EEA Report | No 10/2005

Environment and health

ISSN 1725-9177

EUROPEAN COMMISSION
DIRECTORATE-GENERAL
Joint Research Centre

European Environment Agency

Environment and health

EUROPEAN COMMISSION
DIRECTORATE-GENERAL
Joint Research Centre

European Environment Agency

Cover design: EEA
Front cover photo: © Isabelle Rozenbaum, PhotoAlto Everyday life 18.
Left smaller cover photo: © Michèle Constantini, PhotoAlto Sustainable resources 219.
Right smaller cover photo: © Michèle Constantini, PhotoAlto Sustainable resources 219.
Layout: Brandpunkt A/S

Luxembourg: Office for Official Publications of the European Communities, 2005

ISBN 92-9167-781-7
ISSN 1725-9177

European Environment Agency
Kongens Nytorv 6
1050 Copenhagen K
Denmark
Tel.: +45 33 36 71 00
Fax: +45 33 36 71 99
Web: www.eea.eu.int
Enquiries: www.eea.eu.int/enquiries

Contents

Acknowledgements

This report was prepared in cooperation between the European Environment Agency and the Joint Research Centre (JRC) of the European Commission. The project manager at EEA was Ingvar Andersson with Peter Pärt at JRC as the main author.

EEA acknowledges advice provided at various stages during the preparation of the report by a peer review group consisting of:

Josep M Antó, Universitat Pompeu Fabra, Spain;
Herman Autrup, Aarhus university, Denmark;
Marek Biesiada, Institute of Occupational Medicine and Environmental Health, Poland;
David Briggs, Imperial College, the United Kingdom;
Scott Brockett, DG Environment;
Dafina Dalbokova, WHO, Germany;
Lis Keiding, National Board of Health, Denmark; and
Ann Thuvander, Socialstyrelsen, Sweden.

We have also received valuable comments from Argelia Castaño Calvo, CISA-INIA, Spain, Trine S. Jensen, NERI, Denmark, Francesca Valent, Istituto di Igiene ed epidemiologia, Italy, Andre Zuber and Juergen Helbig, DG Environment, as well as the comments provided by the national focal points of the European environment information and observation network (Eionet).

Abstract

Public concerns, evidence from research and increasing scientific knowledge are all driving widespread discussions on environment and health problems. The issue of environment and health is characterised by multi-causality with different strengths of association. This means that the links between exposures and their health consequences depend on the environmental pollutants and diseases being considered, but are also influenced by factors such as genetic constitution, age, nutrition and lifestyle, and socioeconomic factors such as poverty and level of education.

Air pollution indoors and outdoors are the environmental factors with the greatest impact on health in Europe and are responsible for the largest burden of environment-related disease. Recent estimates indicate that 20 million Europeans suffer from respiratory problems every day. Particulate matter and especially small particles with a diameter less than 2.5 micrometres ($PM_{2.5}$) are associated with increased mortality, especially from cardiovascular and cardiopulmonary diseases.

Asthma is increasing all over Europe, although there is a significant variation between EU-25 countries. The societal cost of asthma has been estimated at EUR 3 billion/year. Clearly, asthmatic persons, and particularly asthmatic children, are sensitive to air quality and several studies show a strong association between exposure to air pollution and the aggravation of asthma. To what extent air pollution initiates asthma is unclear.

Cancer in European children younger than 15 years of age is in general terms rare but remains one of the most common causes of death in children in industrialised countries. Radon exposure is the best-documented environment-related cause of cancer but is localised in geographical areas where radon precursors (uranium) occur naturally in the ground.

A number of chemicals are potentially carcinogenic. Approximately 500 are classified as carcinogens and are legally not allowed to reach the consumer. They may, however, reach the environment via diffuse sources, for instance in accidental cases. Arsenic in drinking water and cadmium from diffuse sources are environmental contaminants of special concern, because of increasing environmental exposure and their suspected carcinogenicity.

Every European citizen is today exposed to electromagnetic fields (EMF) which can be characterised in terms of their frequency and amplitude. Neither experimental studies on animals nor epidemiological studies have come to any conclusive results. On the basis of current scientific knowledge it is not possible to answer 'yes' or 'no' to the question of whether the use of mobile phones poses an increased risk of cancer.

Mercury and lead at concentrations that are sometimes observed in the environment are well known to have neurodevelopmental effects.

Environmental noise is probably the environmental factor that affects the largest number of Europeans. The main health risks of noise are annoyance, interference with social behaviour and speech communication, sleep disturbance and all its consequences, cardiovascular effects, hormonal responses and poor performance at work or school.

Endocrine disruptors are substances that interfere with hormone-dependent functions in the body such as embryonic development, production of sperm, control of the menstrual cycle, the onset of puberty and cancers in hormone-dependent tissues. Worldwide, a decline in semen quality has been observed over the past 50 years but no clear connection to endocrine disrupters has been established.

Every European citizen has man-made chemical contaminants in his or her body. Bio-monitoring of different populations clearly shows an increased body burden of some persistent and bio-accumulative substances, but concentrations of other substances are decreasing.

The global distribution of 'new' metals used in automobile catalytic converters to reduce hydrocarbon pollution is clearly shown in the

Arctic. Concentrations of platinum, palladium, and rhodium in ice and snow in Greenland have increased rapidly since the 1970s.

Arctic human populations are at risk due to the long-distance transport of bio-accumulative substances, with the Arctic as an important sink, and the dependence of indigenous populations on traditional diets exposes them unduly to chemicals accumulated in the food chain. Europe and other developed countries have a clear responsibility for the global body burden of chemicals. This raises issues of equity and global responsibility.

Wildlife can serve as a sentinel to give early warnings of the effects of chemicals at natural exposure levels. Examples include predator birds and mammals with high levels of bio-accumulative, persistent and toxic chemicals.

Links between climate change and health are increasingly being identified, for example 35 000 heat-induced deaths in Europe were registered during the 2003 heat wave, mainly due to dehydration, especially of elderly people. There are possible proactive healthcare measures to combat the impact of such events, which particularly affect vulnerable groups, again raising issues of equity.

Key conclusions

- Public concerns, evidence from research and increasing scientific knowledge are all driving widespread discussions on environment and health problems. The child-focused EU SCALE process (science, children, awareness-raising, legal instruments, evaluation) has initially identified four priority groups of diseases: childhood cancer, childhood respiratory health/asthma, neurodevelopmental and endocrine disorders, but the list of potential pollution-induced diseases is much longer and also includes diseases of adults. The 'European environment and health action plan' will take this into account.

- The issue of environment and health is characterised by multi-causality with different strengths of association. This means that the links between exposures and their health consequences depend on the environmental pollutants and diseases being considered, but are also influenced by factors such as genetic constitution, age, nutrition and lifestyle, and socioeconomic factors such as poverty and level of education. Important elements of exposure and risk assessment are estimation of the body burden of chemicals, combined exposures from multiple sources (food, air and water), the 'cocktail-effect' and the timing of exposures. The topic of 'Late responses to early exposure' needs to be addressed, taking account of individual and generational exposure and its effect. Preventive measures require the development of proactive risk assessment and management responses that can contribute to the formulation of adequate measures, taking into account complexities and uncertainties. Due to the complexity and multi-causality of the links between environment & health, there needs to be wider use of the precautionary principle in order to prevent significant causes of disease and ill health.

- Estimates of the burden of disease attributable to environmental factors vary between different studies, depending on the type of disease, vulnerability, genetics and population group. There are big differences between people living in industrialised and developing countries, and between the east and west of Europe. The environment-related share of the burden of disease also depends strongly on socioeconomic aspects such as income, the share generally being higher in lower-income countries: it is estimated to be 2–6 % of the total burden of disease in the OECD and 13 % in non-OECD countries. Among children aged 0–4 years living in the WHO European region (51 countries), 1.8–6.4 % of deaths from all causes were attributable to outdoor air pollution. Acute lower respiratory tract infections attributable to indoor air pollution accounted for 4.6 % of all deaths and 3.1 % of DALYs (disability adjusted life years). Mild mental retardation resulting from lead exposure accounted for 4.4 % of DALYs.

- Air pollution is the environmental factor with the greatest impact on health in Europe and is responsible for the largest burden of environment-related disease. Recent estimates indicate that 20 million Europeans suffer from respiratory problems every day.

- Particulate matter and especially small particles with a diameter less than 2.5 micrometres ($PM_{2.5}$) are associated with increased mortality, especially from cardiovascular and cardiopulmonary diseases. A WHO evaluation 2004 found that air pollution was responsible for approximately 100 000 deaths and 725 000 years of lost life (DALYs) each year in a selection of European cities within the WHO European region. The more recent estimates of the impact of air pollution made in the European Commission 'Clean air for Europe' (CAFE) programme found that in the EU about 350 000 people died prematurely in 2000 due to the outdoor air pollution caused by fine particulate matter ($PM_{2.5}$) alone. This corresponds to an average loss of life expectancy of about 9 months for every EU citizen.

- In addition, current levels of ozone have severe health implications, such as bringing forward the deaths of more than 20 000 people per year. There

are many examples that show that respiratory health and life quality improve when air quality improves.

- Emissions of ozone precursors as well as of primary PM_{10} have been reduced substantially, by about 20 % between 1996 and 2002, but are not reflected in the ambient concentration trends of these pollutants. PM_{10} concentrations decreased until 2000 but have increased slightly since then. The levels of PM_{10} are expected to exceed the EU annual limit value for PM_{10} of 40 µg/m^3 in force from 2005 in many European urban areas.

- Ozone concentrations generally exceeded the long-term objective to protect human health, 120 µg/m^3 during 8 h, in most EU Member States. The highest concentrations of nitrogen oxides are observed at street level due to traffic emissions — especially in larger cities throughout Europe. Reduced emissions of nitrogen oxides lead to increased ozone levels in the centres of cities.

- Asthma is increasing all over Europe. The societal cost of asthma has been estimated at EUR 3 billion/year. A large portion of the increase is in asthma in children. On average 7 % of European children aged 4–10 have problems with asthma, although there is a significant variation between EU-25 countries. Clearly, asthmatic persons, and particularly asthmatic children, are sensitive to air quality and several studies show a strong association between exposure to air pollution and the aggravation of asthma. To what extent air pollution initiates asthma is unclear, and in general it seems that the association between air pollution and the initiation of children's asthma is weak. Support for this conclusion comes from a number of studies that found allergic disorders (including asthma) to be relatively less frequent in eastern parts of Europe, although levels of many air pollutants were higher than in western Europe. It has to be stressed in this context that the initiation and development of asthma has a multi-causal background including 'lifestyle' and nutritional factors, and a certain genetic predisposition, and it is almost impossible to single out one specific factor as being responsible.

- Poor indoor air quality is the source of a number of health problems, including cancer, allergic symptoms, distress, sleeping and concentration problems, and coughing, wheezing and asthma-like symptoms in children. Many indoor problems are related to increased moisture and humidity, part of which is a consequence of energy-saving policies that have led to reduced rates of air exchange in homes, schools and office buildings. Other indoor air quality problems arise from construction materials, paints, household cleaning agents, environmental tobacco smoke and combustion processes.

- Cancer in European children younger than 15 years of age is in general terms rare but remains one of the most common causes of death in children in industrialised countries. The most common childhood cancers are leukaemia and brain tumours. A small but significant increase in childhood cancers has been noted recently, since the mid-1980s. One reason for this may be better diagnostic methods, but an additional component of environmental exposure cannot be excluded.

- Radon exposure is the best-documented environment-related cause of cancer but is localised in geographical areas where radon precursors (uranium) occur naturally in the ground.

- A number of chemicals are potentially carcinogenic: approximately 500 are classified as carcinogens and are legally not allowed to reach the consumer. They may, however, reach the environment via diffuse sources, for instance in accidental cases. Arsenic in drinking water and cadmium from diffuse sources are environmental contaminants of special concern, because of increasing environmental exposure and their suspected carcinogenicity.

- Every European citizen is today exposed to electromagnetic fields (EMF) which can be characterised in terms of their frequency and amplitude. The fields around power lines and electrical appliances are of low frequency. Mobile telephones and radio transmitters transmit EMF of higher frequency. The explosion in the use of mobile telephones has initiated many research efforts concerning health effects of the higher frequency EMF generated by phones and the base stations. Neither experimental studies on animals nor epidemiological studies have come to any conclusive results. On the basis of current scientific knowledge it is not possible to answer 'yes' or 'no' to the question of whether the use of mobile phones poses an increased risk of cancer. However, mobile phones have only been in widespread use for just over 10 years and more studies over longer periods are needed before any firm conclusions can be drawn.

- Some persistent organic chemicals have been proved to have neurodevelopmental effects during prenatal and post-natal life in humans. Prenatal exposure to polychlorinated biphenyls (PCBs) has been associated with negative effects on cognitive processes, motor development and reflexes in children. PCBs and brominated flame retardants are indicated to interfere with the function of the thyroid hormone, which is crucial for normal neurodevelopment.

- Mercury at concentrations that are sometimes observed in the environment is well known to have neurodevelopmental effects, for example attention problems, reduced learning ability, and slightly reduced IQ in children. Measures are now being taken in Europe to reduce, among other things, prenatal mercury exposure and to ensure that tolerable daily intakes for pregnant women are not exceeded.

- Lead is an established neurodevelopmental toxicant to humans. Recent studies on the effects of lead in humans suggest that a 'safe' exposure level currently cannot be established. More data on lead exposure of European citizens are necessary and are currently being collected. A ban on leaded petrol has been very successful in lowering blood lead levels in children, which clearly indicates a reduced exposure.

- Environmental noise is probably the environmental factor that affects the largest number of Europeans. The main health risks of noise are annoyance, interference with social behaviour and speech communication, sleep disturbance and all its consequences, cardiovascular effects, hormonal responses and poor performance at work or school.

- Endocrine disruptors are substances that interfere with hormone-dependent functions in the body such as embryonic development, production of sperm, control of the menstrual cycle, the onset of puberty and cancers in hormone-dependent tissues. Worldwide, a decline in semen quality has been observed over the past 50 years but no clear connection to endocrine disrupters has been established. Breast and testicular cancers are increasing in Europe but the connection to endocrine disrupters is weak, at least on the basis of current knowledge. Intensive research on this topic is under way. Environmental endocrine disruption in humans is at present far more a matter of speculation than of demonstrated fact. Much of the basis for concern derives from strong evidence of endocrine disruption in wildlife.

- Every European citizen has man-made chemical contaminants in his or her body. Bio-monitoring of different populations clearly shows an increased body burden of some persistent and bio-accumulative substances, but concentrations of other substances are decreasing.

- The global distribution of 'new' metals used in automobile catalytic converters to reduce hydrocarbon pollution is clearly shown in the Arctic. Concentrations of platinum, palladium, and rhodium in ice and snow in Greenland have increased rapidly since the 1970s.

- Arctic human populations are at risk due to the long-distance transport of bio-accumulative substances, with the Arctic as an important sink, and the dependence of indigenous populations on traditional diets exposes them unduly to chemicals accumulated in the food chain. Europe and other developed countries have a clear responsibility for the global body burden of chemicals. This raises issues of equity and global responsibility.

- Wildlife can serve as a sentinel to give early warnings of the effects of chemicals at natural exposure levels. Examples include predator birds and mammals with high levels of bio-accumulative, persistent and toxic chemicals.

- Unintentional releases of pharmaceuticals, personal-care products and bio-wastes into the environment are emerging concerns.

- Links between climate change and health are increasingly being identified, for example 35 000 heat-induced deaths in Europe were registered during the 2003 heat wave, mainly due to dehydration, especially of elderly people. There are possible proactive healthcare measures to combat the impact of such events, which particularly affect vulnerable groups, again raising issues of equity.

- The multi-causality of health impacts is well exemplified by the wider area of health and ecosystems, for example with the severe health effects of disasters such as heat waves, floods and forest fires. Ecological and land-use changes can augment effects when natural or 'ecological' protection has disappeared. Examples are reduced wetland buffering areas, straightening of rivers, forestry and logging, and for tsunamis, reduction of wave protection in the form of coral reefs and mangroves.

1 Introduction

Bald eagles in Florida (1952), river otters in England (late 1950s), mink in Michigan (mid-1960s), pheasants, eagles and ospreys in Sweden (1960s), fish consumers in Japan (1960s), herring gulls in Michigan (1970), western gulls in California (early 1980s), alligators in Florida (1980s), oysters in France (1980s), seals in northern Europe (1988), dolphins in the Mediterranean (early 1990s), and sperm counts in men worldwide (1992); pharmaceutical residues from humans and farm animals, POP contamination in the Great Lakes and in the Baltic Sea and air pollution from sulphur dioxide — these are only a few of the signals that are driving public concerns about the impacts of unsafe and unhealthy environments on ecosystems and human health.

Human health and environmental quality are closely linked. Environmental pollution and other aspects of poor environmental quality have well-established effects on human health and the quality of life. A 2001 OECD review (OECD, 2001) estimated that 5–8 % of all health problems in the high- and middle-income OECD countries, and more than 13 % in non-OECD countries were environment-related. Indoor and outdoor air pollution, hazardous chemicals, noise, food and water contaminants are all major causes of the environment-related burden of disease. Cancer, respiratory diseases, allergies and asthma, cardiovascular disease, neurological effects, and different reproductive and developmental disorders are examples of health outcomes associated with environmental factors (see Table 1). Major sources of air pollution are the transport and energy sectors for outdoor air, and the housing environment (ventilation, construction materials, humidity, mould, mites, and dust) for indoor air. Sources of chemical pollution include industrial activities, households, agriculture, waste disposal and incineration.

A number of 'new' potential health threats have recently been added to the list in recent years. Concern has been expressed over the environmental and health risks associated with nanotechnologies, the impact of electromagnetic and radio-frequency fields, the effects of climate change (increased temperatures, UV radiation) and the consequences to human health of ecological deterioration and imbalances that result in toxic algae blooms, new spreading patterns of pathogens, flooding and severe forest fires.

Public concerns about the interactions between the environment and health, and the pressure to develop preventive and protective strategies put high demands on science and research. There is still a lack of essential information on how environmental factors interfere with human health and well-being. Environmental exposures occur during the whole human lifecycle from the moment of conception, through germ cell differentiation and development, pre- and post-natal development, growth into adulthood and adult life, to aging and senescence. Exposure levels are generally low, but the exposure situation is complex. Environmental stressors are rarely present alone: exposure in our daily lives is usually caused by mixtures of several stressors, often in combination from different sources. This fact is fully appreciated in environment and health risk management today. Exposure assessment, hazard identification, risk estimation and risk communication are elements in an integrated model used by many European Union Member State health authorities. It uses information from all sources from in vitro toxicology, through eco- and conventional toxicology to epidemiology.

Evidence and knowledge about public health and the priority diseases (childhood respiratory disease and asthma, childhood cancer, neurodevelopmental disorders and endocrine disruption) and the contributing environmental factors are the starting point of this report, also seen in the wider context of multi-causality. The exposure of the European population and Europe's responsibility for the global burden of chemicals are exemplified by human bio-monitoring data. The link between climate change and human health is summarised. Although the focus of the report is the 'state of the environment', the 'state of action' is also described, especially in areas with ongoing work on policies and regulation, like air pollution and pesticides. However, action and prevention are not only a matter of policies and regulation. Consumer lifestyles and behaviour are more important to a healthier life (see Figure 1).

The environmental burden of disease is a rather new concept, analysing causality and estimating the importance of different environmental factors to the background of disease. Quantification of the disease burden caused by environmental stressors is important for developing policies and mitigation measures. This concept is applied in Europe as well as in a global context.

The EU 'Environment and health action plan 2004–2010' identifies four groups of diseases or physiological disturbances as the priority for the first implementation phase. These are childhood respiratory disease and asthma, childhood cancer, neurodevelopmental disorders and endocrine disruption. The focus is on these diseases, but some other health outcomes where environmental factors are strongly implicated will be added.

One way of understanding how environmental exposures affect health is to study animals in the wild. Despite many differences, many fundamental physiological processes in animals and humans are identical or very similar. An effect in wild animals carries a strong implication that a similar effect may occur in humans in a similar exposure situation.

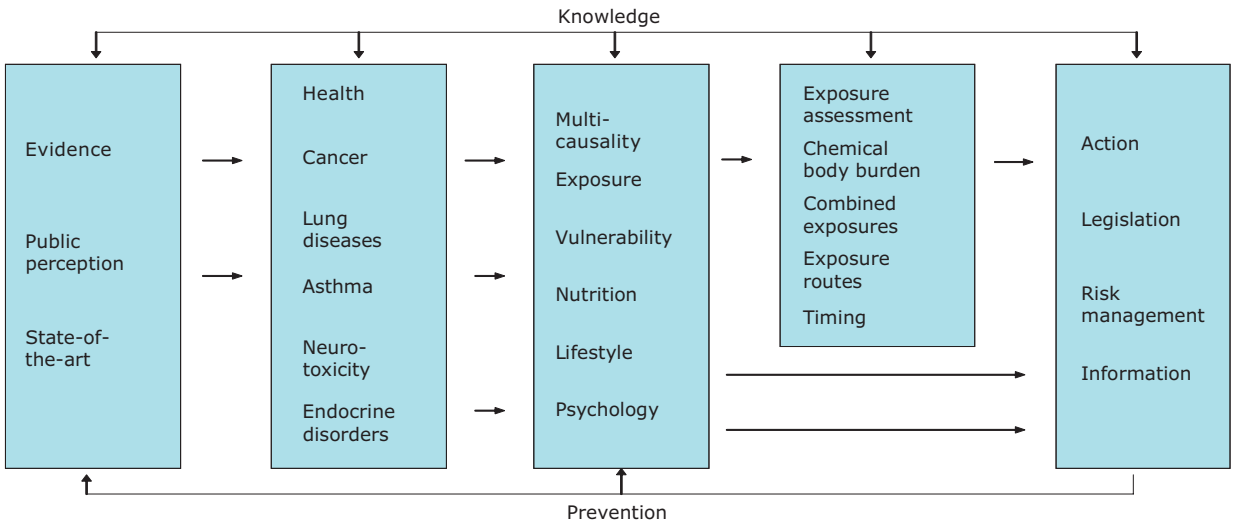
The policy context

Concern for human health has traditionally underlain much of the political priority given to environmental issues within the EU and the Member States. One of the primary drivers for policy actions that target air quality (CAFE — Clean air for Europe), water quality (water framework directives), soil quality and chemicals (REACH, endocrine disrupters, persistent organic compounds, and dioxins) is concern about human health. The direct connection between environmental quality and human health is therefore highlighted as a priority in the sixth environmental action plan of the EU. In 2003, the European Commission developed a proposal for a Community Strategy for Environment and Health (COM(2003) 338), followed in 2004 by the European environment and health action plan 2004–2010 (COM(2004) 416). The Commission is here proposing a holistic approach to address the complex issues of environmental quality and human health. The ambition is to create an integrated environment and health information system where environmental and human health information can be combined.

Children's environment and health is currently a priority issue on several international agendas. WHO-Europe has developed CEHAPE (Children's environment and health action plan for Europe) adopted at the Fourth Ministerial Conference on Environment and Health in Budapest in June 2004; individual EU Member States like Sweden and France have environment and health programmes that target children; and the US EPA is running a specific monitoring programme (America's children and the environment, 2001 and 2003).

Note: Ref: COM(2004) 416 final, 'The European environment and health action plan 2004–2010'.

Figure 1 A framework from knowledge to action



Source: EEA.

2 Environmental burden of disease

An indicator of the 'burden of disease' is the number of 'disability-adjusted life years' (DALYs) (Homedes 2000), which gives an indication how disease can alter the ability of people to live a normal life compared with those with no disease. At the global level, an OECD environment outlook (OECD, 2001) study clearly shows a significant difference in DALYs between OECD and non-OECD countries. The environment-related share of the burden of disease depends strongly on income, with higher environmental shares generally occurring in lower-income countries. In OECD countries, this share is estimated to be 2–6 % of the total burden of disease (OECD, 2001). The contribution of different factors to the burden of disease differs between countries. Most of the burden in non-OECD countries can be attributed to communicable disorders (e.g. infectious, maternal, prenatal), while health loss in the OECD countries is mainly from non-communicable (chronic, degenerative) diseases. Conditions such as heart disease and depression make up a major portion of the burden of disease in OECD countries. Diseases in children under 4 years old account for 50 % of the total burden of disease in non-OECD countries, but significantly less (only 7 %) in OECD countries. The large environmental share of health problems in non-OECD countries (diarrhoea, TBC, etc.) is due mainly to factors related to poverty, such as limited access to proper food, housing, healthcare, and drinking water. Environmental determinants of human health in OECD countries are related more to exposure to air pollutants, mainly in urban areas, and to chemicals in the environment.

Attributing percentage contributions of environmental burden of disease is based on an essentially uni-causal model of the environment and health interactions focusing on a single stressor at a time. However, for environment at health issues the situation is more complex being the result of interactions between environmental stressors, life-style and genetic factors (a multi-causal model).

The effects of exposure to environmental stressors on health vary for different diseases; Table 1 gives a general picture of associations between diseases and exposures through different routes.

The burden of children's diseases attributable to selected environmental factors has also been evaluated in connection with the WHO initiative 'Children's environment and health action plan for Europe' (CEHAPE). The estimates cover outdoor and indoor air pollution, inadequate water and sanitation, lead exposure and injuries among children and adolescents living in European (EU-25), and south-west Asian countries.

Among children aged 0–4 years, 1.8–6.4 % of deaths from all causes were attributable to outdoor air pollution. Acute lower respiratory tract infections attributable to indoor air pollution accounted for 4.6 % of all deaths and 3.1 % of DALYs. Mild mental retardation resulting from lead exposure accounted for 4.4 % of DALYs (Valent *et al.* 2004).

In the 0–14 age group, inadequate water and sanitation leading to diarrhoea caused 5.5 % of deaths and 3.5 % of DALYs. In the 0–19 age group, injuries were the cause of 22.6 % of all deaths and 19 % of DALYs.

The burden of disease was much lower in the western European countries than in the new EU Member States. The study concludes that there is an urgent need for interventions aimed at reducing children's exposure to unsafe water, outdoor and indoor air pollution and preventing injuries. These interventions must be done within the environment and health sector, but also in energy, transport, urban planning and education.

Table 1 Major health impacts and some associations with environmental exposures to chemicals and other environmental stressors and lifestyle factors

Health impact	Associations with some environmental exposures
Infectious diseases	• water, air and food contamination • climate-change-related changes in pathogen life cycle
Cancer	• air pollution (PM), mainly $PM_{2.5}$ or less • smoking and environmental tobacco smoke (ETS) • some pesticides • asbestos • natural toxins (aflatoxin) • polycyclic aromatic hydrocarbons, e.g. in diesel fumes • some metals, e.g. arsenic, cadmium, chromium • radiation (including sunlight) • radon • dioxins
Cardiovascular diseases	• air pollution (carbon monoxide, ozone, PM) • smoking and ETS • carbon monoxide • lead • noise • inhalable particles • food, e.g. high cholesterol • stress
Respiratory diseases, including asthma	• smoking and ETS • sulphur dioxide • nitrogen dioxide • inhalable particles ($PM_{2.5}$ and PM_{10}) • ground-level ozone • fungal spores • dust mites • pollen • pet hair, skin and excreta • damp
Skin diseases	• UV radiation • Some metals, e.g. nickel • pentachlorophenol • dioxins
Diabetes, obesity	• food, e.g. high fat • poor exercise
Reproductive dysfunctions	• polychlorinated biphenyls (PCBs) • DDT • cadmium • phthalates • endocrine disruptors • pharmaceuticals
Developmental (foetal and childhood) disorders	• lead • mercury • smoking and ETS • cadmium • some pesticides • endocrine disruptors
Nervous system disorders	• lead • PCBs • methyl mercury • manganese • some solvents • organophosphates
Immune response	• UVB radiation • some pesticides
Increased chemical sensitivity	• multiple chemical exposures at low doses

Note: Many stressors, like air pollution, POPs, dioxins, pesticides and heavy metals, are under strict regulatory control.

Source: Updated after European Environment Agency (2003), Europe's environment: the third assessment. Environmental assessment report No 10.

Ecosystems and health — examples of multi-causality (extreme weather events and disasters)

Flooding
Flooding extremes that occur within a comparatively short time can severely affect health, directly through diseases, injury or death and indirectly through effects on crops and animals.

In Europe, floods are the most common natural disaster. Since 1990, about 2 000 people have died during floods and some 400 000 have become homeless. The main effects were drowning, injuries, and, perhaps most importantly for floods in Europe, the psychological health effects that result from being flooded. Climate change may increase the risk of both river and coastal flooding.

Environmental factors, including land use in general, forest fragmentation, wetland reduction and straightening of rivers, play a decisive role in causing flooding.

http://www.euro.who.int/ccashh/Extreme/20020610_1 (accessed April 2005).

Heat waves
Heat waves can cause deaths and illnesses, especially among vulnerable people such as the elderly and those with heart or respiratory diseases.

In recent years, there has been an increased frequency of hot days observed in many European countries, as average temperatures have increased. Countries in the Mediterranean and the Balkans are particularly vulnerable to heat waves. People with pre-existing illnesses, especially heart and lung diseases, are also at higher risk.

Examples include:

- 1987: a heat wave in Athens, Greece, caused an estimated 2 000 extra deaths
- 1995: a heat wave in London, United Kingdom, was associated with 180 excess deaths
- 2003: it is estimated that the heat wave in Europe in 2003 caused 14 802 excess deaths in France, 2 045 in the United Kingdom, and 2 099 in Portugal

Multi-causality also exists in heat wave events. Urban heat islands and traffic emissions are reinforcing the serious effects, leading to more deaths and diseases with more hospital admissions.

http://www.euro.who.int/ccashh/HeatCold/20020606_1 (accessed April 2005).

Tsunamis
Although not connected to climate change, the effects of tsunamis are devastating, with thousands of deaths, diseases and suffering. Also here, there are reinforcing environmental factors like coastline exploitation in general, and the destruction of protective coral reefs and mangrove vegetation.

References:

OECD (2001). *Environmental outlook 2001: human health and environment*. OECD Publications ISBN 92-64-18615-8-No 51591.

European Environment Agency (2003). *Europe's environment: the third assessment. Environmental assessment report No 10.*

Valent, Francesca, D'Anna Little, Roberto Bertollini, Leda Nemer, Fabio Barbone and Giorgio Tamburlini (2004). 'Burden of disease attributable to selected environmental factors and injury among children and adolescents in Europe'. *The Lancet* 363: 2032–2039.

Homedes, N. 2000. The disability-adjusted life year (DALY) definition, measurement and potential use. Working paper. World Bank – Human Capital Development and Operations Policy (http://www.worldbank.org/html/extdr/hnp/hddflash/workp/wp_00068.html).

Table 2 Strengths of associations between environmental factors and a selection of diseases, corresponding population impact and prevention possibilities

Disease/pollutant	Strength of association	Qualitative descriptor	Population impact	Prevention possibilities
Cancer/radon neurodevelopment/lead	very likely (90–99 %)	statistical significance: beyond all reasonable doubt	moderate	high
Neurodevelopment/mercury	very likely (90–99 %)	statistical significance: beyond all reasonable doubt	low	high
Respiratory diseases/air pollution	very likely (90–99 %)	statistical significance: beyond all reasonable doubt	high	moderate
Neurodevelopment/POPs	likely (66–90 %)	reasonable certainty: sufficient scientific evidence	moderate	moderate
Asthma causation/air pollution	medium likelihood (33–66 %)	balance of evidence: strong possibility	high	moderate
Cancer/EMF	low likelihood (10–33 %)	scientific suspicion of risk	high	low
Cancer/low level radioactivity	very unlikely (1–10 %)	low risk	moderate	high

Source: Adapted from EEA and the third assessment report from the intergovernmental panel on climate change 'Summary for policymakers', 2001.

2.1 Respiratory disease, asthma and allergies

Air pollution is responsible for the highest burden of environment-related diseases in Europe. Recent estimates indicate that 20 million Europeans a day suffer from respiratory problems. Air pollutants with strongly indicated respiratory health effects are particulate matter ($PM_{2.5}$, PM_{10}), ozone (O_3), nitrogen oxides (NO, NO_2) and sulphur dioxide (SO_2). In addition, chemicals like polyaromatic hydrocarbons (PAH) and benzene from combustion processes contribute to toxicity and potential health effects. The WHO earlier estimated that particulate matter is considered to be responsible for 100 000 deaths and 750 000 life years lost annually in a selection of European cities (WHO, 2004). The more recent estimates of the air pollution impact made within the European Commission 'Clean air for Europe' (CAFE) programme found that in the EU about 350 000 people died prematurely in 2000 due to outdoor air pollution of fine particulate matter ($PM_{2.5}$) alone. This corresponds to an average loss of life expectancy of about 9 months for every EU citizen. The effect is comparable to the loss of life expectancy due to road accidents in the EU. Exposure to PM is also linked with morbidity such as increased frequency of chronic bronchitis, respiratory hospital admissions and restricted activity days.

In addition, current levels of ozone have severe health implications such as bringing forward the deaths of more than 20 000 people (see the CAFE website http://europa.eu.int/comm/environment/air/cafe/index.htm and the new www.cafe-cba.org website). There are many examples that show that respiratory health and life quality improves when air quality improves.

The total health damage cost of air pollution in the EU in 2000 has been estimated by the CAFE programme to be in the range of EUR 305 billion to 875 billion.

This cost of non-action has to be compared with cost of action focused on the different sources of particles: mobile sources (diesel passenger cars and heavy duty vehicles), industrial processes, and domestic wood stoves (see Figure 2).

A new policy to reduce emissions of acid gases, ammonia and fine particles is developed within an EU thematic strategy on air pollution. The aim is to halve the health impact due to PM between 2000 and 2020 which would require action at the Community as well as the national level. The cost of action has been calculated at around EUR 10 billion per year for the EU as a whole if only technical measures are taken. If non-technical measures are also considered the costs may be lower.

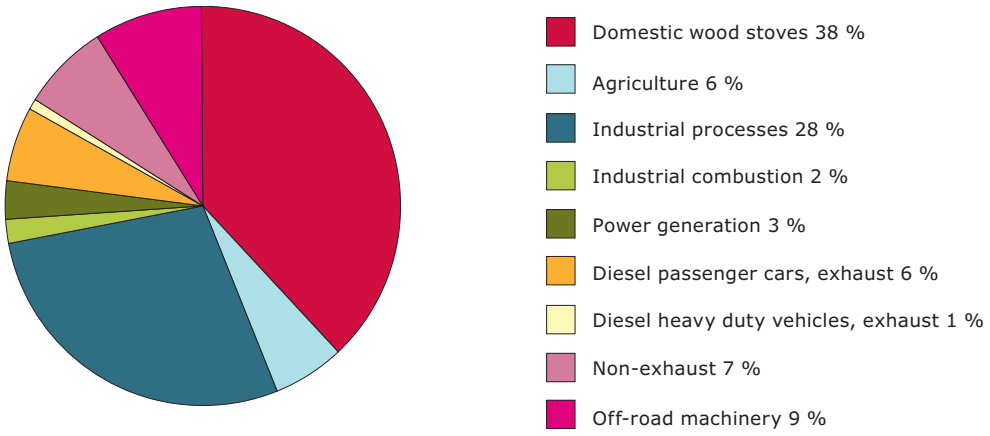
The thematic strategy would also include a revised set of air quality objectives and particularly a shift in focus in EU regulation of particle pollution from PM_{10} to the even smaller and more health-relevant $PM_{2.5}$.

A part of the air-pollution-related disease burden consists of respiratory problems in children, who are exposed to outdoor and indoor air pollutants in their homes, schools and daycare and during travel. The quality of the indoor environment is particularly important since European children spend more than 90 % of their time indoors. Some of the mechanisms by which environmental factors influence children's respiratory health remain unclear and sometimes controversial. This is especially the case with asthma and allergies.

Asthma is now the most common respiratory disease in western European children. The number affected increased markedly between 1970 and 2000. On average 7 % of EU-25 children aged 4–10 have problems with asthma although there is a significant variation between EU countries. The societal cost is estimated at EUR 3 billion per year. The well-documented rise in asthma prevalence has coincided with a general increase in the density of road traffic in most of these countries. A number of studies have therefore investigated the possibility that traffic pollution is responsible for the increase. Clearly, asthmatic individuals are sensitive to air quality and there is a strong association between exposure to air pollution and the exacerbation of asthma. Recent studies indicate that ground-level ozone may be a critical air pollutant in this respect. A study from California has also indicated ozone as a trigger of asthma development in non-asthmatic children, which supports a long-favoured assumption that air pollutants play a role in asthma initiation (McConnel *et al.* 2002). However, in general there is little evidence for air pollution being responsible for the increase in the number of children developing asthma symptoms (asthma prevalence) (WHO, 2004). The basis for this conclusion comes from a number of studies that found allergic disorders (including asthma) to be relatively less frequent in eastern parts of Europe, although levels of many air pollutants were higher than in western Europe. Asthma has a multi-causal background and many factors are involved. Clearly 'lifestyle factors' are important, including the increased level of hygiene in homes and the contribution of nutritional factors. Another important factor is genetic predisposition.

There is a lack of substantial knowledge about the contribution of indoor air quality to respiratory symptoms and whether factors in the indoor environment contribute to the increase in asthma prevalence. Indoor air quality is therefore receiving more and more attention, which is logical in the light of the total time European children and adults spend indoors. Indoor air pollutants can be classified into chemical, biological or physical agents. Many outdoor air pollutants are found indoors, but many indoor pollutants come from specifically indoor sources such as building and construction materials, paints and indoor furnishings (furniture, carpets, etc.). Some indoor sources relate to human activities and habits, such as smoking, cooking and the use of cleaning agents, disinfectants and air cleaners. Open fires (wood, coal or gas) for heating, cooking and pleasure are significant sources. The continuing stress on energy-saving policies has led to reduced air exchange in homes, schools and office buildings. This leads to an increase in indoor humidity, which

Figure 2 Contribution to primary $PM_{2.5}$ emissions in the EU-15, year 2020



Source: IIASA, 2004. (www.iiasa.ac.at/RAINS/CAFE_files/CAFE-baseline-full.pdf).

stimulates the development of biological pollutants such as mites, moulds and bacteria. Ventilation-related humidity problems arise in the warmer climatic zones of southern Europe because of the increased use of air conditioners, as well as in the 'cold' climates of central and northern Europe. Maintenance of ventilation systems is clearly important, particularly regular cleaning or changing of dust filters.

Many acute health problems are connected to the indoor environment, including allergic symptoms, distress, sleeping and concentration problems, and, in children, coughing, wheezing and asthma-like symptoms. Damp and humidity are important factors because they provide a suitable environment for the growth of micro-organisms (mould, bacteria) but also increase the release of chemicals from construction materials. Several reviews find respiratory problems, including asthma, in children from homes with visible damp and mould or smell of mould. Several studies also highlight the importance of the combined effects on children with allergic and asthmatic problems of exposure to emissions from moisture and mould in combination with tobacco smoke, emissions from gas stoves, mites and allergens from house animals. Chemicals like formaldehyde and benzene from construction materials, chemicals from treatment of furniture and decorations, fragrances from cleaning agents and other household products add to the combined burden of the indoor environment. Although the importance of the indoor environment is generally recognised, there is far less knowledge about indoor than outdoor air quality. This is illustrated by the fact that there are several European directives that regulate outdoor air quality but no European guidelines for indoor air quality. In the United States, the Department of Housing and Urban Development has established emission standards for floor underlay as a result of formaldehyde levels.

There is no doubt that pollutants in outdoor air have an impact on respiratory health, as confirmed by a large number of epidemiological studies on both short- and long-term exposure. Many studies show that fine particles (usually measured as $PM_{2.5}$) have serious effects on health, such as an increase in mortality and emergency hospital admissions for cardiovascular and respiratory reasons. Modelling results indicate that $PM_{2.5}$ levels in Europe are now estimated to reduce the statistical life expectancy of the European population by approximately nine months, which is comparable to the impact of traffic accidents. While much information is available for short-term acute exposures, there is little about the effect of long-term exposures. In a study in the former East Germany, an association was found between air pollution levels in the city of residence, the presence of chronic respiratory (especially bronchitic) symptoms and lung function growth. A study conducted in Switzerland also found an increased occurrence of symptoms in children with increased air pollution levels. Several studies in the USA and Canada (6-city study, 12-city and 24-city study) also found increased bronchitic, but not asthmatic, symptoms in children and lower lung function at higher air pollution levels.

Respiratory health improves when air quality improves. This is clearly exemplified by intervention studies. One of the best examples is a labour dispute that shut down a large steel mill in Utah Valley. Respiratory hospital admissions in children were clearly decreased during the strike and returned to pre-strike levels after the dispute ended. In Hong Kong in 1990, a fuel restriction was introduced that required all power plants and road vehicles to use fuel oil with a sulphur content of not more than 0.5 % by weight. Bronchial hyper-reactivity in children living in different polluted districts declined on average from about 25 % to 15 % after the fuel restriction. Another example is the study carried out during the 1996 summer Olympic Games in Atlanta in which the impact of changes in transportation and community behaviour on air quality and childhood asthma was investigated. Implementation of an alternative transport strategy resulted in lower traffic emissions, and hospital admissions of children with acute asthma symptoms fell by 41.6 % during the period of the games. There are also several studies which showed a reduction in respiratory health effects in association with reduced air pollution levels over several years in the former East Germany: a decrease in the prevalence of bronchitis and non-allergic respiratory symptoms and an increase in the mean forced lung capacity and forced expiratory volume per second in children.

Much of the burden of disease resulting from air pollutants relates back to childhood. Air pollutants augment acute respiratory infections in children and disturb the normal development of the lung. More and more scientists and healthcare professionals are focusing on events during foetal life and early childhood. There is growing evidence that these periods are critical for the later development of many diseases that present during child and adult life. Children who grow up in polluted areas, or whose parents grew up in polluted areas, are more likely to develop reduced lung function as adults. Estimates show that the risk of reduced lung function is doubled in children who grow up in urban areas.

Children with asthma are particularly vulnerable but it is uncertain whether air pollutants trigger the onset of childhood asthma. Intervention studies clearly show the health benefits of improved air quality. When considering human health and the quality of life, the most urgent requirement to reduce the environmental burden on health appears to be improvement of the quality of outdoor and indoor air.

References:

International Institute for Applied Systems Analysis (2004). 'Baseline scenarios for the clean air for Europe (CAFE) programme: CAFE scenario analysis report No 1'. http://www.iiasa.ac.at/rains/cafe.html (accessed May 2005).

WHO (2004). Health aspects of air pollution. Results from the WHO project 'Systematic review of health aspects of air pollution'. WHO Europe, June 2004.

SCALE baseline report on respiratory health. (European Commission, DG Environment, 2004) http://www.europa.eu.int/comm/environment/health/finalreports_en.htm (accessed April 2005).

EU Clean air for Europe. http://europa.eu.int/comm/environment/air/cafe/index.htm (accessed April 2005).

McConnell, R., K. Berhane, F.D. Gilliland, S.J. London, T. Islam, W.J. Gauderman, E. Avol, H.G. Margolis and J.M. Peters (2002). 'Asthma in exercising children exposed to ozone'. *The Lancet* 359: 386–391.

2.2 Cancer

Cancer accounted for more than 7 million deaths worldwide (13 % of total mortality) in 2000, and 10 million new cancer cases were diagnosed. More than 60 % of cancer deaths and approximately half of the new cases occurred in the developing regions. Lung cancer was the most common, followed by cancers of the stomach, liver, colon and rectum, and breast. Cancer in all ages is a result of the interaction between genetic and environmental factors. There are significant regional differences in cancer occurrence with respect to type and frequency. Differences in lifestyle and environmental exposures have commonly been assumed to be a major reason for the geographical distribution of cancer. Genetic factors and ethnic variations account for some part of the regional differences.

Childhood cancer and its connection to environmental factors has recently been the focus of several international reviews (EEA-WHO, 2002, European Commission, background documents for SCALE, 2004). Cancer in European children younger than 15 years is in general terms rare but is still one of the most common causes of death in children in industrialised countries. The most common childhood cancers are leukaemia and brain tumours. A small but significant increase in childhood cancers has been noted since the mid-1980s. One reason for this increase may be better diagnostic methods, but an additional component from environmental exposures cannot be excluded. The number of deaths from childhood cancers, however, is falling because of better treatment methods.

Little is still known about the cause of cancers. Genetic factors are important but can only explain a small part of the observed cases. Environmental factors are probably important but the evidence is mostly circumstantial. There are established cancer risks (chemicals, asbestos and radiation) in the occupational environment but since these are generally well known they can be managed by adequate measures. If lifestyle-related environmental exposures like food, alcohol consumption and smoking are excluded, there remains only one environmental factor for which there is a proven connection to cancer development — ionising radiation. There is clear evidence that sufficient doses of radiation can cause many different types of cancer in both adults and children. It is generally assumed that the carcinogenic effect arises through direct damage to the DNA. The assumption of a linear relationship between radiation dose and cancer risk, which is made for radiation protection purposes, implies that any exposure to ionising radiation carries with it a risk to health although at levels normally encountered in the environment, this risk is very small.

Current levels of ionising radiation in Europe are low in general, but there are regional differences because of the presence of radon. Radon is a radioactive gas formed from the radioactive decay of uranium; radon seeps out of the ground in areas with uranium-containing soils and rocks. The most important pathway for human exposure is permeation of radon gas into buildings through the ground, but radon from water, outdoor air and construction materials can also contribute to the indoor burden. Radon decays to radon daughters, some of which emit high energy alpha radiation. Alpha-emitting radon daughters are easily adsorbed onto dust particles and can, when inhaled, cause gene damage and mutations which can lead to cancer development in the lungs. There is a strong relationship between exposure to radon

and the development of lung cancer. A pooled analysis of all European epidemiological studies on domestic exposure to radon shows a clear linear relationship between lung cancer risk and the level of radon exposure. The contribution of radon to the cancer burden is significant. Recent estimates show that radon is responsible for 21 000 cases of lung cancers per year in the USA (US-EPA, 2003). In Sweden (population 9 million), 400–700 cases of lung cancer can be ascribed to radon exposure per year (Socialstyrelsen, 2001, 2005). A recent European study (Darby *et al.* 2005) indicates that 9 % of lung cancer deaths/year in Europe can be ascribed to radon, which out of a total of 330 000 deaths from lung cancer/year (Bray *et al.* 2002) makes approximately 30 000 deaths in Europe/ year due to radon exposure. With these numbers, radon is clearly the environmental risk factor with the highest proven cancer burden. Lung cancers in children are extremely rare and it is not known if childhood exposure increases the risk of lung cancer in adulthood. There are studies suggesting that radon could increase the risk of childhood leukaemia but the findings are inconclusive. Gamma radiation from radon decay in construction materials (concrete) has been indicated as a risk factor for leukaemia in one study, but others have failed to confirm this.

There are also health effects of non-ionising radiation. There is a well established connection between skin cancers and exposure to UV radiation. UV radiation is divided into three groups depending on the wavelength; UV-A (315–400 nm), UV-B (280–315 nm) and UV-C (100–280 nm). UV-B is partly filtered and UV-C totally filtered by the ozone layer in the stratosphere. A thinning of the ozone layer leads to increasing ground levels of UV. UV radiation has both positive and negative health effects. The positive effect is that sunlight and UV-B exposure stimulates the synthesis of vitamin D in the skin. Vitamin D is essential for the metabolism of calcium in the body and vitamin D deficiency leads to de-calcification of the bone (rickets). The negative effect is that UV radiation induces skin cancer and approximately 80–90 % of all skin cancers can be related to UV radiation. UV-B has been considered to be particularly important in this context, but UV-A has also recently received attention, for example in the development of one of the more lethal skin cancers (malignant melanoma) and many sunscreens, while designed to protect against UV-B, do not effectively filter UV-A. The number of skin cancer cases is increasing and an increased intensity of UV radiation may be one of the causes, together with more lifestyle-related factors (sun bathing, time spent in the sun during holidays). Infants are particularly vulnerable to UV radiation because they do not have a fully developed pigment protection. This also applies to children with a low level of skin pigmentation (red-haired, or blond with light skin).

Every European citizen is today exposed to electromagnetic fields (EMF) which can be characterised in terms of their frequency and amplitude. The fields around power lines and electrical appliances are of low frequency. Mobile telephones and radio transmitters transmit EMF of higher frequency. There are well documented acute effects of both types of EMF with high amplitudes, but human exposure is controlled by appropriate protective measures (shields in transformer stations, power generators and radio transmitters). For both types of EMF there has been speculation that long-term, low-dose exposure can cause health effects. Effects have been suggested for low-frequency EMF. Recent pooled studies overarching a number of childhood leukaemia studies have concluded that there is a correlation between low frequency EMF and childhood leukaemia. The evidence is, however, not conclusive. IARC (International Agency for Research on Cancer) classified EMF as a category 2B carcinogen (possibly carcinogenic) in 2001. Currently, the mechanisms by which weak and low-frequency EMF could cause leukaemia are not understood. The explosion in the use of mobile telephones has initiated many research efforts concerning health effects of the higher frequency EMF generated by phones and the base stations. Neither experimental studies on animals, nor epidemiological studies have come to any conclusive results. On the basis of today's knowledge it is not possible to answer 'yes' or 'no' to the question of whether the use of mobile phones poses an increased risk of cancer. But it must be kept in mind that all studies so far have been relatively short-term — mobile phones have only been in widespread use for just over 10 years — and more studies over longer periods are needed before any firm conclusions can be reached. There is public concern about EMF emissions from base stations. The intensity of EMF from a base station is about one thousandth of that from an individual phone. Although base stations result in a long-term, probably lifelong, exposure, they are today not considered to have any demonstrated health impact under current guidelines.

A number of chemicals are carcinogenic. Air pollution includes carcinogenic chemicals such as benzene and polycyclic aromatic hydrocarbons (PAH). Several studies show a positive association between local traffic density and childhood leukaemia. A variety of hazardous industrial sites

may also be a source of carcinogenic chemicals representing a potential risk to children and adults exposed as a result of living nearby, but there are only a limited number of studies evaluating this health risk. Fried and smoked food items may contain carcinogenic substances. Although no specific parental occupational exposure can be definitively established as the cause of childhood cancer, several paternal and maternal occupations have been found to be associated with leukaemia and tumours of the nervous system. The reviews of the chemicals used in these occupations suggest that parental exposure may increase the risk of childhood cancers. An example is the observation of increased risk of brain cancer, which can be related to maternal exposure to high levels of solvents. Paternal exposure to PAH has been associated with an increased, but not dose-related, risk of brain tumours. Several studies indicate that certain pesticides may be carcinogenic. Epidemiological studies have reported associations between childhood cancer, and either parental or childhood exposure to pesticides. The carcinogenic effects of pesticides are carefully taken into consideration in the risk assessment according to Directive 91/414/EEC before authorisation for marketing within the EU. Collectively, these studies suggest that there may be an increase in the risk of several childhood cancer types associated with parental occupational and non-occupational exposure to pesticides.

Many studies suggest that most cancers in children are initiated before birth. In addition, exposure to exogenous carcinogens in childhood may have an important effect on cancer risk in adult life. Post-natal exposures are also likely to play an important role as promoters in children who are predisposed as a result of initiating events that took place before birth. The cancer risks following pre- or post-natal exposure of a child to specific carcinogens or combinations of carcinogens relative to exposure in adults are unknown. For ionising radiation it has been concluded that children in general appear to be at somewhat greater risk when exposed to the same dose as adults. Environmental tobacco smoke, early smoking and PAH in city air are other cancer risk factors for children. In addition, studies of trans-placental carcinogenesis in experimental animals using DNA-alkylating agents have consistently demonstrated a much greater susceptibility of the foetus than adult animals administered the same dose. This greater susceptibility could be predicted for physiological reasons since the foetus and young child are undergoing multiple processes of growth and differentiation. The number of cell divisions per unit of time is considerably greater in the foetus and young child than in adults. The potential for mutations to arise following exposure to a carcinogen is therefore greater in the growing foetus and child. Based on these considerations it is particularly important to protect children from carcinogenic environmental factors. Fortunately, many of the adult risk factors, including smoking and use of chemicals are not very relevant for children. However, children may be exposed to other risk factors or in other situations. Since children spend much of their time indoors, it is clear that the indoor environment should receive more attention from this point of view.

References:

WHO-EEA, 2002. Children's health and environment: A review of evidence. WHO Environmental Issue Report 29.

SCALE baseline report on childhood cancer (2004). (European Commission, DG Environment) http://www.europa.eu.int/comm/environment/health/finalreports_en.htm (accessed April 2005).

Socialstyrelsen, Sweden (2001). Miljöhälsorapport 2001. www.sos.se (accessed April 2005).

Socialstyrelsen, Sweden (2005). Barns Miljö och Hälsa. www.sos.se (accessed April 2005).

Bray, F., R. Sankila, J. Ferlay, and D.M. Parkin (2002). 'Estimates of cancer incidence and mortality in Europe in 1995'. *Eur. J. Cancer* 38: 99–166.

Darby, S. *et al.* (2005). 'Radon in homes and risk of lung cancer: collaborative analysis of individual data from 13 European case-control studies'. *Brit. J. Med.* 330: 223–230.

2.3 Neurodevelopmental disorders

Neurodevelopmental disorders are disabilities in the functioning of the brain that affect a child's behaviour, memory or ability to learn. They include mental retardation, dyslexia, attention-deficit hyperactivity disorder (ADHD), learning deficits, autism and autism-like disorders, and affect between 3 % and 8 % of children in the USA and Europe. Recent epidemiological data seem to indicate that the prevalence of at least two of these disorders (autism and ADHD) has increased in the past two decades. Although such an increase may reflect increased awareness of these disorders and broader diagnostic criteria, there is general concern that environmental factors could contribute to neurodevelopmental disorders.

There is poor understanding of the neural basis and background of most neurodevelopmental disorders characterised by cognitive and behavioural deficits. A large number of environmental factors have been implicated as causes, in addition to genetic factors. The nervous system is vulnerable to a wide range of environmental factors and agents during development. Maternal stress during pregnancy, exposure to neurotoxicants in the prenatal/post-natal phase, and a poor psychosocial environment may all affect the normal development of the brain. For example, there are reports that some forms of autism might arise from toxic insult. Furthermore, the kind of damage that may occur depends on a variety of factors, including the stage of development, the particular agent/environment factor and the magnitude, route and duration of the exposure. Damage of the nervous system may be characterised by anatomical, neurochemical, behavioural and/or cognitive deficits. In some cases behavioural and cognitive alterations may be the only available marker of effect and would thus be important for risk assessment.

Identification of causative factors for neurodevelopmental disorders is extremely difficult because of multi-causality. One must consider the crucial role of socioeconomic variables in exacerbating or compensating for the potential adverse effects of environmental contaminants. In human studies, exposures to mixtures of chemicals or to different risk factors make it difficult to isolate the contributions of the different chemicals/factors to the development of disease. In addition, specific tests for neurodevelopment, e.g. learning and memory, are not required by current legislation in the first tier of toxicology studies for the majority of chemicals. The risk of adverse effects on the developing brain for many potentially hazardous compounds is therefore unknown. Finally, although epidemiological studies show a significant association between a child's neuropsychological outcome and exposure to some neurotoxicants, their mechanisms of toxicity are far from fully understood.

Some persistent organic chemicals have been proved to have neurodevelopmental effects during prenatal and post-natal life in humans. Prenatal exposure to polychlorinated biphenyls (PCBs) has been associated in several studies with negative effects on cognitive processes, motor development and reflexes in children. PCBs, dioxins and probably also brominated flame retardants (polybrominated diphenyl ether, PBDE) are indicated to interfere in the function of the thyroid hormone (thyroxin). Thyroxin regulates a number of genes during development which are critical for the normal development of the brain. PCBs, dioxins and some pesticides (pyrethroids) have in animal experiments been shown to interfere with the neural communication system by affecting neurotransmitters. Background levels of PCBs and dioxins are decreasing but are still too high in several parts of Europe, considering their potential effects on neurodevelopment. The current exposure level to persistent compounds in some areas is substantially higher than the 'tolerable daily intake'.

Pesticides are a very heterogeneous group of substances. They have not been extensively studied with respect to neurodevelopmental effects in humans, and the information available comes from experimental animals. Currently, no neurodevelopmental toxicity effects have been described in humans with a sufficient degree of certainty, although several targeted studies are under way. There are publications that relate effects on learning and aspects of neurobehavior to DDT and other organochlorines, but the studies either do not assess real exposures or do not take confounders into account. In contrast there is much animal data on neurodevelopmental and direct neurotoxic effects of all three major classes of insecticides (organochlorines, organophosphates, pyrethroids), which should be seen as an 'alarm clock' for potential human effects, provided critical exposure levels are reached. As a consequence of such studies that have been evaluated in the framework of the EU pesticides legislation, many organochlorines, organophosphates and pyrethroids have been banned and are (or will be) phased out.

Lead is probably the environmental factor for which we have the strongest evidence for neurodevelopmental effects in humans. Even very low doses have been connected to reduced IQ, learning disabilities, concentration problems and impulsive and unpredictable behaviour. There is evidence for effects on motor coordination, spatio-visual capacity and verbal capacity. Animal experiments show that lead affects differentiation processes in nerve cells and synapses, which are strongly related to developmental stages in the brain. The effects of lead at very low levels in humans suggest that a safe (exposure) level cannot currently be defined. Exposure to lead should therefore be as low as possible. It appears likely that some sub-populations may be at particular risk from lead exposure in older houses (including possible exposure from water coming through obsolete lead pipes), which should be identified and replaced. More data on levels in Europe are necessary and are being collected. A ban on leaded petrol has proved

to be very successful in reducing blood lead levels in children.

Mercury is another metal for which there are well described neurodevelopmental effects, although some studies are contradictory. The toxic form is organic methyl mercury, which easily crosses the blood–brain barrier and the placental barrier. Maternal exposure to methyl mercury has been connected to attention problems, sensoric/motor functions, language development, learning ability and low IQ in children. Animal experiments confirm that methyl mercury has pronounced effects on nerve cell development and differentiation in the prenatal stage. The current levels of tolerable intakes for pregnant women are taking into account these findings. Other neurotoxicants, like cadmium, aluminium, manganese, arsenic and monosodium glutamate, are not known to cause neurodevelopmental disorders in general at current European exposure levels. However, animal experiments with high doses give rise to some concern, and research on whether lower doses have implications for humans is desirable.

Noise is a potentially important influence on neurological development although the evidence is more circumstantial than direct. The physiological role of sleep is still unknown, but it is clear that sufficient long and undisturbed periods of sleep are essential for normal neurodevelopment and normal cognitive processes. Exposure of the pregnant mother to very high noise levels may have a prenatal effect resulting in hearing disabilities later in life. Hearing develops at a late stage of neurodevelopment and it is important to protect the child from major noise impact. Environmental noise is probably the environmental factor that has the greatest daily impact on the European population. It has been estimated that approximately 80 million people suffer from noise levels that scientists and health experts consider as unacceptable. Roughly 20 % of children of 12 years and younger report sleep disturbances because of noise. There are indications that there is a casual chain linking chronically strong annoyance with increased morbidity. Sleep disturbance in adults results in significantly elevated relative risks to the cardiovascular, respiratory and musculo-skeletal systems, and to depression. Many of these diseases increase with age and therefore only appear rarely in children. Significantly elevated relative risks to the respiratory system and of migraines are of great importance to children.

Recent studies highlight the health impact of noise in combination with other environmental stressors. A European Commission research project (NoiseChem) investigates the effects of exposure to noise and industrial chemicals on hearing and balance and also the interaction of noise and chemical 'odour' detection. US studies (Morata 2003) show that when simultaneous exposure to noise and chemicals occur, the hearing loss observed was greater than the expected hearing loss from noise added to that from the chemical. If this synergism is verified in humans, then changes will be required in the limits that are set for occupational hazards in order to prevent occupational hearing loss.

References:

Final report Noise effects and morbidity', WHO LARES, Dr Maschke, Dr Niemann. 2004. (WHO) www.euro.who.int/document/NOH/WHO_Lares.pdf.

SCALE baseline report on neurodevelopmental disorders (2004). (European Commission, Environment DG) http://www.europa.eu.int/comm/environment/health/finalreports_en.htm (accessed April 2005).

Morata, T.C. (2003). 'Chemical exposure as a risk factor for hearing loss'. *J. Occupational Environm. Med.* 45: 676–682.

2.4 Endocrine disruption

The endocrine system, together with the nervous system, is responsible for the coordination and control of body functions. Hormones are released to the blood from endocrine glands on specific stimuli and are carried to the target tissues, where they act through binding to specific receptors on the cell surfaces or within the cell cytosol. They act at very low concentrations, and are present at very low concentrations in the blood, but have strong effects through amplifying mechanisms. Most hormones are peptides (made of amino acids) but a number of them are small chemical molecules. Examples are the sex steroids oestrogen and testosterone, the thyroid hormone thyroxin and the stress hormone cortisol. Receptor binding activates intracellular messengers and finally leads to targeted cellular responses that depend on the physiological function the hormone. The endocrine system controls almost every function of the body, including sexual differentiation before birth, sexual maturation during puberty, reproduction, growth, metabolism, digestion, cardiovascular and respiratory function and excretion. Disruption of, or interference with the endocrine system has widespread effects that

affect fundamental body functions. Many diseases have an endocrine background, for example cancer in hormone-dependent tissues such as the breast, uterus, prostate gland and testicles.

Endocrine disruption is interference in the endocrine system by environmental chemicals and covers basically all aspects of the hormonal cycle from synthesis and release, receptor binding and cellular events, to breakdown and excretion from the blood stream. The subject has received much public and scientific attention for three reasons:

- the fundamental role of the endocrine system in regulating body functions including aspects like sexuality and libido which are sensitive for the individual;
- many industrial chemicals and pesticides have structural resemblances to hormones and could theoretically interfere with the hormone mechanism;
- hormones act at very low concentrations and there is a suspicion that hormone-like chemicals could act at similarly low levels, to which we are exposed in our daily life, and at concentrations that have been found in our bodies.

Several health effects have been ascribed to endocrine disruption mechanisms. One frequently highlighted is sperm quality in men. A number of studies in different countries and regions have shown a reduced sperm count (number of sperm cells/ml ejaculate). This finding has been controversial because of large regional variations, inconsistencies between studies, and the different methodologies between regions and individual studies. However, a meta-analysis concluded that several separate studies confirm a 50 % reduction in sperm quality worldwide over the past 60 years, and also confirmed a large regional variation
(WHO-IPCS, 2002). This variation has been taken as evidence for an environmental influence. The development and maturation of sperm is dependent on the male sex steroid testosterone. Animal experiments have shown that the female sex steroid, oestrogen, reduces sperm quality when given to males. It is also known that men whose mothers were treated during pregnancy with diethylstilbestrol (female sex steroid) against miscarriage, tend to have reduced sperm quality when adults. Recent studies have found correlations between concentrations of PCB metabolites in blood and decreased sperm quality. Other studies have found strong correlations between pesticide metabolites in urine and reduced sperm quality in men from an agricultural area.

However, because of the large regional and culture-associated variations in sperm quality it is difficult to reach a final conclusion. Environmental influences are certainly involved but the extent to which this is due to chemicals or to factors more related to 'lifestyles' remains to be elucidated. These observations point to the role of exposure to estrogenic drugs/substances with respect to sperm quality and have led to the hypothesis that chemicals or other environmental factors with estrogenic activity may be responsible for reduced sperm quality in men. In a wider context, reduced sperm counts and other abnormal testicular effects have been ascribed to a single syndrome, testicular dysgenesis syndrome, whose origins lie in disruption of the development of the foetal testis.

The frequency of breast cancer in European women is increasing. Breast cancer is hormone-dependent (oestrogen) and there has been speculation about the role of hormone-like chemicals in its development. A number of epidemiological studies have been conducted but there is no conclusive evidence of a connection between breast cancer and exposure to environmental contaminants (PCB, DDT). A recent review (Coyle, 2004) concludes that 'although environmental factors have not been convincingly found to influence breast cancer risk, research suggests that environmental exposure in combination with genetic predisposition, age at exposure, and hormonal milieu have a cumulative effect on breast cancer risk'.

Testicular cancer is another hormone-dependent cancer that is increasing in the European population. The cancer appears in young men aged 20–40, but the cancer process probably already starts during the foetal period or the early years of life. One possible risk factor is prenatal oestrogen exposure. For example, a recent epidemiological study based on men from Sweden and Finland shows that environmental exposures early in life, probably via the mother, are likely to be major determinants of this disease (Ekbom *et al.* 2003). The findings are in line with the theory that the origin of testicular cancer is part of the 'testicular dysgenesis syndrome'.

The sexual characteristics of the child are determined during early development and are under the control of oestrogen in girls and testosterone in boys. Under normal circumstances, the sex ratio is higher than one, i.e. more boys than girls are born. Several studies have reported a small but significant decrease in the sex ratio in several European countries. The most important explanatory variable is the socioeconomic situation and the improvements in Europe after the Second World War. However, there are trends in sex

ratios that may be related to hormonal factors and there are also reports of the negative influence of environmental pollution. After the Seveso accident in Italy 1976, which spread dioxin over the surroundings, 48 girls and 26 boys were born in the seven years following the accident in the most exposed families. This was a large deviation from a very stable sex ratio. Dioxin has estrogenic activity. Surprisingly, the effect seemed not to be connected to exposure of the mother, with dioxin exposure of the father appearing to be of importance. Other studies show a similar outcome if the father has been exposed to PCBs. The mechanistic background is not clear.

Thyroid hormones are essential for normal brain development and too little thyroid hormone (hypothyroidism) during development leads to mental retardation. The hormones are initially transferred from the mother to the embryo and the foetus but after approximately 10 weeks the foetus produces its own hormones. Even very small differences in the concentration of thyroid hormones in mothers during pregnancy can lead to changes in intelligence in the children. Several environmental contaminants are known from laboratory studies to interact with the thyroid hormone system. Examples are PCBs, brominated flame-retardants, insecticides, herbicides and phthalates. Exposure of mothers to PCBs and other organic pollutants has also been connected to different neurodevelopmental disorders and although not proved this could result from thyroid hormone disturbances. This is clearly a possibility that merits further investigation. The thyroid hormone system is a very sensitive target for endocrine disruption by environmental chemicals and the consequences for the individual could be far-reaching. The thyroid hormone example again points to the vulnerable relationship between the mother and the developing child.

A recent review of the effects of environmental chemicals on children concludes: 'Right now, endocrine disruption in humans is much more a speculation than a demonstrated fact' (Rogan and Ragan, 2003). This is probably correct on the basis of current knowledge. But much new information is appearing which indicates that it is necessary to view endocrine disruption in a wider context and in a different way. Although effects cannot be proved, they may still exist. We probably have to abandon the classical toxicological dogma of cause/effect relationship at the individual level and extend it to the generational level. More and more studies in the endocrine area show that events at the level of germ cell differentiation, at conception, during embryonic development and during foetal and early life, are important for health outcomes later in life. What is important to consider is the 'environmental heritage' that the parents give to the child. Even slight disturbances in the maternal hormonal balance could have detrimental consequences for the foetus. Chemical contaminants at levels below any measurable health effects in the mother, which for that reason are considered safe according to current standards, may still have an impact on the growing foetus, leading to disease in adult life. The field of endocrine disruption clearly shows the complexity of this area of environment and health, and suggests that the problems should be approached by a more holistic approach than currently offered by classical toxicology, nor should the clear evidence of studies of endocrine disruption in wildlife be ignored (see Section 4).

References:

Coyle, Y.M. 2004. The effect of environment on breast cancer risk. Breast Cancer Res. and Treatment 84, 273–288.

WHO-IPCS (2002). 'Global assessment of the state-of-the-science of endocrine disruptors'. WHO/IPCS/EDC/02.2.

Ekbom, A., L. Richiardi, O. Akre, S.M. Montgomery and P. Sparén (2003). 'Age at immigration and duration of stay in relation to risk for testicular cancer among Finnish immigrants in Sweden'. *Journal of the National Cancer Institute* 95(16): 1238–1240.

Rogan, Walter J. and N. Beth Ragan (2003). 'Evidence of effects of environmental chemicals on the endocrine system in children'. *Pediatrics* 112: 247–252.

SCALE baseline report on endocrine disruption. (European Commission, Environment DG, 2004) http://www.europa.eu.int/comm/environment/health/finalreports_en.htm (accessed April 2005).

3 Body burden of chemicals

Many synthetic chemicals have been introduced during the past 50 years — in consumer products, industrial manufacturing and products, pharmaceuticals and their by-products. These compounds may end up in blood and human tissues, sometimes bio-accumulated, persistent and with toxic properties.

Recent scientific developments have enabled a shortcut in monitoring and risk assessment: instead of measuring concentrations in air, water and food and using them to assess exposures, potentially hazardous substances can be bio-monitored using human samples, thereby estimating the body burden of pollutants and synthetic chemicals.

All Europeans have different concentrations of man-made chemicals in the body, independent of age, lifestyle and region. This is clearly illustrated in the WWF 'Bad blood' study carried out on 14 environment ministers from 13 EU countries.

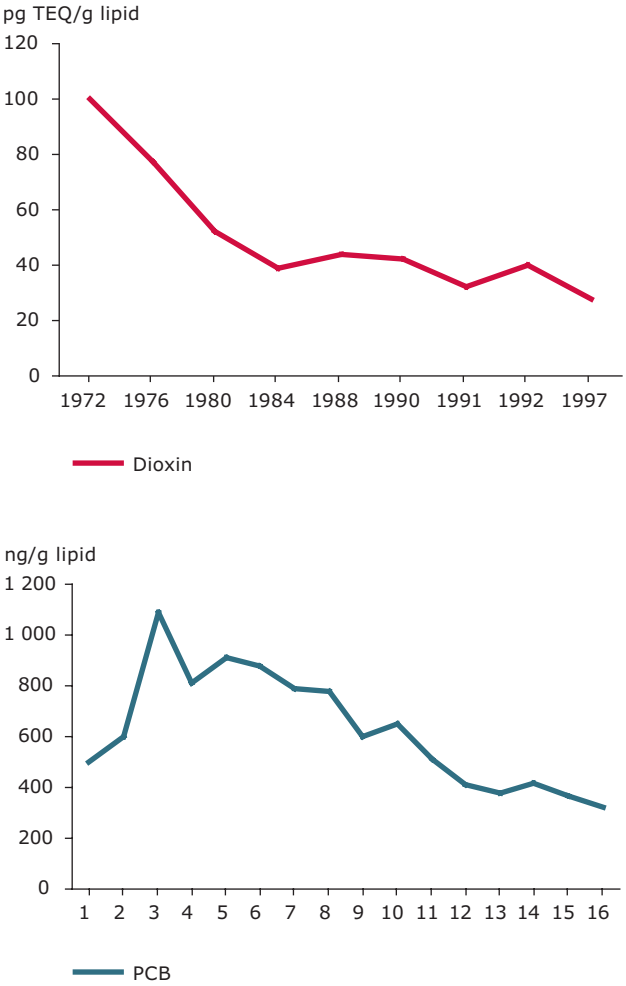
The ministers' blood samples were analysed for a total of 103 different man-made chemicals from seven different chemical families. All were contaminated with PCBs, pesticide residues, brominated flame-retardants, perflourinated chemicals, and most with phthalates and synthetic musk. While many of these chemicals have been banned, many others are still used in everyday products.

The body burden of two main groups illustrate the threat to a sensitive group, European children, and the long-distance transport of chemicals to indigenous people living in the Arctic:

Children

Measurements of the levels of pollutants in children's bodies, and pregnant women, provide direct information about exposures to environmental contaminants. Of special interest is the body burden of persistent organic pollutants (POPs), heavy metals like mercury and lead; and combustion pollutants (PAH, polyaromatic aromatic hydrocarbons) or their metabolites in human tissues.

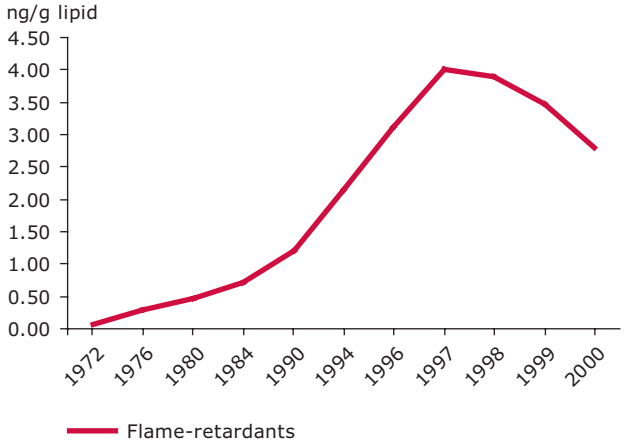
Figure 3 Dioxin and PCB trends in mothers' milk monitored in Sweden



Source: Socialstyrelsen, 2005.

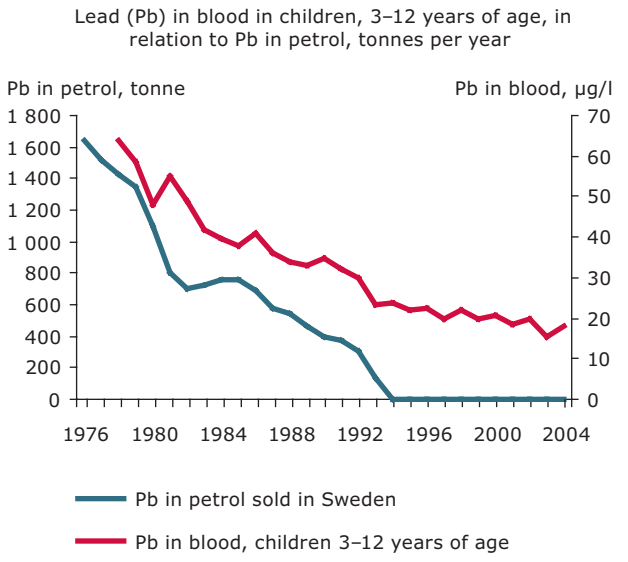
Concentration of chemicals in human breast milk is an extremely relevant biomarker indicating both the mother's exposure to chemicals and the chemical burden to the child. Long time series exist for two compounds with alarming serious health impacts: dioxins, polychlorinated biphenyls (PCBs) and flame retardants. The time series shows the successful bans and other policies leading to decreased concentrations.

Figure 4 **Trends of flame-retardants in mothers' milk monitored in Sweden**



Source: Socialstyrelsen, 2005.

Figure 5 **Lead concentrations in children's blood, and lead in petrol (tonne) sold in Sweden 1976–2003. The children live in two towns in southern Sweden**



Source: Socialstyrelsen, 2005, the Swedish national health-related environmental monitoring programme, http://www.internat.naturvardsverket.se/.

Children are exposed to lead from different sources. Lead concentrations in blood emanates from leaded petrol, lead in soil and dust, but also from fragments of lead-containing paints. This exposure causes the well recognised mental retardation effect. Although the concentration of lead in blood is an important indicator of risk, it only reflects current exposures.

Lead also accumulates in bone. Recent research suggests that concentrations of lead in bone may be more related to adverse health outcomes in children than concentrations in blood. This finding suggests that concentrations in bone may better reflect the net burden of exposure, but blood samples are still the most feasible monitoring method.

The ban on leaded petrol has proved to be very successful in lowering blood lead levels in children. This is clearly shown in many European studies.

Polycyclic aromatic compounds (PAH) emanate from combustion processes, i.e. car engines and wood/coal burning. A human biomarker of PAH is the metabolite 1-OH-pyrene in urine. This biomarker has been used in studying the impacts of decentralised heating and cooking with wood/coal in East Germany, compared with the situation in West Germany. Combustion products have a clear effect on the concentrations of the biomarker (Figure 6).

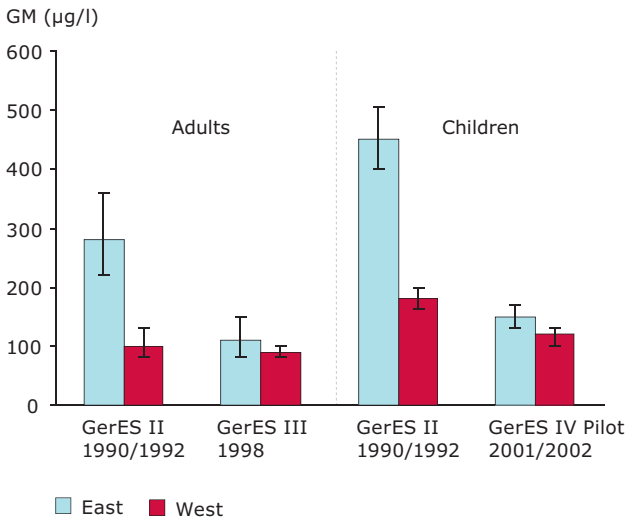
PCB, DDT, brominated flame-retardants and dioxins belong to the POP group but there are many more substances of interest. POPs are a group of specific chemicals regulated under international agreements to reduce or eliminate their use and release to the environment. The CLRTAP POPs protocol lists 16 substances as POPs, and the Stockholm Convention on Persistent Organic Pollutants (2001) identifies a subset of 12 targeted for release reduction or elimination. The manufacture, use or import of 11 POPs has already been banned under EU legislation. Dioxins are by-products of the production of chlorinated compounds, the combustion of waste and some industrial processes. Although PCBs are regulated, there are still problems with hazardous wastes that contain PCBs. DDTs are well known pesticides but are no longer used in Europe.

Indigenous people living in the Arctic

Mercury and other metals released to the atmosphere can travel long distances, as clearly shown by the high concentrations in the Arctic and other remote places far from the sources: industrial processes, waste burning and consumer products. The industrial use of inorganic mercury and dentistry are also important sources. Humans are exposed to methyl mercury, which is the toxic form of mercury, mainly through eating fish.

Concentrations of platinum, palladium, and rhodium in ice and snow in Greenland have increased rapidly since the 1970s. These elements are used in automobile catalytic converters to reduce hydrocarbon pollution. The toxicity and

Figure 6 **Concentrations of most PAH metabolites in 1990/1992 were higher in East than in West German adults, but by 1998 East Germans had approached the level of West Germans**



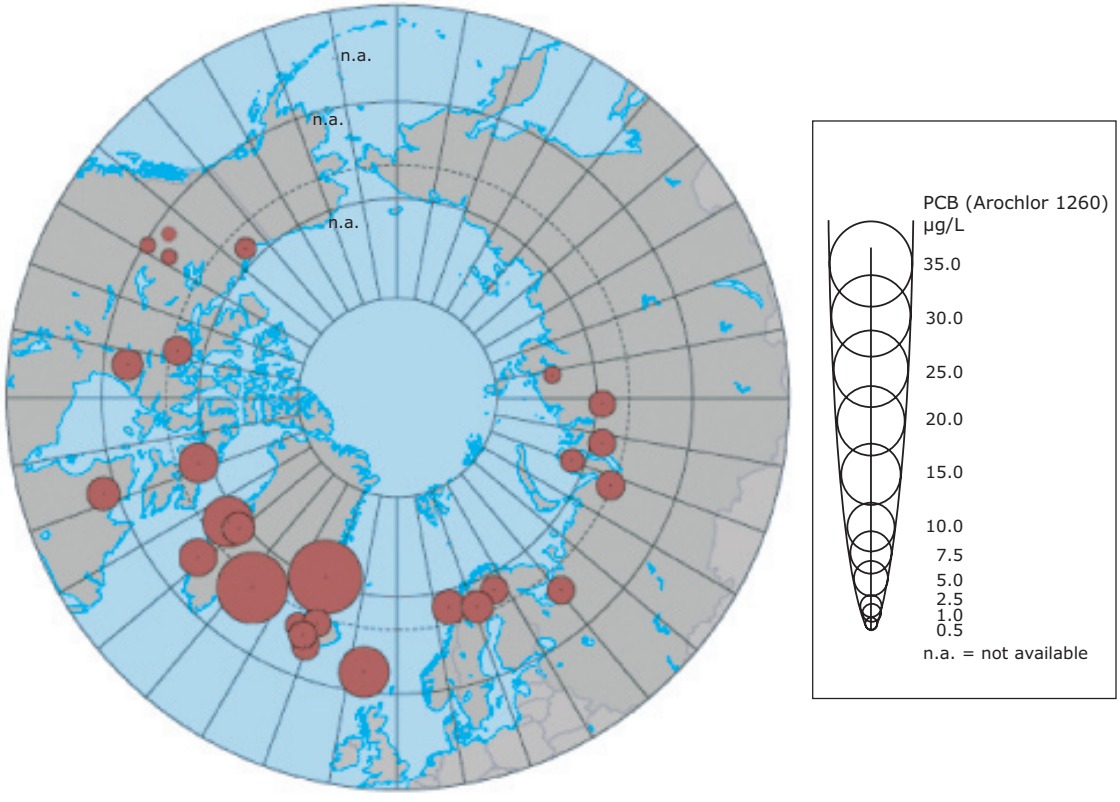
Source: German Environmental Survey, 2003.

bioaccumulation potential of these elements are largely unknown.

The burden of chemicals in the Arctic is caused partly by long-distance transport of pollutants from Europe. An assessment clearly shows that Inuit living in the Arctic (Greenland and Canada) have high exposures to several POPs and mercury, linked mainly to the consumption of marine species as part of traditional diets. Concentrations of mercury are increasing but no trends in human exposures to POPs are as yet apparent.

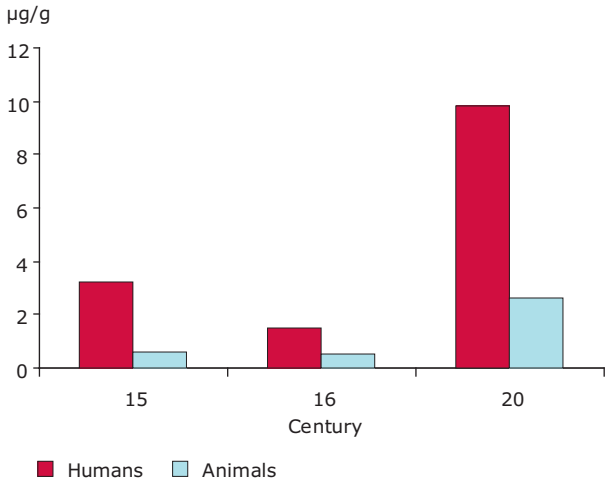
Subtle health effects are occurring in certain areas of the Arctic due to exposure to contaminants in traditional food, particularly mercury and PCBs. The greatest concern is for the foetus and the newborn. Human intake of substances with dioxin-like effects is a matter of concern in the Arctic, as confirmed by results from Greenland. Increasing human exposure to chemicals currently in use has been documented, also for brominated flame retardants. Polychlorinated naphthalene (PCN) is expected in human tissues, possibly adding to total dioxin activity.

Figure 7 **PCB concentrations in blood of mothers and women of childbearing age**



Source: AMAP, 2003.

Figure 8 Changes in mercury concentration in human and animal hair from Greenland



Source: Hansen, Personal communication, 2005.

It has been clearly established that diet is the exposure route for most contaminants in the Arctic. Dietary intake of mercury and PCBs exceeds established national guidelines in a number of communities and there is evidence of neurobehavioral effects in children in some areas. In addition, lifestyle factors have been found to influence the body burden of some contaminants, for example cadmium exposure from smoking. The physiological and nutritional benefits of traditional food support the need to base dietary recommendations on risk–benefit analyses.

From the human health perspective, it is of the utmost importance that considerations of global action against POPs and mercury take into account the concerns for human health in the Arctic (Figure 8).

The transport of POPs and heavy metals to the Arctic is strongly influenced by climate variability and global climate change. It is influenced by factors such as temperature, precipitation, winds, ocean currents, and snow and ice cover. Pathways within food webs and the effects of exposure on humans may also be modified by changes to climate. Global change scenarios have indicated the potential for substantial changes in atmospheric and oceanographic pathways that carry contaminants to, within, and from the Arctic. These effects mean that climate-related variability in recent decades may be at least partly responsible for some of the trends observed in contaminant levels.

Figure 9 Some results from the German Environmental Specimen Bank

Source: www.umweltprobenbank.de.

Bio-banks are important tools for retrospective studies of long-term changes and the effects of policy measures and for making regional comparisons. The advantages of bio-banks are that one can use the latest analytical methods on traditional and new contaminants, and that 'monitoring overload' can be prevented. Several countries in Europe have established bio-banks and use them for bio-monitoring. A European bio-bank network is under consideration.

The German Environmental Specimen Bank (ESB) was established in 1985 as a permanent institution for the systematic collection, processing, characterisation and storage of environmental samples from marine, freshwater and terrestrial ecosystems as well as human samples. Blood and other human specimens have been collected since 1981 from a group of about 100 unexposed persons in defined peripheral conditions. The subjects have to complete standard questionnaires about family and health status, occupational exposure, nutrition, smoking and drinking habits and the use of medicine.

Pentachlorophenol (PCP) is a broad-spectrum biocide which has been used mainly as a fungicide. PCP is a chemical product with a high risk for the environment because of its toxicity, its contamination with dioxin and its wide range of uses. PCP levels in the environment and in humans have been decreasing in Germany in recent years, mainly as a result of a legal ban since 1989 (Figure 9). The data are from blood plasma samples from male and female students (age 20–29) and represent background PCP contamination in Germany. After the legal ban, the sources of exposure to PCP that remain are the uncontrolled use by private persons of old products that contain PCP, imported leather goods, textiles, carpets, old military tarpaulins and rucksacks.

References:

SCALE baseline report on biomonitoring. (European Commission, Environment DG, 2004) http://www.europa.eu.int/comm/environment/health/finalreports_en.htm (accessed April 2005).

US Environmental Protection Agency (2003). 'America's children and the environment — measures of contaminants, body burdens and illnesses'.

Socialstyrelsen, Sweden (2005). Miljö och Hälsorapporten.

Norén, K. and D. Meironyté. 'Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20–30 years'. *Chemosphere* 40: 1111–1123.

Meironyté Guvenius, D (2002). 'Organohalogen contaminants in humans with emphasis on polybrominated diphenyl ethers'. Akademisk avhandling, Karolinska Institutet.

AMAP (2003). 'AMAP assessment 2002: human health in the Arctic'.

Umweltbundesamt (2003). German Environmental Survey. http://www.umweltbundesamt.de/survey-e/index.htm (accessed April 2005).

German Environmental Specimen Bank (2005). www.umweltprobenbank.de (accessed April 2005).

4 Wildlife as early warning signals for human impacts

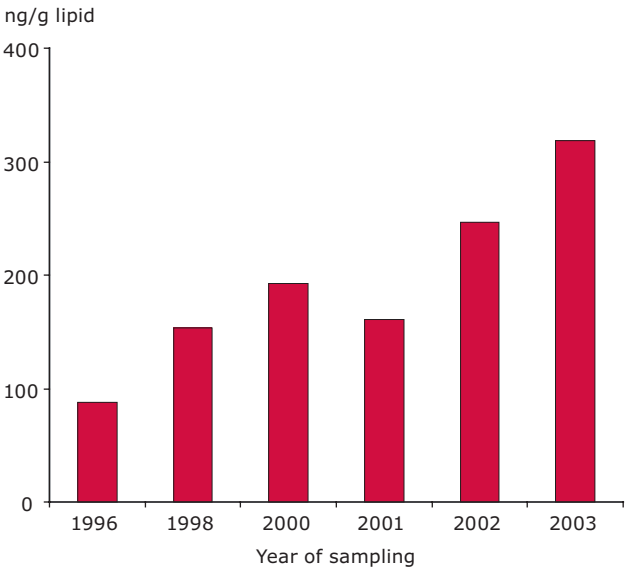
One of the most difficult aspects of 'environment and health' is the realistic assessment of the health impacts of environmental exposures. Humans are exposed to low levels of contaminants in their daily lives, but exposures may be over long, often lifelong, periods. The exposure situation is usually complex, with environmental contaminants being present in mixtures or coming from different sources. Current methodological approaches in toxicology have problems dealing with these complex situations, and there are several examples of failure to realistically assess and predict the impact of environmental exposures on human health and well-being. Wild animals in the environment can serve as sentinel species, indicators and 'early warners' of the impacts of environmental exposure since they are exposed for their whole lifecycle in their natural environment. High trophic-level organisms (at the top of the food chain) occupy a similar niche to many human consumers and are often exposed to similar types of environmental contaminants through daily dietary intake. Many fundamental physiological functions in animals and humans are similar. If a chemical at a certain concentration is causing a certain effect in a wild animal, it could be taken as an indication of the same effect occurring in humans. A substance that is neurotoxic to birds is very likely to be so to humans and a substance that is estrogenic in fish is likely to be so in humans.

Persistent organic contaminants (POPs) in the environment and their effects on wildlife provide a good example of this. The persistent, lipophilic and bioaccumulative nature of POPs causes them to bio-magnify to extremely high concentrations from water in aquatic systems to the tissues of top predators. Evaluating the patterns, levels, trends and effects of POPs in higher trophic-level consumers contributes to our understanding of both the contamination of ecosystems and the risks posed to the health of humans and wildlife. Even when the concentrations are below the limit of detection in water they may easily be measured in animals. Wildlife examples have shown that some human groups may be at increased risk because of their preferential habit of consuming fish and other aquatic food.

Top marine mammalian predators like seals, whales, dolphins and polar bears have been shown to have high levels of POP residues in their bodies, obtained through the food chain. A number of physiological effects have been observed in these animals (Table 3) including infertility, immunodeficiency and different types of tissue malformation. There is additional information from non-mammalian species like birds and fish from POP contaminated areas and waters that shows reproductive disturbances, retarded embryonic development and different types of malformation, which supports the conclusions from mammalian observations. Several studies of exposed human populations confirm the observations from wildlife. Similar effects are observed in humans when they reach body burdens of POPs that approach the levels that cause effects in wildlife.

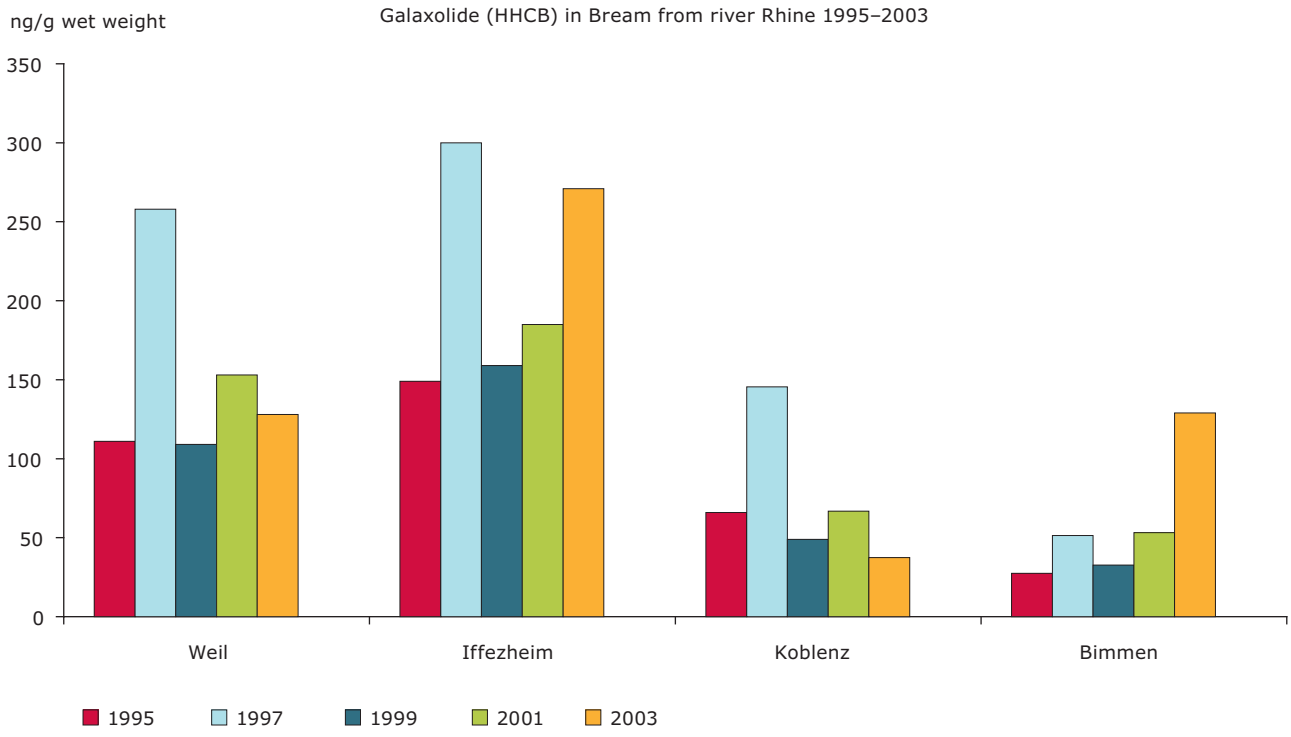
Endocrine disruption is another area where observations from wildlife have proved to be

Figure 10 Methyltriclosan (MTCS) concentrations in bream *(Abramis brama)* muscles from the river Rhine near Bimmen 1996–2003



Source: www.umweltprobenbank.de.

Figure 11 Polycyclic musks (in this case galaxolide) in bream muscles from the river Rhine



Source: www.umweltprobenbank.de.

valuable. As mentioned in the section on endocrine disrupters there is, as yet, very little evidence of endocrine disruption in humans caused by environmental factors. For wildlife, however, the situation is different. There is a wealth of information (Table 3) that industrial chemicals, pesticides and natural hormone-like substances found in different types of food are interfering with the endocrine systems of birds, fish and amphibians. Many of the substances in Table 3 are regulated or have been withdrawn from the market. Vital hormone-controlled functions like reproduction, metamorphosis and behaviour are affected. Wildlife studies have also shown the significance of hormone residues released to the environment with municipal sewage waters or from farms with intensive animal husbandry. There are clear signs of endocrine disruption in fish populations in waters that receive effluents from such installations. Wildlife has signalled that industrial effluents such as those from pulp and paper mills contain substances with endocrine activity. Studies of natural fish populations have pointed to the environmental risk of pharmaceuticals released to the environment and potential effects with respect to reproduction. Metabolites of ethynyl-estradiol from contraceptive pills have been found in fish downstream of municipal sewage treatment plants with signs of impact on the reproductive system (vitellogenin induction, intersex).

It can be concluded that observations from wildlife in the area of endocrine disruption have provided valuable information about the potential exposure to endocrine-active substances that may also affect humans. The wildlife studies have also very clearly shown that substances with endocrine activity are contaminating the environment. They have alerted us to the possibility that these substances can be recycled to human consumers via the food chain, for example in drinking water and food.

Pharmaceutical residues in the environment have recently received considerable attention. Although carefully tested for human use, they can be re-circulated to human consumers and reach groups for which they are not aimed, like children. Currently our sewage treatment facilities are not optimised to degrade and prevent pharmaceuticals from household wastewater from reaching the environment. Pharmaceuticals are not only a health risk for humans but can also have serious effects on the environment. One current example is an unusually high mortality of vultures in India and Pakistan, where populations have declined by more than 95 % since 1997 as a result of exposure

to diclofenac, a widely-used painkiller and anti-inflammatory drug. Vultures are natural scavengers, cleaning up carrion from wildlife and domestic livestock. The birds are exposed to the drug when they feed on the carcasses of farm animals which have been treated with the drug. For hitherto unknown reasons the drug is poisonous to the birds and the wide use of the drug in livestock is now threatening local populations of vultures. Diclofenac is widely used in Europe both for humans and livestock but no impacts on European bird populations have yet been described.

Personal-care products such as soaps, shampoos and different types of cosmetic products contain substances that are not degraded in sewage treatment systems and can therefore reach the environment. Many substances are persistent, lipophilic and bio-accumulating. Triclosan is an antimicrobially active substance used for many purposes such as disinfectants, preservatives and personal-care products. Triclosan is transformed to methyltriclosan in the environment through a path which is not fully understood. This substance persists in the environment and accumulates in organisms. Methyltriclosan concentrations in fish are increasing at all sampling sites in Germany, but data about the toxicity and action of methyltriclosan are largely missing (Figure 10).

Polycyclic musk compounds such as galaxolide are used as perfume substitutes for the more expensive original musk in personal-care products. Unlike the original scent, synthetic musks persist in the environment and accumulate in aquatic organisms. Synthetic musk compounds are only mildly toxic but inhibit cellular defence against other xenobiotic substances. Although some perfume and consumer product companies began phasing out their use of polycyclic musks in Europe in the mid-1990s, no general decrease in concentrations in aquatic organisms can be found in German rivers (Figure 11).

Table 3 Examples of reproductive and developmental abnormalities attributed to endocrine disruption

Species	Impact	Substance
Mammals/Humans	impotence, decreased libido, reduced sperm counts and motility, menstrual cycle irregularities	DDT, kepone, oral contraceptive exposure, stilbene derivatives
cattle	infertility	coumestrol
sheep	infertility	isoflavonoids, coumestans
seals	impaired reproductive functions	PCBs
mink	population decline, developmental toxicity, hormonal alterations	PCBs, dioxins
rabbits	infertility, failure of ovulation, failure of implantation	isoflavonoids
guinea pigs	infertility	isoflavonoids, coumestans
mice	infertility, inhibition of estrus, inhibition of ovulation	DES, isoflavonoids
Birds		
Japanese quail	abnormal reproductive behaviour, haematology, and feather morphology	o,p'-DDT
gulls	abnormal development of ovarian tissue and oviducts in male embryos	o,p'-DDT
waterbirds	eggshell thinning, mortality, developmental abnormalities, growth retardation	DDE, PCBs, AhR agonists
Reptiles		
alligators	abnormal gonads, decreased phallus size, altered sex hormone levels	o,p'-DDT, p,p'-DDE, dicofol
red-eared slider turtle	anomalous reproductive development	trans-nonachlor, cis-nonachlor, arochlor 1242, p,p'-DDE, chlordane
Fish		
mosquito fish	abnormal expression of secondary sex characters, masculinisation	androstenedione
roach	hermaphroditism, vitellogenin in males, altered testes development	sewage effluent mixture
lake trout	early mortality, deformities, blue sac disease	dioxin, related AhR agonists
white sucker	reduced sex steroid levels, delayed sexual maturity, reduced gonad size	bleached kraft pulp, mill effluent mixtures
flatfish	decreased hormone levels, reduced ovarian development, reduced egg/larvae viability	PAHs
Invertebrates		
snails	masculinisation, imposex, formation of additional female organs, malformed oviducts, increased oocyte production	tributyltin, bisphenol A, octylphenol
marine copepods	stimulate sexual maturation and egg production	bisphenol A
Daphnia magna	delayed moulting time	PCB29, arochlor 1242, diethyl phthalate

Abbreviations: AhR, aryl hydrocarbon receptor; DES, diethylstilbestrol; DDE, dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane.

References:

McLachlan, J.A. (2001). 'Environmental signaling: what embryos and evolution teach us about endocrine disrupting chemicals. *Endocrine Reviews* 22(3): 319–341.

German Environmental Specimen Bank (2005) www.umweltprobenbank.de (accessed April 2005).

5 Climate change and health

Climate change is likely to affect human health, either directly through the physiological effects of heat and cold, or indirectly, for example through the spread of vector-borne pathogens. An increase in such impacts has been observed during recent decades in Europe (the summer heat waves in 2003 alone resulted in more than 35 000 excess deaths). Such impacts are projected to escalate further with changing climate.

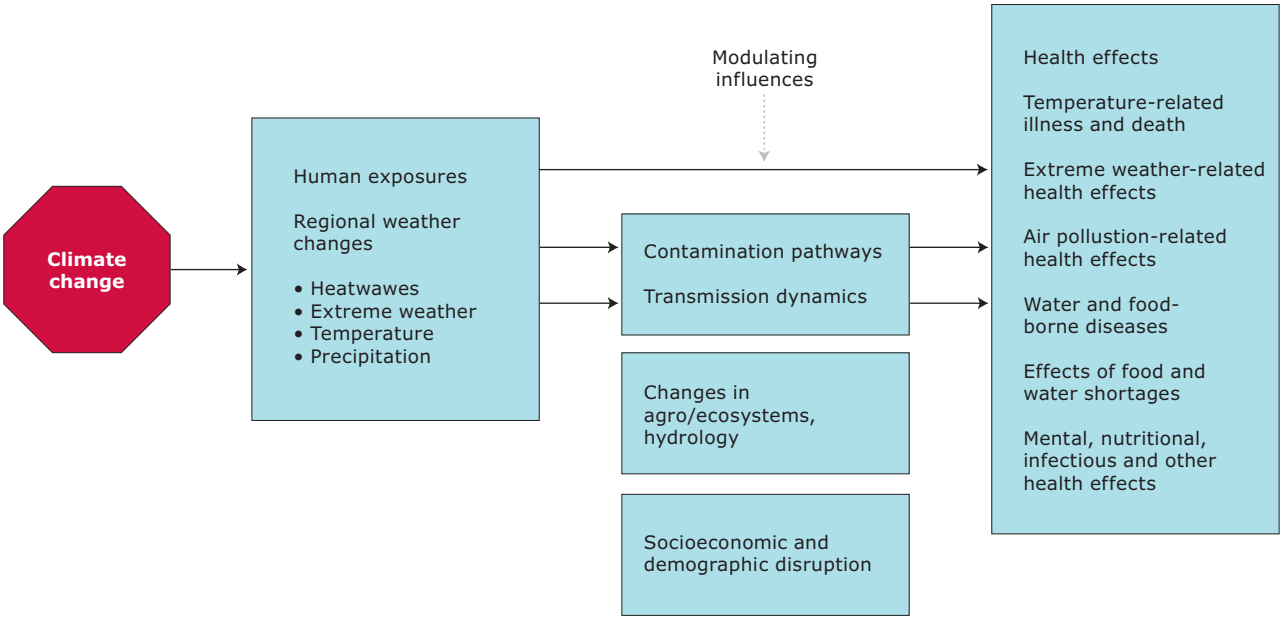
Direct impacts on human health are associated mainly with heat waves and floods. Extreme hot or cold conditions can be detrimental to many human body functions and exposure to them has an important effect on daily mortality. Heat waves are projected to become more frequent and intense, and hence to increase the number of deaths. The risk of excess winter death seems to be widely distributed among the elderly in Britain. Cold spells are also a risk in warmer regions when they occur in conjunction with ecological disturbances, catastrophes, or social conflicts such as the war in Bosnia. But fewer cold spells under warmer climate conditions are likely to reduce the number of cold-related deaths.

Increasing intensity of heavy rainfall is likely to make extreme floods more frequent in some areas of Europe. The number of deaths can be particularly high during sudden flash floods. Flood events cause physical (e.g. injuries) as well as mental (stress and depression) disorders.

Water and food-borne diseases may increase with climate change, particularly when water availability decreases and high temperatures affects the quality of food.

Climate change is also likely to result in changes in trans-boundary pollution effects.

Figure 12 Climate change and human health — risks and responses



Source: WHO, WMO, UNEP, 2003. www.who.int/globalchange/climate/en/ccSCREEN.pdf.

The seasonality of allergic disorders may change as a result of a shift in the flowering period of plants.

The projected rise in temperature is likely to increase the geographical extent of ticks and the infestation of areas that are currently tick-free. Tick-borne diseases are of increasing importance, mounting to epidemic proportions in many parts of Europe. An increase in cases of tick-borne diseases has been observed since the 1980s in the Baltic and central European countries and in Russia. However, because of the lack of knowledge on causal relationships between climate change and increases in ticks, projections of the rates of tick activity and infection have not been possible. Climate-induced changes in the potential distribution of malaria is projected, mainly in poor and vulnerable regions; localised outbreaks in Europe are expected in areas where the disease has been eradicated but vectors are still present.

The impacts of climate change on human health are being exacerbated by synergies and interactions with environmental quality: ambient air and aero-allergens, water and food shortages, decreases in natural ecosystems and their recreational capacities, changes in agrochemical use as a response to new agro-climatic conditions, and the influence of demographic, economic and social dislocations.

References:

EEA (2005). 'Vulnerability and adaptation to climate change impacts in Europe: a scoping report' (in preparation). EEA Technical report no 7/2005.

WHO, WMO, UNEP (2003): Climate change and human health — risks and responses. (www.who.int/globalchange/climate/en/ccSCREEN.pdf).

European Environment Agency

Environment and health

2005 — 35 pp. — 21 x 29.7 cm

ISBN 92-9167-781-7

TH-AL-05-010-EN-C

Price (excluding VAT): EUR 15.00

ISBN 92-9167-781-7

European Environment Agency
Kongens Nytorv 6
1050 Copenhagen K
Denmark

Tel.: +45 33 36 71 00
Fax: +45 33 36 71 99

Web: www.eea.eu.int
Enquiries: www.eea.eu.int/enquiries

Publications Office

European Environment Agency