3.10. Human health issues

Main findings

The environment in which people live, work and play is an important determinant of health and well-being, but the extent of its importance in developed economies is difficult to quantify.

The most common diseases in the EU – heart and circulatory diseases, cancer, respiratory diseases, stress and related symptoms – have many causes which are often interconnected; including genetics, the condition people are in (via diet, exercise etc.), and the environmental circumstances to which they are exposed.

Identifying cause-and-effect relationships is therefore very difficult, especially if the impact of the environment on health is delayed, or is the product of many, perhaps small, environmental factors acting together.

There is a serious lack of data and information on exposures, effects and biological models that connect them. Therefore considerable uncertainty surrounds many issues of concern, such as air pollution, noise, water contamination, waste, climate change, chemicals (including endocrine disruptors and antibiotics) and non-ionising radiation.

In many cases, however there is sufficient evidence to take preventive action, particularly where the impacts may be serious, large-scale and irreversible – circumstances which merit the use of the precautionary principle. Preventive action on many of the environmental hazards covered in this chapter is being taken, but more integrated and effective action is being proposed to reduce threats to health and well-being.

1. Introduction

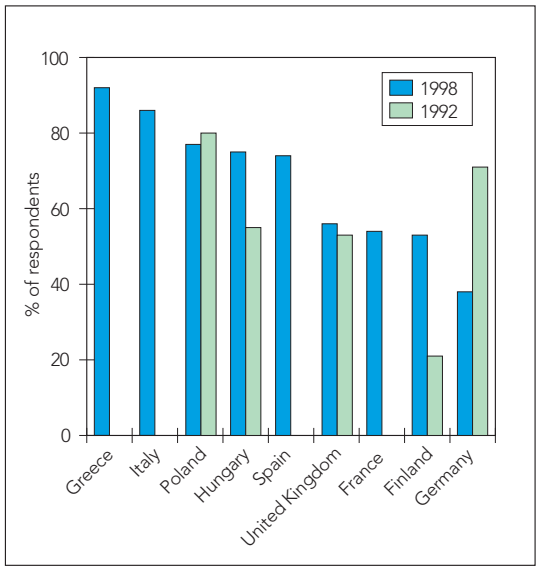
'The environment is everything which isn't me.'
— Albert Einstein

People are at the centre of 'their' world, as Einstein observed, but they are also part of the environment, and play a significant role in shaping it, as Chapter 2.1. and other chapters have shown. But the relationship is not just unidirectional: the environment 'shapes' people by the impact it has on their health.

This is not just in the obvious ways of sustaining life, through the provision of food, water and shelter, but also through the less visible impact it has on genes, cells, organs and biological systems which together can cause disease. In the more developed economies – such as the EU – where basic supplies of clean water and sewage facilities are generally available, environmental impacts on health are often less obvious – and more insidious – than in developing countries. However, people feel very concerned about the links between their environment and their health, and more so now than in the early 1990s when environmental issues were much higher on their and the media's agenda (Figure 3.10.1). Politicians have reflected this concern and declared that human health is a key objective of environ-

Opinions: Proportion of respondents who believe environmental problems affect their health a great deal/fair amount in 1992 and 1998

Figure 3.10.1



Source: Environics International, 1998

mental policies (Box 3.10.1). However, unravelling the less obvious connections between the environment and health in developed economies is not easy.

Damage to health is the result of many factors acting in various combinations, over different time periods, to a diverse range of people, of varying sensitivities, and at different stages of their lives (Figure 3.10.2). Understanding the complexities of what causes ill health is clearly going to be difficult – and, very often the more we know, the more we realise what we don't know. It is not surprising, therefore, that scientific and public controversies over environment and health have been or are currently common within scientific and public circles (e.g. leaded petrol and brain damage in children, or antibiotic growth promoters in animal feed and increased human resistance to antibiotics). Public policy decisions on environmental *hazards* (potential damage) and *risks* (probable damage) are difficult to make and evaluate. However, understanding the types of information needed for environmental health decision-making (as well as its use and limitations) will contribute to a wider appreciation of the reasons for public concerns, differences in expert opinions and the action (or inaction), of governments.

Environmental stresses for which there are reasonably good exposure and effect data are estimated to be a major factor in an estimated 5% of disease, according to preliminary report prepared for the WHO on the basis of Dutch data (WHO, 1999a; De Hollander *et al.*, in press). The main components of this environmental fraction are: external air pollution, which accounts for most of the total environment related health loss in the Netherlands (in terms of reduced life expectancy, the quality of life and number of people affected); environmental noise and indoor air pollution, including radon, damp and environmental tobacco smoke. Lead in drinking water is also significant. Traffic and domestic accidents, which together would bring the total environmental fraction of disease causation from 5% to 12% are very important public health hazards, but are not normally considered as environmental health issues.

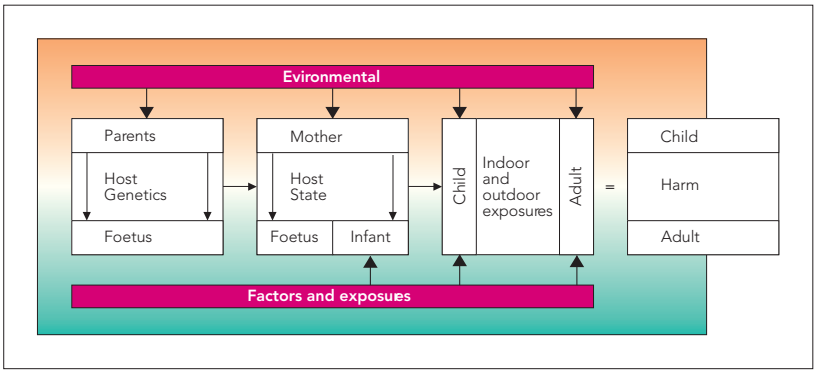
Box 3.10.1. Health and the environment: key declarations

The environment should be regarded as a resource for improving living conditions and increasing well-being (Frankfurt Conference, WHO 1989).

Human beings are at the centre of concern for sustainable development. They are entitled to a healthy and productive life in harmony with nature (Earth Summit, Rio de Janeiro, UNCED 1993).

We have a shared goal before us: to improve the living and health conditions of the present generation, to ensure that the carrying capacity of nature is not exceeded and that the right of the future generations to a satisfying and productive life is safeguarded (Helsinki Conference, WHO 1994).

Figure 3.10.2 **Environment, people and health: some key relationships**



Environmental factors (e.g. overcrowding, diet, climate, stress) and exposures (e.g. from air, food, drink, surfaces) play a part in causing and/or aggravating disease and ill health, both directly and via parents.

Source: EEA

2. Some dominant environmental health issues in Europe

2.1. Air pollution

Atmospheric pollution is a major cause of exposure to substances which are hazardous to health: the causes and effects of air pollution are discussed in chapters 3.3 and 3.4.

2.1.1 Exposure of European population to ambient air pollutants

Exposure data on suspended particulate matter is poor and is still measured by different methods throughout Europe, and the measurements of size-fractionated particulate matter of health relevance (PM10 or PM2.5, i.e. particles up to 10 and 2.5 microns in size respectively) has been introduced in only a few countries. This scarce data has to be extrapolated which increases the uncertainty of the analysis. Therefore the estimates presented below provide just an indication of the possible magnitude of the effects (Figure 3.10.3).

Box 3.10.2. Scope of the chapter

The scope of this chapter, which summarises some key environmental health impacts, is limited to a selection of those environmental stresses to which people may be exposed at home or out of doors, and which illustrates a range of health impacts and knowledge about their links to the environment. The selected information presented is designed to illustrate some general points about the links between environment and health, rather than being a comprehensive review of the literature, which is beyond the scope of this chapter. Where the issue is very well covered in other publications, such as climate change (WHO, 1999b), relatively little space is devoted to it.

The chapters on waste, hazardous substances, transboundary air pollution, climate change, stratospheric ozone, urban areas and water stress provide background information on the driving forces, pressures and associated exposures that are linked to health problems.

This chapter does not cover occupational impacts on health in much detail, despite its significant influence on public health. Fully integrated approaches to health need to include potential stresses from all parts of the environment. This is not only because human lungs and livers do not discriminate between pollutants that come from the factory or the street. but also because the sum of the exposure to stresses from all sources may be either additive, synergistic (more than the sum of the parts) or antagonistic (less than the sum of the parts), and therefore need to be included in any integrated assessment of environmental health risks (La Dou, 1998).

Knowledge of the distribution of environmental health impacts between e.g. social groups, geographical areas and generations is critical for undertaking fully integrated assessments, but apart from a few references to geography, age, class and future generations, these equity issues are beyond the scope of this chapter (Luhmann *et al.*, 1998).

A more comprehensive view of this field is available from the report 'Overview of Environment and Health in Europe in the 1990s' prepared by WHO for the 3rd European Conference on Environment and Health held in London in June 1999 (WHO, 1999a).

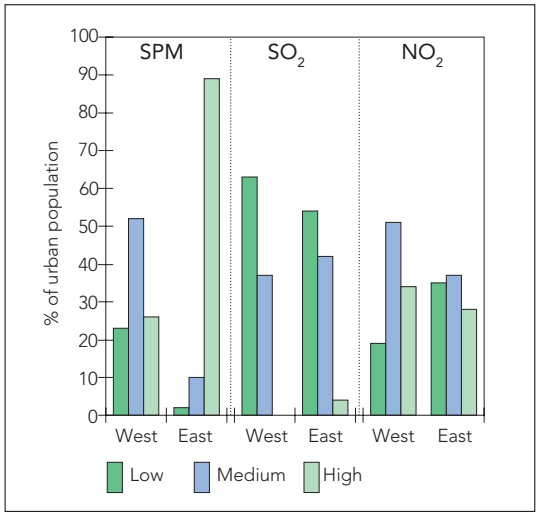
Over 24% of EU urban residents live in cities where the annual mean concentration of particulate matter (SPM) exceeds 30 µg/m^3. In the eastern part of Europe for which the data was available, close to 90% of the population live in cities with such relatively high concentration of particles. Trends in SPM were better in EU countries than in the rest of Europe, with 35% of people in EU experiencing more than a 5% per year reduction in SPM levels (21% in central Europe) and only 12% experiencing a more than 5% per year increase in SPM concentration (27% for central Europe) (for further information on urban air pollution, see Chapter 3.12).

Most of the population of Europe now lives in cities with low or medium concentrations of SO_2. In EU, 97% of urban residents have enjoyed reductions in SO_2 concentrations, whereas in the central part of Europe, almost of 20% of urban residents experienced increasing concentrations of SO_2.

More people in the western cities were exposed to medium and high levels of NO_2 than in central Europe. However, the trends were mostly stable (for 60% of urban residents) or decreasing (15%) in the EU countries, while NO_2 concentration increased for 43% residents of cities in central Europe.

Concentration of lead in ambient air has been decreasing over the recent decade, mainly due to the phasing out of lead from petrol. Several 'hot spots' were still observed in eastern Europe at the end of 1980s, mainly due to the poorly controlled emissions from industrial point sources. Monitoring data indicates that relatively high lead concentrations were also measured in the proximity of busy roads in several large cities in western Europe (Zaragosa, Toulouse, Lyon) in the early 1990s. More recent data indicates that concentration of lead in ambient air was decreasing even in those highly-polluted locations in the 1990s.

Exposure to common air pollutants Figure 3.10.3



Data on concentration of the most commonly-monitored air pollutants (suspended particulate matter, SO_2 and NO_2) is shown for 1995 or later.

In total, information on one or more pollutants is available from 110 cities in 24 countries, including 64 cities in 13 EU countries with close to 58 million residents.

Source: EEA-ETC AQ and WHO/ECEH

2.1.2. Estimates of health impacts of some ambient air pollution in the EU

The most common of the well-known air pollutants (suspended particulate matter and ozone) are associated with ill health even at relatively low concentrations of the pollutants frequently experienced by people in Europe. This observation comes from a number of studies on the effects of daily changes in pollution levels conducted in many parts of the world, including Europe, as well as from a few studies on the health effects of longer term exposures, most of which have been conducted in the United States. The results of these new studies have been used in the revision and update of the WHO Air Quality Guidelines, which, in turn, provide a basis for the work on the new so-called 'Daughter' Directives to the EU Air Quality Framework Directive (96/62/EC), which will set revised limit values for the main air pollutants.

The most important message from these studies is the health significance of particulate matter at low levels. The effects include short-term impacts on pulmonary function, increased incidence of respiratory symptoms, and increased mortality implying considerable reductions in life expectancy. However, there is still discussion on the applicability of the results from long-term studies conducted in the United States to European conditions. One of the reasons for doubts are the possible differences in the composition of the pollution mix in the European and American cities.

In combining the information from epidemiological studies with the data on ambient concentration of main air pollutants, it is possible to calculate a proportion of health problems which can be associated with the exposure (Krzyzanowski, 1997).

The effects of *long-term* exposure to suspended particulate matter are the most important health effect of ambient air pollution in Europe, and are involved in perhaps 41 000 to 152 000 extra deaths of respiratory diseases per year in the EU cities. These effects occur at various concentration levels, including concentrations considered as 'low' and reduce life expectancy in middle age people (Brunekreef, 1997). The precise magnitude of the effects of long-term exposure is uncertain, within a wide range of estimates which reflects the weakness of the scientific evidence available.

Short-term variation of population health associated with the daily changes in air pollution levels is better documented. Air pollution with particulate matter is associated with more deaths (22 000 to 47 000 a year) or hospitalisation (4 000 to 8 000 admissions) than exposures to SO_2 and ozone which together are responsible for 3 000 to 6 000 deaths and 400 to 1 600 hospital admissions a year in the EU. It can be assumed that the health problems attributed to the pollution and registered through hospital admissions could have been avoided in the absence of the pollution. However, this interpretation is not valid for mortality (McMichael *et al.,* 1998). While there is an association between the daily number of deaths and air pollution level, it is not certain to what extent the life of the affected individuals is shortened by the exposure.

In summary, the available data from the 1990s indicates that a significant reduction in population exposure to sulphur dioxide has occurred in the last decade, and that this air pollutant remains a problem only in a limited number of cities in central Europe. However, the levels and trends of pollution with particulate matter are still of concern, and there is little improvement with respect to ambient levels of NO_2 or ozone. These components contribute to significant adverse impacts on public health, including increased mortality and reductions in life expectancy. The economic costs of these air pollution health impacts are considerable (WHO /EEA, 1997; Maddison, 1998).

2.1.3. Respiratory allergies and asthma

Outdoor air pollution also plays a role in the aggravation, and possibly the causation of asthma and other allergic responses, which are increasingly prevalent diseases, especially in children. Approximately 70% of outdoor air pollution penetrates indoors (WHO, 1999a) so that an integrated approach to both outdoor and indoor air pollution is needed. Other key components of indoor pollution which have been associated with respiratory and allergic responses are dust mites, spores from pets, damp, environmental tobacco smoke and NO_x from gas ovens.

The prevalence of asthma in children of school age varies from 4% to 27% in different parts of Europe. Wide geographical variation in asthma prevalence is also noted in adults. There is an indication that the prevalence rates have increased over the last decade. The frequency of asthma attacks, sometimes requiring medical assistance or hospitalisation, has been shown to be associ-

Box 3.10.3. Environment and immunity

There is increasing evidence that the fine ambient air particles involved in respiratory and cardio-vascular diseases impact on health via the immune system. Other examples of environmental stresses that have a negative impact on the immune system are:

- ultraviolet radiation, which is known to have effects on the immune system at doses that are currently encountered outdoors;
- natural and manmade chemicals, for which a large data base on laboratory animals shows effects on the immune system, suggesting that chronic exposures to even low concentrations may potentially have an impact on humans; and
- combinations of immunotoxic agents, such as in food, e.g. natural toxins, heavy metals etc.

However, except for allergies resulting from sensitisation by pollutants directly, there is little information or understanding about the link between negative effects on the immune system and adverse health effects in the individual. There are apparently large 'reserve capacities' in the immune system that can absorb negative effects without adverse effects on health. However, for individuals whose immune response system is already adversely affected by others stresses (e.g. infections), and for populations that contain susceptible people (e.g. the sick and the elderly), the reserve capacity may not be sufficient to prevent adverse health effects, such as allergies (skin and respiratory), or cancers. Therefore 'any deviation from the normal situation is considered undesirable: this 'precautionary principle' point of view is aimed at the prevention of adverse effects in the population' (European Commission, 1996). Small increases in the incidence and duration of frequently occurring diseases may have large social and economic impacts.

Further research is needed into identifying bio-markers that are relevant to adverse health effects, especially in sensitive groups such as children, pregnant women, the elderly and people with genetic pre-dispositions to immune system impacts.

Source: EEA, based on European Commission, 1996

ated with air pollution levels. However, it is not clear if environmental conditions can cause the onset of the disease or only make the symptoms worse. Moreover, it is not known to what extent the geographical variation in asthma levels and trends is related to environmental factors. Diet (e.g. less omega-3 fatty acids and antioxidants) or compromised immune systems (Box 3.10.3) are also implicated in the development of asthma. However, current data prompts more questions than answers (Strachan, 1995; UCB, 1997). Figure 3.10.10 in section 4 below illustrates the multi-causal chain of factors implicated in childhood asthma.

Radon is another indoor air pollutant that is responsible for several thousand lung cancer deaths a year in the EU, confined to particular localities where geological formations give off the radioactive gas into confined spaces of houses (WHO, 1999a).

2.2. Water

Water quality is a significant factor in exposure to health risks. In general, water pollution has declined in the EU, although concerns remain over localised quality problems, and particularly nitrate contamination of groundwater resources (see Chapter 3.5).

2.2.1. Quality of water

A Europe-wide assessment of drinking water quality and estimation of related health risks faces serious difficulties due to scarcity and comparability of appropriate data (Box 3.10.4). These problems are common to both EU and Accession Countries.

2.2.2 Drinking water contamination and some health effects

The detection systems across the EU for water-borne disease are generally poor and only the larger outbreaks are detected in practice. Outbreaks affecting less than 10-20% of the supplied population are rarely detected. Individual cases of gastrointestinal disease, even if registered by medical care systems, are impossible to link directly to water quality.

Inadequate microbiological water quality and occasional outbreaks of water-borne diseases are reported across the EU, even

Box 3.10.4. Problems with data for European assessment of health risks related to water quality

- Often limited to information on the coverage by services.
- Focused on operational control by water supply agencies and for compliance assessment by regulatory agencies.
- Limited availability if collected by suppliers from private sector.
- Not suitable for statistical analysis and international comparisons.
- Different drinking water quality standards resulting in non-comparability of percentage compliance data.
- Different approaches to laboratory analysis and poor inter-laboratory comparability.

from countries with high standards of supply (and notwithstanding the often limited sensitivity of surveillance systems). For example, 3 to 6 outbreaks of waterborne gastro-enteritis have been reported by Sweden each year in the 1990s (WHO/EEA, 1998). Contamination of drinking water by faecal coliforms is detected in 1-4% of samples analysed in many European countries. Microbiological pollution is especially prevalent in small supply systems, and in some countries private supplies are not subject to such stringent standards as public supplies. In up to 33% of water samples taken from small private water supply systems in Ireland, faecal coliforms were present in amounts exceeding the standard level in 1995.

Increasing chemical water pollution from agriculture is a significant problem in Europe. Nitrate concentrations in groundwater are generally low in northern Europe, but high in several western and eastern countries.

Increased contents of nitrate pose a risk of methaemoglobinaemia to infants, a potentially serious, life-threatening disease. However, the total number of cases of methaemoglobinaemia reported are low and from only a few countries, mainly in eastern Europe.

Old water distribution systems, using leaded pipes, may be a significant source of population exposure to lead, which, in turn, may affect neurobehavioural development of children (see Section 3.6. below). This exposure can be markedly reduced by adequate treatment of water before its distribution, to reduce the solvency and bioavailability of lead. In Glasgow for example, effects of exposure-reduction measures have been shown (Moore *et al.*, 1998). Increasing water alkalinity and adding organophosphate to the water supply reduced the concentration of lead (Pb) in drinking water, which in turn lead to parallel decreases in maternal-blood lead. Part of the observed reduction of lead blood levels is attributed to a decrease of lead exposure from non-water sources, such as lead in petrol, food cans etc.

Pesticides and their degradation products are, in some areas, found in drinking water

Box 3.10.5. Pharmaceutical substances in water, sewage etc.

Pharmaceutical substances, like pesticides, are designed to have a biological effect. As they are widely used as medicines, (up to a tonne/day in some countries) and as growth promoters and veterinary medicines in animals, their presence in the environment may be significant, following human and animal excretion and other routes of exposure. They have not received much attention, partly because exposure levels were thought to be too low to be of concern. However, as the effects of endocrine-disrupting substances can be observed at very low levels, similar levels of exposure to pharmaceuticals in the environment may be significant for human and ecological health.

About 70% of antibiotics used in fish farming are released into the environment (Schneider, 1994). Several studies have identified antibiotics in sediment cores beneath fish farms (Samuelson, 1992a; 1992b), in groundwater (Eckel et al., 1993; Hohm et al., 1995; Stan et al., 1994; Feuerpfeil et al., 1999), and in manure (Macri et al., 1998).

Modelling of exposure pathways and potential doses has indicated possible worst-case scenarios of 30 µg/kg for olaquindox and 70 µg/kg for tylosine, two pig-growth promoters (Jorgensen et al., 1998). Information about possible eco-toxic effects is rare, though some rather potent effects have been demonstrated for metronidazole and other antibiotics on green algae (Lanzky and Halling-Sørensen, 1997; Holten-Lutzhoft et al., 1999). There are few, if any, studies on possible impacts on endocrine or hormonal functions in either humans or wildlife (Halling-Sørensen et al., 1997; Andersen et al., in press). However, there is increasing evidence that the use of antibiotics as growth promoters in cattle, pigs and poultry can lead to increasing antibiotic resistance in both animals and humans via the food chain (Swedish Ministry of Agriculture, 1997). For example, Denmark has a higher frequency of resistance to enterococci in pigs (55-84%) than does Sweden (14-15%) which banned antibiotics as growth promoters in 1986. The transfer of antibiotic resistance from animals to humans is possible but there is as yet little or no data on the extent of the problem in humans caused by antibiotics from growth promoters in the food chain (Edqvist, 1997). However, vancomycin-resistant enterococci (VRE), which are associated with the use of avoparcin for growth promotion, have been identified in non-hospitalised humans who eat meat, but not in vegetarians). There is also a risk of the development of cross resistance involving several strains of bacteria. For example, Feuerpfeil found cross resistance in 8 types of microbes. The WHO recommends the reduction in the use of antibiotics as growth promoters and the EU has recently (Dec. 1998) banned four antibiotics (virgianmycin, spiranycin, tysolin phosphate and bacitracin zinc) and is investigating four others. However, the evidence on animal growth promoters is not clear: the European Federation of Feed Additives Manufacturers thinks there is insufficient scientific evidence for an EU ban (Swedish Ministry of Agriculture, 1997).

No new chemical class of antibiotics has been developed in the past 20 years, despite extensive research. This provides opportunities for increased resistance. It takes at least 10-20 years to find and clinically test new antibiotics, a time lag within which antibiotic resistance could increase without opposition from new drugs.

or in groundwater (see Chapter 3.7). Triazine herbicides are the pesticides most frequently detected in groundwaters and several countries have introduced bans or restrictions on the use of products containing the active ingredients. There has been a significant overall downward trend in the contamination of groundwater by triazine herbicides and their breakdown product in most countries, although this is not the case with all pesticides (see Chapter 3.5).

Data on microbiological quality of recreational waters is collected in some countries, principally for compliance assessment by regulatory agencies. EU Member States cooperate to produce an annual assessment of bathing water quality but despite many attempts to collate and compare data from different locations (nationally or internationally), the quality of such data has severe limitations regarding its value in assessing hazards to human health, primarily due to different approaches to analysis and poor inter-laboratory comparability.

The quality of freshwater sites designated for bathing is considerably worse than those of coastal sites in the EU although the overall quality trend appears to be improving (Figure 3.10.4).

Other low-level contaminants of water may be a threat to health in some areas (WHO/EEA, 1999).

A possibly emerging threat are trace residues of pharmaceuticals, including antibiotics (Box 3.10.5), though there is little data and few studies available.

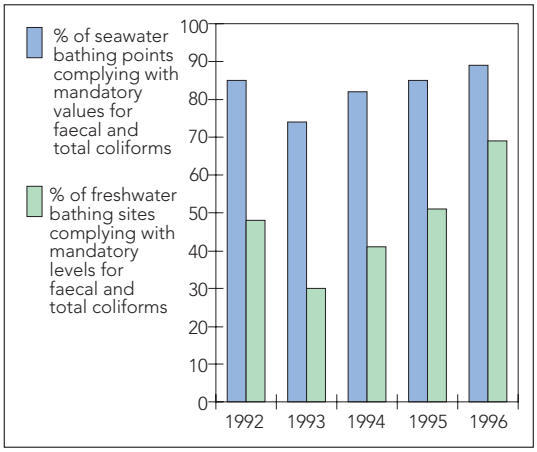
2.3. Noise

Noise can have a variety of effects which depend on the type, duration and timing of the noise and the susceptibility of the recipient (Box 3.10.6).

Reports from recent scientific research on the precise health effects of nocturnal traffic noise reveals that night-time traffic noise not only disturbs sleep but also encourages psychosomatic illnesses, shortens the period of deep, dream-rich REM (rapid eye movement) sleep, lengthens the phase of light slumber and may cause cardio-circulatory problems.

There may be some segments of the population at greater risk of adverse effects of noise. Young children, (especially during language acquisition), the blind, the hearing-impaired and hospital patients are examples of higher risk groups.

Tests of recreational water quality Figure 3.10.4



Source: WHO

Noise affects more than our health and quality of life; it even influences social behaviour and cognitive development. In 1997, studies carried out around Munich airport found that children exposed to frequent aeroplane noise do not learn to read as well as other children. Excessive background noise caused the children to tune out human voices and interfered with their language acquisition. The psychologists who conducted the study speculated that as a result of noise pollution, parents and teachers were also less willing to speak or read aloud.

Community noise needs to be assessed with respect to risks for both human health and well-being. Intensity, frequency, reversibility and avoidability are pertinent criteria for the severity of noise effects.

The knowledge about harmful impacts of noise exposure has to be transformed into environmental standards. There is also limited evidence for noise impacts on birth weight, congenital effects, and the immune system (Ministère des affaires sociales, de la santé et de la ville, 1995). However, estimated thresholds are available for only a limited range of noise impacts for which there is more substantial evidence of noise causation (Table 3.10.1).

3. Other environmental hazards of concern

Besides the recognised and relatively well-understood issues described in the previous

Box 3.10.6. Noise: some exposure/effect relationships

Exposures

Noise remains a serious environmental problem: estimates of its costs range from 0.2 to 2.0% of GDP (Quinet, 1993). It is estimated that about 32% of the EU population (approx. 120 million people) are exposed to road noise levels over 55 Ldn dB(A) at house facades and that approx. 3 million people are exposed to aircraft noise (see Chapter 3.12). Perceptions of the various types of transport noise differ between individuals and impacts can depend on the type of noise, e.g. from rail or aircraft.

Effects: Public

- Annoyance;
- Interference with speech communication;
- Sleep disturbance effects (more than 'awakenings');
- Effects on performance and productivity (reading acquisition, learned helplessness, etc.);
- Effects on residential and social behaviour (opening windows, use of dwelling area, etc.);
- Psychophysiological effects (the stress complex, hypertension, ischaemic heart disease, aggressiveness, etc.);
- Mental health effects (hospital admissions, etc.);
- Dose-effect for joint effects (e.g. annoyance + sleep disturbance + hypertension?);
- Vulnerable groups (children, hearing-impaired).

Effects: occupational

- Noise-induced hearing dysfunctions (e.g. tinnitus, temporal threshold shifts, deafness, 'impulse sounds')

Source: EEA

Table 3.10.1. **The long-term effects of noise exposure for which there is sufficient evidence**

	Observation threshold			
Effect	**Situation**	**Noise metric**	**Level in dB (A)**	**Inside/outside**
Hearing damage	work	Laeq, 8hr	75	inside
	sport	Laeq, 24hr	70	inside
Hypertension	work	Laeq, 8hr	<85	inside
	home	LAeq, 6-22hr	70	outside
Ischaemic heart	home	LAeq, 6-22hr	70	outside
Annoyance	home	Ldn	42	outside
Awakening	sleep	SEL	55	inside
Sleep stages	sleep	SEL	35	inside
Self-reported sleep quality	sleep	LAeq, night	40	outside
School performance	school	Laeq, day	70	outside

Source: Health Council of The Netherlands, 1994

sections, a number of other environmental factors create potential risk for health and cause public concerns (Table 3.10.2). In most cases, the information and scientific basis for assessment of the risk is not sufficient to confirm or deny the existence of the risk. The list of relevant issues is long, and this section provides selected examples which illustrate some general points about the identification and management of environmental health.

3.1. Chemicals and endocrine-disrupting chemicals

Low doses of some chemicals (EEA/UNEP, 1998) are associated with cancer, cardiovascular disease, respiratory diseases, neurotoxicity, and chemical sensitivity, with varying strengths of evidence, but information about exposure /effect relationships is often poor or non-existent (Box 3.10.8).

A broad class of chemicals present in the environment, such as PCDDs, PCDFs, PCBs, persistent pesticides, some detergents and some compounds used in the plastic industry, are known to have a capacity to interfere with hormonal regulation mechanisms (Toppari *et al.*, 1996; EU Scientific Committee, 1999). The Weybridge Report (EUR, 1997) concluded that while there was increasing evidence about rising trends in reproductive ill health in wildlife and humans, there were still great uncertainties about the causes of the reproductive ill health (Box 3.10.7). However, exposure reduction to endocrine disrupting chemicals was recommended in line with the 'precautionary principle'. Since then, reports by the European Parliament and others have repeated the call to reduce exposures.

3.2. Chemicals from waste disposal and treatment

Part of the still growing volume of waste generated and disposed in Europe is hazardous to health via exposure to hazardous chemicals or microbiological pollution.

Several epidemiological studies conducted in the United States have suggested a small increase in risk of a range of health impacts associated with the hazardous waste landfills, but a UK review concluded that 'The epidemiological evidence that these substances represent a cancer risk at much lower environmental levels either does not exist or is equivocal. However, data on the effects of background environmental exposures on combinations of chemicals is absent, making it difficult to assess any health impact resulting from relatively small additional exposures from incinerators (MRC, 1997). A

recently published European study adds to the suspicion that the landfill operations may contribute to a small increase in risk of certain birth defects (Dolk *et al.*, 1998). However, the present studies are not powerful enough to indicate a particular characteristic of the landfill which may cause a risk, and a weakness of the exposure assessment in those studies makes any causal relation between disease and landfills difficult to establish.

An analysis of cancer incidence patterns around municipal solid waste incinerators in the UK revealed that the observed slightly increased overall incidence of cancers in the proximity of the incinerators is related to a combination of confounding factors, and not to the waste treatment operations (Elliot *et al.*, 1996). However, the need for a further study on a still unexplained incidence of liver cancer in the vicinity of incinerators was proposed.

Technical requirements of design and operation of waste treatment at such facilities aim at the elimination, or radical reduction, of the risk to population health. Whilst there is a decline in population exposure to hazardous chemicals which may be emitted from incinerators such as dioxins, the average exposure of Europeans in industrialised countries to dioxins is significant in relation to what is now known about their likely effects (see Chapter 3.3) which include cancer, reproductive disorders, neurotoxicity and heart disease (WHO, 1997d and 1998 a).

3.3. Climate change and ozone depletion-future burdens?

The potential consequences of climate change include increases in sea level, more

Box 3.10.8. Breast cancer: an 'integrated' disease?

Breast cancer rates are rising in Europe. Some risk factors are known (genetics and family history, use of the contraceptive pill etc.) and others are suggested such as some occupational and environmental causes, such as pesticides, radiation and endocrine-disrupting chemicals but these account for only 30-40% of cases (Kristensen, 1991; Davis, 1993; Woolff, 1993; Hulka, 1995; Cantor *et al.* 1995; Rachel's Environment and Health Weekly, 1997; Wallerson, 1995; McPherson, 1994; Hoyer *et al*, 1998). However, the links with occupational and environmental factors may be small and the evidence for this is disputed. Disentangling the relative contributions of several factors in an inter-dependent causal chain is always going to be difficult, and prevention calls for an integrated, holistic approach, based on the precautionary principle (Davis, 1997).

Major health impacts and some associations with environmental exposures Table 3.10.2.

Health impact	Associations with some environmental exposures
Infectious diseases	• water, air and food contamination • climate change
Cancer	• smoking and environmental tobacco smoke (ETS) • some pesticides e.g. phenoxy herbicides • asbestos • natural toxins • food, e.g. low fibre, high fat • polycyclic aromatic hydrocarbons, e.g. in diesel fumes • some metals e.g. cadmium, chromium • radiation (incl. sunlight) • several hundred other animal carcinogens
Cardiovascular diseases	• smoking and ETS • carbon monoxide (CO) • lead • inhalable particles • food, e.g. high cholesterol • stress
Respiratory diseases, including asthma	• smoking and ETS • sulphur dioxide • nitrogen dioxide • inhalable particles • fungal spores • dust mites • pollen • pet hair, skin and excreta • damp
Skin diseases	• some metals, e.g. nickel • some pesticides, e.g. pentachlorophenol • some foods (allergies)
Diabetes, obesity	• food, e.g. high fat • poor exercise
Reproductive dysfunctions	• polychlorinated biphenyls (PCBs) • DDT • cadmium • pthalates and other plasticisers • endocrine disruptors
Developmental (foetal and childhood) disorders	• lead • mercury • smoking and ETS • cadmium • some pesticides • endocrine disruptors
Nervous system disorders	• lead • PCBs • methyl mercury • manganese • aluminium • some solvents • organophosphates
Immune response	• UVB radiation • some pesticides
Chemical sensitivity?	• trace amounts of many chemicals?

Note: Most diseases are the result of several causes. These include:

- inherited vulnerability,
- factors which are related to poverty, e.g. diet, housing quality and location, stress, alcohol and substances abuse, smoking, low birth weight etc.; work; unemployment; climate, and
- other environmental exposures arising from air, water, soil and surfaces.

The link between environmental exposures and health impacts varies from known causal relationships such as inhalable particles and respiratory-system damage to suggestive but unproved associations, such as between some cancers and exposure to low levels of some pesticides. Poor diet plays a key role in the 'diseases of affluence', such as cancer, heart and circulatory diseases.

Source: EEA

Box 3.10.7. The 'Weybridge Report' on endocrine disruptors

There is increasing evidence and concern about rising trends of reproductive ill health in wildlife and humans, and some substances have been implicated, but there are great uncertainties about the causes of reproductive ill health.

Key conclusions are:

- Sufficient evidence exists that testicular cancer rates in humans are increasing.
- The apparent decline in human sperm counts in some countries was likely to be genuine.
- There is insufficient evidence to definitely establish a causal link between the health effects seen in humans with exposure to chemicals.
- The major route of exposure to Endocrine Disrupting Substances (EDS) is usually by ingestion of food, and to a lesser extent water. It is valid for terrestrial animals, birds and mammals, including humans.
- Compared with the situation in the US, there are few cases of reproductive ill-health in wildlife in the EU where the effects could be definitely associated with EDS.
- However, some cases exist within the EU area where adverse endocrine effects, or reproductive toxicity, in birds and mammals coincide with high levels of anthropogenic substances shown to have endocrine-disrupting properties in some test systems.
- The considerable uncertainties and data gaps could be reduced by research and monitoring into exposure and effects in wildlife and humans.
- Current eco-toxicological tests, studies and risk assessments are not designed to detect endocrine-disrupting activities.
- Meanwhile, consideration should be given to reducing the exposure of humans and wildlife to endocrine disrupters in line with the 'precautionary principle'.

Source: European Commission *et al.*, 1997

frequent and intensive storms, floods and droughts, changes in biota and food productivity. Changes in ecosystems may affect the growth, transmission and activity of vector-borne or infectious diseases, such as malaria and dengue fever. Human health is likely to be adversely affected, either directly or indirectly, through complex interactions of ecological systems (McMichael, 1996a, WHO, 1999b). The direct effects may result from changes in exposure to thermal extremes, and be expressed by an increase in heat-related disease and death, but also by a decrease in cold-related disease. Other extreme weather events can lead to psychological disorders, disease or death, indirectly causing an increase in morbidity. Although there are some signs of these climate effects already beginning to happen, such as shifting geographical range and longer seasons of some vector born diseases (WHO, 1999 b), much of the burden of ill health from climate change will be on our children and grandchildren. However, climate change policies based on avoiding these health impacts will have considerable secondary benefits of avoiding shorter term health impacts from fossil fuel combustion (WRI 1997).

Similarly, stratospheric ozone depletion is expected to cause increased UV radiation and thereby increased skin cancer sometime in the next century (Figure 3.10.5). The relation of UV radiation with some forms of skin cancer is well-established, though not always with respect to the specific wavelength, exposure-response or individual susceptibilities. Though the current increase in skin cancer in Europe (3 to 5% per year since the 1960s, for malignant melanoma, WHO, 1999) seems mostly related to more frequent sunbathing and other lifestyle factors, the depletion of the protective layer of ozone in the stratosphere will increase the likelihood of increased skin cancer in the future, despite the reductions in the production of CFCs and other ozone-layer-depleting substances. However, the implementation of the Copenhagen amendments to the Montreal protocols (see Chapter 3.2) on the banning and phased reduction of ozone-depleting substances has greatly reduced the future excess incidence of skin cancer.

Increased UV-radiation also reduces the response of the immune system (see Box 3.10.3), and causes eye cataracts and other impacts. It can also be beneficial, by providing extra vitamin D.

Environmental-health hazards that impact in the future via long latent periods, such as asbestos and other carcinogens, present difficult issues of public health policy that require considerations other than good science, such as appropriate levels of proof (see section 4). Decisions sometimes need to be based on 'early warnings', which often come from the world of work, where exposures are usually higher and where the monitoring and the identification of impacts is often easier. Any integrated assessment therefore needs to embrace occupational exposures, which in any case add to the sum of stresses on the body.

3.4. The occupational environment

'It is a sordid profit that is accompanied by the destruction of health'
— Bernardino Ramazzini, 'father' of Occupational Medicine, 1713

A full-time employee spends about one-half of waking time in the workplace; the other

half is spent on domestic or leisure activities, and one-third of the 24 hours is spent sleeping. It follows that many environmental contributions to all health will be found in and around the workplace. This is why Bernardino Ramazzini, the father of occupational medicine, advised doctors to always ask their patients: 'What work do you do?' (Ramazzini, 1713).

A European survey found that 23% of the EU workforce were absent from work during the previous 12 months due to work-related ill health (European Foundation, 1996), and the WHO has identified a range of occupational stresses (Box 3.10.9).

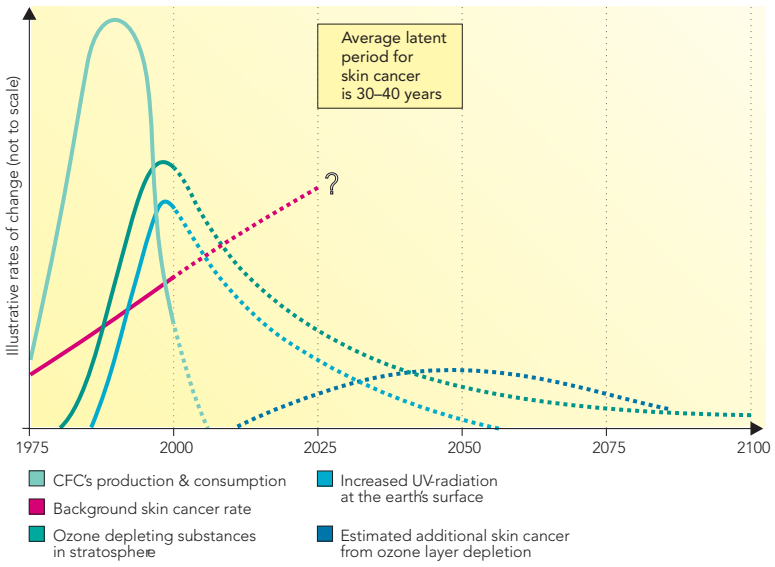
The workplace is also an effective place through which to focus efforts on health promotion, embracing occupational, environmental and 'lifestyle' factors, such as smoking, alcohol, diet and exercise. The WHO considers that 30-40% of the total disease and ill-health burden in Europe can be tackled effectively through workplace activity on either occupational factors, or on lifestyle/environmental factors that can be addressed through employee or employer activity (WHO, 1999a). Occupational accidents and ill health cost between 0.4 and 4.0 % of GNP in the EU (EASHW, 1998).

The monitoring, identification and 'proof' of the occupational origins of disease are as controversial as identification of the environmental contributions to ill health. The 'occupational' fraction of cancer has been estimated at 4-5%, or up to 25% (WHIN, 1998), but as with all diseases that have long time lags ('latent periods') between exposure and harmful effect, the conditions of exposure will always have changed by the time 'proof' of causality can be provided, some 20-40 years after exposure first began. This then affords opportunities to argue that current conditions are now harmless, and the point can only be 'proven' one way or the other some 20-40 years later (Box 3.10.10).

Both unemployment, via its link to poverty, alcohol, loss of self-esteem, etc. and overwork can cause disease and ill health.

Many environmental diseases are first identified in the higher exposure, more easily monitored world of work, e.g. 95% of the 24 known lung carcinogens and over half of all causes of cancer were identified in workplace studies, according to the WHO's

CFCs, skin cancer and time lags Figure 3.10.5



This graph illustrates the approximate time lags between CFC production, the resulting depletion of the stratospheric ozone layer and subsequent extra penetration of UV radiation and the impact this will eventually have on increasing the background rate of skin cancer, given the 30-40 year average latent period for such cancers. Reality is far more complex than this schematic illustration. For example, there are other ozone-depleting chemicals (HCFCs, HFCs and methyl bromide); the ozone hole varies with latitude, time of the year and meteorological conditions; the increased UV radiation varies between different wavelengths and with latitude and cloud cover; and the skin cancer excess comes on top of a rising background rate of skin cancer, with differential effects on the different types of skin cancer, such as malignant melanoma and non-malignant skin cancers. Human behaviour is also a determining effect in skin cancer. Health effects also include cataracts and immune response suppression. However, the figure illustrates the main relationships and time lags between CFC production and skin cancer, and the 'success' in stopping CFC production and averting much more skin cancer from ozone depletion than what is now expected. (Slaper, *et al.*, 1996).

Source: EEA

Box 3.10.9. The dangerous world of work

The World Health Organisation says:

- Some 50 physical factors, 200 biological factors and 20 adverse ergonomic conditions, plus an innumerable number of psycho-social factors, have been identified as creating hazardous working conditions. These contribute to the risk of occupational injuries, diseases and stress reaction, as well as to job dissatisfaction and the absence of physical and mental well-being.
- The risk of cancer from work and workplace exposure is of particular concern. Approximately 300-350 different chemical, physical and biological factors have been identified as occupational carcinogens. They include benzene, chromium, nitrosamines, asbestos, ultraviolet radiation, ionizing radiation and aflatoxins. The most common cancers occurring as a result include lung, bladder, skin and bone cancer and sarcomas.
- Allergenic factors are also a growing cause of occupational illness. An estimated 3 000 allergens have been catalogued which can cause dermatoses and respiratory diseases (e.g. asthma).
- Approximately 30-50% of workers in industrialised countries complain about psychological stress and overload. Such psychological factors have been associated with sleep disturbance and depression, as well as with elevated risks of cardiovascular diseases, particularly hypertension.
- Only 20-50% of workers in industrialised countries (with few exceptions) have access to adequate occupational health services.

Source: WHO, 1997a

Box 3.10.10. Asbestos and disease 1898-1998: A 100 years of 'early' warnings...

An astute observation by a lady factory inspector in 1898 concluded: 'The evil effects of asbestos dust have also attracted my attention. A microscopic inspection clearly revealed the sharp, glass-like, jagged nature of particles and ... the effects have been found to be injurious, as might have been expected' (ARCI, 1898).

Her fears were confirmed 30 years later. A government-funded study in 1929 found that one-third of asbestos workers had asbestosis, a form of pneumoconiosis. By 1955, a study of workers by Sir Richard Doll showed that asbestos also caused lung cancer, and by 1964 other cancers, including the most deadly, mesothelioma, were added to the list of 'evil effects of asbestos dust.' Table 3.10.3 summarises the history of asbestos as it moved from the harmless substance of the 1880s to the recognised killer of the 1990s, now being responsible for about 10 000 deaths a year in western Europe. Poorly-controlled asbestos use expanded right up until the 1980s, by which time it had killed thousands of people, and condemned thousands of others to die in the next 20-60 years as a result of their past exposure. The costs of failing to control asbestos early enough are not just health costs – dealing with compensation and asbestos in buildings is costing billions of pounds and was partly responsible for the bankruptcy of some Lloyds insurance underwriters in the early 1990s.

The latest study on the extent of asbestos-induced deaths 'in the pipeline' concludes that some 250 000 men (mainly) will die of asbestos-related cancer in western Europe over the next 35 years, following a doubling of the current annual total of deaths from the main asbestos cancer, mesothelioma, from 5 000 a year in 1998 to 9 000 a year by 2018 (Peto *et al.*, 1999). The study was based on the cancer registries of six European countries (France, Germany, Italy, the Netherlands, Switzerland and the UK, which account for 72% of the population of western Europe). Asbestos use in Europe remained high until about 1980, and as mesothelioma, a cancer of the lung or stomach lining, has a latent period of 30-60 years, deaths will peak around 2020 and decline slowly over the following decades.

Workers not directly employed with asbestos, such as electricians, carpenters, plumbers and maintenance men, are also at risk. Although the non-occupational risk from asbestos is very much smaller, the possibility of 24-hour exposure, and of children's exposure, contributes to a significant risk for some 'public' groups, e.g. those living in the houses of asbestos workers, where contaminated clothing has caused mesothelioma in wives, sisters and children and those living and playing in the streets near asbestos plants (Camus *et al.*, 1998).

Although there have been 'early warnings' about asbestos for 100 years, effective preventative measures were not taken until it was too late to stop deaths 'in the pipeline' of the latent period. And even accurate monitoring of mesothelioma, lung cancer and of their relationship (which may be 1:1 or 1:3 or 4) is still poor. 'It is unfortunate that the evolution of the epidemic of asbestos-induced mesothelioma, which far exceeds the combined effects of all other known occupational industrial carcinogens, cannot be adequately monitored.' (Peto, 1999).

Smoking and asbestos together have a strong synergistic effect causing a 50 fold excess of lung cancer while their separate effects are 'only' a 10 and five-fold excess for smoking and asbestos respectively (Hammond, 1979).

Synergy from smoking and other pollution is not confined to asbestos. The WHO (1998b) has concluded that smoking and other workplace contaminants can also act together to 'amplify the severity of adverse effects beyond what could be expected from smoking or the toxic hazard alone'.

Table 3.10.3.

Exposed group	Asbestosis	Lung cancer	Mesothelioma cancer
Occupational			
Workers	(1898-1929)	1955	1960s
Mates	1964	1964	1964
Environmental			
Relatives	1960s	?	1960s
Public	?	?	1980s

Note: Asbestos also causes other cancers, e.g. cancer of the larynx

Source: EEA based on Gee, 1995

International Agency for Research on Cancer. Many 'early warnings' of environmental health hazards will therefore continue to come from workplace studies (Wegman, 1996). For example, the potential human health effects of non-ionising radiation were first identified in occupational studies (Box 3.1.11).

3.5. Diet

Healthy eating plays a crucial role in disease prevention. For example, in addition to genetics, occupational and environmental factors, diet plays a key role in cancer causation, perhaps 30-40% of all cancers.

Recommendations on balanced diets have been available for many years (Figure 3.10.6). However, advice can vary, depending on scientific knowledge. Poor consumer labelling can make it difficult to make the right choice, assuming the consumer already has physical and financial access to healthy food. Contamination with chemicals, such as antibiotics and pesticides (Box 3.10.12) can diminish some of the value of healthy eating, but, as with breast feeding when the milk may be contaminated with very low levels of dioxins or PCBs, the other benefits of a healthy diet usually overwhelm the costs from micro-contaminants. Achieving a healthy diet and contaminant-

Box 3.10.11. Electromagnetic fields: an emerging occupational, environmental and consumer hazard?

The World Health Organisation (WHO) has said that research into possible adverse health effects of electromagnetic fields (EMF) should be a priority for the next four years.

The WHO's EMF project will be co-ordinating and encouraging research into the possible associations between low-frequency EMF (less than 300 Hz) and childhood leukaemia, breast cancer and diseases of the central nervous system.

WHO also recommended further research into possible associations between exposure to radio frequency fields (300 Hz – 300Ghz) and leukaemia/ lymphoma and brain and cancers.

Dr Paul Kleihues, Director of WHO's International Agency for Research on Cancer, has observed that, 'with an estimated 15 million new cancer cases each year by the year 2020, we must know if exposure to EMF is contributing to any significant extent to the incidence of disease'.

The controversial theory that electric fields like those around power lines can cause cancer has received some support from a National Institute of Health scientific panel in the US. 'This report does not suggest the risk is high', and 'The risk is probably quite small compared to many other public health risks', said Michael Gallo, chairman of the group and a professor at the university of Medicine and Dentistry of New Jersey-Robert Wood Medical School, Pistacaway.

The report comes from a National Institutes of Environmental Health Sciences panel convened to review scientific research on electromagnetic fields.

The group voted 19-9 in June 1998 that electromagnetic fields should be regarded a potential cause of cancer, using the International Agency for Research on Cancer criteria for cancirogenicity.

Eight members said that, because of conflicting studies, they could not decide whether electrical fields were potential cancer causes. One said they probably were not.

The new finding is at odds with a 1996 report by a National Research Council panel of scientists who evaluated about 500 studies on the health effects of high voltage power lines and found 'no conclusive and consistent evidence' that electric and magnetic fields cause any human disease. Studies of the incidence of disease analysed by the new NIH group found a slight increase in childhood leukaemia risk for children whose homes are near power lines and an increase in chronic leukaemia in adults working in industries where they are exposed to intensive electric fields.

The group said that there wasn't enough evidence to link household exposure to power lines to cancer in adults, or to associate electromagnetic fields to such diseases as Alzheimer's, depression and birth defects.

They found no evidence of abortion from video display terminals and no evidence of illness other than leukaemia in children (WHIN, 1998).

free food and drink may be possible if both sustainable agriculture and the reduced use and exposure to hazardous chemicals are pursued in an integrated approach to ecological and human health.

Box 3.10.12. Lindane safety re-assessed

A re-assessment of the organochlorine insecticide lindane has concluded that consumer safety limits can be exceeded by over 12 times.

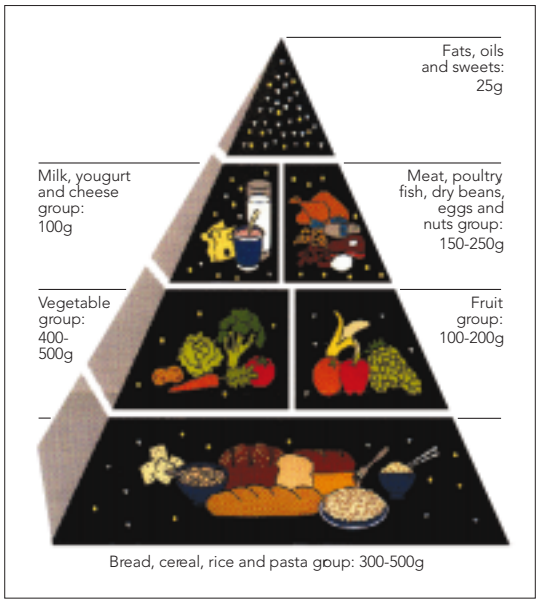
The joint FAO/WHO Food Standards Programme Codex Committee on Pesticide Residues has set a more stringent acceptable daily intake (ADI) for the insecticide lindane of 0.001mg/kg body weight.

For a 60 kg adult, therefore, the maximum daily dose should not exceed 0.06 mg in total. The ADI is the amount of pesticide that can be consumed every day for a lifetime without harm.

Codex data discussed at a recent meeting shows that any person consuming an average local diet in any region of the world could theoretically exceed the ADI for lindane by between 3.8 and 12 times if foods containing the maximum lindane residues were consumed.

Source: FAO/WHO, IN WHIN 1998

Healthy eating Figure 3.10.6



Source: CECHE, 1998

3.6. Children

As has been seen with several health hazards (air pollution, noise, skin cancer, allergies etc.) children can be particularly sensitive to environmental stresses. They are a 'biomarker' for environmental threats that require special protection, not only because they are more at risk but because they can also provide early warnings of hazards to others, as well as being effective points of intervention for the prevention of disease in their later lives.

Chemical pollutants that may affect reproductive health and new-born children include certain metals (e.g. lead and methyl mercury; Box 3.10.13), pesticides (e.g. DDT), industrial chemicals (e.g. PCBs), solvents and other substances (Foster and Rousseaux, 1995; CJPH, 1998). Exposures can occur through placenta and breast milk (Jensen, 1996; Rogan, 1996), and some may cause small abnormalities of the immune response system. However, WHO and others conclude that the benefits for breast-feeding outweigh the risks of pollutants in breast milk (Weisglas-Kuperus *et al.*, 1996; WHO, 1996b).

Children may be particularly at risk from chemicals because of their greater biological sensitivity and greater exposure to environmental pollution relative to body weight (NRC, 1993; McConnell, 1992; Bearer, 1995). Their physiological and intellectual development may be impaired by exposure to chemicals (Rodier, 1995; Rylander *et al.*, 1995; Jacobson 1996; Grand Jean *et al.*, 1997). Low-level pesticide contamination of food (infants consume eight times more food per kilogram of body weight than adults, making this a more significant exposure pathway; CICH, 1997), and of residential surfaces and toys in the UK and US, is being reported (Pesticides Trust, 1998; Gurunathan *et al.*, 1998). Some regulatory authorities are giving special attention to the higher levels of risk to children from pollution (USEPA, 1996). For example, the Food Quality Protection Act in

Box 3.10.13 Children and lead

'Lead makes the mind give way.'
— Greek physician, 2000 BC

- Lead is brought into the environment through human activities in 300 times greater amounts than through natural processes (Unicef, 1992).
- People, particularly children, may be exposed to lead from car emissions through leaded petrol, water contaminated by lead pipes, some factories (e.g. metal polishing and smelters; old paintwork in houses), contaminated soil (e.g. nurseries built on old petrol station sites), certain cultural practices (e.g. use of folk medicines containing lead), use of improperly glazed lead ceramic ware for cooking and food storage, and use of lead-contaminated cosmetics such as surma and kohl.
- Children absorb up to 50% of lead taken into their bodies, compared to 10-15% in adults. Children may receive three times the dose of adults because they have a larger surface-to-volume ratio.
- Lead in dust and dirt can be ingested via children's hands and toys, for example by thumb-sucking or by putting objects in their mouths.
- Even in the world's most developed countries, it is estimated that a large proportion of children suffer from lead poisoning. It is the most common, chemical-related, environmental child health problem. It is especially pronounced in economically-disadvantaged sections of the population. Poverty can cause malnourishment or physical stress, which intensifies disabilities caused by lead absorption.
- At low levels, i.e. 10-25 µg/dl (indicating the amount of lead in a tenth of a litre of blood) lead poisoning in children causes:
  - reduction in IQ and attention span;
  - reading and learning disabilities;
  - hyperactivity and behavioural problems;
  - impaired growth and visual and motor functioning; and
  - hearing loss.
- Exposure to these levels in maternal and umbilical-cord blood is associated with low birth weight and prematurity. The body can store lead for more than 20 years and then release it during pregnancy, harming the foetus (lead can move across the placenta with ease).
- At higher levels, i.e. 60-100 µg/dl, lead poisoning in children causes:
  - anaemia; and
  - brain, liver, kidney, nerve and stomach damage.
- According to the World Bank, countries can save five to ten times the cost of converting to unleaded petrol in health and economic savings due to reduced health costs, savings on engine maintenance and improved fuel efficiency.

Source: UNEP and UNICEF, 1997

the US requires the government to add an extra margin of safety to the risk assessment of chemicals that children may be exposed to.

Cancer in children in the US appears to be increasing (Pogoda, 1997; EHP, 1998; Rachel's EHW, 1998), and a large-scale study of childhood leukaemia and other cancers in the UK has found them to be associated with living close to industrial plants, particularly where fossil fuels were being used or processed (Knox and Gilman, 1997).

4. Approaches to environment and health

4.1. Multifactoral causes of disease

As has been seen in earlier sections much ill health and many diseases are multifactoral (Figure 3.10.7). Identifying the causes of ill health in populations is therefore very difficult and quantifying the contributions of environmental exposures to adverse health impacts is even more so, particularly at the level of the individual. Adverse health impacts are the results of varying combinations of host genetics, host state (including 'lifestyle' factors such as smoking, alcohol, diet parents etc.) and exposures to other environmental stresses, both indoors and outdoors. All these factors can operate at different times, influencing each other in various ways, and causing changes in cells, tissues and functions that may or may not lead to adverse health impacts. The same 'dose' of air pollution for example does not have the same impact because of differences between people, with sensitive groups, such as the elderly, the sick children, and pregnant women responding more than less sensitive groups. The same 'exposure' may not lead to the same 'dose' because of biological and activity differences, e.g. children and joggers who have higher breathing rates.

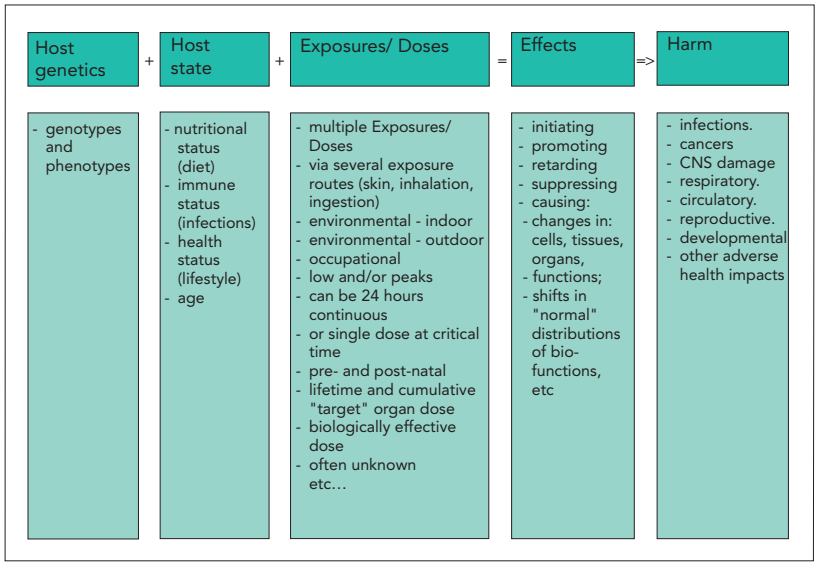
Several key questions need to be addressed in dealing with environmental health issues:

- what is the nature and strength of the evidence for an adverse impact and for the role of the environment in that impact,
- what is the nature of the impact (trivial or serious, reversible or irreversible, immediate or long term, large or small numbers affected etc.),
- what level of proof is to be used in making a decision, particularly about whether an *association* between an environmental stressor and an adverse impact is actually *causation* (Box 3.10.14),
- are exposure or effects avoidance measures available, and actors identifiable and willing to take action,
- the cost and benefits of action and inaction, and their distribution between classes, races, sexes, the regions and generations,
- how uncertainties are to be handled ,
- how informed consent and public involvement in 'acceptable risks' can to be achieved,
- and how the consequences of action/ inaction are to be evaluated?

The answers to these questions require good information for effective decision-making, but in practice, a lack of data, information or understanding, or disagreements about the interpretation of the information can lead to delays in preventing public ill health. For example, one of the main weaknesses of animal evidence is the difference between the healthy young rats used in experiments (which breathe through the nose) and a population of mixed age and health status humans, who partly breathe through their mouths. These three differences (age, health status and mouth-breathing) are the main reasons why experts 'dramatically' underestimated the health impacts on humans of fine particles in air pollution in 1987 compared to 1997 (WHO, 1997b).

The level of proof used in decision-making is crucial, and it can vary from very high to low, depending on the issue being addressed. For 'sound science', a high level is required,

Multifactoral disease causation Figure 3.10.7



Source EEA:

Box 3.10.14. Association and causality

It is often fairly easy to show that a measure of ill-health, e.g. the number of admissions to hospital per day, is associated with a possible cause, such as the day-to-day variation in levels of air pollutants. To show that a causal relationship exists, a number of guidelines or tests have been developed. These include the consistency of results between different studies, the way in which the results of different studies fit together (coherence); whether there is a 'dose-response relationship' between the proposed causal factor and the effect; and whether the sequence of events makes sense, i.e. the cause always preceding the effect.

Proof of causality is often very difficult, but by the application of these and other criteria, an expert judgement as to whether an association is likely to be causal can often be made. Where effects are likely to be serious and/or irreversible, then a low level of proof, as in the 'precautionary principle', may be sufficient to justify action to remove or reduce the probable causes (EEA/WHO, 1997).

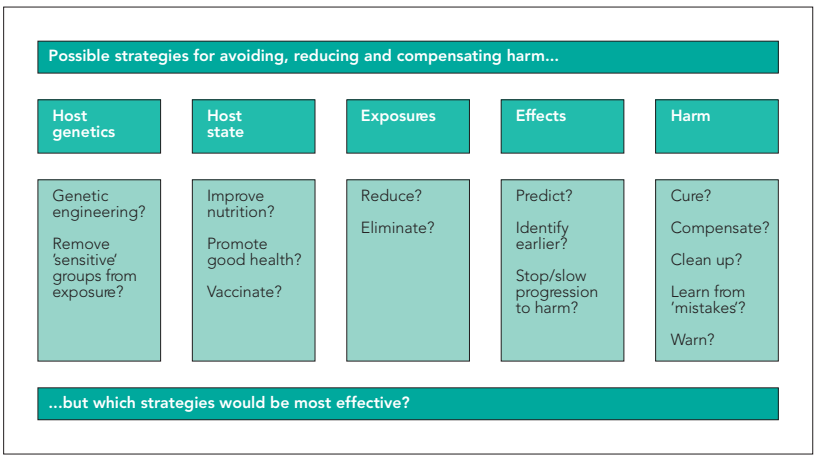
Box 3.10.15 Precaution

This principle featured in the 1992 Rio Declaration on Environment and Development (as Principle 15):

'In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.'

The precautionary principle permits a lower level of proof of harm to be used in policy-making whenever the consequences of waiting for higher levels of proof may be very costly and/or irreversible; the UN Intergovernmental Panel on Climate Change recently used the precautionary principle in concluding that 'the balance of evidence … suggests a discernible human influence on global climate' (IPCC, 1995).

Figure 3.10.8 **Strategies: points of intervention**



Source: EEA

such as 'beyond all reasonable doubt'. This means that the costs of being 'wrong' in failing to reach the high level of proof (such as new and correct, scientific hypotheses being initially dismissed, called 'false negatives'), is considered by society to be less costly than being 'wrong' in the other direction when using a lower level of proof i.e. the 'false positive' of incorrect scientific hypotheses being accepted as correct. Similarly, in criminal trials, where the 'cost' of being wrong in one direction i.e. innocent people being jailed (or sometimes executed), is regarded as being worse than being wrong in the other direction (i.e. guilty people going free), a high level of proof is also used.

For other purposes in society, such as compensating injured people through the courts, a lower level of proof, such as 'the balance of probabilities', is generally used. In this case society considers that the costs of being 'wrong' in reaching the lower level of proof, i.e. the 'false positive' of compensating injured people for injuries that were not caused by the negligence of others, is less costly than being 'wrong' in the other direction, i.e. the 'false negative' of not compensating people for the injuries that were caused by the negligence of others. Another example of the use of a low level of proof, or probability, is disaster insurance where the cost of being wrong when no disaster happens is generally considered more acceptable than the cost of being wrong in the other direction, i.e. where no insurance premiums are paid and disaster strikes. 'It is better to be safe than sorry ' is the popular expression of this sentiment.

For public health policy-making, where there may be serious and irreversible health impacts, the use of a lower level of proof than used in good science is recommended in various international agreements, via the 'precautionary principle' (Box 3.10.15).

4.2. Integrated approaches to prevention

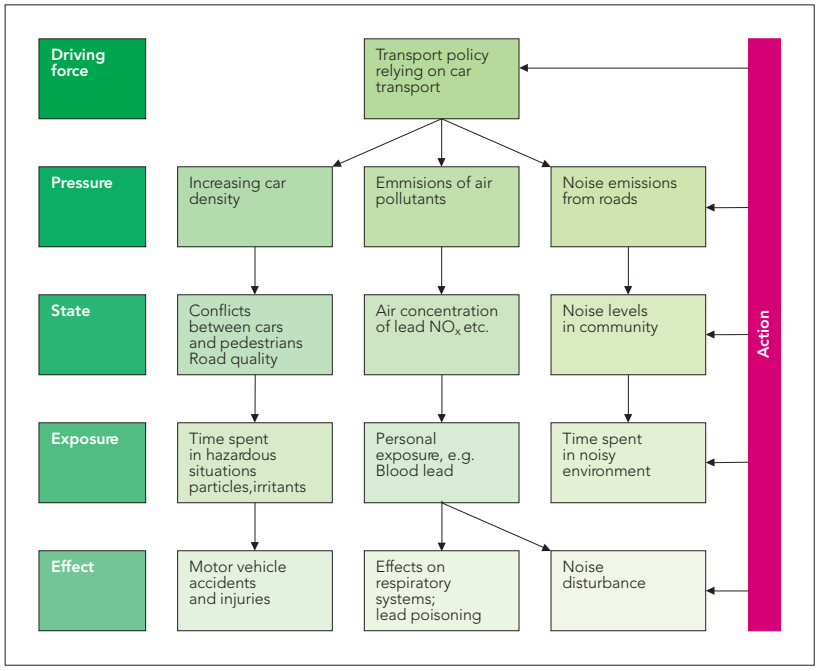
The multi-causal disease process also offers several points at which strategies for avoiding reducing or compensating harm can be focused (Figure. 3.10.8). However, identifying and implementing the most effective strategy is difficult, and involves questions of feasibility (technical, economic and practical), cost-effectiveness and ethics. Responses can also be focused on the individual (behaviour change or medical intervention), or at the community and its environmental exposures. When the response strategy is

focused on reducing exposures, say to traffic fumes, there are many points of policy intervention, involving both 'upstream', e.g. the 'driving forces' of transport policy, and 'downstream', e.g. noise barriers (Figure 3.10.9). In general, strategies-focused 'upstream' will be more effective than those focused 'downstream', partly because of the need to take an integrated approach that embraces the linkages between different parts of transport policy. An integrated approach will also take into account the full range of benefits and costs of policy responses, and allow for adaptation to a modified transport system. For example, policies designed to reduce air pollution from traffic by reducing traffic volumes will also yield substantial benefits from reduced noise, accidents (Box 3.10.17), congestion, less divided communities and increased freedoms to play, walk and cycle in safety. Such holistic approaches can help counter the common 'tendency to over-estimate the costs and under-estimate the benefits' of policy action (WHO, 1997c).

There may also be differences between causes of ill health that are most important from a scientific point of view, and causes that may be most important from a policy response point of view. Figure 3.10.10 illustrates the differences between 'scientific' and 'social intervention' causes in multi-factoral disease processes, such as asthma in children. Whilst genetic pre-disposition, respiratory hyper-sensitivity from pre-natal exposures, diet or indoor air pollution from damp or mites, may be the most important scientific causes of asthma in children, the relatively minor role of traffic pollution may be the most important 'social intervention' cause, given the secondary benefits of a reduction in traffic growth, and the impact of removing one link in a multi-causal chain.

In practice, given the multi-causal nature of diseases like asthma, policy responses are needed in several areas: single cause approaches can not reduce more than a proportion of disease. Integrated approaches to prevention (BMA, 1998) and hazard exposure reductions, as well as more research on the links between environment and health (ESF, 1998) are needed to achieve improved health and wellbeing.

Transport: multi-causality in transport hazards Figure 3.10.9



Source: WHO

Box 3.10.17. Traffic accidents

Road traffic accidents are 1.4% of all deaths (some 45 000 deaths in 1994 in the EU) and 20% of all accidental deaths in the European Region of WHO. About 1 in every 3 deaths involves people younger than 25 years. Due to the high proportion of young victims, it is estimated that on average people killed in traffic accidents die about 40 years earlier than their life expectancy.

From 1993, it appears that the decreasing trend is levelling-off, especially in western countries, where there has been little progress in achieving a further reduction in mortality over the past few years.

The reduction in the number of fatalities has not been paralleled by a proportional reduction in the number of traffic accidents with injuries, which since 1993 have increased slightly.

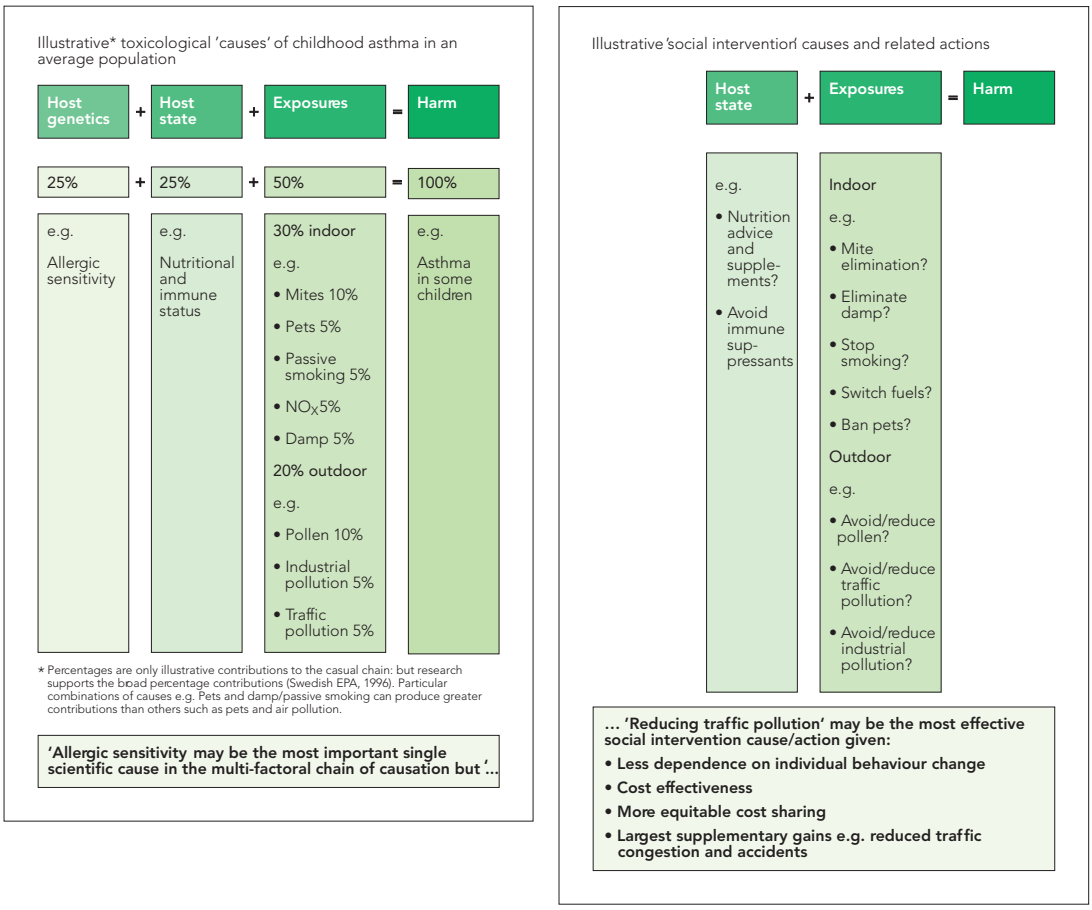
More pedestrians are killed per 1 000 accidents with injury than other road user categories.

Pedestrians account for around 13% of casualties (dead and injured) and 22.5% of deaths by road traffic accidents in the 26 countries of the ECMT. Pedestrians report the second highest number of fatalities among road users in all OECD countries, with the exception of the Netherlands, where cyclists account for more fatal accidents than pedestrians (OECD, 1998).

Cyclists are more likely to have an accident than other road users and they will sustain a greater proportion of head injuries than other road users (OECD, 1998). At least two-thirds of the cyclists killed in accidents had head injuries which contributed to or resulted in death. However, both cycling and walking have very beneficial health effects. WHO estimates that half an hour's walking and cycling a day could reduce the prevalence of heart disease, obesity and diabetes by 50% (WHO, March 1999, press release).

Figure 3.10.10 **Scientific causes and social intervention causes for childhood asthma**

Source: EEA



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