that authorities have to know everything. At the same time the authorities are not responsible for everything. However, they usually have the greatest influence over matters.

### ***10.2.3 Essential Elements of Risk Communication***

There are a range of elements identified in risk communication and in risk perception. In 2009 Frederic Bouder (2009) developed for the UK Risk and Regulation Advisory Council a science-informed “‘survivors’ guide” to help the institutional senders of risk messages cope with the risk communication challenge. The so-called “five As” of public risk communication suggest to consider five elements:

1. Assembling the evidence
2. Acknowledgement of public perspectives
3. Analysis of options
4. Authority in charge
5. Interacting with the audience

### **10.2.3.1 Assembling the Evidence**

The first step for message senders is to “assemble the evidence” about a given risk. Concretely, this means that risk communicators need to demonstrate that they understand the science behind the risk and that their decisions will be based on credible evidence. This does not mean that a thorough risk assessment can always be conducted, but this means that basing decisions on mere judgment or discarding important new information when it does not fit with pre-established conceptions is unlikely to fare well with stakeholders.

### **10.2.3.2 Acknowledgment of Public Perspectives**

Studying people’s perceptions and paying proper attentions to people’s concerns is also essential. The perception of risks differs from individual to individual, yet these variations are not irrational or random. Forty years or so of risk perception studies has uncovered a number of perception drivers, such as degree of control, catastrophic potential, familiarity, impact on children and fertility etc (Fischhoff et al. 1978; Slovic 1987): see Table 10.2. Personal experience is also important. If people do not believe they are in danger, for instance, or do not understand that they are at risk they are less likely to be receptive to risk communication information. On the other hand, people that have personally experienced the impact of crises or similar events will be much more receptive to risk advisories and communication than those who did not share this experience (Fitzpatrick-Lewis et al. 2010).

### **10.2.3.3 Analysis of Options**

Consider a broad range of options and the associated trade-offs. Based on the evidence that you have assembled and public perspectives on the risk, you need to develop and analyse a broad range of options. Each option for managing the public risk that you consider will have costs and benefits, and these will often be different for different groups. You will need to understand, and explain, the trade-offs that need to be made in choosing particular options. You will need to show that your decisions are fair and justifiable.

**Table 10.2** Risk perception (adapted from Fischhoff et al. 1978; Slovic 1987; Corvello 1998)

Factor	Increase public concern	Decrease public concern
Catastrophic potential	Fatalities and injuries grouped in time and space	Fatalities and injuries scattered and random
Controllability (personal)	Uncontrollable	Controllable
Manifestation of effects	Delayed effects	Immediate effects
Effects on children	Children specifically at risk	Children not specifically at risk
Familiarity	Unfamiliar	Familiar
Media attention	Much media attention	Little media attention
Origin	Caused by human actions or failures	Caused by 'Acts of God'
Reversibility	Effects irreversible	Effects reversible
Trust in institutions	Lack of trust in responsible institutions	Trust in responsible institutions
Uncertainty	Risks unknown	Risks known
Understanding	Mechanisms or processes not understood	Mechanisms and processes understood
Voluntariness of exposure	Involuntary	Voluntary

The methods used to analyse the options will depend on the context. Where appropriate, and where time allows, technical methods such as risk-benefit, cost-benefit analyses or multi-criteria decision analysis may help in reconciling trade-offs, including conflicting objectives and goals in different groups. Such analyses may be a part of a formal impact assessment. These technical methods can be supplemented by, for example, consultative, or deliberative, techniques. Optimum technical solutions are not necessarily perceived as the best solutions by the public and specific groups of risk actors, who will bring societal and special interests to bear on the solution. The technical and societal interests will need to be reconciled if the solution is to be generally accepted.

#### 10.2.3.4 Authority in Charge

Define the nature of your involvement with the risk. There are several ingredients of open risk communication. These ingredients for open communication have been described in a report of RIVM (2013) on communication on environmental incidents:

1. Recognition or giving meaning to the message
2. Limitation of the damage
3. Providing information

The media focus often on the information and the way authorities are controlling or not the damage that has occurred. However, it is very important that the responsible person for the authorities or any other organisation that is providing risk communication, show that they take the situation very seriously, offer recognition and compassion for any victim. The messenger has the personal role to be a connecting factor to the affected population so it feels itself well listened to.

### 10.2.3.5 Interaction with the Audience

A common approach to obtain success in risk communication is to know that the message has been convincing the receiving stakeholders and that they feel that their opinion has been heard and taken into account. People's attitudes in this respect are influenced by a variety of factors and experiences. One factor is heuristics (Tversky and Kahneman 1974), i.e. mental shortcuts. For instance people may use heuristics to compare a new situation to a more familiar one. Talking about familiar situation will reinforced known conceptions that people can relate to. Affect (Finucane et al. 2000) and risk-as-feeling (Loewenstein et al. 2001) are other key factors that have been uncovered. Risk communication is not just about rationally exposing facts but about also about understanding and addressing people's affective motivations, including dealing with fears and sensitivities. We can distinguish three elements:

1. The ratio: the message should be understandable from the point of view of the receiving stakeholder, while it is also clear that this is the latest state-of-the-art knowledge of science.
2. The emotion: the receiving stakeholder should have a good feeling about the message. This has to do with believing in the messenger and believing in the message.
3. The ethics: The question has to be answered if the issue at hand is allowable to society, that it has to do with reliable common practice or that there is moral justice in what the message conveys. This latter issue is influenced by the moral attitude of people based on someone's societal position, culture or political ideas.

Under the Aarhus Convention (UNECE 1998), and through numerous 'right to know-initiatives' at local and national level information about risks has become more transparent. Examples include the many 'in my backyard' websites that now offer ready access to environmental information. Another somewhat unusual example, because of its focus on health effects (cancer risk) of pollutants, is the

‘right to know-website’ in the Netherlands (Ragas et al. 2006). This includes a public forum, where opinions on the release of such information to the broader public have been aired.

Chess et al. (1988) argue for citizen involvement in risk communication:

- people are entitled to make decisions about issues that directly affect their lives;
- input from the community can help the agency make better decisions;
- involvement in the process leads to greater understanding of—and more appropriate reaction to—a particular risk;
- those who are affected by a problem bring different variables to the problem-solving equation; and
- cooperation increases credibility.

The use of focus group may be considered in situations with stakeholders that have low influence but high interest. The focus group can serve the purpose of collective information about a specific subject or area of concern. It is a useful method to gather information on risk perceptions. Focus groups are also used to assess needs, preferences and attitudes of different stakeholders. The collected information can be used to formulate risk messages, to determine the appropriate channel of communication, to choose the best communicator and to frame the risk information in an acceptable way. The advantages of a focus group are multiple. This form allows participants to discuss a subject openly and in detail. The setup and conducting focus groups can be done quickly in a couple of weeks. This can be followed by quick implementation. This form is far less intimidating or frustrating than other forms of risk communication. The anxiety of the individuals is lessened in the group context.

#### ***10.2.4 Practical Recommendations for Risk Communication***

We end this section with a practical overview of recommendations for risk communication in general (Covello 2003; Fagerlin et al. 2011; Verroen et al. 2013; modified):

- Accept and involve stakeholders as legitimate partner as early as possible in the process
- Listen to people and their peers, build trust
- Be honest, transparent, open
- Coordinate and collaborate with other credible sources

(continued)

(continued)

- Meet the needs of the media
- Communicate clearly by using a language that the stakeholders understand
- Plan thoroughly and carefully
- Enhance levels of efficacy beliefs in the message
- Present data using absolute risks and using frequencies
- Recognize that comparative risk information is persuasive and not just informative
- Be aware that the order in which risks and benefits are presented can affect risk perception
- Experienced communicator with empathy, trustworthiness, good speaker, eye contact, identification with audience

### **10.3 Two-Way Risk Communication Advisory Practice: How to Deal with Amateurism?**

After this theoretical guided tour to risk communication, we will now turn to practice. We mainly focus on practice regarding collaborative approaches advising experts in the direction of more problem solving and two-way risk communication. We draw on our own experiences, in EU and national environmental health projects, sketching efforts and highlighting challenges that after decades are still prominent.

#### ***10.3.1 Analytical Deliberative Work in Belgium on Human Biomonitoring***

Between 2001 and 2011 in Flanders (the Dutch speaking part of Belgium) human bio-monitoring research was being carried out, investigating the relation between environmental pollution and human health by measuring pollutants and health effects in human beings, using biomarkers. The project was carried out in the scope of the Flemish Centre of Expertise for Environment and Health (CEH), funded and steered by the Flemish government. In the CEH, environmental health experts from all Flemish universities and from two research institutes cooperate. The CEH combines natural (Schoeters et al. 2012) and social scientific research (Keune et al. 2014).

In two decision support case studies a multi-criteria group decision support method was applied. First the action-plan (2005–2007): together with medical and environmental scientific experts and policymakers, an action-plan for setting policy priorities with regard to the bio-monitoring results was developed (Keune et al. 2009).



Second the hotspot selection procedure (2007–2008): in the CEH we experimented with the input of a diversity of actors with regard to setting research priorities (Keune et al. 2010). Both approaches were inspired by the analytical deliberative approach (Stern and Fineberg 1996), an approach that combines scientific complexity and social complexity by linking expert analysis and debate with social deliberation. In practice it concerned close interdisciplinary cooperation: the general approach had to be negotiated between totally different disciplinary backgrounds and natural and social scientific data were combined. It also concerned close transdisciplinary cooperation with policy representatives: the research had to be policy relevant, which puts totally different demands on research than just scientific ones. Furthermore, both external experts and stakeholders were involved.

How did we get there? The general analytical deliberative approach resulted from a long and intense transdisciplinary dialogue in which social scientists gradually were able to bring this perspective into the discussion and into practice. The process of policy interpretation of the data was initially seen as an essentially scientific one; a working group was established, comprising mainly of environmental and medical experts both from science and policy. With the right group of experts, it was assumed, interpretation with regard to policy priorities would follow automatically. Once attempts were made to translate the scientific conclusions into policy priorities, however, it became evident that none of the environmental or medical experts involved dared to claim the necessary and overarching knowledge needed to prioritize. This was especially clear when incorporating other aspects considered relevant from a policy perspective, such as economics, social preferences or political feasibility. This resulted in openness to an analytical deliberative approach in which expert elicitation was combined with stakeholder consultation as a basis for advice for both the government (Keune et al. 2009) and the CEH (Keune et al. 2010). The ambition of the transdisciplinary team became one of open arms, embracing a broad diversity of actors and factors. Moreover the ambition stretched the horizon of scientific research to concrete policy action plans.

An essential element of the development towards an analytical deliberative way of working was a strategic approach. An important strategic move in the conceptual design phase that proved to be of decisive importance was an active listening approach: the use of an internal reflective questionnaire. At first the practical relevance of an analytical deliberative way of working was not recognized by the colleagues from natural science and policymaking. However, when in an in-group questionnaire questions were asked such as who are relevant actors and factors, elements of an analytical deliberative approach came to the fore. This led to a breakthrough in the conceptual development process and formed the basis for an approach in which questions of openness to relevant actors and factors were pragmatically dealt with.

So far, so good. Trying to bring ambitions into practice however created new dynamics causing a boomerang effect. Application in practice created pressure on the work of the colleagues: e.g. time pressure, pressure on their role as experts, practical pressure by complicating their own or the joint effort. As such the initial

enthusiasm of natural scientists and policy makers was overshadowed by concern for practical and analytical constraints. Without the social scientists being concretely involved, the analytical deliberative elements probably would have had a hard time to survive in real practice. It is therefore crucial that the analytical deliberative perspective is represented by ambassadors of such approach at the methodological decision table.

By joining conceptual discussions on policy interpretation of scientific research outcomes and reflecting on the ambitions of both natural scientists and policy representatives step by step from an active listening approach the role of the social scientist evolved to one of more central importance. One of the senior natural scientists involved, saying she (on the level of ambition) approved of the social scientific contribution, but she sometimes felt like an object of some social scientific experiment. Colleagues with natural scientific background sometimes react as if they feel lured into unexpected complexity, unknown to their expertise, difficult to handle and sometimes confrontational, and they either question its usefulness or appear to be unable to articulate the benefits themselves. This is also reflected in the often heard concern of the natural scientists and their counterparts in policy making that the analytical deliberative approach is relevant and interesting but should not stand in the way of the research or policy agenda and should not complicate the already complicated research and policy endeavour.

As part of the analytical deliberative approach, all external actors contributing to the project were asked for their feedback on the project. The vast majority evaluated openness to outsider perspectives and diversity of actors to be worthwhile. This is of course a bonus for those organizing such processes and for the end-user of the outcomes (e.g. policymakers). Simultaneously this can be perceived both as a stimulus and a pressure for prolonging such openness.

### ***10.3.2 A Problem Solving Turn in Environmental Health Expert Elicitation in HENVINET***

As mentioned in paragraph 2, the question of how to deal with limited knowledge on important societal issues in relation to environmental health is critical for risk communication. It is not always possible to prove unambiguously that a causal relationship exists between environmental pollution and specific health effects. Scientific assessment of environmental health risks is faced with large (partly irreducible) uncertainties, knowledge gaps, and imperfect understanding, out of which may arise deep-seated conflicts and controversies. The EU HENVINET project (Bartonova et al. 2012) had the ambition to synthesize scientific information available on a number of topics of high relevance to policy makers in environment and health: brominated flame retardants, phthalates, the impacts of climate change on asthma and other respiratory disorders, the influence of environment health stressors on cancer induction, the pesticide CPF and nano particles. At first it was the

ambition to focus mainly on the state of the art scientific knowledge, with a special interest in gaps of knowledge. By means of expert elicitation the gaps of knowledge were highlighted by using confidence levels for assessment of current scientific knowledge.

During the work in progress a complementary focus developed through interdisciplinary reflections (Keune et al. 2012): after long and intense discussions more traditional scientists opened up to the idea that the challenge of gaining knowledge about complexity is as important as the challenge to act based on limited knowledge. This resulted in extending the horizon from a mere scientific to the problem solving policy perspective: interpreting the synthesized available knowledge from a policy perspective, addressing the question which kind of policy action experts consider to be justifiable based on the identified state of scientific knowledge. As such the expert elicitation approach became helpful in overcoming the policy action impasse caused by the search for perfect scientific evidence. It did so by constructively discussing the weight of existing knowledge for potential policy action, thus stressing more the societal importance of the issues under study and considering to take action, rather than aiming for ever more scientific knowledge. Both parts of the expert elicitation, the assessment of state of the art scientific knowledge by means of confidence levels and the problem solving interpretation by means of a qualitative questionnaire and a workshop discussion, were quite challenging for all experts involved, as it did not relate easily to mainstream environment and health scientific practice.

The problem solving turn from mainly focusing on overcoming gaps in science to overcoming gaps between science and policymaking was strategically triggered by pointing at ambitions. The social scientist involved in the project while trying to introduce a problem solving perspective, realized it was not easy to convince the principal coordinator of the case study. The potential benefits of a problem solving approach were countered by pointing out practical complexities that would put further pressure on what in itself was already quite a challenging pioneering endeavour, let alone put pressure on the loyalty to the expert elicitation project of the natural scientists in the team. The social scientist used reference to ambitions that were part of the initial project aims, be it mainly dormant, and ambitions from the professional background of the principal coordinator of the project as persuasive arguments. He pointed out the initial ambition of policy relevance of the project as an argument for integrating a problem solving perspective. Also he referred to two grand old men in the field of environment and health for whom he knew the coordinator had high respect, and who promote a problem solving turn in the field of environment and health: Grandjean (2005) and Briggs (2008). Being part of the project one of them in fact had criticized the absence of a clear problem solving perspective in the early phases of the project. The fact that idealistic ambitions are often not easily applied in practice thus does not withhold them from being used as persuasive arguments: from a dormant or Ten Commandments' status to becoming seeds of practical change and inspiration. This case also exemplifies how an outsider perspective can be helpful: the social scientist joined the project at a later stage, thus as a newcomer could reflect on the work in progress from some distance.

How did the scientists perceive the experiment? In general most experts were positive, and sometimes very positive about the approach. A main negative critique to the approach seems to be that it is not sophisticated enough with respect to complexity, especially regarding technical issues and as such, according to several experts, it is too superficial for what is commonly understood as proper risk assessment. A challenge was that experts were expected to be an expert in all aspects of issue relevant complexity, when in fact they were not, or at least did not always feel at ease with this. The following statement illustrates this position:

Cannot agree that this is a group of experts, we were selected as guinea pigs, but not as a risk assessment group. You must make a distinction on how far you can go. I do not feel comfortable in serving policy makers conclusions.

Still, not all experts responded in the same way. Some were more self-confident in being able to give policy advice, and in fact in the end the majority of experts felt confident enough to be acknowledged in the policy briefs that were the output of the project. Even the expert being quoted above, after intense consultation on the content of the policy brief, changed position from not wanting to be acknowledged to wanting to be acknowledged.

One expert questioned whether such approach will indeed come up with new knowledge. Also to some experts lack of clarity about the process and their role was a problem. Positive critique is pointed to the fact that it was an innovative approach that was considered interesting and promising. In particular the opportunity to widen one's own horizon and to interactively exchange knowledge and debate with a diversity of experts seemed to be well appreciated in this approach. Different parts of the approach also helped in focusing on specific relevant aspects of scientific knowledge, and as such can be considered of reflective value. Moreover the combination of experts offered the opportunity to learn from and discuss diversity of interpretation from different perspectives. Diversity of expertise is considered important because of the complexity of the combination of relevant aspects, but is difficult to oversee for individual specialists. Also it was considered important to organize a good balance between different fields of expertise. Transparency about the background of experts is a related issue: it should be clear e.g. if experts have a relation with industry. More in general does any composition of expert panels run the risk of bias because of over- or underrepresentation of specific types of expertise. With respect to the involvement of experts it was suggested to recruit a large panel so as to ensure that enough will remain even when some drop out.

In comparison with risk assessment some stated this approach to be of complementary benefit: "Reports after risk assessments often take long time to write and may not reflect the latest data. We should not put this group aside. This is an intermediate stage. You will get different answers depending on who you ask; public, scientists, risk assessors." One expert even considered the approach better than risk

assessment as it seems to be more up to date on scientific information than most risk assessment documents.

When asked about the possibility of a stakeholder workshop, most experts welcomed the idea, even though (to some) certain aspects were unclear. E.g. the question who would be relevant stakeholders was unclear: policymakers and risk assessors were mentioned by one expert, industry by another. One expert pointed out that a balance of views is important. One expert was explicitly negative, stating this will probably lead to ‘prestige-filled confrontations’, thus questioning the relevance.

### ***10.3.3 Role-Play and Social Learning in HENVINET***

HENVINET conducted a role-play session at one of the project annual meetings (Van den Hazel et al. 2012). The role-play aimed to strike a balance between respect for the complexity of environment and health issues which the role play aims to discover and discuss, and the feasibility of the participants being able to fulfil their roles in the role play without too much difficulty, while simultaneously being able to facilitate social learning. In order to make the role-play easier to perform but also sufficiently illustrative of the complexity of reality, the discussion agenda was narrowed to one simple question. At the same time the diversity of actor roles involved in the discussion aimed to create the potential for the discussion to mirror the complexity of environment and health. As such the complexity of the situation was hidden behind the different social perspectives on what could be viewed, at first sight, as a simple issue. The participants had to play roles, in small groups of 2–4 persons, representing stakeholders from different organisations such as national authorities, scientific organisations (as consultants), industry, public health authorities and NGO’s. On the agenda of the role-play was a discussion on the meaning of a policy brief on the environment and health risks of a pollutant: the role-play discussion by a diversity of actors aimed to provide the authorities with advice on measures to be taken regarding the pollutant, based on the expert advice in the policy brief. This followed in the slip stream of the previously described expert elicitation case study which was also part of HENVINET (Keune et al. 2012). In that case study the step to stakeholder involvement was a bridge too far for extending the horizon in an analytical deliberative spirit. The role-play tried to address this challenge in a safe (experimental) and non-demanding (in terms of resources such as time) setting.

The aim of the role-play was on the one hand to test how a stakeholder discussion on such a policy brief evolved, and on the other hand to introduce stakeholder involvement to the participating experts. It thus aimed to perform a learning experience in different respects. Two moderators introduced the topic and the structure of the role-play. The roles were distributed among the participants of the session. These roles were randomly distributed. The roles were allocated to five different groups: local government, local residents, industry, non-governmental organisations, and public health authorities. The diversity in roles aimed to ensure that the complexity of the issues under discussion would be highlighted by the different perspectives and

stakeholders. The moderators provided role-information at the start of the session. Most participants could use their own experience and knowledge to fit their role. In this session the participants learnt from each other the lessons that emerged, and how each group supported its own arguments. The subgroups easily adopted the stereotype role of the stakeholder they represented. Industry was defensive, NGOs greatly opposing industry views, experts requesting more research, and local authorities waiting for a decision. In the evaluation it was stated that the views of different social perspectives were most valuable. The scientists performing the role of the NGO discovered how simple it was to use their own scientific knowledge to attack the polluter, the industrial representative. While the national authority representatives found it hard not to allow their scientific knowledge to prevail over the other issues they had to address including economic and social issues. The public health authorities were easily manoeuvred into the position of defending the general public's interest and health, although internally they had difficulties in agreeing the level of scientific proof. As a result they became less interesting partners for both the national authorities and the NGO's. Finally, the industrial representatives became defensive and deployed all available arguments concerning lack of scientific certainty to avoid any responsibility or claims of harm done.

The role play session illustrated the usefulness of stakeholder involvement in procedures that aim to provide policy advice based on scientific expertise. The social complexity of environment and health issues was clearly illustrated during the role play, indicating the added value for policy makers to be informed not only about scientific aspects of environment and health issues, but also about social aspects from a diversity of actor perspectives. The role play moreover was able to convince most of the participating experts of the usefulness of stakeholder involvement. One of the more sceptical experts in the end became one of the main defenders, and as a spokesman for the group vigorously presented the benefits both of the role play and stakeholder involvement to the non-participating experts from HENVINET. Moreover some participating experts indicated that the use of a method like the role play would have been beneficial to their perception of their involvement in the HENVINET project development, as it gave them the opportunity to better express their opinion in an interactive and cooperative manner.

## 10.4 Conclusions: Unmasking Zorro?

Where are we with risk communication development, after decades of analysis and advice pointing in the direction of a need for more problem solving and two-way risk communication (Par. 2)? One main conclusion is that the tendency for use of one-directional risk communication still is dominant. Another conclusion is that a large part of the effort regarding risk communication is still on collection of ever more detailed and stronger evidence of information about the (potential) problems, the risks at stake, and far lesser effort going to a solution focus knowledge effort and

far less effort going to collaborative analytical deliberative approaches. Does this mean that risk experts are doing a bad job with bad intentions? Or are they mainly like scientific Zorros: trying to do good, but not very transparent or open to dialogue and collaboration? Let us assume the latter and reflect to some lessons learned from collaborative practical efforts where risk communication experts tried to find a more modern approach together with these scientific Zorros (Par. 3).

Creating knowledge as such is another challenge than creating societally relevant knowledge and is another ambition than developing problem solving actions. Part of the answer is in the discussion amongst those who are in the driving seat: the cocktail of actors involved will create specific dynamics affecting the process. Professional contexts differ in professional tradition. Obvious examples are differences between quantitative and qualitative scientific approaches, between a focus on knowledge and a focus on action, between natural sciences and social sciences, between science and policy. The teams cooperating in several of the presented cases had to undertake a lot of negotiation during the process, the importance of which is often underestimated both in terms of impact on the process and its output, but also in practical complexity. The richness of dialogue can be very beneficial to a broader and more integrated view on complexity, but it is not always easy. The mind-sets of actors from specific contexts remain largely influenced by and focussed on their home-base contexts, and only to a lesser extent to the new collaborative context. This is beneficial from the point of view of specific expertise, and this is needed. But it can become problematic in the perception of other expert contexts: one is full of one's own expertise and related complexity, and has only limited sight of the complexity of other expertise, and in fact often underestimates this. This to a large extent cannot be avoided, as experts are often overloaded and are constantly attracted to context specific interests, rewards, challenges. This also means that the openness towards other forms of expertise is limited, as they only have limited attention for it and only limited interest. The transferability of expertise from one context to the other is possible of course, but will be more difficult once experts' contexts differ more. This poses the question whether we should invest in transfer of context specific expert knowledge to other expert contexts, or that we should focus on cooperation in well balanced inter- and transdisciplinary teams. From our experience it can be concluded that teamwork currently is absolutely necessary. Even after years of intense cooperation, natural scientific colleagues often still do not have clear sight of the complexity social science deals with. This would make a plea for constant and direct involvement of social scientists and in fact to the notion of the old saying: 'Let the cobbler stick to his last'. This also holds true for transdisciplinary cooperation between scientists and policy makers.

The *epistemological divide* between the traditional and alternative approaches largely sticks to ambassadors safeguarding either approach. Without ambassadors of either paradigm at the table where crucial methodological choices are being made, especially in practice and under resource constraints such as time pressure, the dominant approach will largely steer the process. This does not mean that there can be no cross boundary figures. This also does not mean that for example the traditional experts are not open to alternative approaches or that they do not see the

value of it. In the example of the CEH clearly there was good will regarding opening up, especially at the level of ambition, before concrete practical methodological choices had to be made. Still, along the way, the open arms appeared to be accompanied by closed mindsets amongst the traditional experts. Without the social scientists being concretely involved, the shift to a more collaborative approach probably would have had a hard time to survive in real practice. It is therefore crucial that the diversity which is considered to be relevant in the process in one way or the other is represented by either ambassadors of diversity as such or representatives of specific (e.g. experts and stakeholder) diversity at the methodological decision table.

Science should no longer hide behind expertism, elitist attitudes and non-transparent scientific black box approaches. Despite good intentions of environmental health experts to help society tackle risks, unmasking these scientific Zorros is crucial to take practice and its practitioners and stakeholders serious. It is time therefor for real professional risk communication experts to be involved in order to apply involved in two-way directional and problem solving collaborations.

## References

- Arvai J, Rivers L (eds) (2014) *Effective risk communication*. Routledge, Oxon and New York
- Bartonova A, Koppe JG, Fucic A, Gutleb AC, van den Hazel P, Keune H (2012) The health and environment network and its achievements. *Environ Health* 11(Suppl 1):S1
- Bennett P, Calman K (eds) (1999) *Risk communication and public health*. Oxford University Press, Oxford
- Bouder F (2009) *A practical guide to public risk communication. The five essentials of good practice*. Risk and Regulation Advisory Council, UK
- Bouder F, Löfstedt R (eds) (2013) *Risk, critical concepts in the social sciences*. Routledge, Oxon and New York
- Briggs DJ (2008) A framework for integrated environmental health impact assessment of systemic risks. *Environ Health* 7:61
- Chess C, Hance BJ, Sandman PM (1988) *Improving dialogue with communities: a short guide to government risk communication*. New Jersey Department of Environmental Protection, Trenton, NJ
- Cilliers P (1998) Complexity and postmodernism. *Understanding complex systems*. Routledge, London
- Corvello VT (1998) Risk communication. In: Callow P (ed) *Handbook of environmental risk assessment and management*. Blackwell, Oxford, pp 520–541
- Covello VT (2003) Best practice in public health risk and crisis communication. *J Health Commun* 8:5–8
- Davies SR (2008) Constructing communication: talking to scientists about talking to the public. *Sci Commun* 29:413–434
- EU (2004) *Aid delivery methods - project cycle management guidelines Vol 1*, Brussels
- Fagerlin A, Zikmund-Fisher BJ, Ubel PA (2011) Helping patients decide: ten steps to better risk communication. *J Natl Cancer Inst* 103:1436–1443
- Fewtrell F, Bartram J (eds) (2001) *Water quality: guidelines, standards and health*. IWA, London. ISBN: 1 900222 28 0
- Finucane ML, Alhakami A, Slovic P, Johnson S (2000) The affect heuristic in judgment of risk and benefit. *J Behav Decis Mak* 1–6
- Fischhoff B (1995) Risk perception and communication unplugged: twenty years of process. *Risk Anal* 15(2):137–145



- Fischhoff B (2013) The sciences of science communication. *Proc Natl Acad Sci U S A* 110(Suppl 3):14033–14039
- Fischhoff B, Slovic P, Lichtenstein S, Read S, Combs B (1978) How safe is safe enough, a psychometric study of attitudes towards technological risks and benefits. *Policy Sci* 9:127–152
- Fitzpatrick-Lewis D, Yost J, Ciliska D, Krishnaratne S (2010) Communication about environmental health risks: a systematic review. *Environ Health* 9:67–82
- Frewer L (2004) The public and effective risk communication. *Toxicol Lett* 149(1-3):391–397
- Funtowicz SO, Ravetz JR (1990) Uncertainty and quality in science for policy. Kluwer, Dordrecht
- Grandjean P (2005) Non-precautionary aspects of toxicology. *Toxicol Appl Pharmacol* 207(Suppl 2):652–657
- Holland JH (1998) Emergence: from chaos to order. Basic Books, New York
- Kauffman S (1995) At home in the universe. Oxford University Press, Oxford
- Keune H (2012) Critical complexity in environmental health practice: simplify and complexify. *Environ Health* 11:S19
- Keune H, Bert M, Johan S, Ilse L, Gudrun K, Ann C, van Campenhout K et al (2009) Policy interpretation of human biomonitoring research results in Belgium: priorities and complexity, politics and science. *Environ Pol Gov* 19(2):115–129
- Keune H et al (2010) Open the research agenda: participatory selection of hot spots for human biomonitoring research in Belgium. *Environ Health* 9:33
- Keune H et al (2012) We're only in it for the knowledge? A problem solving turn in environment and health expert elicitation. *Environ Health* 11:S3
- Keune H, Springael J, De Keyser W (2013) Negotiated complexity: framing multi-criteria decision support in environmental health practice. *Am J Oper Res* 3(1A):153–166
- Keune H, Koppen G, Morrens B, Colles A, Springael J, Loots I, Teughels C, Chovanova H, Van Campenhout K (2014) Extended peer evaluation of an analytical deliberative decision support procedure in environmental health practice. *Eur J Risk Regul* 1:25–35
- Kuneman H (2010) Ethical complexity. In: Cilliers P, Allen R (eds) Complexity, difference and identity. Springer, Dordrecht
- Laswell HD (1948) The structure and function of communication in society. In: Bryson L (ed) The communication of ideas: a series of addresses. Cooper Square, New York, pp 32–35
- Leiss W (1996) Three phases in the evolution of risk communication practice. In: Kunreuther H, Slovic P (eds) Challenges in risk assessment and risk management. Special Issue of the Annually of the American Academy of Political and Social Science, vol 545, pp 84–95
- Loewenstein G, Weber EU, Hsee CK, Welch N (2001) Risk as feelings. *Psychol Bull* 127(2):267–286
- Löfstedt R, Boudier F, Wardman JK, Chakraborty S (2011) The changing nature of regulation and communication of risk in Europe. *J Risk Res* 14(4):404–429
- McComas KA (2006) Defining moments in risk communication research: 1996–2005. *J Health Commun* 11(1):75–91
- Morin E (2008) On complexity. Perfect Paperbacks, New York
- Ragas A, Huijbregts M, van Kaathoven E, Wolsink J, Wemmenhove J (2006) Development and implementation of a right-to-know web site that presents estimated cancer risks for air emissions of large industrial facilities. *Integr Environ Assess Manag* 2(4):365–374
- Renn O (2008) Risk governance: coping with uncertainty in a complex world. Earthscan, London
- Renn O, Levine D (1991) Credibility and trust in risk communication. In: Kasperson RE, Stallen PJM (eds) Communicating risks to the public. Kluwer, Dordrecht, pp 175–214
- RIVM (2013) Communicatie over milieu-incidenten. RIVM Bilthoven, The Netherlands
- Rosenhead J (ed) (1989) Rational analysis for a problematic world. Problem structuring methods for complexity, uncertainty and conflict. Wiley, West Sussex
- Sand PH (2003) Information disclosure as an instrument of environmental governance. *Heidelberg J Int Law* 63:487–502
- Schoeters G, Den Hond E, Colles A, Loots I, Morrens B, Keune H, Bruckers L, Nawrot T, Sioen I, De Coster S, Van Larebeke N, Nelen V, Van de Mierop E, Vrijens J, Croes K, Goeyens K, Baeyens W (2012) Concept of the Flemish human biomonitoring programme. *Int J Hyg Environ Health* 215(2012):102–108

- Slovic P (1987) Perception of risk. *Science* 236:280–285
- Slovic P (2001) The risk game. *J Hazard Mater* 86:17–24
- Stern PC, Fineberg HV (eds) (1996) Understanding risk: information decisions in a democratic society. National Research Council, National Academy Press, Washington, DC
- Tversky A, Kahneman D (1974) Judgment under uncertainty. *Heuristics Biases Sci* 185:1124–1131
- UNECE (1998) Convention on access to information, public participation in decision-making and access to justice in environmental matters
- Van den Hazel P, Keune H, Randall S, Yang A, Ludlow D, Bartonova A (2012) The challenge of social networking in the field of environment and health. *Environ Health* 11(Suppl 1):S15, <http://www.ehjournal.net/content/11/S1/S15>
- Van Kleef E, Houghton JR, Krystallis A, Pfenning U, Rowe G, Van Dijk H, Van der Lans I, Frewer LJ (2007) Consumer evaluations of food risk management quality in Europe. *Risk Anal* 27(6):1565–1580
- Verroen S, Gutteling JM, De Vries PW (2013) Enhancing self-protective behavior: efficacy beliefs and peer feedback in risk communication. *Risk Anal* 33:1252–1264
- Wynne B (1996) Misunderstood Misunderstandings: social identities and public uptake of science. In: Irwin A, Wynne B (eds) *Misunderstanding Science? The public reconstruction of science and technology*. University Press, Cambridge, pp 19–46

# Chapter 11

## Citizen Participation Approaches in Environmental Health

Sonja Grossberndt and Hai-Ying Liu

**Abstract** Environmental health is a very complex topic, as a whole range of factors are involved, many with high risks and uncertainty. The combination of complexity and uncertainty requires a different approach to support decision-making than traditional science has been offering to date. The last decades have shown that in order to support knowledge-based decision-making and to solve problems, knowledge and experience from different actors have to be taken into account through participatory approaches, including both traditional science and the general public.

The overall objective of this chapter is thus to demonstrate the role of public participation in the field of environment and health. We define the role of and present common approaches for public participation as well as elaborate further on two rather new approaches: Citizen Science and Citizens' Observatories. At the end, we discuss some of the challenges involved and identify the development needs in public participation.

**Keywords** Public participation • Citizen science • Citizens' observatories • Environment and health • Environmental governance • Decision-making

### 11.1 Why Public Participation?

Health can be subject to a range of factors. The World Health Organization (WHO) defined the following determinants of health that can influence the health condition of each individual to a stronger or lesser degree: income and social status, education, physical environment, social support networks, genetics, health services, and gender (WHO 2015a). We know that a diversity of environmental factors can harm human health, such as air pollution from burning fossil fuels and biomass (WHO 2015b), noise from transportation (WHO 2015c) or use of pesticides in agriculture (WHO

---

S. Grossberndt (✉) • H.-Y. Liu  
Department of Environmental Impacts and Economics (IMPEC),  
NILU – Norwegian Institute for Air Research, Kjeller, Norway  
e-mail: [sg@nilu.no](mailto:sg@nilu.no)

2015d), etc. On the other hand, different environmental factors have the potential to influence our health into the opposite direction, such as the benefit of urban green spaces (WHO 2015c) or the intake of fruit and vegetables (WHO 2015d).

Environmental health is a very complex topic, as a whole range of factors with to some extent high risks and the predominance of uncertainty determinate it. The combination of complexity and uncertainty requires a different approach to both communicate scientific results, risks and to support decision-making than traditional science is offering.

Over hundreds of years, scientists have erected an ivory tower, keeping the ignorant public with their negative attitude towards science outside (e.g., Frewer 2004; Ahteensuu 2012). Instead, the public should be fed only with bits and pieces of what scientists consider “objective scientific knowledge” to become aware of and tackle problems. Wynne (1996) refers to this phenomenon as deficit model. This deficit is not only symptomatic for science, but also for policy. During the last decades, such deficit has been dealt with in different political contexts. One of them is the so-called Aarhus Convention, a document that meets the needs of the public towards the “right to know”: the convention grants the public rights to access information and participate in decision-making. It focuses on the interaction between the public and public authorities and is an important milestone towards considering the “right to know” as democratic right (UNECE 1998). Thus, environmental health requires a rethinking on both ends, the scientific and the policy-making. This is where participation comes into play.

Science cannot always be communicated simple, certain or easily (Ahteensuu 2012), especially where “facts are uncertain, values in dispute, stakes high and decisions urgent”, as it is the case in environmental health (Funtowicz and Ravetz 1991). Efforts have been made to move away from the traditional one-layer top-down to multi-layers bottom-up communication processes towards more participation in decision-making (Frewer 2004; Liu et al. 2014). However, still more endeavour has to be made from the academic side to find out about what kind of knowledge is actually relevant in certain situations and to what extend (Ahteensuu 2012). At the same time, decision-makers have to align their decision-making strategies to adopt to changing circumstances and create a sustainable planning and stewardship of environmental resources by including a diversity of knowledge and expertise from different stakeholders (Litke and Day 1998; Reed 2008). This includes not only scientists, but also citizens and other stakeholder groups (Fiorino 1990).

In their White Paper on Environmental Governance, the European Commission (EC) is calling upon different actors to cooperate jointly in the context of environmental governance. This includes both, problem framing, generating and evaluating of problem-solving options, and coming to a joint conclusion. This White Paper is not only calling upon decision-makers and scientists, but also requires including representatives from civil society (EC 2001). This approach is also described as inclusive governance, a concept that actively engages different stakeholders in the decision-making process, including their expertise and ideas. Inclusive governance in relation to decision-making requires the main aspects of the following (Renn and Schweizer 2009):

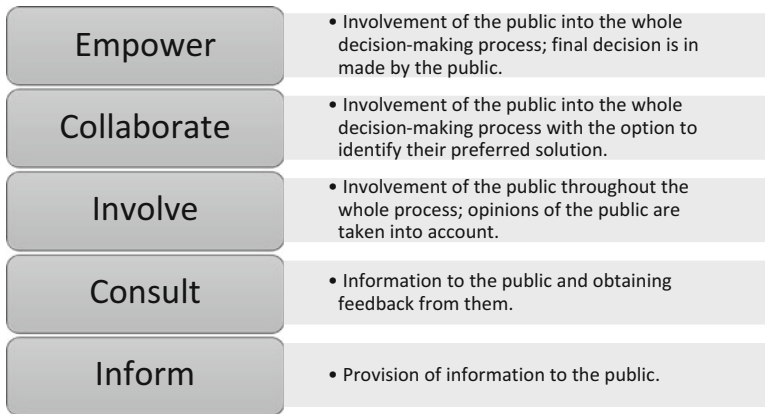
- Involvement of representatives of all relevant stakeholder groups;
- Empowerment of all participants towards an active and constructive participation;
- Co-design in problem framing through a dialogue with participants;
- Generating a common understanding of the problem, potential solutions and (expected) consequences based upon each individual's experience;
- Conducting a forum for decision-making that provides equal and fair opportunities for all stakeholders to express their opinions and views;
- Establishing a connection between the participating decision-makers and the political implementation of the process outcome.

In order to support knowledge-based decision-making and to solve problems, knowledge and experience from different actors have to be taken into account through participatory approaches. This chapter provides a general introduction into public participation in environmental health governance. We do not have the ambition to draw a complete picture of the whole participation landscape. Rather, we will provide a sketch of the whole picture and highlight two approaches that have become popular amongst scientists during the last decades: Citizen Science and Citizens' Observatories.

## 11.2 Public Participation in Environmental Governance: A Process

Public participation is generally described as the involvement of the public in policy making through taking part in setting the agenda, making decisions and forming policies (Rowe and Frewer 2005). However, this description does not define to what extent the public is actually involved in the participation process, and in fact, different degrees of active involvement can be distinguished. Arnstein's ladder of participation was the first visualisation of different levels of participation from almost non-participatory forms on the lower rungs all the way up to citizen power/control on the higher rungs (Arnstein 1969). This classification has been modified several times, leading to more recent visualisation, as for example by the International Association for Public Participation (IAP2) (Fig. 11.1). Similar distinctions can for example be found in Shirk et al. (2012) and Conrad and Hilchey (2011).

Based on the categorisation made by IAP2, we can see that the first level is a more passive one for the public. Here, they only receive *information* without having the opportunity for feedback or any further form of involvement. At the next level, the public acts as sort of *consultant*. Depending on the information they obtain, the public can provide their feedback. The level of actual *involvement* enables the public to participate throughout the whole process, taking into consideration their opinions and questions. Nevertheless, it is not before reaching the level of *collaboration*



**Fig. 11.1** Levels of public participation (modified with permission from IAP2 2014)

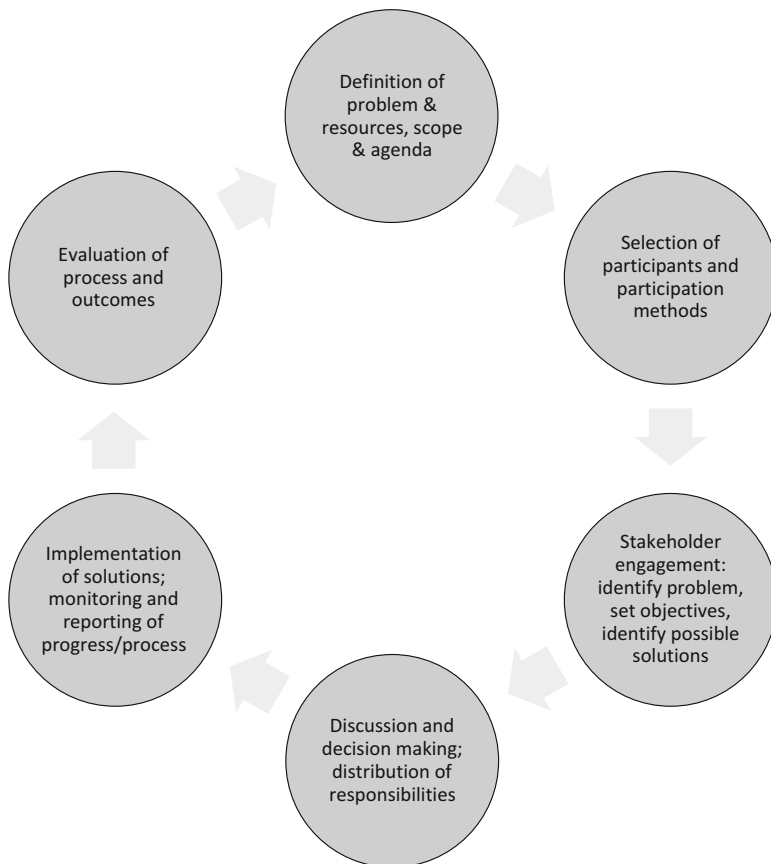
that they are also involved into the completely decision-making process with the option to identify their preferred solutions. At the level of *empowerment*<sup>1</sup> then, the public is also making the final decision.

Participation that involves citizens more actively is a dynamic process that comprises of the following elements (Fig. 11.2):

A participatory process consists of six major parts. It starts with the *problem definition*, an analysis of the current state, where problems, resources, scope and agenda of the participation process should be defined. After a thorough analysis, the *selection of stakeholders and participation methods* is the next action, followed by the actual *stakeholder engagement*. Here, the topic of discussion has to be identified, objectives made clear and potential solution scenarios should be created. During the *discussion and decision making* phase, all participants should have the opportunity to define their position and actively engage in the actual discussion process. After agreeing on a solution, responsibilities have to be clearly defined and distributed, to guarantee a smooth *implementation*. Here, monitoring of the process and reporting of any progress are the key actions that have to be *evaluated* in the next step. Depending on the result, the process will either be finalised, or lead to another round to improve the situation.

The next paragraphs will provide additional information for each phase.

<sup>1</sup>The term “Empowerment“ describes a process that enables people to make decisions and gain greater control over their personal life (WHO 1998). We can find the concept of empowerment in different settings, ranging from empowering of employees in organisations to legal empowerment, economic empowerment, and community empowerment or in the context of health (e.g., Wilkinson 1998; Piper 2010; Open Society Foundations 2014; UNDP 2015). The Ottawa Charter for Health Promotion identified already back in 1986 community empowerment as one of the central concepts in health promotion to enable citizens to set priorities, make decisions, plan and implement strategies, that would ultimately lead to improved health for each individual (WHO 2015e).



**Fig. 11.2** Design of a participatory process (modified with permissions from Richards et al. 2007)

**Problem Definition** The first step in the participatory process is to define the topic of discussion and decide what kind of decision-making process should be applied. A participatory approach might not always be required and not all stakeholders would expect or demand to be involved. Some people might have made negative experience attending previous participative processes that result in consultation fatigue. Sometimes it might be a question of available resources, since actively engaging different stakeholders requires both time, personal resources and monetary support (Richards et al. 2007; Haklay 2015).

**Selection of Participants and Methods** After deciding for a participatory method, the next decision will be who to involve. Generally, all interested and affected parties should be represented, with special efforts on including those groups that are difficult to reach (such as young or elderly people, people with migration background, homeless, etc.) (Richards et al. 2007; Roy et al. 2012). In order to be most effective, participatory processes should include citizens with the strongest interest

and goals in the results (Reed 2008). One has to ensure that the participants are included as early as possible in the participation process and have not only the power to actually influence decisions, but also the technical means to do so (Reed 2008; Renn 2008). Actively involving citizens in decision-making can also help them developing confidence and capacity to engage further in the political and community life in their society (Richards et al. 2007).

Several other conditions have to be met in order to increase the probability of a positive result of the participation process. First, stakeholder communication and participation have to be based on mutual trust. Trust first of all in the facilitator (or moderator) who should be perceived as trustworthy, honest and impartial, able of handling challenging situations and participants and managing a reflective discourse, but also between the participants and stakeholders involved in that process (Richards et al. 2007). The facilitator has to create an environment of openness and equality within the group, taking into consideration each individual expertise and background, gender, age and if possible, personality. This implies a good knowledge of the participants, the process, and expected outcomes and requires a thorough preparation phase, especially when identifying the different stakeholder groups (i.e., participants) (Reed et al. 2009). Depending on the available resources, the conduct of a stakeholder analysis and their relationships can be more or less extensive (for different stakeholders analysis methods, see e.g., Reed et al. 2009).

The appropriate choice of participation methods is a crucial precondition to guarantee a successful outcome of the engagement discourse. Rowe and Frewer (2005) have carried out an extensive literature review on activities that have been used in the context of “participation”, going back until 1975. Their list compiles 102 different mechanisms, ranging from “Act Create Experience” to “Whole System Development”. By now, there will certainly be more. Rowe and Frewer (2005) argue that this multitude and diversity create confusion and uncertainty regarding their use and application. In order to ensure effectiveness, they suggest to categorize engagement activities based on their information flow, and propose the following categories: communication, consultation, and participation. Participation methods also have to be aligned according to the decision-making context and socio-cultural and environmental factors (Reed 2008).

**Stakeholder Engagement** Another precondition for a successful outcome of the participatory process is the clear definition of its objectives. This might cause at first disagreement between the participants who might have different views on the topics of discussion. If however, the creation of an environment of fairness, openness and mutual trust is successful, the controversy can be a fertile ground for a so-called analytic-deliberative (participative) process (Reed 2008; Renn 2008; Richards et al. 2007). This approach underlines the importance of combining analysis and deliberation, leading to the more acceptable decisions of the involved stakeholders. Thus, it is crucial to include different experts and perspectives in the decision-making process (Renn 2008).



If the participation process is leading towards a clear framing of problems and objectives, it is more likely that the participants will take ownership of the process, build partnerships and stay motivated to be actively engaged throughout the whole time. In this context, the participants will have a better understanding of potential limitations (often due to legislation) and that not all their expectations can be fulfilled (Richards et al. 2007). Needless to say that an active participation and inclusion of all stakeholders will be beneficial for the outcome, since the results will be relevant for their own needs and priorities (Reed 2008). According to Lennox et al. (2011), effective stakeholder participation will be granted if the following preconditions are met:

1. Adequate time spent in preparation of participants, help them understanding their roles and responsibilities, the input they will provide and the communication with each other;
2. Ensure that stakeholders input will be treated confidential and promote in-depth, relevant and useful discussions;
3. Provide proof that the process will actually provide meaningful and practical outcomes and is not just tokenistic.

**Discussion and Decision-making** Decision-making requires adequate information. One has to ensure that the participants have all necessary information available in a way that is understandable for them (Lennox et al. 2011). As stated earlier, problems related to environment and health are rather tricky. It is a very complex topic where many interdependencies, uncertainty and high risks are involved. Processes based on analytical methods only provide facts and value judgements, the choice of decision criteria, and facilitate evaluation. Participatory processes, on the other hand, making explicit divergences, facilitate a consensus with high legitimacy, and can initiate a dynamic learning process. Applying an analytical-deliberate approach means to contribute to both, scientific and non-scientific knowledge. This combination seems to be a suitable solution to overcome deficiencies of traditional decision-making tools (Rauschmayer and Wittmer 2006).

**Implementation and Monitoring** Rather challenging will be the implementation of the outcomes that have been agreed upon in the previous phase. Especially public authorities are known for slow reaction towards changes. This is why the implementation and monitoring phase is so crucial.

Monitoring describes the continuous process of assessing the status of implementation in relation to the agreed implementation plan and available resources, with the aim to effectively manage outputs (UNEP 2015). Good monitoring includes the following main aspects (UNDP 2002 quoted in UNEP 2015):

- Focussing on results and follow-ups;
- Communication carried out by the facilitator on a regular basis;
- Regular analysis of project-related reports;
- Participatory monitoring mechanisms should be applied to ensure both commitment, ownership, follow-up, and feedback on the project performance;

- (Objective) assessment of progress and performance by criteria and indicators designed and agreed upon by all participants;
- An active generation of lessons learned.

The facilitator should also report all activities for documentation purposes and make sure that all project reports are submitted on time. Reports do not only fulfil the purpose of presenting results and outcomes, but also to document progress, successes and failures of the initiative in the light of evaluation and as reference for future initiatives (UNEP 2015).

**Evaluation** Evaluation of participation practices is considered an important step, albeit the complexity and diversity of issues that have to be taken into account in environmental health governance. Different meaningful attempts have been promoted and tested in the last decades to evaluate public participation activities. Since there are different perspectives on different participation initiatives, it is impossible to define “the one and only” or “the best” evaluation framework. One more general example is the framework proposed by Ran (2012). It comprises of the four criteria equity, effectiveness, efficiency and social learning. For each criterion, both process and outcome of public participation are evaluated. Ran (2012) considered this framework as a tool for policy-makers to apply a more structured and comprehensive way of evaluating public participation.

We should not only evaluate the outcome, but also the participation process itself. Bickerstaff et al. (2002) conducted a survey of English highway authorities and a content analysis of policy documents, applying four criteria that they considered “key principles” of a participation process, namely inclusivity, transparency, interactivity and continuity. These criteria can be amended by a list from Wittmer et al. (2006) who was evaluating information, legitimacy, social dynamics and costs.

Since the objectifying of evaluation criteria is especially challenging in analytical deliberative participation processes due to their complexity, Rauschmayer et al. (2009) proposed to involve different actors in the design of evaluation criteria by applying a participatory evaluation approach. The outline of a participatory evaluation will vary, depending on the underlying understanding of participation and the different participants. However, all stakeholders involved should agree upon the design of the evaluation process after the concept of the participation process has been defined. This will not only provide more clarity to all participants, but can also help avoiding unwanted shifts within the participation process (Nitsch et al. 2013). Participatory evaluation should be applied in every stage of the participation process. Nitsch et al. (2013) developed a framework to plan and analyse the individual phases of a participatory process. They distinguished it in six phases: (1) Evaluation design; (2) Data collection; (3) Data analysis; (4) Developing recommendations; (5) Reporting on findings; and (6) Dissemination of findings. Every phase is evaluated based on three indicators (apart from phases one and four, where the operative performance aspect is missing): Social input (decision/power, consultation and information), factual input (deliberation, voice perspectives, inquiring perspectives) and operative performance. Application of this framework can contribute to knowledge sharing and building of evaluation skills of all participants and thus strengthen participation throughout the whole process.

### 11.3 Citizen Science: A Novel Old Approach

So far, we have only focused on public participation in the context of decision-making processes. However, involving members of the public can also be beneficial within scientific research. First, it brings advantages for the scientists who often suffer from a constraint of time and resources to collect large data (Dickinson et al. 2010). Engagement of citizens in data collection can also be the only practical way to achieve the geographic reach required for data collection and documentation (Tulloch et al. 2013). With regard to benefits for the citizens, involving citizens in science will lead to an increased awareness of problems related to their immediate environment and can result in a larger interest and increased engagement activities in these issues. Citizens' engagement can also have educational effects and increase science literacy (Evans et al. 2005; Haklay 2015).

Literature provides an array of different terms to describe the participation of citizens in science. Shirk et al. (2012) suggest the term *Public participation in scientific research* (PPSR) to summarise the different initiatives in this field. However, the term *citizen science* has gained more popularity during the last decade. Citizen science is commonly defined as “scientific work undertaken by members of the general public, often in collaboration with or under the direction of professional scientists and scientific institutions” (Oxford English Dictionary 2014).

Observations of nature, carried out by volunteers have been carried out for centuries. Already back in 1874 joint efforts have been made internationally to collect data from all around the world under the Venus transit (The Royal Society 2015). Another more popular example is the traditional Christmas Bird Count that has been initialised in 1900 and has been attended by more than 70,000 volunteers in 2013/2014 (National Audubon Society 2015). Nowadays, a large number of initiatives have been initiated and are still ongoing, covering different fields such as for example ornithology (e.g., The Cornell Lab of Ornithology; [www.birds.cornell.edu](http://www.birds.cornell.edu)), astronomy (e.g., Galaxy Zoo; [www.galaxyzoo.org](http://www.galaxyzoo.org)), environmental monitoring (e.g., mosquito atlas; [www.mueckenatlas.de](http://www.mueckenatlas.de); Open Air Laboratories Network (OPAL); [www.opalexplorenature.org](http://www.opalexplorenature.org)), or geography (e.g., OpenStreetMap; [www.openstreetmap.org](http://www.openstreetmap.org)). In fact, the list of citizen science programmes is endless.

During the last decade, citizen science activities have experienced quite a boom and have become fairly advanced. The main reasons for this phenomenon were the rapid changes in the development of and access to the internet (especially the emergence of Web2.0 systems and social media that simplified the engagement of the public), the improvement and simplification of data collection/management, and the introduction of smart phones and other mobile devices. Smart technologies have opened new modes in collecting and reporting data and enabling communication, by e.g., taking pictures and collecting other data on site and sending them through wireless technologies to places on the other side of the world (examples of these initiatives can be found at Haklay 2015). On the other hand, societal changes such as increased public educational levels, more leisure time and increased understanding of scientific concepts have also contributed to the emergence of citizen science.

### 11.3.1 *Policy Dimensions*

Citizen science does not only serve scientific purposes, but also decision-making, since the collected data can contribute to informed policy-making. In addition, citizens can benefit by addressing the environmental issues that affect them directly in the context of participatory decision-making. In this way, the value of lay knowledge should not be underestimated (Science Communication Unit 2013).

According to Haklay (2015), the following three policy dimensions can be distinguished: (1) level of geography; (2) policy domains; and (3) level of engagement and type of citizen science activity. Citizen science initiatives can influence policy decisions in a specific geographical area, i.e., local, regional, national and international. Usually, problems that affect the direct environment of people lead to more engagement since people are more concerned (Haklay 2015). This potential can be used to build upon observation activities through citizen science initiatives. Local citizen science is often linked to environmental activism and supports community management by working towards effective and meaningful management planning, management and stewardship (Conrad and Hilchey 2011). Local citizen science can also apply the so-called community-based monitoring approach (CBM). CBM describes a process where concerned citizens, public authorities and further stakeholders collaborate to monitor, track and respond to issues that arise from common community concern (Whitelaw et al. 2003).

Environmental issues are very complex and would actually require additional resources. Thus, there is an increasing need for communities to fall back on citizen science approaches (or CBM respectively) and include different stakeholders with their diverse knowledge and experience into the decision-making processes (Conrad and Daoust 2008). Next to the decision-making bodies' saving time and money, the societal benefits of CBM will be creating environmental democracy, social capital, and increase in scientific literacy and inclusion in local issues (Conrad and Hilchey 2011).

Policy areas can be manifold and partially overlapping. For example, city-scale infrastructure contains public transport, environmental quality, education, infrastructure and public health. Thus, cities can be a canvas for a potpourri of local monitoring activities, originating from different concerns but using the accumulated data to see the bigger picture. Moving citizen science projects to regional, national or even international level is likely to meet even more challenges than there already are. Since bottom-up initiatives usually dispose of limited budgets only, it will be less likely to find community science approaches with an active involvement of citizens in all parts of the participation cycle, i.e., citizens will rather only be asked to share observations or viewpoints on certain issues. Nevertheless, national and even international initiatives including citizen science are possible and do exist. Projects funded by the EC and formation of international organisations like the European Citizen Science Association (<http://ecsa.biodiv.naturkundemuseum-berlin.de/>) provide frameworks for national initiatives and Non-Government Organizations (NGOs) to create synergies to promote citizen science on larger scale and call on international institutions such as the European Environmental Agency (EEA) to promote citizen participation also on international level (Haklay 2015).

### 11.3.2 Types of Involvement

However, how can people actually be involved in citizen science? Haklay (2015) distinguishes different levels of engagement within citizen science (Fig. 11.3). As we can see, these categories resemble very much those participation levels presented earlier in this chapter. The first level is called *passive sensing* and describes a process where information is collected without any effort on the participants' part through their own devices. *Volunteer computing* is a method where participants allow scientists to use their unused computing resources on, e.g., their pc or smartphone for complex computer models while the device is not in use. The next level, *volunteer thinking* makes use of the citizens' cognitive abilities not used during passive leisure activities, e.g., people contribute recognizing patters while watching TV. The obtained data will then be further used in scientific projects. *Environmental and ecological observation* describes the traditional form of citizen science, where volunteers monitor and/or observe their personal environment, often based on protocols that are designed by scientists. In *participatory sensing* activities, the citizens are more actively involved in designing both data collection and analysis. The last activity type can be summarised as *civic/community science*. In this bottom-up approach, the

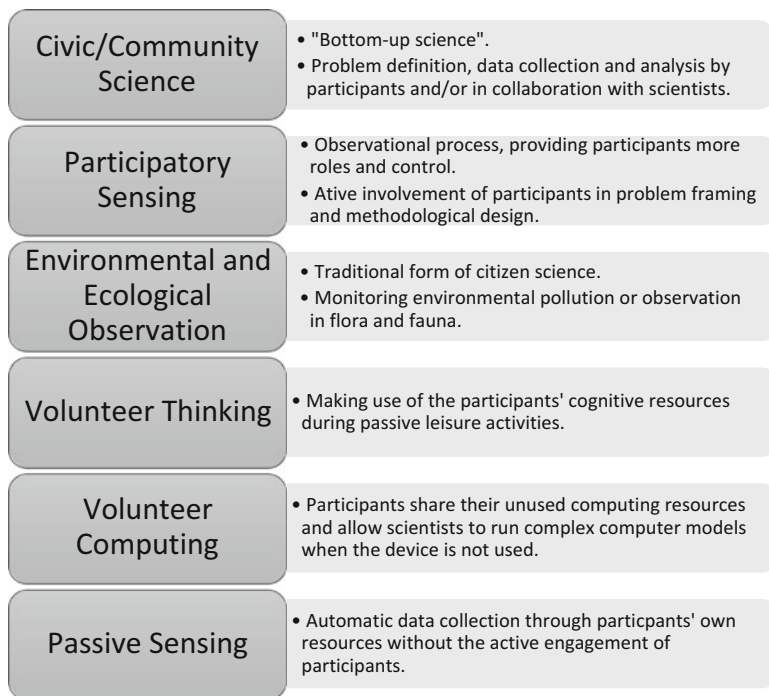


Fig. 11.3 Types of citizen science, modified from Haklay (2015)

participants can choose their level of engagement and can even be involved in the analysis and interpretation of the results and their publication/utilization. This level of engagement does not necessarily require the involvement of scientists.

Citizen science initiatives should be tailored to match both interest and skills of the participants. This is crucial in order to develop successful initiatives. Although motivations vary widely, we find that enjoyment and enthusiasm for the goals of the initiative are two of the main drivers. As known from other research areas, the feeling of control over the scientific process can also lead to long-lived engagement (Roy et al. 2012).

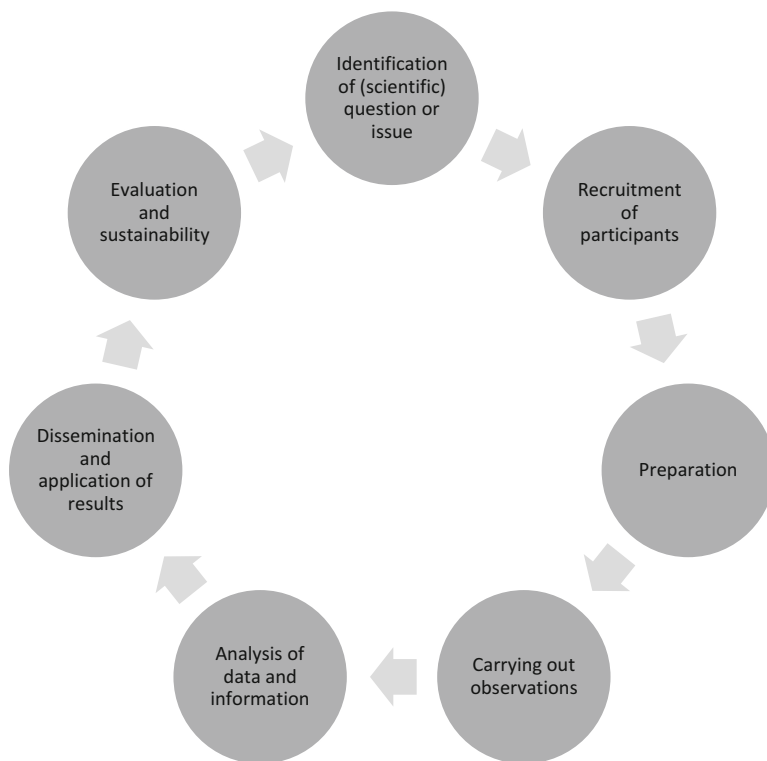
### 11.3.3 *Framework for Citizen Science Initiatives*

A number of frameworks have been created for the design and implementation of public participation in scientific research. Figure 11.4 is one example, merging the ideas from Bonney et al. (2009) and Shirk et al. (2012). Again, we can see the similarity to the process we described earlier with regard to public participation in environmental governance.

As public participatory initiatives per definition include collaborative endeavours, the project design should consider both, scientific and public interests during the *identification of issues*. The design can have major impact on the process outcome. After designing and developing a *recruitment* campaign, the *preparation* of protocols, forms and training material has to go hand in hand with the training of participants before *carrying out observations*. This part of the process has two outcomes: one being the observed data and information, the other one being the increase in personal skills, knowledge and experience the participants can harvest. After a thorough *analysis of obtained data and information*, they can be *disseminated* to the scientific environment, the public or any other interested stakeholders. In addition, the results should find *application* in legislation, policy-making, education or other areas. The last actions should be undertaken with regard to *evaluation*, especially in terms of maintaining participants and keeping the project *sustainable* (Bonney et al. 2009; Shirk et al. 2012).

## 11.4 **Citizens' Observatories: A New Approach**

In recent years, there has been a boom in citizen science projects, which allow members of the public to play an active part in monitoring and recording environment across the globe (See Sect. 11.3). At the same time, the novel term 'Citizens' Observatories' (COs) is emerging as an increasingly essential tool that provides an approach for better observing, understanding, protecting and enhancing our environment (Liu et al. 2014). However, there is no consensus yet on how to develop such a system, nor is there any agreement on what a Citizens' Observatory (CO) is



**Fig. 11.4** Design of a participatory process in citizen science (modified from Bonney et al. 2009 and Shirk et al. 2012)

and what results it could produce. The increase in the prevalence of COs globally has been mirrored by a growth in the number of variables that are monitored, the number of monitoring locations and the different types of participating citizens. This calls for a more integrated approach to handle the emerging complexities involved in this field; but before this can be achieved, it is essential to establish a common foundation for COs and their usage. There are many aspects related to a CO. One view is that its essence is a process that involves environmental monitoring, information gathering, data management and analysis, assessment and reporting systems. Hence, it requires the development of novel monitoring technologies and of advanced data management strategies to capture, analyse and survey the data, thus facilitating their exploitation for policy and society. Practically, there are many challenges in implementing the COs approach, such as ensuring effective citizens' participation, dealing with data privacy, accounting for ethical and security requirements, and taking into account data standards, quality and reliability. These concerns all need to be addressed in a concerted way to provide a stable, reliable and scalable COs programme. On the other hand, the COs approach carries the promise of increasing public awareness for risks in their environment, which has a corollary economic value, and enhancing data acquisition at low or no cost.

### 11.4.1 *What Is a Citizens' Observatory?*

There is no clear definition of a CO available yet. Ciravegna et al. (2013, p. 1) first defined a CO as

a method, an environment and an infrastructure supporting an information ecosystem for communities and citizens, as well as emergency operators and policymakers, for discussion, monitoring and intervention on situations, places and events.

In the broadest sense, a CO for supporting community-based environmental governance can be defined as

the citizens' own observations and understanding of environmentally related problems and in particularly as reporting and commenting on them within a dedicated ICT (Information and Communication Technology) platform (Liu et al. 2014, p. 4).

As such, the CO promotes communication and supporting sharing of technological solutions (e.g., sensors, mobile apps, web portals) and community participatory governance methods (e.g., aided by various social media streams) among citizens. This definition reveals three core components that underpin some of its objectives, i.e., raising the citizens' environmental awareness; enabling dialogue among citizens, scientists and policy- and decision-makers; and supporting data exchange among citizens, scientists and other stakeholders (Liu et al. 2014; Kobernus et al. 2015). It also promotes a more active role for the community concerning understanding of the environment, since citizens are traditionally considered merely consumers of information services at the very end of the information chain and not as data providers (Copernicus 2015). Furthermore, COs support open and democratic programmes, enabling the possibility for anyone who is interested or willing to contribute to and participate in earth observation and environmental conservation (ELLA 2013; Holohan 2013).

Building on the countless ongoing activities in this area, The EC has seen the opportunity to deliver improvements to European citizens, and at the same time increase observational capabilities. The EC has supported five projects with the aim to build COs in the fields of marine water quality, flood protection, odour nuisance reduction, biosphere reserve management, and urban life quality targeting air pollution, comfort and school indoor environment. For example, the Omniscientis project combined the active participation of the citizens with the implementation of innovative technologies to improve the governance of odour nuisance (Omniscientis 2012–2014). CITI-SENSE aims at empowering citizens to participate in environmental governance by developing various COs supporting services related to outdoor air quality, indoor school air quality and environmental perception in public spaces of societal concern (CITI-SENSE 2012–2016; Castell et al. 2014; Engelken-Jorge et al. 2014; Liu et al. 2014). COBWEB aims to create a test bed environment that will enable citizens living within Biosphere Reserves to collect environmental data using mobile devices (COBWEB 2012–2015). Citclops aims at developing an observatory based on citizens' science applications for the bio-optical monitoring of coast and ocean (Citclops 2012–2015). WeSenseIt (WeSenseIt 2012–2016) is a

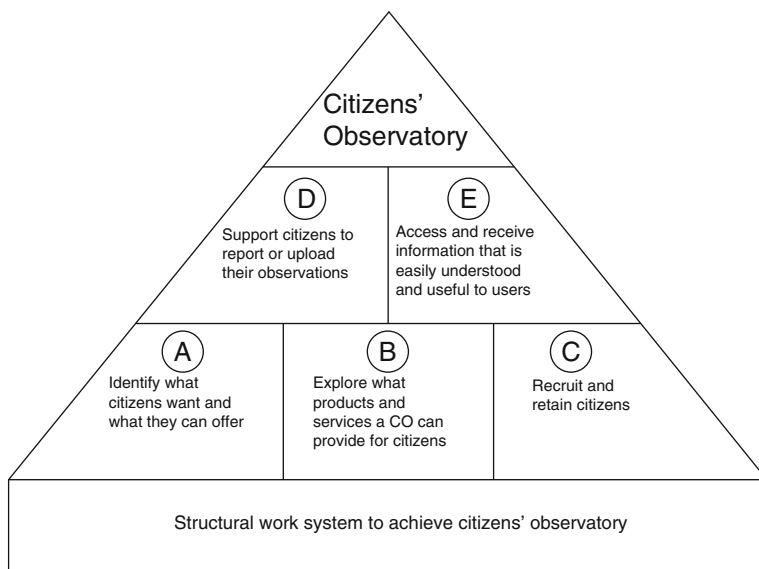


project that puts an emphasis on enabling citizens to become active stakeholders in information capturing, evaluation and communication for the water environment including flood risk (Ciravegna et al. 2013; Lanfranchi et al. 2014).

### 11.4.2 Citizens’ Observatories Framework

To establish a CO and to make it useful for society, there is need for researchers to collaborate with citizens, citizen groups and their representatives, and with the representatives of the local authorities, to identify interests and needs (Liu et al. 2014). In a CO, all parties shall be engaged as active participants, to create knowledge about the environmental situation in a participatory manner and to contribute to dealing with the situation (Lanfranchi et al. 2013). One of the main aspects of a CO is the need to effectively address citizens participation in data collection, data interpretation and information delivery. According to Liu et al. (2014), this can be expressed as the following set of five sequential aspects that underlie the COs’ skeleton and support effective citizen participation (Fig. 11.5):

- (A) Citizens’ participation in identifying what citizens want and what a CO can offer to provide information and knowledge in response to public concerns. This is achieved mainly by a dialogue among the stakeholders;



**Fig. 11.5** Sequential aspects of a Citizens’ Observatory programme, reproduced from Liu et al. (2014), operating under the terms of the Creative Commons Attribution License

- (B) Citizens’ participation in exploration of what products and services a CO can provide for the citizens. This involves systematizing and structuring citizens-created content to make it appealing for use by citizens during their normal daily life;
- (C) Recruiting and retaining citizens to participate in and contribute to environmental governance: further clarify the purpose, scope and expected impact of the CO, identify motivations that will promote citizens to contribute to and take part in the CO, and encourage public participation in data collection and interpretation;
- (D) Obtaining public participation in the relevant decision making and/or in changing their related personal priorities and behaviour by gaining access to environmental data, knowledge and experience, and by using tools that can support citizens to report or upload their objective/subjective observations, inference and concerns;
- (E) Supplying tools to access/receive timely information on the relevant environmental issues in a manner that is both easily understood and useful to the users.

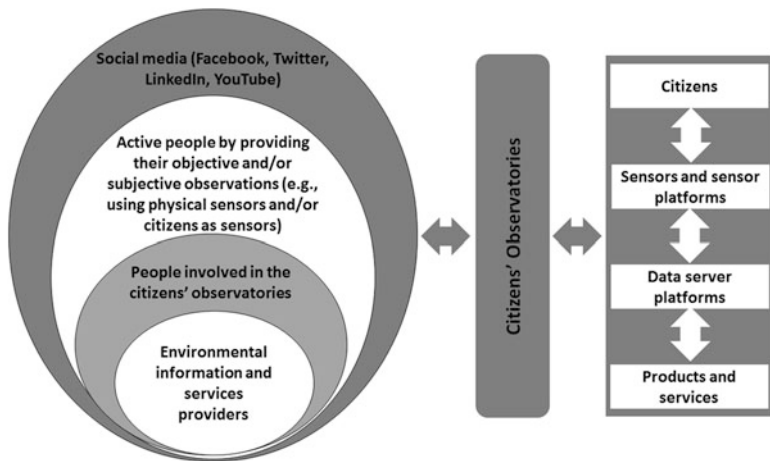
### ***11.4.3 Citizens’ Observatories Model in Practice***

A key concept in the operation of COs is the idea of a campaign that specifies stakeholders’ concerns by defining the types of data that need to be collected and by describing the goal, expected feedback and analysis outcomes in terms of maps, statistical results, etc. The *raison d’être* of the observatory then is to “run and orchestrate” a campaign in order to assure that sufficient data is gathered, both qualitatively and quantitatively, and to enable non-expert stakeholders to define, monitor and analyse campaigns in a way understandable to them (D’Hondt et al. 2014; Zaman et al. 2014).

Figure 11.6 illustrates a common model of COs: A set of concentric circles that are characterised by different types of information needs and of information gathered and shared (left part). The circles represent different types of stakeholders: environmental information and services providers and people actively involved in COs (e.g., public, researchers, policy-makers, small and medium-sized enterprises (SMEs), etc.). In addition, the circles also represent different tools that different stakeholders may use to provide their observations and share their data and information, e.g., innovative low-cost physical sensors, citizens as sensors, and social media, etc. The aim of the COs approach is to support all stakeholders by designing various tools and applications that support co-participation.

In addition to the different stakeholders and the way to engage such stakeholders, Fig. 11.6 also illustrates the four core components of the data flow model in various COs projects (right part).

- Citizens: Different types of stakeholders, including the general public, COs participants (i.e., involved either in monitoring through sensors or by using and co-developing products and services, or by both means, and other groups using COs products and services.



**Fig. 11.6** A common citizens’ observatories model in practice (modified with permissions from Lanfranchi et al. 2013 and Liu et al. 2014)

- Sensors and sensor platforms: Technologies for environmental monitoring (i.e., static and portal sensors and sensors platforms).
- Data server platforms: Information and communication technologies (e.g., web-featured service).
- Products and Services: Information products and services, i.e., mobile apps, web applications, social media networks, public environmental survey, etc.

The first three components (citizens, sensors and sensor platforms, data server platforms) are the main elements of participatory sensing, reflecting the concept of communities or other groups of people contributing information in the form of data and knowledge (Dua et al. 2009). The fourth component (products and services), linked to the usage of obtained data and knowledge, is designed and expected to be used by various stakeholders for different purposes. These components can be considered as the four pillars that support various COs in practice (Engelken-Jorge et al. 2014). They relate to the stakeholders’ experience, expertise and expectations and are clearly important. Within these four pillars, various actors may use the tools or instruments to produce raw data that leads to a range of new products or services as well (Liu et al. 2014). Here, we present a common model of COs which is being translated into practice in several existing COs projects, i.e., Citclobs (<http://www.citclops.eu>), CITI-SENSE (<http://www.citi-sense.eu>), Citi-Sense-MOB (<http://www.citi-sense-mob.eu>), COBWEB (<https://cobwebproject.eu>), EVERYAWARE (<http://www.everyaware.eu>), Omniscientis (<http://www.omniscientis.eu>) and WeSenseit (<http://www.wesenseit.com>). All of these projects put an emphasis on delivering highly innovative technologies to support citizens, communities and authorities by getting ‘near real-time’ and/or ‘up-to-date’ data about environmental conditions (e.g., air quality, water quality) and situations (e.g., flooding), while respecting the different information needs and actions respond. On

a technical level, these projects consists of a combination of crowdsourcing and custom applications designed to empower and foster participation with the objective of creating an enriched information and knowledge base to facilitate decision making while increasing opportunities for citizen engagement in their community (Omniscientis 2012–2014; Ciravegna et al. 2013; Lanfranchi et al. 2013; Liu et al. 2014). These projects base data capture on one or several of the following tools:

- A set of innovative low cost sensors for monitoring environment that are designed and can be used directly by both professionals and citizens alike (CITI-SENSE 2012–2016; WeSenseIt 2012–2016);
- A set of public environmental perception surveys (“citizens as sensors”) designed to be used by citizen participants (CITI-SENSE 2012–2016);
- Tools that enable the exploitation of the citizens’ collective intelligence through crowdsourcing (e.g., Volunteered Geographic Information (VGI)) (CITI-SENSE 2012–2016; WeSenseIt 2012–2016) and social media platforms (e.g., Facebook page, Twitter account) analysis (CITI-SENSE 2012–2016; EVERYAWARE 2012–2016; WeSenseIt 2012–2016).

## 11.5 Challenges of Participation Approaches

Both citizen science initiatives and COs require a strict data management. Since most of the citizen science initiatives take place outside organisational frameworks, special efforts have to be made to guarantee the quality of the obtained data. This can be reached through either controlling the quality of data during their acquisition or subsequently after their acquisition by comparing them with reference data (Goodchild and Li 2012). The first approach requires the introduction into and training of data collection and/or interpretation methods. Here, participants receive instructions and guidelines (e.g., provision of standardized equipment, instruction sheets, online training, etc.) on the use of the measuring device and how to collect data. In the case of VGI, a certain quality assurance is guaranteed when several participants deliver the same GPS (Global Positioning System) coordinates for the same object. In some occasions, some participants can also be asked to monitor and validate the data collected by participants with less experience, as for example in bird watching activities or monitoring of invasive species. Another example is Wikipedia, where a group of individuals with special rights acts as moderators or gatekeepers to avoid vandalism, remove copyrighted materials and resolve conflicts. In other occasions, it can be valuable to use existing knowledge of citizens to evaluate the validity of the data from volunteers. For example, by using existing knowledge about the fauna in Norway, it is very unlikely that a giraffe will be spotted in the streets of Oslo (Goodchild and Li 2012; Haklay 2015).

Next to data issues, there are a number of further challenges for the implementation of public participation. In their review of citizen science and community-based initiatives, Conrad and Hilchey (2011) provide a list of challenges of CBM. Apart from data issues mentioned above, these challenges include:

- Lack of interest of the volunteers;
- Lack of opportunities for networking;
- Funding difficulties;
- The participants, inability to get access to appropriate information and expertise;
- Insufficient experimental design;
- Utility of CBM data for, e.g., decision-making.

Another aspect that we have to bear in mind is the fact that even though international agencies, such as the EEA, promote approaches of inclusive governance processes, this does not mean that these advices are followed automatically on national, regional or even local level. Not only the willingness, but also the readiness of decision-makers has to be taken into consideration when designing and implementing any participative initiative. In this context, it should also be mentioned that even if the participative process has been carried out successfully, it should be strived to translate the prepared work into action (Litke and Day 1998).

Although the use of COs is becoming a more common practice for environmental management, challenges still exist also here regarding how to deal with the quality of the data collected as well as how to use it for environmental policy (Hanahan and Cottrill 2004; Goeschl and Jürgens 2012).

Such challenges suggest that the following areas should receive careful consideration (Liu et al. 2014):

- Data quality (i.e., accuracy and uncertainty)—especially when comparing crowd-sourced and reference data;
- Data privacy and security—sharing of data and information requires strong ethical and security considerations;
- Data interpretation—qualitative indicators such as “quality of life”, “wellbeing”, “happiness”, etc., should be developed in parallel with more quantitative indicators that are based not only on individual perception, but on an integrated sensor network;
- Systematisation and structuring of citizens-created content and feedback—establishing a viable model(s) to support decisions and empower the public (Engelken-Jorge et al. 2014);
- Involving and maintaining a broad spectrum of society—implementing various location-specific and target group-tailored tools in recruiting and sustaining citizens’ participation in environmental monitoring (Fernandez-Gimenez et al. 2008).

In practice, various COs all share a similar structure, yet in the current status, constructing a new CO for a new type of campaign (e.g., air pollution, mobility patterns of users of public transportation, biodiversity, climate change, etc.) requires all software infrastructure to be rebuilt from scratch (Liu et al. 2014). The lack of a systematic, easy and reusable methods for setting up new COs and for defining new campaigns poses an unsurmountable hurdle for communities and organisations as they usually lack the specific technical ICT-skills and programming knowledge to create the necessary server infrastructure and mobile applications. This often forces organisations to opt for a non-technological approach (i.e., pen and paper) or to spend big chunks of their restricted budget on external ICT-consultants (D’Hondt et al. 2014; Zaman et al. 2014).

In order to ensure a usable CO and to maintain citizens in a CO in practice, Liu et al. (2014) have addressed the following development needs:

- The need to adequately promote the COs platform and tools for raising awareness, recruiting and sustaining citizens' participation;
- The need of a good understanding of citizens' demographics in order to develop the COs' platform to meet their needs, especially as they change;
- The need to build a long lasting infrastructure that uses open standards, is easily exploitable through an open Application Programming Interface (API), can be widely accessed, extended and maintained, and is seen as a generic environmental enabler rather than a project specific outcome;
- The need to address and evaluate Citizens' Voice (Citizen' Views on certain environmental issues and its related environmental actions) and government Accountability (governments that can be held accountable for their environmental action) in the social and political context in which COs are embedded (DFID 2008; Fernandez-Gimenez et al. 2008), to actively promote the Citizens' Voice and Accountability concepts as important dimensions of good environmental governance (DFID 2008), to address COs potential role to influence environmental equity and to improve social justice (Kamar et al. 2012);
- The need to develop particular channels and mechanisms that can underpin the sound environmental-social-political actions in which COs are addressed, in a manner that facilitates citizens to influence environmental governing priorities and processes.
- The need to explore and further develop technologies, which deal with data collection and analysis by:
  - Building necessary technical capacity and overcoming the 'digital divide' for environmental monitoring, data exchange, visualizing and communicating results back to the broader users (DFID 2008; Brabham 2009);
  - Managing and analysing increasing data volumes, variety and velocity (Zikopoulos et al. 2011);
  - Reducing measurement uncertainties;
  - Developing reliable and fast quality assurance/quality control (QA/QC) tools that can work in real-time;
  - Increasing need for interdisciplinary use of data, integration of different types of data.

## 11.6 Conclusion

With this chapter, we have provided insight into a topic that creates ambivalent feelings for many, i.e., public participation. Based on our information from literature and practice we can see that during the last years, policy makers are beginning to value citizen science approaches as important tool in their decision-making process. The fact that issues related to environment and health are usually complex and uncertain calls for a

paradigm shift. Integrating knowledge and experience of different citizen groups and other stakeholders will help authorities in their decision-making process.

As we know, not one approach fits all. The choice of both participants and participation methods are always context-dependent and require a thorough preparation phase. Thus, time and personnel resources are crucial preconditions. Additionally, we see a need in fostering bottom-up approaches to a larger degree as it has been the case so long. In this context, we see COs as an increasingly essential tool for public participation in environmental monitoring. This approach assists citizens in observing and understanding environmental related problems, as well as reporting and commenting on them by help of advanced ICT.

We discovered that different ongoing COs and COs-related programmes in the environmental domain are sharing a common model that is significantly relevant for the outcome of the participatory process. It consists of the following elements: (1) Citizens are playing an active role in observing environment by using novel sensor-technologies and citizen as sensor approach; (2) Unique virtual places are created to gather and share data from a variety of sources; (3) Extraction and making use of relevant citizens-related data and providing multimodal services for citizens, communities and authorities; (4) Raising citizens' environmental awareness to form a strong public voice; and (5) Enabling dialogue among citizens, researchers, policy-makers and other stakeholders.

To be successful, we recommend the following sequential steps: (a) Identifying what citizens want and what citizens can offer; (b) Exploring what products and services a CO can provide for the citizens; (c) Recruiting and retaining citizens to participate in and contribute to environmental governance; (d) Providing tools that support citizens to report their observations, inference and concerns; and (e) Supplying tools to access/receive timely information on the environment in a manner that is both easily understood and useful.

With these elements in mind, a successful performance and sustainable implementation should not be impossible.

## References

- Ahteensuu M (2012) Assumptions of the deficit model type of thinking: ignorance, attitudes, and science communication in the debate on genetic engineering in agriculture. *J Agric Environ Ethics* 25:295–313
- Arnstein SR (1969) A ladder of participation. *AIP J* 35:216–224
- Bickerstaff K, Tolley R, Walker G (2002) Transport planning and participation: the rhetoric and realities of public involvement. *J Transp Geogr* 10:61–73
- Bonney R, Cooper CB, Dickinson J, Kelling S, Philipps T, Rosenberg KV, Shirk J (2009) Citizen science: a developing tool for expanding science knowledge and scientific literacy. *Bioscience* 59:977–984
- Brabham DC (2009) Crowdsourcing the public participation process for planning projects. *Plan Theory* 8(3):242–262
- Castell N, Kobernus M, Liu H-Y, Schneider P, Lahoz W, Berre AJ, Noll J (2014) Mobile technologies and services for environmental monitoring: The Citi-Sense-MOB approach. *Urban Climate*. doi:10.1016/j.uclim.2014.08.002

- Ciravegna F, Huwald H, Lanfranchi V, When de Montalvo U (2013) Citizen observatories: the WeSenseIt Vision. Proceeding of the infrastructure for spatial information in the European community (INSPIRE 2013), Florence, Italy, 23–27 June 2013, pp 1–2
- Citclops (2012–2015) Citizens' observatory for coast and ocean optical monitoring. <http://www.citclops.eu>. Accessed 2 July 2015
- CITI-SENSE (2012–2016) Development of sensor-based citizens' observatory community for improving quality of life in cities. <http://www.citi-sense.eu>. Accessed 2 July 2015
- COBWEB (2012–2016) Citizen observatory. <http://cobwebproject.eu>. Accessed 3 July 2015
- Conrad TC, Daoust T (2008) Community-based monitoring frameworks: increasing the effectiveness of environmental stewardship. *Environ Manag* 41:358–366
- Conrad CC, Hilchey KG (2011) A review of citizen science and community-based environmental monitoring: issues and opportunities. *Environ Monit Assess* 176:273–291
- Copernicus (Europe's Eye on Earth) (2015) Global monitoring for environment and security. <http://www.copernicus.eu>. Accessed 6 July 2015
- D'Hondt E, Zaman J, Philips E, Gonzalez Boix E, De Meuter W (2014) Orchestration support for participatory sensing campaigns. Proceedings of the 2014 ACM international joint conference on pervasive and ubiquitous computing (UbiComp 2014), ACM, New York, 13 Sept 2014, pp 727–738
- DFID (Department for International Development) (2008) Citizens' voice and accountability—evaluation mozambique country case study (final report). [http://diplomatie.belgium.be/en/binaries/evaluation\\_cva\\_mozambique\\_en\\_tcm312-64788.pdf](http://diplomatie.belgium.be/en/binaries/evaluation_cva_mozambique_en_tcm312-64788.pdf). Accessed 6 July 2015
- Dickinson JL, Zuckerberg B, Bonter DN (2010) Citizen science as an ecological research tool: challenges and benefits. *Annu Rev Ecol Evol Syst* 41:149–172
- Dua A, Bulusu N, Feng W, Hu W (2009) Towards trustworthy participatory sensing. In: Proceedings of the 4th USENIX conference on hot topics in security (HotSec'09). USENIX Association, Berkeley, CA, pp 8–8
- ELLA (Evidence and Lessons from Latin America) (2013) Citizen participation in Latin America: innovations to strengthen governance. <http://ella.practicalaction.org/node/1131>. Accessed 6 July 2015
- Engelken-Jorge M, Moreno J, Keune H, Verheyden W, Bartonova A (2014) CITISENSE consortium: developing citizens' observatories for environmental monitoring and citizen empowerment: challenges and future scenarios. In: Parycek P, Edelmann N (eds) Proceedings of the conference for e-democracy and open government (CeDEM14), Danube University Krems, 21–23 May 2014, pp 49–60
- European Commission (EC) (2001) European governance—a white paper. 2001/C 287/01
- Evans C, Abrams E, Reitsma R, Roux K, Salmonsén L, Marra PP (2005) The neighborhood nest-watch program: participant outcomes of a citizen-science ecological research project. *Conserv Biol* 19:589–594
- EVERYAWARE (2012–2016) Enhance environmental awareness through social information technologies. <http://www.everyaware.eu>. Accessed 6 July 2015
- Fernandez-Gimenez ME, Ballard HL, Sturtevant VE (2008) Adaptive management and social learning in collaborative and community-based monitoring: a study of five community-based forestry organizations in the western USA. *Ecol Soc* 13:4
- Fiorino DJ (1990) Citizen participation and environmental risk: a survey of institutional mechanisms. *Sci Technol Hum Values* 15:226–243
- Frewer L (2004) The public and effective risk communication. *Toxicol Lett* 149:391–397
- Funtowicz SO, Ravetz JR (1991) A new scientific methodology for global environmental issues. In: Costanza R (ed) *Ecological economics: the science and management of sustainability*. Columbia University Press, New York, pp 137–152
- Goeschl T, Jürgens O (2012) Environmental quality and welfare effects of improving the reporting capability of citizen monitoring schemes. *J Regul Econ* 42:264–286
- Goodchild MF, Li L (2012) Assuring the quality of volunteered geographic information. *Spat Stat* 1:110–120
- Haklay M (2015) *Citizen science and policy: a European perspective*. Woodrow Wilson International Center for Scholars, Washington, DC



- Hanahan RA, Cottrill C (2004) A comparative analysis of water quality monitoring programs in the southeast: lessons for Tennessee. <http://isse.utk.edu/wrcr/programs/projects/pdfs/mainbook.pdf>. Accessed 6 July 2015
- Holohan A (2013) Community, competition and citizen science: voluntary distributed computing in a globalized world. Ashgate, Farnham, Surrey
- International Association for Public Participation (IAP2) (2014) IAP2's public participation spectrum. <http://www.iap2.org.au/resources/iap2s-public-participation-spectrum>. Accessed 19 June 2015
- Kamar E, Hacker S, Horvitz E (2012) Combining human and machine intelligence in large-scale crowdsourcing. In: International Foundation for Autonomous Agents and Multiagent Systems (eds) Proceedings of the 11th international conference on autonomous agents and multiagent systems (AAMAS 2012), Valencia, 4–8 June 2012, pp 467–474
- Kobernus M, Berre AJ, Gonzalez M, Liu H-Y, Fredriksen M, Rombouts R, Bartonova A (2015) A practical approach to an integrated citizens' observatory: the CITI-SENSE framework. In: Berre AJ, Schade S (eds) Proceedings of the workshop 'Environmental Information Systems and Services—Infrastructures and Platforms 2013' (ENVIP 2013), Neusiedl am See, 10 Oct 2013
- Lanfranchi V, Wrigley SN, Ireson N, Ciravegna F, When U (2013) Citizens' observatories for situation awareness in flooding. In: Hiltz SR, Pfaff MS, Plotnick L, Shih PC (eds) Proceedings of the 11th international ISCRAM conference, University Park, PA, May 2014
- Lanfranchi V, Wrigley SN, Ireson N, Ciravegna F, and Wehn U (2014) Citizens Observatories for Situation Awareness in Flooding. In Proceedings of the 11th International ISCRAM Conference (pp 145–154). University Park, Pennsylvania, USA
- Lennox J, Proctor W, Russell S (2011) Structuring stakeholder participation in New Zealand's water resource governance. *Ecol Econ* 70:1381–1394
- Litke S, Day JS (1998) Building local capacity for stewardship and sustainability: the role of community-based watershed management in Chilliwack, British Columbia. *Environments* 25:91–109
- Liu H-Y, Kobernus M, Broday D, Bartonova A (2014) A conceptual approach to a citizens' observatory—supporting community-based environmental governance. *Environ Health* 13:107
- National Audubon Society (2015) History of the Christmas bird count. <http://www.audubon.org/conservation/science/christmas-bird-count>. Accessed 10 June 2015
- Nitsch M, Waldherr K, Denk E, Griebler U, Marent B, Forster R (2013) Participation by different stakeholders in participatory evaluation of health promotion: a literature review. *Eval Program Plann* 40:42–54
- Omniscientis (2012–2014) Odour monitoring and information system based on citizen and technology innovative sensors. <http://www.omniscientis.eu>. Accessed 1 July 2015
- Open Society Foundations (2014) Legal empowerment. <http://www.opensocietyfoundations.org/projects/legal-empowerment>. Accessed 19 June 2015
- Oxford English Dictionary (2014) New words notes June 2014. <http://public.oed.com/the-oed-today/recent-updates-to-the-oed/june-2014-update/new-words-notes-june-2014/>. Accessed 10 June 2015
- Piper S (2010) Patient empowerment: emancipatory or technological practice? *Patient Educ Couns* 79:173–177
- Ran B (2012) Evaluating public participation in environmental policy-making. *J US-China Public Adm* 9:407–423
- Rauschmayer F, Wittmer H (2006) Evaluating deliberative and analytical methods for the resolution of environmental conflicts. *Land Use Policy* 23:108–122
- Rauschmayer F, Berghöfer A, Omann I, Zikos D (2009) Examining process or/and outcomes? Evaluation concepts in European governance of natural resources. *Environ Pol Gov* 19:159–173
- Reed MS (2008) Stakeholder participation for environmental management: a literature review. *Biol Conserv* 141:2417–2431
- Reed MS, Graves A, Dandy N, Posthumus H, Hubacek K, Morris J, Prell C, Quinn CH, Stringer LC (2009) Who's in and why? A typology of stakeholder analysis methods for natural resource management. *J Environ Manage* 90:1933–1949

- Renn O (2008) Risk governance. Coping with uncertainty in a complex world. Earthscan, London
- Renn O, Schweizer PJ (2009) Inclusive risk governance: concepts and application to environmental policy making. *Env Pol Gov* 19:174–185
- Richards C, Balckstock LK, Carter CE (2007) Practical approaches to participation. SERG Policy Brief No 1. Macauley Land Use Research Institute, Aberdeen
- Rowe G, Frewer LJ (2005) A typology of public engagement mechanisms. *Sci Technol Hum Values* 30:251–290
- Roy HE, Pocock MJO, Preston CD, Roy DB, Savage J (2012) Understanding citizen science and environmental monitoring. Final report on behalf of UK environmental observation framework. NERC Centre for Ecology and Hydrology and Natural History Museum
- The Royal Society (2015) Transit of venus—1874 transit. <https://royalsociety.org/exhibitions/transit-venus/1874/>. Accessed 10 June 2015
- Science Communication Unit, University of the West of England, Bristol (2013) Science for environment policy in-depth report: environmental citizen science. Report produced for the European Commission DG environment, Dec 2013, Issue 9. [http://ec.europa.eu/environment/integration/research/newsalert/pdf/IR9\\_en.pdf](http://ec.europa.eu/environment/integration/research/newsalert/pdf/IR9_en.pdf). Accessed 22 June 2015
- Shirk JL, Ballard HL, Wilderman CC, Philipps T, Wiggins A, Jordan R, McCallie E, Minarchek M, Lewenstein BV, Krasny ME, Bonney R (2012) Public participation in scientific research: a framework for deliberate design. *Ecol Soc* 17:29–48
- Tulloch AIT, Possingham HP, Joseph LN, Szabo J, Martin TG (2013) Realising the full potential of citizen science monitoring programs. *Biol Conserv* 165:128–138
- United Nations Development Programme (UNDP) (2015) Women's empowerment. <http://www.undp.org/content/undp/en/home/ourwork/womenempowerment/overview.html>. Accessed 19 June 2015
- United Nations Economic Commission for Europe (UNECE) (1998) Convention on access to information, public participation in decision-making and access to justice in environmental matters. <http://www.unece.org/env/pp/treatytext.html>. Accessed 8 June 2015
- United Nations Environment Programme (UNEP) (2015) UNEP project manual: formulation, approval, monitoring and evaluation. [http://www.unep.org/pcmu/project\\_manual/chapters.asp](http://www.unep.org/pcmu/project_manual/chapters.asp). Accessed 23 June 2015
- WeSenseIt (2012–2016) Citizen water observatories. <http://www.wesenseit.com>. Accessed 1 July 2015
- Whitelaw G, Vaughan H, Craig B, Atkinson B (2003) Establishing the Canadian community monitoring network. *Environ Monit Assess* 88:409–418
- WHO (World Health Organisation) (1998) Health promotion glossary. WHO/HPR/HEP/98.1. Geneva
- WHO (World Health Organisation) (2015a) The determinants of health. Introduction. <http://www.who.int/hia/evidence/doh/en/>. Accessed 2 June 2015
- WHO (World Health Organisation) (2015b) The determinants of health. Energy. <http://www.who.int/hia/evidence/doh/en/index6.html>. Accessed 19 June 2015
- WHO (World Health Organisation) (2015c) The determinants of health. Transport. <http://www.who.int/hia/evidence/doh/en/index2.html>. Accessed 19 June 2015
- WHO (World Health Organisation) (2015d) The determinants of health. Food and agriculture. <http://www.who.int/hia/evidence/doh/en/index3.html>. Accessed 19 June 2015
- WHO (World Health Organisation) (2015e) The Ottawa charter for health promotion. <http://www.who.int/healthpromotion/conferences/7gchp/track1/en/>. Accessed 28 May 2015
- Wilkinson A (1998) Empowerment: theory and practice. *Pers Rev* 27(1):40–56
- Wittmer H, Rauschmayer F, Klauer B (2006) How to select instruments for the resolution of environmental conflicts? *Land Use Policy* 23:1–9
- Wynne B (1996) Misunderstood misunderstandings: social identities and public uptake of science. *Public Underst Sci* 1:281–304
- Zaman J, D'Hondt E, Gonzalez Boix E, Philips E, Kambona K, De Meuter W (2014) Citizen-friendly participatory campaign support. Proceedings of 2014 IEEE international conference on pervasive computing and communications work in progress (PerCom WiP'14), Budapest, 24–28 Mar 2014, pp 232–235
- Zikopoulos IBMP, Eaton C, Zikopoulos P (2011) Understanding big data: analytics for Enterprise Class Hadoop and streaming data. McGraw-Hill, New York

# Chapter 12

## Environment and Health Research Funded by the European Union (EU) Research Framework Programmes: Increasing Scientific Knowledge and Building a Solid Evidence-Base for Policy Making

Tuomo K. Karjalainen

**Abstract** The European Union has a significant funding programme for environment and health research. The projects funded by its Framework Programmes for Research and Technological Development, also called Framework Programmes, have contributed to building a knowledge base which is needed to make informed policy decisions in Europe and beyond.

**Keywords** Environment and health research • EU framework research framework programme • Environmental stressors • Environment and health policy • EU-funded projects

In response to public concerns about environmental health issues and the need to support evidence-based policies, a specific key action ‘Environment and health’ was first introduced under the Fifth Framework Programme (FP5). Over the period 1998–2002, this key action initiated more than 100 transnational research projects addressing a number of targeted environmental and health issues, especially related to chemicals, air pollution, noise and electromagnetic fields. Its follow-up programme, the Sixth Framework Programme (FP6), saw a slight increase in annual funding for environment and health projects for the period 2002–2006. The areas of research ranged from the contribution of environmental stressors to health end-points such as cancer or allergies/asthma, with funding of large networks of excellence, to the examination of the risks of waterborne stressors and nanoparticles. A specific emphasis was put on supporting EU policies. Numerous projects were also

---

T.K. Karjalainen (✉)  
DG Research and Innovation, Health Directorate, European Commission,  
1049 Brussels, Belgium  
e-mail: [Tuomo.Karjalainen@ec.europa.eu](mailto:Tuomo.Karjalainen@ec.europa.eu)

funded to improve the methods of integrated risk assessment, health impact assessment and in vitro testing of chemicals. The momentum was maintained in the Seventh Framework Programme (FP7—2007–2013), with the funding of over 160 projects and attracting participants world-wide. Many of these projects are on-going. These projects have increased our knowledge on health issues of concern to a large number of European citizens, ranging from assessing the potential health impacts of nanomaterials or chemicals to quality of air in our homes, schools and workplaces. In addition, the European Exposome Cluster was launched in 2012, introducing a new era in understanding life-course exposures and potential health outcomes.

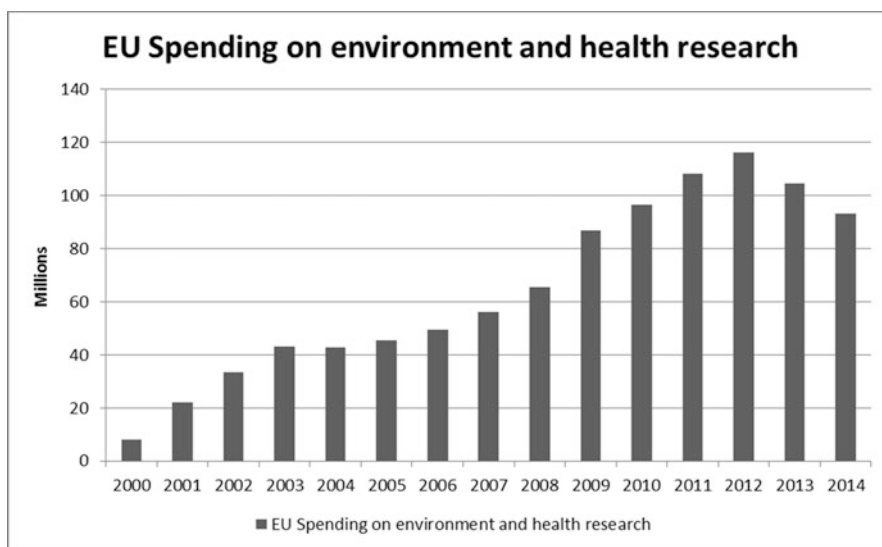
This chapter will analyse some of the funding trends observed, gives some details on the projects funded and address their implications. We believe that relevant information should be useful not only for the scientific community, but also to regulatory authorities and policy makers.

## 12.1 Funding Trends and Drivers: The Beginnings

Since their launch in 1984, the Framework Programmes have played a lead role in multidisciplinary research and cooperative activities in Europe and beyond. Environment and health research funded by the Fifth Framework Programme (FP5) was the first EU research framework programme in which a dedicated environment and health research activity emerged (European Commission 2007). This was due to the increasing public concerns for environment and health issues as well as the need to support evolving EU policies in this area.

Although the framework programme started in 1998 and ended in 2002, the 102 projects funded from this FP effectively received EU funding during the period 2000–2006. The average annual spending during this FP was around €39 million, starting with a modest €8 million in the year 2000 but rising rapidly in subsequent years (Fig. 12.1).

This first period was characterised by funding of targeted small-scale research projects focusing especially on exposures to various classes of chemicals (39 projects), air pollution (20 projects), non-ionising (8 projects) and ionising radiation (14 projects), noise (5 projects) and their potential health effects. The health end-points covered ranged from allergy/asthma for the air pollution-related projects to cancer and neurodevelopmental or endocrine-mediated effects such as declining sperm counts for projects investigating chemical exposures. Projects that have received the media attention from this period beyond the scientific community include INTERPHONE (Box 1), RANCH (Box 2) and the CREDO cluster (Box 3).



**Fig. 12.1** Annual average EU contribution to environment and health projects

### **Box 1: The INTERPHONE Project (International case Control Studies of Cancer in Relation to Mobile Telephone Use) Funded by FP5**

Due to the expanding use of mobile phones at the end of the 1990s and concerns about health and safety, the INTERPHONE project was initiated as an international set of case–control studies to determine whether mobile phone use increases the risk of tumours in tissues that most absorb radiofrequency (RF) energy emitted by mobile phones. The 13-country study was the largest case–control study to date investigating risks related to mobile phone use and to other potential risk factors for the tumours of interest. The results showed that, overall, no increase in risk of glioma or meningioma was observed with use of mobile phones. There were suggestions of an increased risk of glioma at the highest exposure levels, but biases and error prevented a causal interpretation. Partially based on INTERPHONE findings, International Agency for Research on Cancer has classified radiofrequency electromagnetic fields as possibly carcinogenic to humans (Group 2B) (INTERPHONE Study Group 2010, 2011; World Health Organization 2013a).

## **12.2 Funding Trends and Drivers: The Period of Acceleration**

As seen in Fig. 12.1, the period 2004–2012 during which the Sixth Framework Programme (FP6—2002–2006) projects received EU funding, the annual spending increased steadily although the number of projects funded per annum was lower (15) than in FP5 (25). A total of 61 projects were funded, receiving around €275

**Box 2: The RANCH Project (Road Traffic and Aircraft Noise Exposure and Children's Cognition and Health: Exposure-Effect Relationships and Combined Effects) Funded by FP5**

The RANCH project investigated the effects of environmental noise on children, revealing negative effects of aircraft noise on several aspects of cognition, including reading comprehension and memory. RANCH studied 2844 children in 2001–2003, aged between nine and ten, attending 89 primary schools near major airports in the UK, the Netherlands and Spain (Clark et al. 2006).

**Box 3: The CREDO Cluster of Projects (The Cluster of Research Into Endocrine Disruption in Europe) Funded by FP5**

The CREDO cluster, consisting of four separate projects encompassing 63 laboratories in Europe, was launched as a direct response to the call to enhance research efforts by the European Commission's Strategy for Endocrine Disruptors adopted in 1999. The results of the cluster showed, among others, the conventional approach of estimating no-observed-effect-levels for chemicals is inadequate for capturing low-dose effects of endocrine disruptors. Endocrine disruptors of relatively low potency and at low exposure levels can still work together to produce significant combination effects when they are present in sufficient numbers. Furthermore, a wide variety of endocrine disrupting chemicals were found together in human tissue specimens, and in specimens of wild fish. The reproductive and developmental effects of certain endocrine disruptors are comparable across phyla, from invertebrates to mammals (European Commission 2005).

million from the EU, representing 612 participating institutions from 58 countries from all over the world (Table 12.1) (European Commission 2012).

The availability of extra resources and policy support allowed the concentration and re-focusing of efforts to build up a European Research Area in specific fields of environmental health science. The research/policy interface was enhanced by the introduction in the FP6 of a specific 'Scientific Support to Policy' activity, which allowed close exchanges of ideas between research and policy-makers and the discussion of policy options and policy implications of the research undertaken.

To tackle larger-scale issues and to promote the building of a European Research Area on particular issues, FP6 saw the introduction of two new funding schemes, namely integrated projects and networks of excellence. The former was an instrument to support objective-driven research, where the primary deliverable was new knowledge. The projects were quite large, comprising up to 69 participating institutions in environment and health-related projects, with a substantial budget and medium-long duration (usually 5 or 6 years). The latter were designed to strengthen

**Table 12.1** Number of project funded focused on environmental determinants as indicated in FP5, FP6 and FP7

	FP5	FP6	FP7	Total
Industrial chemicals	39	21	35	95
Multiple or undefined factors	3	17	19	39
Nanomaterials	3	7	42	52
Lifestyle factors	2	1	17	20
Ionising radiation	14	4	15	33
Air pollution	20	1	9	30
Climatic factors	2	5	14	21
Biological hazards	7	3	7	17
Non-ionising radiation	8	2	8	18
Noise	5	1	2	8

scientific and technological excellence on a particular research topic by integrating at European level the critical mass of resources and expertise needed to provide European leadership and to be a world force in that topic. This expertise was networked around a joint programme of activities aimed principally at creating a progressive and durable integration of the research capacities of the network partners while, at the same time, advancing knowledge on the topic.

The main policy driver for environment and health research since the end of FP5 was the European Environment and Health Strategy (European Commission 2003) and the associated Action Plan (European Commission 2010), adopted in 2004, which finished at the end of 2010. Projects funded under the FP5 contributed to the formulation of this Strategy. The strategy and the action plan aimed to improve our understanding of the links between environmental factors and health and were partially inspired by the need to link policy with research results.

The Action Plan has served as a catalyst to increase research spending at the EU level, especially under FP6. It had four research-focused actions amongst its 13 actions, three of which in particular resulted in the funding of significant large-scale integrated projects and networks of excellence:

- *Action 6—Targeting research on diseases, disorders and exposures:* The aim of this action was to improve knowledge of the links between environmental exposures and four priority diseases/disease mechanisms, namely childhood respiratory diseases, neuro-developmental disorders, cancer and endocrine disrupting effects. A number of research initiatives were launched, including the GA<sup>2</sup>LEN network of excellence (Box 4), bringing together allergy and asthma researchers in Europe into a network; ECNIS, improving and structuring research on environmental determinants of cancer risks (European Commission 2013a); and CASCADE, networking scientists around the thematic of endocrine disrupting chemicals (European Commission 2013b).
- *Action 7—Research aiming at developing methodological systems to analyse interactions between environment and health:* Substantial support—over €100 million—was provided in FP6 to projects devoted to the development of inte-

**Box 4: The GA<sup>2</sup>LEN Network of Excellence (Global Allergy and Asthma European Network) Funded by FP6**

The GA<sup>2</sup>LEN Network of Excellence was created to combat fragmentation in the European research area, ensuring excellence in EU allergy and asthma research by bringing together institutions and researchers from across the EU. The vision of GA<sup>2</sup>LEN was—and still is—to reduce the burden of allergic diseases in Europe by improving the health of European with allergic diseases, increasing the competitiveness and boosting the innovative capacity of EU health-related industries and businesses while addressing health issues including emerging allergies. Work carried out by GA<sup>2</sup>LEN has established the EU as one of the leaders in the field of allergy and asthma research and clinical care. GA<sup>2</sup>LEN has achieved sustainability and is continuing as a non-profit network, building on the foundation provided by the 6 years of FP6 funding (GA2LEN 2012).

grated risk assessment methodologies and models for evaluating health effects of multiple environmental stressors or mixtures of pollutants, including research on risk/benefit analyses and environmental health economics. Examples of projects include INTARESE (European Commission 2011), which developed a methodological framework and a set of tools and indicators for integrated assessment that can be applied across different environmental stressors such as air pollution, noise and chemicals, exposure pathways and policy areas; and HEIMTSA (HEIMTSA 2012), which developed an integrated methodology for health impact assessment and cost benefit analysis.

- *Action 8—Research to ensure that potential hazards on environment and health are identified and addressed:* Two new areas of research emerged in FP6: research on health impacts of climate change and environmental and human health impacts of nanomaterials. The EDEN project (Box 5) could be considered the flagship initiative as regards the former, investigating the impacts of environmental change on the spatial and temporal distribution of vector-borne diseases including tick-borne and rodent-borne diseases. The latter is exemplified by the establishment of the EU NanoSafety Cluster, including several FP6 projects, and later FP7 projects, to maximise the synergies between projects addressing many aspects of nano-safety including toxicology, ecotoxicology, exposure assessment, mechanisms of interaction, risk assessment and standardisation (EU Nanosafety Cluster 2015).



**Box 5: The EDEN Integrated Project (Emerging Diseases in a Changing European Environment) Funded by FP6**

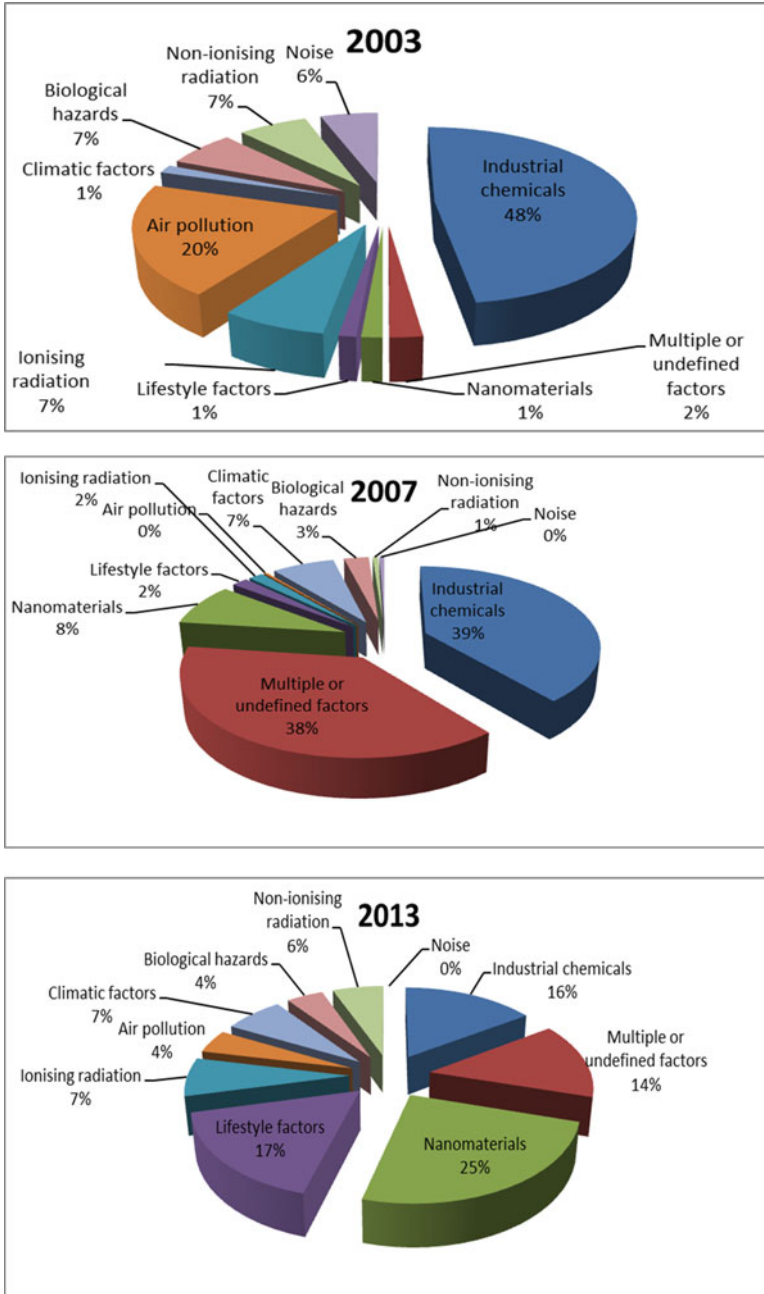
The 49-partner EDEN project identified and evaluated European ecosystems and environmental conditions linked to global change, which can influence the spatial and temporal distribution and dynamics of pathogenic agents. It worked on providing predictive emergence and spread models including global and regional preventive, early warning, surveillance, and monitoring tools and scenarios. Diseases were selected according to the vectors and hosts involved in their epidemiology: tick-borne encephalitis, haemorrhagic fever with renal syndrome (rodents), leishmaniasis (sand-flies), West Nile and malaria (mosquitoes). Also, African sources of West Nile and Rift Valley fever viruses were studied to improve control for the benefit of African populations, and investigate the risk of introduction in Europe. The results showed, in general, climate change alone cannot explain the upsurge or emergence of vector-borne diseases in Europe. This was demonstrated for the case of tick-borne encephalitis in Baltic countries and Central Europe, for which socio-economic factors and human behaviour are tightly related to the disease risk. Parts of the work continued in the FP7-funded EDENEXT project (EDENEXT biology and control of vector-borne infections in Europe 2011).

### 12.3 Funding Trends and Drivers: The Period of Consolidation and Re-Assessment

As seen in Table 12.1, the Seventh Framework Programme (FP7—2007–2013) funded 167 environment and health-related projects, with an EU contribution of around €638 million (average €87 million per annum) (European Commission 2011, 2014a). On an annual basis, this represents a substantial increase as compared to FP5 and a modest increase as compared to FP6 (Fig. 12.1). Many projects are ongoing, with the more recently funded projects estimated to finish in 2018.

Sustained funding by the Framework Programmes to environment and health research can be speculated to be attributable to several factors, including continued public concern for environment and health issues, the continued impetus provided by the European Environment and Health Action Plan which run until 2010, and the acknowledged need to provide scientific support for specific policy actions or programmes.

The largest proportion of the FP7 environment and health budget was allocated to research projects related to environmental and human exposures and possible effects of nanomaterials (Table 12.1; Fig. 12.2). These projects and their accomplishments are described in detail on a dedicated website on EU Nanosafety cluster (EU nanosafety cluster 2015). This was closely followed by projects devoted to chemical risks (e.g., food safety issues, detection, alternative testing methods, life cycle assessment, health impacts, etc.) and lifestyle factors. The array of chemicals addressed in FP7 projects is wide, including (*inter alia*): pesticides with neurotoxic



**Fig. 12.2** Snapshots of funding allocations to allocated to various stressors from 2003 (FP5), 2007 (FP6) and 2013 (FP7)

effects—e.g., the DENAMIC project, focused on the impact of these chemicals on learning and developmental disorders in children (DENAMIC developmental neurotoxicity assessment of mixtures in children 2015); pharmaceuticals—e.g., the CYTOTHREAT project, investigating cytostatic drugs released in the environment and their potential long-term effects (CYTOTHREAT fate and effects of cytostatic pharmaceuticals in the environment and identification of biomarkers for an improved risk assessment on environmental exposure 2015); cosmetics (e.g., the SEURAT-1 cluster (Box 6); and food contaminants/additives—e.g., the FACET project, focused on estimating exposure to flavours, additives and food contact materials in Europe (FACET 2015). The projects funded in this area have the potential to contribute to many policies at the EU level such as the REACH regulation, legislation on food contaminants or that relevant to the Three Rs (replacement, reduction, replacement) principle in animal experimentation (EUR-LEX 2015).

In addition, there has been a greater emphasis on projects examining the role of life-style and behavioural factors in health and disease in FP7 (Table 12.1). For example, the EPI-MIGRANT project (EPI-MIGRANT identification of epigenetic markers underlying increased risk of T2D in South Asians 2015) focuses on the identification of epigenetic risk factors underlying the increased rates of type-2 diabetes (T2D) amongst South Asians in their home countries, migrants to Europe and other parts of the world.

Another emerging trend—albeit less eminent than that related to nanomaterial research—was increased funding to projects dealing with various health-related aspects of climate change (Table 12.1). These projects focused, *inter alia*, on risks of emergence of pollen-induced allergies and asthma due to climate change—e.g., ATOPICA (ATOPICA atopic diseases in changing climate, land use and air quality 2015).

**Box 6: The SEURAT-1 Cluster of Projects (Towards the Replacement of In Vivo Repeated Dose Systemic Toxicity Testing) Funded by FP7**

The SEURAT-1 cluster (SEURAT-1 2015) is a research initiative created through a call for proposals by the European Commission with matching funds from the Cosmetics Europe industry to make a total of EUR 50 million available to try to fill current gaps in scientific knowledge and accelerate the development of non-animal test methods. The initiative focuses on the complex area of repeated dose toxicity. It is a first step to addressing the long term strategic target of “Safety Evaluation Ultimately Replacing Animal Testing (SEURAT)”. It is called “SEURAT-1”, indicating that more steps have to be taken before the final goal will be reached. SEURAT-1 will develop knowledge and technology building blocks required for the development of solutions for the replacement of current repeated dose systemic toxicity testing in vivo used for the assessment of human safety. The initiative is composed of six research projects, which started in 2011 and will run for 5 years.

FP7 continued to fund projects related to ionising radiation, the largest one being DOREMI (DOREMI low dose research towards multidisciplinary integration 2015), a network of excellence aiming to promote the sustainable integration of low dose risk research in Europe. Support was also allocated to projects looking at potential health risks related to exposure to electromagnetic fields (non-ionising radiation). In the context, and as a follow-up of the INTERPHONE project funded under FP5, the MOBILKIDS project (MOBI-KIDS 2015) is carrying out a prospective epidemiological case-control study of brain tumours diagnosed in young people in potential relation to EMF exposure from mobile phones and other sources of radiofrequency fields in 11 EU and non-EU countries. It is the largest study in the world on childhood brain cancer.

It is estimated that air pollution is one of the major environmental stressors human populations are exposed to in Europe, both indoors and outdoors. This environmental challenge was addressed by nine projects, focusing both on health impacts of ambient air quality and indoor air quality (Table 12.1). An example of the former is the ESCAPE project (ESCAPE 2015), which has increased substantially the knowledge base of various health risks related to air pollution exposure, including childhood asthma, arteriosclerosis, cerebrovascular events and lung cancer. The latter is exemplified by the HITEA project (European Commission 2015), which focused on moisture damage and dampness and microbial exposures in schools and homes and possible health consequences. Moisture problems were found to be relatively common, with 20–41 % of school buildings being affected.

Although now acknowledged as being an increasingly important environmental stressor, there was only one noise-focused coordination action funded in FP7—the ENNAH network. It brought together 33 European research centres to establish future research directions and policy needs for noise and health in Europe. These are available from the project website (ENNAH European network on noise and health 2008).

## 12.4 Overall Appraisal and Picture of 15 Years of EU-Funded Environment and Health Research (2000–2015)

The European Union has invested massively in environment and health research since 2000. The 325 projects funded have received over a billion euros from the three framework programmes concerned. The investment in this area is even more significant as the EU spending is complemented by spending in the various member states.

The outreach of the programmes is equally far-reaching: In FP6 612 institutions participated from 58 countries whereas 68 countries worldwide have participated in FP7, including 1190 unique institutions (participants).

As shown in Fig. 12.2 and Table 12.1, the majority of funding has been allocated to projects dealing with exposure and effects of chemicals, nanomaterials or combinations of environmental factors. This reflects the significant uncertainties in science that remain in these areas of environment and health research.

Factors that could explain the sustained funding in this area of science include:

- Public concern for environment and health issues as these are perceived as personal: numerous opinion surveys of European populations, including those carried out by the Eurobarometer, as regards the significance of environmental factors for their health, have shown that citizens are concerned—rightly or wrongly—about the impacts of various pollutants on their health. The 2014 survey ‘Attitudes of European Citizens towards the Environment’ (European Commission 2015), for example, showed that almost all Europeans say that protecting the environment is important to them personally, and over half say it is very important. Half or more of Europeans said that they are worried about air pollution and water pollution, while over four in ten were worried about the impact on health from chemicals in everyday products and the growing amount of waste. Over three-quarters of respondents feel that environmental problems have a direct effect on their daily lives. This level of concern has translated into support for this area by politicians, e.g., those in the European Parliament, by policy-makers at national and European level, and by non-governmental organisations, leading also to requests for increased funding of research in this area. In addition, the European Commission’s scientific committees (European Commission 2015) have identified areas where more research could be carried either at national or EU level.
- The transnational nature of environment and health policies: overarching environmental health policies, due to the cross-border nature of pollution-related issues, are adopted at the European level and require scientific support for their justification and updating. As many problems are complicated and wide-reaching, it is logical that they be tackled also scientifically at the European level to have the critical mass and resources necessary. Almost all sub-areas of environment and health research have the potential to underpin one or several sectorial policies or more global initiatives. The former include, *inter alia*, numerous directives and regulations on specific categories of chemicals such as plant protection products or environmental stressors such as noise. The latter encompasses initiatives and strategies of various size and scope, prominent ones being the European Environment and Health Strategy and the associated Action Plan, the European Strategy for Nanotechnology and the Nanotechnology Action Plan, The Community Strategy for Endocrine Disruptors, and the Thematic Strategy on Air Pollution.
- The European environment and health process managed by the World Health Organization: in parallel with the environment and health-related activities in the European Commission, the main political driver of the European environment and health process has been the World Health Organization European region (WHO Europe) in the past 20 years. The 2004 Budapest and 2010 Parma ministerial conferences especially have set commitments and targets to reach in the wider European region, which also have helped in setting research priorities and stimulated international scientific cooperation. In addition, outside this process the WHO has reviewed the current state of science and provided recommendations for research and related policy in several areas, including electromagnetic fields, noise (World Health Organization 2011), or air quality (World Health Organization 2013b).

- Distinct environment and health activity in the framework programmes: core funding has been ensured to environment and health research by the fact that each framework programme—FP5, FP6 and FP7—has had a separate environment and health activity with its own funding. However, it should be noted that while in FP5 this accounted for over 90 % of the funding allocated to this area, in FP7 the activity only received around €20 million per year, the rest coming from various other programmes and activities relevant to environment and health. This reflects the fact that even before the adoption of Horizon 2020, the environment and health area has become very cross-cutting and multidisciplinary and no longer reliant on this particular area alone.

#### ***12.4.1 Conclusions of an Impact Assessment Study Carried Out in 2010***

A study carried out by an external impact assessment body on the longer-term impact of European Union funding of research in the field of Environment and Health was funded by DG Research and Innovation in 2010 ([European Commission 2010](#)). The study concluded, inter alia, that:

- There is objective evidence that projects funded by the EU framework programmes have contributed to numerous EU policies;
- An important longer-term impact of the EU funding is the fact that the environment and health research has created an important common European research platform with a very high degree of legitimacy among the actors;
- EU funding of environment and health research has stimulated research and other activities at national level, and has contributed to restructuring, integration and networking within many sub-areas of environment and health.

#### ***12.4.2 Looking into the Future***

Under the current framework programme, Horizon 2020 (2014–2020), environment and health research is embedded in the societal challenge ‘Health, demographic Change and Wellbeing’. The total budget available for the first calls (2014–2015) was approximately EUR 1.2 bn, while the total budget allocated to this challenge over 7 years is around EUR 7.5 bn.

The new approach taken under Horizon 2020 aims to look at societal challenges in a holistic, challenge driven and integrated manner. It is acknowledged that promotion of health, active ageing, well-being and disease prevention depend on an understanding of the determinants of health (including environmental and lifestyle factors), on effective preventive tools, such as vaccines, on effective health and disease surveillance and preparedness, and on effective screening programmes.

As a result of the challenge-driven approach, the topics in the calls for proposals are wider. For example, in the call 2014–2015 there were two topics which in particular were relevant for the environment and health research community, namely ‘*Understanding health, ageing and disease: determinants, risk factors and pathways*’, and ‘*Health promotion and disease prevention: improved inter-sector co-operation for environment and health based interventions*’, which each received a considerable number of proposals. The analysis of the results of the selection process shows that even in this highly competitive environment excellent environment and health projects are selected for funding, such as EDC-MICRISK, which aims at further developing chemical risk assessment through improved understanding of the mechanisms and health effects of endocrine disrupting chemicals, in particular mixtures.

For the call 2015–2016, the challenge of understanding the health impacts of population exposures to a number of chemicals in our surroundings will be tackled by a proposed topic on ‘The European Human Biomonitoring Initiative’ (HBM4EU). The objective is to create a European joint programme for monitoring and scientific assessment of human exposures to chemicals and potential health impacts in Europe. HBM4EU should be achieved through coordination of HBM initiatives at national and EU level, with a special focus on linking research to evidence-based policy making. The HBM4EU should build on European excellence in the field and promote capacity building and the spread of best practice. The HBM4EU should provide a platform through which harmonised and validated information and data collected at national level can be accessed and compared. It should support research and innovation in various ways, e.g., by improving underlying methods and procedures (e.g., for sampling, sample analysis, data analysis, and data management), by improving the understanding of the impact of the exposure on human health (e.g., development of validated exposure and effect biomarkers and establishing correlation between biomarker levels and health risks) and by improving the use of HBM data in risk assessment of chemicals. The acquired knowledge should support informed decision taking and policy making in a wide variety of sectors, one of the most important being the EU chemicals legislation under REACH. The initiative is planned to be co-funded through Horizon 2020 and member state contributions, and the planned commencement date is in the first half of 2017.

In recent years, and especially under FP7, new scientific approaches have emerged that are likely to shape and guide environment and health research in years to come. These include:

- A strong emergence of projects developing personalised exposure assessment approaches, which is likely to become even more prominent in the future within the frame of the exposome approach (Box 7);
- Increased development of exposure and early effect biomarkers based on ‘omics’, which poses some challenges for risk assessment;
- Studies exploring the impact of early exposures (e.g., *in utero*) and the risks of developing diseases later in life—the so-called developmental origins of disease concept;

**Box 7: The European Exposome Cluster Funded by FP7**

In preparation for the challenge-driven approach offered by the EU Framework for Research and Innovation (2014–2020)—Horizon2020—the EU Exposome Cluster (European Commission 2014b) was launched in 2012. At its most complete, the exposome encompasses life-course environmental exposures (including lifestyle factors), from the prenatal period onwards; thus to understand it is a formidable scientific challenge to overcome. This large-scale initiative, having a total budget of around €38 million and an EU contribution of €29 million until 2018, is the largest ever initiative in the environment and health research area in the EU until the end of FP7. It includes three projects: EXPOSOMICS (Enhanced exposure assessment and omic profiling for high priority environmental exposures in Europe), HELIX (The human early-life exposome—novel tools for integrating early-life environmental exposures and child health across Europe) and HEALS (Health and environment-wide associations based on large population surveys). Since this launch, the exposome concept is gaining in popularity and recognition, and it is possible that new actions will be undertaken to further our understanding of the concept.

It is possible that the EU General Environmental Action Programme—‘*Living well, within the limits of our planet*’, adopted in 2013—will be one of the main policy drivers for environment and health research in Europe in the years to come (EUR-LEX 2015). It has three key objectives: (1) to protect, conserve and enhance the Union’s natural capital; (2) to turn the Union into a resource-efficient, green, and competitive low-carbon economy; and (3) to safeguard the Union’s citizens from environment-related pressures and risks to health and wellbeing.

## 12.5 Conclusions

Environment and health research is rightly placed at the EU level because it tackles common European problems that are not directly dependent on specific Member State conditions. EU environmental health policy is very extensive and requires significant scientific support. This necessitates approaches that maximise synergies between various disciplines and sectors.

The environment and health-related policy areas are likely remain important in the foreseeable future. In this area of science and policy many scientific uncertainties and challenges remain. These include predicting health impacts of global change, assessing exposures to mixtures of chemicals/environmental stressors, and evaluating risks and benefits of new technologies, which are all scientific issues that require coordinated research and policy actions at the EU level.



## 12.6 Legal Notice

Neither the European Commission nor any person acting on behalf of the Commission is responsible for the use which might be made of the following information.

The views expressed in this publication are the sole responsibility of the author and do not necessarily reflect the views of the European Commission.

## References

- ATOPICA atopic diseases in changing climate, land use and air quality (2015) Project website. [www.atopica.eu](http://www.atopica.eu). Accessed 20 Apr 2015
- Clark C, Martin R, van Kempen E et al (2006) Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. *Am J Epidemiol* 163:27–37
- CYTOTHREAT fate and effects of cytostatic pharmaceuticals in the environment and identification of biomarkers for an improved risk assessment on environmental exposure (2015) Project website. <http://www.cyothreat.eu>. Accessed 20 Apr 2015
- DENAMIC developmental neurotoxicity assessment of mixtures in children (2015) Project website. [www.denamic-project.eu](http://www.denamic-project.eu). Accessed 20 Apr 2015
- DOREMI low dose research towards multidisciplinary integration (2015) Project website. <http://www.doremi-noe.net>. Accessed 20 Apr 2015
- EDENEXT biology and control of vector-borne infections in Europe (2011) Project website. <http://www.edenext.eu>. Accessed 20 Apr 2015
- ENNAH European network on noise and health (2008) Project website. [www.ennah.eu](http://www.ennah.eu). Accessed 20 Apr 2015
- EPI-MIGRANT identification of epigenetic markers underlying increased risk of T2D in South Asians (2015) Project website. <http://www.epimigrant.eu>. Accessed 20 Apr 2015
- European Study of Cohorts for Air Pollution Effects (ESCAPE) (2015) Project website. <http://www.escapeproject.eu>. Accessed 20 Apr 2015
- EU Nanosafety Cluster (2015) Project website. <http://www.nanosafetycluster.eu>. Accessed 20 Apr 2015
- EUR-LEX (2015) Access to European Union Law. <http://eur-lex.europa.eu/browse/summaries.html>. Accessed 20 Apr 2015
- European Commission (2003) Communication from the commission to the council, the European Parliament and the European Economic and Social Committee: a European environment and health strategy. Brussels. <http://eur-lex.europa.eu/legal-content/EN/TXT/HTML/?uri=CELEX:52003DC0338&from=EN>. Accessed: 20 Apr 2015
- European Commission (2010) Commission staff working document—progress report on the implementation of the “European Environment and Health Action Plan 2004-2010”. Brussels. [http://ec.europa.eu/health/healthy\\_environments/policy/health\\_environment/actionplan\\_en.htm](http://ec.europa.eu/health/healthy_environments/policy/health_environment/actionplan_en.htm). Accessed 20 Apr 2015
- European Commission (2011) CORDIS—Community Research and Development Information Service. INTARESE—Integrated Assessment of Health Risks from Environmental Stressors in Europe. [http://cordis.europa.eu/project/rcn/78406\\_en.html](http://cordis.europa.eu/project/rcn/78406_en.html). Accessed 20 Apr 2015
- European Commission (2013a) CORDIS—Community Research and Development Information Service. ECNIS—Environmental cancer risk, nutrition and individual susceptibility. [http://cordis.europa.eu/project/rcn/74967\\_en.html](http://cordis.europa.eu/project/rcn/74967_en.html). Accessed 20 Apr 2015
- European Commission. (2013b) CORDIS—Community Research and Development Information Service. CASCADE—Chemicals as contaminants in the food chain: an NOE for research, risk assessment and education. [http://cordis.europa.eu/project/rcn/74150\\_en.html](http://cordis.europa.eu/project/rcn/74150_en.html). Accessed 20 Apr 2015

- European Commission (2015) CORDIS—Community Research and Development Information Service. HITEA health effects of indoor pollutants: integrating microbial, toxicological and epidemiological approaches. [http://cordis.europa.eu/project/rcn/87924\\_en.html](http://cordis.europa.eu/project/rcn/87924_en.html). Accessed 20 Apr 2015
- European Commission (2015) Directorate-General for Health and Food Safety. Website on scientific committees. [http://ec.europa.eu/health/scientific\\_committees/index\\_en.htm](http://ec.europa.eu/health/scientific_committees/index_en.htm). Accessed 20 Apr 2015
- European Commission (2005) Directorate-General for Research and Innovation. An overview of endocrine disrupter projects financed by the European Commission under the 4th and 5th framework programmes. Brussels. <http://ec.europa.eu/research/quality-of-life/pdf/endocrine-projects.pdf>. Accessed: 20 Apr 2015
- European Commission (2007) Directorate-General for Research and Innovation. EU research on environment and health—results from projects funded by the Fifth Framework Programme. Brussels. <http://bookshop.europa.eu/en/eu-research-pbKINA22816>. Accessed 20 Apr 2015
- European Commission (2010) Directorate-General for Research and Innovation. Study on the longer-term impact of European Union funding of research in the field of environment and health. Brussels. [http://ec.europa.eu/research/environment/pdf/cowi\\_study.pdf](http://ec.europa.eu/research/environment/pdf/cowi_study.pdf). Accessed 20 Apr 2015
- European Commission (2011) Directorate-General for Research and Innovation. European research on environment and health funded by the seventh framework programme, vol 1. Brussels. <http://bookshop.europa.eu/en/eu-research-pbKINA24641>. Accessed 20 Apr 2015
- European Commission (2012) Directorate-General for Research and Innovation. EU research on environment and health funded by the sixth framework programme—snapshots of final results. Brussels. <http://bookshop.europa.eu/en/eu-research-pbKINA25049>. Accessed 20 Apr 2015
- European Commission (2014a) Directorate-General for Research and Innovation. Bridging the science-policy gap. EU-funded research for better environmental health. Brussels. <http://bookshop.europa.eu/en/bridging-the-science-policy-gap-pbKI0114345>. Accessed 20 Apr 2015
- European Commission (2014b) Directorate-General for Research and Innovation. Environment and health. [http://ec.europa.eu/research/environment/index\\_en.cfm?pg=health](http://ec.europa.eu/research/environment/index_en.cfm?pg=health). Accessed 20 Apr 2015
- European Commission. Eurostat. Special Eurobarometer 416 attitudes of European citizens towards the environment. Brussels. [http://ec.europa.eu/public\\_opinion/archives/ebs/ebs\\_416\\_en.pdf](http://ec.europa.eu/public_opinion/archives/ebs/ebs_416_en.pdf). Accessed 20 Apr 2015
- Flavours, Additives and Food Contact Material Exposure Task (FACET) (2015) Project website. <http://www.ucd.ie/facet>. Accessed 20 Apr 2015
- Global Allergy and Asthma European Network (GA2LEN) (2012) Project website. <http://www.ga2len.net>. Accessed 20 Apr 2015
- Health and Environment Integrated Methodology and Toolbox for Scenario Assessment (HEIMTSA) (2012) Project website. <http://www.heimtsa.eu>. Accessed 20 Apr 2015
- INTERPHONE Study Group (2010) Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. *Int J Epidemiol* 39:675–694
- INTERPHONE Study Group (2011) Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. *Cancer Epidemiol* 35:453–464
- MOBI-KIDS (2015) Risk of brain cancer from exposure to radiofrequency fields in childhood and adolescence Project website. <http://www.crealradiation.com/index.php/en/mobi-kids-home>. Accessed 20 Apr 2015
- SEURAT-1 (2015) Towards the replacement of in vivo repeated dose systemic toxicity testing. Project website. <http://www.seurat-1.eu>. Accessed 20 Apr 2015
- World Health Organization (2011) Burden of disease from environmental noise. Quantification of healthy life years lost in Europe. Copenhagen. [http://www.euro.who.int/\\_\\_data/assets/pdf\\_file/0008/136466/e94888.pdf](http://www.euro.who.int/__data/assets/pdf_file/0008/136466/e94888.pdf). Accessed 20 Apr 2015
- World Health Organization (2013a) IARC monographs on the evaluation of carcinogenic risks to humans. Vol. 102: Non-ionizing radiation. Part 2: Radiofrequency electromagnetic fields. Lyon. <http://monographs.iarc.fr/ENG/Monographs/vol102/index.php>. Accessed: 20 Apr 2015
- World Health Organization (2013b) Health effects of particulate matter. Policy implications for countries in Eastern Europe, Caucasus and central Asia. Copenhagen. [http://www.euro.who.int/\\_\\_data/assets/pdf\\_file/0006/189051/Health-effects-of-particulate-matter-final-Eng.pdf?ua=1](http://www.euro.who.int/__data/assets/pdf_file/0006/189051/Health-effects-of-particulate-matter-final-Eng.pdf?ua=1). Accessed: 20 Apr 2015

# Index

## A

Air pollutants, 122, 175, 176  
Air pollution, 30, 31, 33, 34, 118, 175  
  control policies, 118  
  effectiveness of measures, 130  
  efficient measures, highest net benefit, 130  
  impact pathway approach, 120–125  
  in transport sector, policies and reducing  
    measures, 127–131  
  utility losses, 127  
Air Quality Directive, 118  
Airborne particulate matter, 85  
Aluminium (Al), 105, 107  
Alzheimer's disease (AD), 98, 108  
AMAP Human Health Assessment Group, 54  
AMAP Ring Test, 55  
Amyotrophic lateral sclerosis (ALS), 98  
Analytical deliberative approach, 215, 216  
ArcRisk project, 86, 90, 92  
Arctic food chains, 52  
Arctic Health Risks, 86  
Arctic Monitoring and Assessment Program  
  (AMAP), 52, 54, 85  
Arsenic (As), 103, 104  
Arsenic exposure, 37, 38  
Arsenobetaine, 111  
Arsenolipids, 104, 111  
Arsenosugars, 104, 111  
Asthma  
  allergens, 42  
  cleaning products, 41  
  dampness and mould, 42  
  diet and physical activity, 39, 40  
  exposures and risk factors  
    air pollution, 30, 31, 33, 34

    arsenic exposure, 37, 38  
    metals, 36, 37  
    noise exposure, 35, 36  
  external exposome, 31  
  non-allergic, 30  
  phthalates, 40, 41  
Attention deficit hyperactivity disorder  
  (ADHD), 60, 61  
Avertive expenditures, 138

## B

Benefit transfer methods, 119  
Best environmental practices (BEP) concept,  
  18, 20  
Biogeochemical cycling, 8–10, 23, 24  
Biomass burning, 5  
Bio-monitoring networks, 89–90  
Breast cancer, 70–71  
British Committee on the Medical Effects of  
  Air Pollutants (COMEAP), 34  
Brominated flame retardants (BFRs), 54  
Bubble bursting, 5

## C

Cadmium, 13, 93  
Carbon capture and storage (CCS), 190  
  CFD, 188  
  energy intensive technology, 190  
  geochemical reactions, 189  
  GHG emissions, 196  
  LCA, 192–194  
  nitramines, 192  
  nitrosamines, 192

- Carbon capture and storage (CCS) (*cont.*)  
 photosynthetic and respiratory processes, 189  
 power plant emissions, 191  
 public and policy makers, 185  
 quantitative risk assessment, 193
- Carbon dioxide (Co<sub>2</sub>)  
 CCS, 188–189  
 crop productivity, 173  
 emissions, 174  
 GHGs, 169
- Cardiovascular diseases (CVD), 29  
 diet and physical activity, 39, 40  
 exposures and risk factors  
 air pollution, 30, 31, 33, 34  
 arsenic exposure, 37, 38  
 metals, 36, 37  
 noise exposure, 35, 36  
 external exposome, 31
- Cardiovascular effects  
 breast cancer, 70–71  
 carcinogenic effects, 69  
 endocrine effects, 65–66  
 epigenetics, 73  
 hypothalamo-pituitary-gonadal axis, 66  
 hypothalamo-pituitary-thyroid axis, 67–68  
 lung cancer, 70  
 mercury, 64–74  
 ovarian cancer, 71  
 pancreatic cancer, 71  
 POPs, 68–69  
 prostate cancer, 71  
 type 2 diabetes, 68–69
- CFD. *See* Computational fluid dynamics (CFD)
- CGE models, 158–159
- CHICOS project, 86, 87
- Chukotka studies, 57
- Citclobs, 241
- CITI-SENSE, 238, 241
- Citi-Sense-MOB, 241
- Citizen science  
 definition, 233  
 frameworks, 236  
 involvement types, 235–236  
 policy dimensions, 234  
 types, 235
- Citizens' observatories (COs)  
 common model, 241  
 Creative Commons Attribution License, 239  
 data flow model, components, 240, 241  
 definition, 238  
 development needs, 244  
 framework, 239, 240  
 issues, 237
- CLEAR project (Climate Change, Environmental contaminants and Reproductive), 59, 86, 87
- Climate change challenge, 74–75
- CO<sub>2</sub> pipelines, 188
- COBWEB, 238, 241
- Cohorts in Faroe Island, 59–60
- Computable General Equilibrium (CGE) models, 158
- Computational fluid dynamics (CFD), 188
- Contingent valuation (CVM), 141
- COPHES/DEMOCOPHES projects, 86, 88
- Cost benefit analysis (CBA), 152
- Cost-of-illness (COI), 140
- Counterfactual scenario design  
 dimensions, 155  
 environmental policy, 154–155  
 gross effects, 156  
 net effects, 156
- CoZMoMAN model, 93
- Creative Commons Attribution License, 239
- CREDO cluster, 252
- D**
- Damage function approach, 135–136
- Dampness and mould, 42
- Decision support system (DSS), 20–23
- Decision-making, 226
- DEHP, 40, 41
- Diesel exhaust particulate (DEP), 34
- Dietary ingestion, 38
- Disability-adjusted life years (DALYs), 85, 145, 146
- Discrete choice experiments (DCE), 141
- Disruptive Behavior Disorders Rating Scale (DBD), 61
- Disutility, 138
- Docosahexaenoic acid (DHA), 60
- Driving Force-Pressure-State-Exposure-Effect-Action (DPSEEA), 171, 172
- E**
- E3ME modelling framework  
 expected macroeconomic outcomes, 165–167  
 forming model inputs, 163  
 healthcare costs, 163–164  
 health-economic impacts, 162  
 labour market participation, 164  
 labour productivity, 165  
 modules, 163  
 mortality rates, 164

- Earth's global cycle of lead, 11  
 Earth's natural global metal cycles, 12  
 Economic models, consumer behaviour, 133  
 ECOSENSE, 121  
 EDC-MICRISK, 261  
 EDEN integrated project, 255  
 EEA, 243  
 EFSA, 106  
 Electric arc (EA) process, 17  
 Electrostatic precipitators (ESPs), 7, 18  
 Emission inventory, 8  
 Emission sources, 13, 22, 23  
 Empowerment, 228  
 Endocrine-disrupting chemical, 65  
 Endocrine disrupting compounds (EDC), 89  
 ENRIECO project, 86, 88  
 Environment and health policies, 259  
 Environment and health research, 260  
 Environmental Burden of Disease in European countries (EBoDE) project, 84  
 Environmental contaminants and human health, research projects  
     ArcRisk project, 86  
     CHICOS, 87  
     CLEAR project, 87  
     COPHES/DEMOCOPHES projects, 88  
     ENRIECO project, 88  
     OBELIX project, 89  
     PHIME project, 89  
 Environmental governance, public participation  
     decision-making, 231  
     evaluation phase, 232  
     IAP2 categorisation, 227  
     implementation and monitoring phase, 231  
     participants and methodsselection, 229, 230  
     problem definition, 229  
     stakeholder engagement, 230  
 Environmental hazards and health impacts, 27  
 Environmental health, 216–219, 226  
 Epigenetics, 73  
 EPI-MIGRANT project, 257  
 Epistemological divide, 221  
 Epstein-Barr virus (EBV), 69  
 EU-funded environment and health research, 258–262  
 EU-funded research projects, 86  
 EU General Environmental Action Programme, 262  
 EU Nanosafety cluster, 255  
 European Community Respiratory Health Survey II (ECRHS II), 41  
 European Environmental Agency (EEA), 234  
 European Exposome Initiative, 262  
 European Food Safety Authority (EFSA), 98  
 European Human Biomonitoring Initiative (EHBMI), 261  
 EVERYAWARE, 241  
 Expert elicitation approach, 217  
 Exposome, 28  
 Exposure science, 28  
 External quality assessment scheme (EQAS), 54  
 ExternE, 146  
 ExternE projects, 145  
 ExternE's IPA method, 136
- F**
- Fabric filters (FFs), 18  
 Fifth Framework Programme (FP5), 250, 253  
 Flue gas desulfurisation (FGD), 18  
 Flue gas desulphurization (FGD), 7  
 Fluidized bed combustion (FBC), 17  
 FP6 and FP7, 253  
 Framingham Heart Study for heart disease, 29  
 Full chain approach, 120  
 Funding trends and drivers  
     action plan, 253  
     consolidation and re-assessment period, 255–258  
     environmental stressors, 256  
     EU contribution, 251  
     first period, 250  
     period of acceleration, 251–254
- G**
- GA2LEN Network of Excellence, 254  
 Global emissions of metals, 9  
 Global warming, 169–171, 196  
 Glutathione (GSH), 98  
 Greenhouse gases (GHGs)  
     abatement cost curve, 182, 183  
     agriculture and forestry, 178  
     air pollution, 175  
     CCS, 196  
     emissions, 126–127  
     emission reduction, 130
- H**
- Health benefits  
     metals' exposure reduction, 146  
     quality of life indices, 145–146  
 Health end-points, 124  
 Health impacts  
     economic valuation  
         cost components, 137, 139

- Health impacts (*cont.*)  
 damage function approach, 135–136  
 methods (*see* Valuation methods)  
 WTP, 136  
 monetary valuation  
 cancer risk, 143  
 characteristics, 142  
 OECD meta-analysis, 142  
 Hedonic price approach, 140  
 HENVINET  
 expert elicitation approach, 217  
 role-play and social learning, 219–220  
 Hexabromocyclodecane (HBCD), 54  
 Hexachlorobenzene (HCB), 53, 62  
 $\beta$ -Hexachlorocyclohexane ( $\beta$ -HCH), 53  
 Horizon 2020, 260  
 Household production approach, 141  
 HRAPIE project, 123  
 Human biomonitoring, 214–216  
 HYENA study of noise, 35
- I**  
 Impact assessment guidelines, 152  
 Impact pathway, 120  
 Impact pathway analysis (IPA), 135  
 Impact pathway approach, 120–125  
 Impaired health, 134  
 Indigenous Arctic population, 71, 72  
 Input-output and multiplier analysis, 157  
 Integrated assessment (IA), 118  
 International Agency for Research on Cancer (IARC), 103  
 INTERPHONE project, 251  
 INUENDO project, 58  
 Inuit Cohort (Canada), 58  
 Iron (Fe), 107, 108
- K**  
 Kola Lapland study 2001–2006, 57
- L**  
 LCA. *See* Life cycle assessment (LCA)  
 LCIA approach, 146  
 Lead (Pb), 101–103  
 Lead exposures, 37  
 Life cycle assessment (LCA), 192–194  
 Life cycle impact assessment (LCIA)  
 methodology, 146  
 Life expectancy, 139  
 Long-chain polyunsaturated fatty acids (LCPUFA), 99
- Loss of productivity, 138  
 Low carbon economy, 169, 185–195  
 air pollutants, 174–177  
 application, 179–180  
 cold-related mortality, 173  
 CSS (*see* Carbon capture and storage (CCS))  
 energy intensity, 179–180  
 fossil fuels, 182  
 GHGs (*see* Greenhouse gases (GHGs))  
 Kyoto Protocol propose, 178  
 plant and animal species, 172  
 renewable energy, 181  
 vulnerability, 174  
 water resources and agriculture, 172  
 Lung cancer, 70
- M**  
 Macroeconomic modelling  
 CGE models, 158–159  
 counterfactual scenario (*see* Counterfactual scenario design)  
 coverage, 160  
 health care costs, 153  
 health effects, 153, 160–161  
 input-output and multiplier analysis, 156–157  
 labour market participation, 154  
 labour productivity, 154  
 model capabilities, 162  
 mortality rates, 153  
 parameters, 160  
 policy analysis, 152  
 public benefits, 154  
 Main model linkages, 167  
 Manganese (Mn), 108, 110  
 Man-kind mobilization of metals, 8  
 Marginal avoidance costs (MAC), 127  
 Mass burn/waterwall (MB/WW) type, 17  
 Mercury, 4  
 biological guideline values, 91  
 cardiovascular effects, 64–74  
 neurological effects, 60–64  
 toxicity, central nervous system, 92  
 toxicokinetics, 92  
 Metal emission control, DSS, 20–23  
 Metals  
 anthropogenic sources, 5–9  
 aquatic emissions, 7  
 atmospheric emissions, 6–8  
 biogeochemical cycling, 9–12  
 CVD and asthma, 36, 37  
 discharges into soils, 7–8  
 DSS, emission control, 20–23

- emissions of, 4–9
  - global emission to environment, 8–9
  - harmful pollutants, 2–4
  - local scale flows, 15–16
  - man-kind mobilization, 9
  - measures to reduce anthropogenic emissions
    - non-technological measures, 18–20
    - technological measures, 16–18
  - natural sources, 4–5
  - perturbations of geochemical cycles,
    - exposure pathways and health concern, 3
  - public health concern, 2
  - regional scale flows, 12–14
  - soil forming processes, 4
- Methylmercury (MeHg), 92, 97–99, 111
- MFA diagram, Cd, 14
- Minamata convention, 4, 18
- MISA study of Northern Norway, 55–56
- Municipal sewage sludge, 8, 14
  
- N**
- Net effects, 156
- Neurological effects
  - immunological effects, 62–63
  - lead and mixed metal exposures, 61
  - mercury, 60–64
  - persistent organic pollutants, 62
  - reproductive effects, 63–64
- Noise pollution, 35, 36
- Non-allergic asthma, 30
- Non-market valuation, 140
- Non-transferrin-bound iron (NTBI), 108
- Northern Finland 1966 Birth Cohort, 56–57
- Nunavik Child Development Study (NCDS), 58, 60
- Nunavik Health Survey Qanuippitaa, 58
  
- O**
- OBELIX project, 86, 89
- Omniscientis, 241
- Open hearth (OH) processes, 17
- Organochlorines (OCs), 62
- Organotin, 99–101
- Ovarian and prostate cancers, 71
- Ozone exposure, 32
  
- P**
- Pancreatic cancer, 71
- Parkinson's disease (PD), 109
- Perfluorinated compounds (PFCs), 60
- Perfluorooctane sulfonate (PFOS), 54
- Perfluorooctanoic acid (PFOA), 54
- Persistent organic/toxic pollutants (POPs), 85, 93
- Persistent toxic substances (PTS), 57
- PHIME project, 86, 89, 92, 93
- Phthalates, 40, 41
- Pollutant-health pairs, 145
- Polybrominated diphenyl ethers (PBDEs), 54
- Polychlorinated biphenyls (PCBs), 52, 62, 93
- Polycyclic aromatic hydrocarbons (PAHs), 31, 34
- Polyunsaturated fatty acids (PUFA), 65
- Polyvinyl chloride (PVC) heat stabilizer, 99
- Potentially disappeared fraction (PDF) of species, 125
- Problem solving collaborations, 222
- Public participation
  - citizen science design, 237
  - data issues, 242, 243
  - decision-making, 231
  - EEA, 243
  - evaluation phase, 232
  - human health
    - decision-making strategies, 226, 227
    - environmental factors, 225
  - IAP2 categorization, 227
  - implementation and monitoring phase, 231, 232
  - levels, 228
  - participants and methodsselection, 229, 230
  - problem definition, 229
  - process design, 229
  - stakeholder engagement, 230
- Public participation in scientific research (PPSR), 233
  
- Q**
- QALY, 146
  
- R**
- RANCH project, 252
- Rare Earth Elements (REE), 24
- Rawlsian approach, 134
- Renewable energy, 181
- Residue desulphurization (RDS), 17
- Resource costs, 137
- Revealed preference approach, 141
- Risk assessment, 218
- Risk communication
  - deficit model, 204
  - environmental health, 206
  - issues, 204

Risk management, 205  
 Risk perception, 211  
 Rural and urban background concentrations,  
 122

## S

Sea-salt production processes, 5  
 Second hand smoke (SHS), 34  
 Selenium, 65  
 Semi-volatile compounds (SVOCs), 31  
 Serum steroid hormone-binding globulin  
 (SHBG) levels, 64, 66  
 SEURAT-1 cluster, 257  
 Seventh Framework Programme, 255  
 Social welfare, 134  
 Stock–flow model approach, 12  
 Substance flow analysis (SFA), 12

## T

Tetraalkyllead, 101  
 Tetrabromobisphenol A (TBBPA), 54  
 Tetraethyllead, 111  
 Tobacco-specific nitrosamines (TSNA), 72  
 Toxic metals, 9  
 Toxicokinetic modelling, 93  
 Treatment/direct costs, 138  
 Triethyltin (TET), 100, 111  
 Trimethyltin (TMT), 100, 111  
 Trisenox, 103  
 Tromsø Study, 56  
 Two-way risk communication  
 amateurism, analytical deliberative work,  
 214–216

collective decision-making, 209  
 elements, 209–213  
 history, 205–206  
 risk management, 207  
 senders and receivers roles, 207  
 senders of information, 209  
 stakeholders, 208

## U

Ultrafine particle (UFP) concentrations, 33  
 Uncertainties, 125–126  
 UNFCCC. *See* United Nations Framework  
 Convention on Climate Change  
 (UNFCCC)  
 Unified EMEP model, 121  
 United Nations Framework Convention on  
 Climate Change (UNFCCC), 170

## V

Valuation methods  
 benefit transfer method, 141  
 CVM and DCE, 141  
 non-market valuation, 140  
 Value Of Life Year (VOLY), 139, 143  
 Volatile organic compounds (VOCs), 31

## W

Welfare economics, 134  
 Welfare loss due to premature death, 139  
 WeSenseIt, 241  
 Willingness-to-pay (WTP), 123, 136, 144  
 World Health Organization (WHO), 36, 103